



uOttawa

L'Université canadienne
Canada's university

FACULTÉ DES ÉTUDES SUPÉRIEURES
ET POSTDOCTORALES



FACULTY OF GRADUATE AND
POSTDOCTORAL STUDIES

Natasha Flemming Kyle

AUTEUR DE LA THÈSE / AUTHOR OF THESIS

M.Sc. (Human Kinetics)

GRADE / DEGREE

School of Human Kinetics

FACULTÉ, ÉCOLE, DÉPARTEMENT / FACULTY, SCHOOL, DEPARTMENT

Muscle Activation Patterns During Gait Initiation

TITRE DE LA THÈSE / TITLE OF THESIS

G. Robertson

DIRECTEUR (DIRECTRICE) DE LA THÈSE / THESIS SUPERVISOR

CO-DIRECTEUR (CO-DIRECTRICE) DE LA THÈSE / THESIS CO-SUPERVISOR

EXAMINATEURS (EXAMINATRICES) DE LA THÈSE / THESIS EXAMINERS

R. Balasubramaniam

N. Paquet

Gary W. Slater

LE DOYEN DE LA FACULTÉ DES ÉTUDES SUPÉRIEURES ET POSTDOCTORALES /
DEAN OF THE FACULTY OF GRADUATE AND POSTDOCTORAL STUDIES

MUSCLE ACTIVATION PATTERNS DURING GAIT INITIATION

by

NATASHA FLEMMING KYLE

B.Kin., McMaster University, 2001

THESIS

Submitted to the Faculty of Graduate and Postdoctoral Studies
in partial fulfillment of the requirements for the degree of
Master's of Science in Human Kinetics

University of Ottawa

© Natasha Flemming Kyle, Ottawa, Canada, 2006



Library and
Archives Canada

Bibliothèque et
Archives Canada

Published Heritage
Branch

Direction du
Patrimoine de l'édition

395 Wellington Street
Ottawa ON K1A 0N4
Canada

395, rue Wellington
Ottawa ON K1A 0N4
Canada

Your file *Votre référence*

ISBN: 0-494-14920-5

Our file *Notre référence*

ISBN: 0-494-14920-5

NOTICE:

The author has granted a non-exclusive license allowing Library and Archives Canada to reproduce, publish, archive, preserve, conserve, communicate to the public by telecommunication or on the Internet, loan, distribute and sell theses worldwide, for commercial or non-commercial purposes, in microform, paper, electronic and/or any other formats.

The author retains copyright ownership and moral rights in this thesis. Neither the thesis nor substantial extracts from it may be printed or otherwise reproduced without the author's permission.

AVIS:

L'auteur a accordé une licence non exclusive permettant à la Bibliothèque et Archives Canada de reproduire, publier, archiver, sauvegarder, conserver, transmettre au public par télécommunication ou par l'Internet, prêter, distribuer et vendre des thèses partout dans le monde, à des fins commerciales ou autres, sur support microforme, papier, électronique et/ou autres formats.

L'auteur conserve la propriété du droit d'auteur et des droits moraux qui protègent cette thèse. Ni la thèse ni des extraits substantiels de celle-ci ne doivent être imprimés ou autrement reproduits sans son autorisation.

In compliance with the Canadian Privacy Act some supporting forms may have been removed from this thesis.

Conformément à la loi canadienne sur la protection de la vie privée, quelques formulaires secondaires ont été enlevés de cette thèse.

While these forms may be included in the document page count, their removal does not represent any loss of content from the thesis.

Bien que ces formulaires aient inclus dans la pagination, il n'y aura aucun contenu manquant.


Canada

TABLE OF CONTENTS

TABLE OF CONTENTS	ii
LIST OF TABLES	iii
LIST OF FIGURES.....	iv
ABSTRACT	v
ACKNOWLEDGEMENTS	vi
INTRODUCTION.....	1
Objectives	3
Hypothesis.....	3
Limitations	4
Delimitations.....	5
Rationale	5
REVIEW OF LITERATURE	6
Gait Initiation Variables in Young Healthy Subjects	7
Electromyographic Studies	8
Kinematic and Kinetic Studies.....	13
Gait Initiation Variables of Pathological Gait	15
Summary.....	17
METHODOLOGY	18
Participants.....	18
Instrumentation	18
Experimental Set-up and Protocol	18
Data Reduction and Statistical Analysis	19
RESULTS	22
DISCUSSION.....	34
CONCLUSIONS AND RECOMMENDATIONS	43
REFERENCES	45
APPENDIX A.....	49

LIST OF TABLES

Table 4.1 Muscle onset times during gait initiation.....	22
Table 4.2 Frequencies of muscle activation occurrence	23
Table 4.3 Muscle onset times of the grand ensemble averaged LE-EMGs	33

LIST OF FIGURES

Figure 3.1 Left tibialis anterior EMG signal from one subject. Raw signal (top) and linear envelope signal (bottom)	20
Figure 4.1 Ensemble mean (\pm SD) of the lead (top) and trail (bottom) side erector spinae from 10 trials for subject eight.....	24
Figure 4.2 Ensemble mean (\pm SD) of the lead (top) and trail (bottom) leg tensor fasciae latae from 10 trials for subject eight.	25
Figure 4.3 Ensemble mean (\pm SD) of the lead (top) and trail (bottom) leg adductor magnus from 10 trials for subject eight	26
Figure 4.4 Ensemble mean (\pm SD) of the lead (top) and trail (bottom) leg tibialis anterior from 10 trials for subject eight.....	27
Figure 4.5 Ensemble mean (\pm SD) of the lead (top) and trail (bottom) limb vertical ground reaction force from 10 trials for subject eight throughout gait initiation.	28
Figure 4.6 Grand ensemble LE-EMGs (\pm SD) for lead (top) and trail (bottom) erector spinae throughout gait initiation	29
Figure 4.7 Grand ensemble LE-EMGs (\pm SD) for lead (top) and trail (bottom) tensor fasciae latae throughout gait initiation.....	30
Figure 4.8 Grand ensemble LE-EMGs (\pm SD) for lead (top) and trail (bottom) adductor magnus throughout gait initiation	31
Figure 4.9 Grand ensemble LE-EMGs (\pm SD) for lead (top) and trail (bottom) tibialis anterior throughout gait initiation	32

ABSTRACT

Gait initiation is a temporary movement between upright posture and steady-state gait. The activation of several postural muscles has been identified to precede changes observed in vertical reaction force. Previous research examining gait initiation has concentrated on the electromyographic activity of muscles of the lower limbs. Few studies, however, have looked at recruitment patterns of the muscles of the thigh and trunk. This study was conducted to determine the recruitment patterns and the roles of certain muscles of the trail and lead lower limbs and trunk for the duration from quiet stance to trail leg toe-off. Eleven healthy participants initiated gait with their right leg. Electromyographic data were collected bilaterally from the erector spinae, tensor fasciae latae, adductor magnus and tibialis anterior muscles. In addition, force platform data were recorded for the duration of quiet stance to toe-off of the trail limb. For each subject, onsets and offset timings of all eight muscles were calculated from time-normalized, ensemble-averaged data. The patterns of muscle activity across subjects were generally consistent. The earliest activation was consistently found to be the lead limb tibialis anterior, followed by the lead limb tensor fasciae latae. The trail limb tibialis anterior was the next muscle to become active, which was followed by the trail limb tensor fasciae latae and then the trail limb adductor magnus. There was a tendency for the muscle activity to be more variable during the middle of the gait initiation process. There were notable inconsistencies between subjects for the order of the fifth and sixth muscle activations. Specifically, it was unclear as to whether the activation of the trail limb adductor magnus preceded the lead limb adductor magnus. The last two muscles to become active were consistently found to be the erector spinae of the trail limb side followed by the erector spinae of the lead limb side.

ACKNOWLEDGEMENTS

There are several people whom without their support the completion of this document would not have become a reality. First I would like to express my sincere gratitude to my supervisor Dr. Gord Robertson. Thank you for all of the long hours you put in to this project. Without your guidance and support I would have given up a long time ago. I would also like to thank the members of my committee, Dr. Ramesh Balasubramaniam and Dr. Nicole Paquet, each provided insight and feedback from his/her unique perspective that resulted in the thesis being more complete and comprehensive. Thank you to Ms. Lise O'Reilly for always keeping an eye out for me. The project would not have been possible were it not for the individuals who acted as study participants.

I would like to thank all of my friends for all of their encouragement and interest in the progress of my thesis. A special note of gratitude goes to my parents. You have always encouraged me to pursue and attain my ever changing goals. Without both of your endless support and encouragement I would not be where I am today. I am so lucky to have you both as my parents. Lastly, a special thank you must go to Jon who was always there to help me when I needed him, sometimes at the expense of his own work. You are my best friend and without your support and encouragement I would never have completed this thesis. Words cannot express how grateful I am to you. I love you with all my heart.

CHAPTER I

INTRODUCTION

A key component of our independence as human beings is mobility (Winter, 1995). With the increase in our aged population and the increase in life expectancy of our elderly, the importance of maintaining mobility is becoming ever more critical (Winter, 1995).

Walking consists of three major components: gait initiation, steady state walking and gait termination. Gait initiation has been defined as the transition from quiet stance to steady state gait (Brunt, Liu, Trimble, Bauer & Short, 1999), which challenges the balance control system as the individual moves from a stable static balance to an initially unstable gait (Winter, 1995). The geriatric population and those with certain pathological conditions, which decrease dynamic balance control, may find this task challenging (Winter, 1995).

Gait initiation requires an individual to achieve forward movement while, at the same time, maintaining body stability (Tokuno, Sanderson, Inglis & Chua, 2003). During steady state gait, these responsibilities are shared equally and alternately between the two lower limbs. During gait initiation, however, this does not occur, as different demands are placed on each limb (lead versus trail limb) depending on its role.

The gait initiation process has been divided into two consecutive phases: a preparatory (or postural) and a stepping (or movement) phase (Vaugoyeau, Viallet, Mesure & Massion, 2003; Mickelborough, van der Linden, Tallis & Ennos, 2004). It has been shown that within the preparatory phase there exists a dynamic phase that occurs prior to step execution, where anticipatory postural adjustments (APA) are made (Brenière & Do, 1986). A forward and contralateral acceleration of the centre of mass

(COM) and a backward and ipsilateral displacement of the centre of pressure (COP) with respect to the lead limb are the earlier changes in posture identified as the APA (Couillandre, Brenière & Maton, 2000). This aids in reducing the need for the lead limb to maintain body support and, as a result, assists in generating a portion of the initial forward thrust. At the same time the trail limb, accepts the load that was previously supported by the lead limb (Tokuno *et al.*, 2003). This is the onset of the stepping phase.

When the lead limb returns towards the stance phase, the trail limb begins to apply a greater amount of horizontal force in the posterior direction to achieve the required forward movement for the second step (Tokuno *et al.*, 2003). The lead leg now becomes responsible for absorbing most of the shock generated from the impending heel-strike. Once the trail limb completes its initial step, steady state gait is set in motion.

Kinematic, kinetic and electromyographic (EMG) data have been collected for initiation of gait for young and elderly individuals, stroke and Parkinson's patients as well as for persons with certain disabilities, such as below the knee amputations. From several of these studies, the muscles of the lower extremities have been identified to act stereotypically during initiation of gait (Mann, Hagy, White & Liddell, 1979; Brenière, Do, & Sanchez, 1981; Winter, 1995). These studies have documented an initial inhibition of the gastrocnemius/soleus that had been active during quiet stance. This event was followed by the onset of the tibialis anterior of both the lead and trail limbs (Crenna & Frigo, 1991; Winter, 1995). Several authors have speculated that the inhibition and activation of these aforementioned muscles results in the changes observed in the COM and COP. It seems plausible, however, that more powerful muscles such as the hip abductors and adductors would facilitate these changes. Few studies, however, have

looked at recruitment patterns of the muscles of the thigh and trunk (Mickelborough *et al.*, 2004). The existence of a dynamic phase has been shown to precede motion, where changes in ground reaction forces (GRF) and COP trajectory have been identified (Brenière & Do, 1986). By looking at the tensor fasciae latae and the adductors of the thigh in addition to the muscles of the trunk, one will get a more comprehensive understanding of the role and order of muscle activation in the earliest stages prior to the onset of step execution.

Objectives

The objectives of this research are:

1. To determine the recruitment patterns of the muscles of the trail and lead limbs and trunk for the duration from quiet stance to trail leg toe-off.
2. To elaborate on the roles of the muscles during gait initiation.

Hypothesis

The hypotheses are based on the results found from the following studies: Carlsöö, 1966 White & McNair, 2002; and Mickelborough *et al.*, 2004.

- As the vertical reaction force begins to increase below the lead leg, both the lead and trail leg tibialis anterior muscles will become active, followed by activity of the lead leg tensor fasciae latae and lead leg adductor magnus. Additionally, the trail side erector spinae will become active. There will also be a simultaneous decrease in the vertical reaction force beneath the trail limb.
- The lead and trail leg tibialis anterior will remain active until trail leg toe-off.

- As the vertical reaction force begins to decrease beneath the lead leg and increases below the trail leg, the trail tensor fasciae latae, and the trail leg adductor magnus will become active. Additionally, the lead side erector spinae will become active. While the lead tensor fasciae latae and lead leg adductor magnus will become inactive.
- At lead limb toe-off, both the lead and trail tensor fasciae latae as well as the trail adductor magnus will become active again.
- Both the lead and trail leg tensor fasciae latae as well as the lead adductor magnus will become active at trail leg toe-off.

Limitations

The proposed investigation has several inherent limitations. Firstly, the use of a convenience sample may not be an accurate representation of the general population. Secondly, the signal-to-noise ratio may influence the reliability of the signal. Noise may come from various sources: inherent noise in the electronic equipment, ambient noise, as well as motion artifacts (DeLuca, 2002). With an electrode unit that provides minimal distortion and the highest signal-to-noise ratio, the accuracy of the EMG signal can be maximized. Furthermore, the susceptibility of the equipment to crosstalk may affect the EMG signal. Crosstalk can be minimized by selecting the proper electrode size, interelectrode distance and location of electrodes over the muscle (Winter, Fuglevand & Archer, 1994; DeLuca, 2002). Lastly, ensemble averaging may obscure both the amplitudes and temporal pattern of muscle activity if there is high between trial variability for a given subject. To remedy this, all trials will be visually inspected prior to ensemble averaging.

Delimitations

There are several restrictions placed on this research project. Firstly, the use of subjects without pathologies. Secondly, all subjects will be asked to walk at a self-selected speed. Lastly, all subjects will be asked to start with their right leg, irrespective of the participant's usual choice of lead limb.

Rationale

Both ageing and disease cause changes to the nervous, muscular and skeletal systems. These changes, in turn, may affect the control of balance. Many falls in the elderly occur during transitions such as the initiation and termination of gait, changes in walking direction, negotiating stairs or ramps and obstacle avoidance (Winter, 1995). Given that gait initiation is a risky activity for elderly and that the population is rapidly ageing, the underlying mechanisms warrant further investigation. Furthermore, problems with gait initiation can be exacerbated in pathologies such as Parkinson's disease or because of limb prostheses or neuromuscular disability. Through a more in-depth analysis of the temporal activation of muscles of the lower and upper leg of both the lead and trail limb in addition to the muscles of the back, one can achieve a comprehensive understanding of the order and role of muscle recruitment involved in the initiation of gait. This primarily descriptive analysis may, in turn, lead to improved rehabilitation treatment strategies to reduce the risk of falling and aid patients encountering difficulties in executing their first step. Moreover, knowledge of the recruitment patterns will provide normative data against which abnormal muscle patterns of gait initiation may be compared.

CHAPTER II

REVIEW OF LITERATURE

Gait is a complex behaviour that involves the entire body and therefore requires the integration of neural mechanisms and muscular activity coupled with the internal and external applied forces. Mobility not only includes the ability to walk, but also includes the capability to initiate and terminate locomotion, adapt gait to avoid obstacles, and change speed and direction as needed.

Most studies have described the initiation of gait by using trajectory, acceleration and velocity of the body COM, GRFs, and COP of the feet as well as angular displacements and EMG activities of the muscles of the lower limb (Carlsöö, 1966; Mann *et al.*, 1979; Brenière & Do, 1986; Winter & Yack, 1987; Brunt, Lafferty, Mckeon, Goode, Mulhausen, & Polk, 1991; Crenna & Frigo, 1991). Many of these studies, however, used only one force platform and analyzed only one limb, which has caused difficulty in separating the role of individual limbs. Furthermore, several of these studies did not look at the events prior to the stepping phase. Additionally, the EMG signal has been shown to be influenced by a number of factors including instrumentation and the choice of data analysis, which introduces difficulty in comparing the results of different studies (Craik & Oatis, 1995).

The following section reviews the various definitions of the phases of gait initiation from the literature. This will be followed by a review of the electromyographic, kinematic and kinetic analyses that have attempted to explain the process of gait initiation in healthy subjects. Subsequently, a brief review of the aforementioned measurements of

locomotion in elderly persons, Parkinson's disease patients and stroke patients will be provided.

Gait Initiation Variables in Young Healthy Subjects

In healthy subjects gait initiation is an automatic transition from stance into walking with a consistent pattern of lower limb muscle activity (Elble *et al.*, 1994). Many authors divide gait initiation into two phases: a phase prior to step execution and an execution or stepping phase. How these phases are defined in themselves, however, differs from one study to another. Brenière, Do, & Bouisset (1987), Brunt *et al.* (1999) and Gélat and Brenière (2000) defined the anticipation phase as the dynamic phase prior to heel-off of the stepping limb, while Mickelborough *et al.*, (2004) and Jian, Winter, Ishac, & Gilchrist (1993) defined the preparatory phase as the onset of a dynamic movement until the toe-off of the swing foot.

Mickelborough *et al.*, (2004) further divided the preparatory phase into two sub-phases: a release phase and an unloading phase. During the release phase, the COP was found to move posterolaterally towards the lead foot, increasing the vertical and horizontal GRF components, thus accelerating the COM forwards. The release phase lasts until the furthest point of posterolateral COP movement (COPmax), when the COP abruptly changes direction. This marks the beginning of the unloading phase. During this phase, the COP moves rapidly across to the trail foot, unloading the lead foot for toe-off. Other authors have defined the preparatory phase as the anticipatory phase, starting with the deactivation of the soleus, followed by activation of the tibialis anterior, backward shift of the COP and backward shear force (Herman, Cook, Cozzens, & Freedman, 1973; Crenna & Frigo, 1991; Dietrich, Brenière, Do, 1994; Brunt *et al.*, 1999). Lead leg toe-off

marks the start of the stepping phase according to Mickelborough *et al.*, (2004). Jian *et al.*, (1993), on the other hand, renamed the stepping phase the “take-off phase” and defined it in agreement with Mickelborough *et al.*, (2004) as the time from lead toe-off to trail toe-off.

Mickelborough *et al.*, (2004) subdivided the stepping phase into single- and double-support phases. The single-support phase lasts from lead toe-off to initial contact of the lead foot, with the double-support phase lasting from initial contact of swinging foot to toe-off of the original stance foot. Gélat and Brenière (2000) defined the execution phase as analogous to the stepping phase but beginning at heel-off of the lead limb and ending at the instant of foot contact of the lead limb. A later double-stance phase was described as beginning at foot contact of the leading limb and ends at the instant of toe-off of the trailing limb. Jian *et al.*, (1993) went further in describing a stabilizing period, defined as the time from lead leg toe-off to the second trail leg toe-off in which steady-state gait begins to be achieved.

Electromyographic Studies

Surface EMG has been increasingly used over the past several decades for the assessment of muscle activity of normal and pathological gait (Winter & Yack, 1987). Several studies have shown that activation of several postural muscles occurs before vertical force variations are seen in gait initiation (Herman *et al.*, 1973; Brunt *et al.*, 1991). Over the years researchers have developed typical EMG patterns during different phases of the gait cycle. The majority of studies have focused on the lower extremities and on steady-state gait. Studies of thigh and trunk muscle EMG activity during gait are limited (Craik & Oatis, 1995).

Muscle activity is required to initiate gait from a standstill. The predominant muscle activation prior to any detectible movement of the body has been well documented as a bilateral inhibition of gastrocnemius/soleus activity, closely followed by a bilateral burst of tibialis anterior activity (Hermann *et al.*, 1973; Mann *et al.*, 1979; Brunt *et al.*, 1991; Crenna & Frigo, 1991; Elbel, Moody, Leffler, Sinha, 1994; Mickelborough *et al.*, 2004). The coordinated muscle activity of the tibialis anterior and inhibition of the gastrocnemius/soleus are believed to contribute to the acceleration of the COM forwards and towards the trail leg side while the COP is directed initially posterolaterally towards the lead leg and then across to the trail leg (Mann *et al.*, 1979; Brenière & Do, 1986; Crenna & Frigo 1991). Some authors, however, have speculated that the hip abductors and adductors are much more powerful muscles and would seem more plausible that it would be these muscles responsible for this lateral COP motion.

In steady-state-gait the plantarflexors (medial and lateral gastrocnemius, soleus and peroneus longus) have been found to have peak activation at push-off (Winter & Yack, 1987). More specifically, Craik & Oatis (1997) found that the gastrocnemius and soleus muscles are active for approximately 10 to 50% of the gait cycle. Even with the plantarflexor muscle activity lasting until 50% of the gait cycle, Sutherland (1980) has suggested that the last 10 to 12% of stance phase, during which the foot continues to plantarflex, occurs without corresponding activity in the plantarflexors (Craik & Oatis, 1997). Burleigh, Horak, & Malouin (1994) found a marked asymmetry in the level of gastrocnemius activation, (more reduced in the trail leg) whereas the soleus response was reduced bilaterally. These findings correspond to the fact that the initiation of gait from quiet stance requires bilateral inhibition of the soleus for forward movement of the COM

followed by asymmetrical activation of the lead limb gastrocnemius for the propulsion during heel-off (Winter, 2005). Mickelborough *et al.*, (2004) found that gastrocnemius activity in the lead leg did not continue to lead leg toe-off and found a clear offset of the trail leg gastrocnemius activity prior to trail leg toe-off. The authors proposed that perhaps the final stage of toe-off is not actively propulsive but rather is caused by the forward momentum of the COM. Nolan and Kerrigan (2003) found that the duration of gastrocnemius inhibition in the lead limb was shorter, and there was an increase in the magnitude of the gastrocnemius activity in the lead limb when initiating gait from toe-standing compared to heel-toe standing. The authors suggested that the increased activity of the gastrocnemius and biceps femoris muscle activity cause the greater amount of forward momentum generated during toe standing. In contrast to Mickelborough *et al.*, (2004), for heel-toe gait initiation the authors speculated that the gastrocnemius and biceps femoris muscle activity in the trail limb, and tibialis anterior and rectus femoris muscle activity in the lead limb, may be related to the generation of forward momentum.

It has been found that the weight-accepting muscles (tibialis anterior, extensor digitorum longus, rectus femoris, vastus lateralis, hamstrings, gluteus maximus and medius) have their major peak in the first 15% of stride during steady-state-gait (Winter & Yack, 1987). The tibialis anterior muscle shows biphasic activity first from toe-off through mid-swing then again at heel strike (Craig & Oatis, 1997). In agreement with Mickelborough *et al.*, (2004) voluntarily assuming a forward lean before initiating gait has been shown to progressively reduce the amplitude of the lead limb tibialis anterior because the forward position of the body has already been achieved (Crenna & Frigo, 1991). Brunt *et al.*, (1991) found that for all subjects, force plate data indicated that the

tibialis anterior onset preceded force plate activity, and trail tibialis anterior activity was noted before the onset of lead tibialis anterior. The results of the study by Mann *et al.*, (1979), Crenna & Frigo (1991) and Mickelborough *et al.*, (2004) did not show this trend.

Crenna and Frigo (1991) found that by changing the velocity of gait initiation, the amplitude of soleus inhibition and tibialis anterior burst were gradually enhanced, to become maximal at the highest velocities. Brunt *et al.*, (1999) found that regardless of initiation speed, the timing interval between the inhibition of the soleus and onset of the tibialis anterior were the same for both self-paced and fast paced initiation speeds (mean values were between 46 ms and 51 ms). With an increase in the speed of initiation, however, the duration of the tibialis anterior activity during this period increased by 42% and soleus activity decreased by 41%. The authors proposed that coupling the soleus inhibition with tibialis anterior activation at a higher velocity would produce sufficiently rapid COP displacement, enabling the movement to be initiated at a higher velocity.

The differences in the soleus to tibialis anterior latency reported by Crenna and Frigo (1991) compared to the data reported by Elble *et al.*, (1994) and Brunt *et al.*, (1999) could be due to the detection of soleus inhibition. Crenna and Frigo (1991) used the onset of the inhibitory component of soleus to determine soleus to tibialis anterior latency, whereas Elble *et al.*, (1994) and Brunt *et al.*, (1999) used complete inhibition of soleus.

With respect to the thigh musculature, Winter & Yack (1987) have proposed that the adductor longus and magnus have slightly different patterns during swing-the adductor longus peaks at 70% of stride, whereas the adductor magnus peaks at 50% of stride. Both these muscles, however, serve to control the lateral movement of the swinging lower limb. Over the course of stance, both adductors have a moderate but

decreasing activity, which acts to stabilize the hip joint against the action of the hip abductors.

Carlsöö (1966) found that the tensor fasciae latae of the trail leg was inhibited simultaneously with an increase in the activity in the tensor fasciae latae of the lead leg at the onset of gait initiation. It has been suggested that the hip abductors contribute to the control of frontal plane motion of the COM and the lateral loading/unloading mechanism during the stance phase (Winter et al., 1993; Winter et al., 1996). The hip extensors and knee flexors (gluteus maximus and hamstrings) have increasing activity in late swing to stop the forward movement of the swinging lower limb (Winter & Yack, 1987).

Waters and Morris (1972) reported that the abdominal muscles either remained active throughout the gait cycle or showed biphasic bursts—one in stance and one in swing phase. White & McNair (2002) found similar results for the internal obliques. Biphasic activity was observed with maximal values occurring during the mid to late stance phase of both legs. The external obliques, however, were found to produce a biphasic pattern of muscle activity with peaks occurring close to foot strike. The erector spinae did not show the swing-stance transition burst but rather had bursts prior to and after heel contact (Craig & Oatis, 1995; White & McNair, 2002). The erector spinae has been shown to peak at 10% of stride in order to control forward rotation of the trunk during weight acceptance (Winter & Yack, 1987). A second, smaller burst was identified after heel contact of the contralateral limb at 60% of the gait cycle. The authors have speculated that this was likely for the aforementioned reason as when the ipsilateral limb is accepting weight. This is similar to the results found by White & McNair (2002) in which the erector spinae activity was observed close to foot-strike. In contrast, Shiavi (1990)

suggested that these muscles contract bilaterally, one eccentrically and the other concentrically, to balance the torso and pelvis in preparation for the swing phase.

It has been identified that the distal muscles show less variability and are most active, whereas the more proximal muscles show more variability and are least active (Winter & Yack, 1987). This can be explained by considering the heavy and regular use of the support muscles at the ankle (soleus, gastrocnemius and tibialis anterior). The roles of the more proximal muscles are twofold: support and balance, which results in more stride-to-stride variability. Winter and Yack (1987) found that the peak amplitudes of these proximal muscles were less than the ankle plantarflexors, resulting in a lower level of mean activity and a higher coefficient of variation calculation. The authors found a wide range of peak amplitudes registered for each subject's ensemble average. They found that it is possible for normal subjects to exhibit almost identical kinematics but with a wide range of combinations of activity of muscles such as the vastus lateralis and biceps femoris. Furthermore, the authors found that two muscles of the same group can have a different coefficient of variation. Such differences indicate that one muscle can have a more variable function than another muscle of the same muscle group.

Kinematic and Kinetic Studies

The paths of the COP and of the COM during gait initiation have been well documented in the literature (Mann *et al.*, 1979; Brenière & Do, 1986; Jian *et al.*, 1993, Burleigh *et al.*, 1994; Elble *et al.*, 1994; Winter, 1995). The COM measurements follow body position while the COP measurements reflect weight shifts and muscular control during dynamic posture changes such as gait initiation (Chang & Krebs, 1999).

In normal healthy subjects the initiation of gait begins with a posterolateral movement of the COP toward the lead foot. Crenna and Frigo (1991) found that the backward shift of the COP started from 100 to 200 ms after the onset of soleus inhibition and was maximal after the peak burst of the tibialis anterior. The backwards movement of the COP has been suggested to be the result of a reduction in the plantarflexor moment (Jian *et al.*, 1993). The lateral movement towards the lead leg has been suggested to be due to a temporary loading of the lead limb and unloading of the trail limb (Mickelborough *et al.*, 2004). Soon after lead limb toe-off the COP quickly moves towards the trail leg, which continues to accelerate the COM forward and starts to accelerate it away from the trail leg. In the period from lead limb toe-off to trail limb toe-off the major task is to achieve a speed close to steady-state gait and to develop a nearly steady-state trajectory of the COM. During double support the COP moves rapidly ahead of the COM (Jian *et al.*, 1993).

The duration of gait initiation was found to be independent of gait velocity (Brenière & Do, 1991). However, the higher the gait velocity, the longer the duration of the anticipatory phase (Brenière *et al.*, 1987) and the step length (Herman, Wirta, Bampton & Finley, 1976). Furthermore, the amplitude of the initial backward shift of the COP was significantly larger with an increase in gait velocity (Brenière *et al.*, 1987).

Nolan and Kerrigan (2003) showed that when gait was initiated from toe-standing, there was a qualitatively similar movement of the COP towards the lead foot and then the trail foot, although little backwards displacement was observed.

The vertical GRF patterns have been well documented. The functional role of variation in these forces has been described as a means of facilitating the transition from

bipedal to monopodal support (Patchay & Gahery, 2003). Patchay and Gahery (2003) demonstrated that the vertical force variations are anticipatory to any body segment movement. The overall pattern of vertical GRF beneath both feet was identical, in which a momentary marked increase beneath the lead foot and a corresponding drop beneath the trail foot was observed (Brunt *et al.*, 1991; Patchay & Gahery, 2003). Brunt *et al.*, (1999), found that prior to heel-off of the trail limb, the timing and amplitude measures of the vertical GRF increased with initiation speed.

There is much disagreement throughout the literature as to when steady-state-gait is reached. Some authors speculate that steady-state walking is reached within the first step (Brenière & Do 1986), while others argue that it is not until the end of the second step (Jian *et al.*, 1993), or perhaps even in approximately three steps (Mann *et al.*, 1979).

Gait Initiation Variables of Pathological Gait

Virtually all neuromusculoskeletal disorders result in some deterioration in the balance control system (Winter, 1995). Mickelborough *et al.*, (2004) examined muscle activity during gait initiation in normal elderly people. The muscle activity was found to be, in general, similar to those previously found for young adults. Some differences, however, were found. The expression of the tibialis anterior and medial gastrocnemius pattern at onset was less consistent in elderly people. A failure of the medial gastrocnemius to be consistently inhibited at gait initiation onset has been shown to be more common in elderly people (Polcyn, Lipsitz, Kerrigan & Collins, 1998), Parkinson's patients (Elble, Cousins, Leffler & Hughes, 1996) and in stroke patients (Brunt, Vander Linden & Behrman, 1995). This continued tendency to fire the medial gastrocnemius throughout gait initiation would tend to reduce the initial backward displacement of the COP and the

forward acceleration of the COM. This results in a significantly lower amount of forward momentum generation (Polcyn *et al.*, 1998). Elderly people and Parkinson's patients have been shown to have a smaller COM velocity at the end of the preparatory phase than young adults (Gantchev, Viallet, Aurenty & Massion, 1996; Halliday, Winter, Frank, Patla & Prince, 1998). The medial gastrocnemius activity throughout, or immediately after, gait initiation may act as a safety strategy, in that initial anterior COM acceleration is controlled or reduced. A similar strategy has been demonstrated in stroke patients (Brunt *et al.*, 1995). Besides abnormalities in the activity of the medial gastrocnemius and tibialis anterior, Crenna, Frigo, Giovannini & Piccolo (1990) observed a delay between the soleus inhibition and the corresponding tibialis anterior activation in Parkinson's patients. Since the preparatory phase showed more variability than the stepping phase, this suggests that the preparatory phase be a particular source of difficulty in patients with gait disorders (Mickelborough *et al.*, 2004).

Ditz and Colombo (1998) showed that the load sensitivity decreased with age and was seriously affected in patients with Parkinson's disease. The authors suggested that this decrease would contribute to impaired gait in elderly subjects and in Parkinson's patients. Patchay, Gahery & Serratrice, (1997) and Patchay, Gahery and Serratrice, (2002), found that elderly patients with *la marche à petits pas* and elderly normal subjects had smaller values and impaired amplitude of the vertical GRF than the young normal adults, although both elderly groups and the young adults showed symmetry of the distribution of the vertical forces under both feet before they were asked to start walking. Similarly, all groups showed an increase in vertical forces under the lead foot and a corresponding decrease under the trail foot during gait initiation. The patients showed

much larger backward reaction force under the lead foot. The reaction forces that act in the direction opposite to that of progression could be related to start hesitations and to the foot remaining stuck on the floor when the patient starts to walk (Patchay *et al.*, 1997).

Summary

Despite the vast body of knowledge on gait there continues to be a need for further investigation in the area of gait initiation. The majority of the EMG research on gait initiation has focused primarily on the muscles of the lower leg with very little research done on the medial muscles of the upper thigh and trunk. In addition most studies have focused on steady-state gait. The predominant muscle activation prior to any detectible movement of the body has been documented as a bilateral inhibition of gastrocnemius/soleus activity, closely followed by a bilateral burst of tibialis anterior activity. The coordinated muscle activity of these muscles has been proposed to generate a horizontal GRF component to accelerate the COM forward and toward the trail leg side while the COP is directed initially posterolaterally toward the lead leg and then directed toward the trail leg. With further investigation, however, other muscles may be identified as being activated and/or inhibited before the tibialis anterior and gastrocnemius/soleus that may be responsible for the aforementioned kinetic and kinematic variables identified during the initiation of gait. By looking at the tensor fasciae latae and the medial adductors of the thigh in addition to the trunk one will get further insight into the mechanisms that may contribute to the observed kinematic and dynamic changes.

CHAPTER III

METHODOLOGY

Participants

Eleven healthy participants (4 male and 7 female), having neither anatomical nor gait abnormalities or a history of lower extremity injury were recruited from the University of Ottawa population (e.g., student, staff, faculty). All subjects signed an informed consent form in accordance with the University of Ottawa ethical policy.

Instrumentation

Two force platforms (Kistler) were used to measure vertical, horizontal and mediolateral ground-reaction forces, at 1040 Hz, from quiet stance to toe-off of the trail limb. In addition, 8 channels of electromyographic data (Octopus, Bortec) were collected at 1040 Hz for each muscle. EMG electrodes were placed bilaterally over the erector spinae, the tensor fasciae latae, the adductor magnus and the tibialis anterior muscles. All analog signals were synchronously recorded by SIMI Motion software (SIMI Motion Systems GmbH).

Experimental Set-up and Protocol

Prior to electrode placement, the areas under the electrodes were shaved and cleansed of the underlying skin to reduce skin resistance. Silver/silver chloride electrodes were spaced 2 cm centre-to-centre on the aforementioned muscles of interest in line with the muscle fibres over the area of greatest muscle bulk. The electrodes were 3 cm in diameter with a hydrogel centre (Meditrace 230 General Monitoring Electrode). To ensure proper resolution, a maximal voluntary contraction of each muscle of interest was used to set the

gain for each channel. Participants were then asked to stand with one foot on each of the force plates, distributing their weight equally onto both limbs. After a go command was given by the researcher, the participant began walking briskly. Electromyographic data were collected for three steps, beginning at quiet stance on the two force platforms, while force platform data were recorded for the duration of quiet stance to toe-off of the trail limb. Ten trials were collected for each subject. Trials were discarded when equipment malfunction resulted in data loss or because gait was initiated prior to the go command. Trials were not excluded for other reasons. Each subject was tested during a single session and walked barefoot.

Data Reduction and Statistical Analysis

Force platform data were filtered with a zero-lag, second-order, critically damped, low-pass filter with a cut-off frequency of 20 Hz. To remove low frequency motion artefacts the raw electromyographic data, were high-pass filtered with a cut-off frequency of 8 Hz (Figure 3.1, top) (Robertson & Dowling, 2003). Electromyographic data were full-wave rectified. Using a second-order, critically damped, low-pass filter with a cut-off frequency of 5 Hz, a linear envelope was calculated (Figure 3.1, bottom) (Robertson & Dowling, 2003). The filtered EMG and force data were clipped at trial leg toe-off and 1.5 seconds prior to this event.

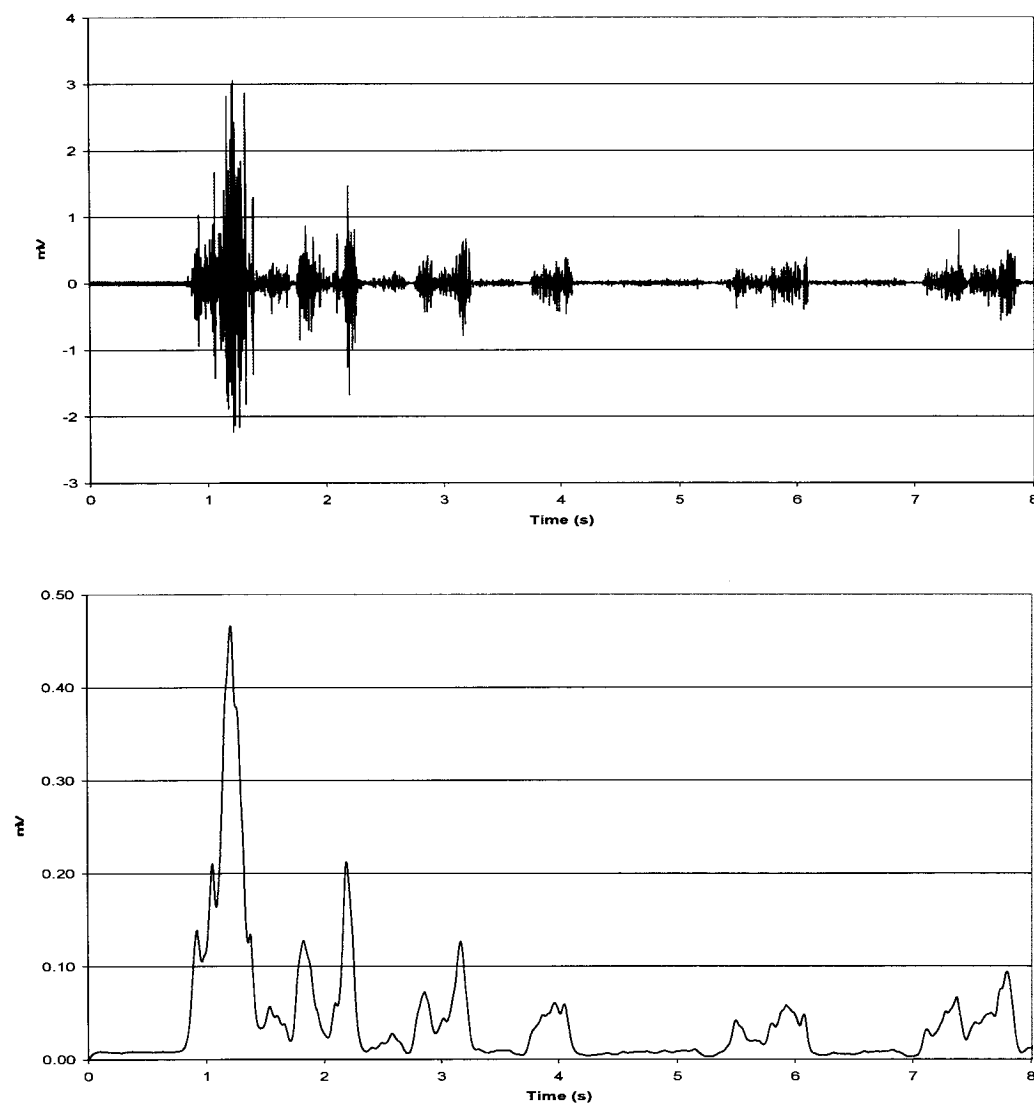


Figure 3.1: Left tibialis anterior EMG signal from one subject. Raw signal (top) and linear envelope signal (bottom).

The determination of the start and end of muscle activity was obtained by an amplitude threshold criterion. The amplitude threshold was based on three times the standard deviation of the resting EMG baseline for each muscle, estimated from the quietest 100 ms periods of each EMG trace. This was obtained from the beginning of the trial while the participant was in quiet stance. Any activity above three standard

deviations from the mean was taken as activation, and any activity below three standard deviations from the mean was taken as relaxation.

Ensemble averages for 9 subjects were calculated for each muscle using BioProc1 (Robertson, 2005). Timings of all eight muscle onsets and offsets were recorded from time-normalized, ensemble-averaged data for each subject. The standard deviation from these ensemble-averaged data will show the variability in muscle activity between trials for each subject. Lastly, all subjects' ensemble averaged curves were normalized to percent maximum amplitude using BioProc1 and a grand ensemble average for each muscle was calculated. Timings of all eight muscle onsets and offsets were recorded from amplitude normalized grand ensemble-averaged data. The standard deviation from the grand ensemble average will show the variability in muscle activity between subjects.

CHAPTER IV

RESULTS

The muscle onset times for 9 subjects are given in Table 4.1. Two subjects were not included due to problems with the equipment or because the subject anticipated the go command. The mean and standard deviation for the onset times of each muscle is also found in Table 4.1. On average, the earliest recruited muscles were the lead leg tensor fasciae latae (16.3%) and tibialis anterior (16.3%) with the trail leg tibialis anterior not far behind at 20.0%. Furthermore, the trail and lead leg erector spinae muscles were recruited latest and nearly simultaneously (44.6% and 47.7%, respectively).

Table 4.1: Muscle onset times during gait initiation

	L-ES	T-ES	L-TFL	T-TFL	L-ADD	T-ADD	L-TA	T-TA
Subject1	51	84	18	22	49	35	16	19
Subject2	43	25	20	35	79	24	13	15
Subject3	51	75	21	36	50	39	17	30
Subject4	45	36	12	18	17	30	11	20
Subject5	41	35	20	28	24	34	19	23
Subject6	63	40	16	12	18	19	32	28
Subject7	46	35	11	16	33	18	10	15
Subject8	41	39	15	34	26	28	12	17
Subject9	48	32	14	39	21	18	17	13
Mean	47.7	44.6	16.3	26.7	35.2	27.2	16.3	20.0
St Dev	7.35	20.4	3.64	9.94	20.5	7.92	6.63	5.94

Note: Muscle onset timings are all expressed as a percentage of total gait initiation (0-100%). L-ES & T-ES are the lead leg and trail leg erector spinae; L-TFL & T-TFL are the lead leg and trail leg tensor fasciae latae; L-ADD & T-ADD are the lead leg and trail leg adductor magnus; L-TA & T-TA are the lead leg and trail leg tibialis anterior.

The frequencies of occurrence for the predominant bursts of muscle activity, from quiet stance to trail leg toe-off, are given in Table 4.2. The patterns of muscle activity for gait initiation were generally consistent. However, there were some noticeable inconsistencies between subjects. The most variability in the patterns of muscle activity appears to occur in the middle of the gait initiation cycle. Only 44.4% of the subjects activated their trail leg tensor fasciae latae fourth. Only 44.4% of the subjects activated

their trail leg adductor magnus fifth, while in 33.3% of subjects this muscle was activated fourth. Furthermore, only 33.3% of the subjects activated their lead leg adductor magnus sixth, while in 22.2% this muscle was either activated third or fourth. The most consistent patterns of muscle activity were found in the lead leg tensor fasciae latae (88.8%), the lead leg tibialis anterior (77.8%) and the erector spinae on the lead limb side (66.7%).

Table 4.2: Frequencies of muscle activation occurrence

	Muscle Order								Total
	1	2	3	4	5	6	7	8	
L-ES							3	6	9
T-ES					1	1	5	2	9
L-TFL		8	1						9
T-TFL	1			4	1	2	1		9
L-ADD			2	2	1	3		1	9
T-ADD				3	4	2			9
L-TA	7		1			1			9
T-TA	1	1	5		2				9

Note: L-ES & T-ES are the lead leg and trail leg erector spinae; L-TFL & T-TFL are the lead leg and trail leg tensor fasciae latae; L-ADD & T-ADD are the lead leg and trail leg adductor magnus; L-TA & T-TA are the lead leg and trail leg tibialis anterior. The numbers within the body of the table indicate in how many subjects that muscle was active.

The time normalized ensemble averaged EMG curves, from 10 trials, for one subject are plotted in Figures 4.1 to 4.4. The ± 1 SD curves demonstrate graphically the within-subject variability of the muscle activity. The ensemble averaged vertical ground reaction force curves, from 10 trials, for one subject are plotted in Figure 4.5. For comparison, Figure 4.6 is a plot of the grand ensemble averages ± 1 SD for 9 subjects for eight muscles, showing the between-subject variability of muscle activity.

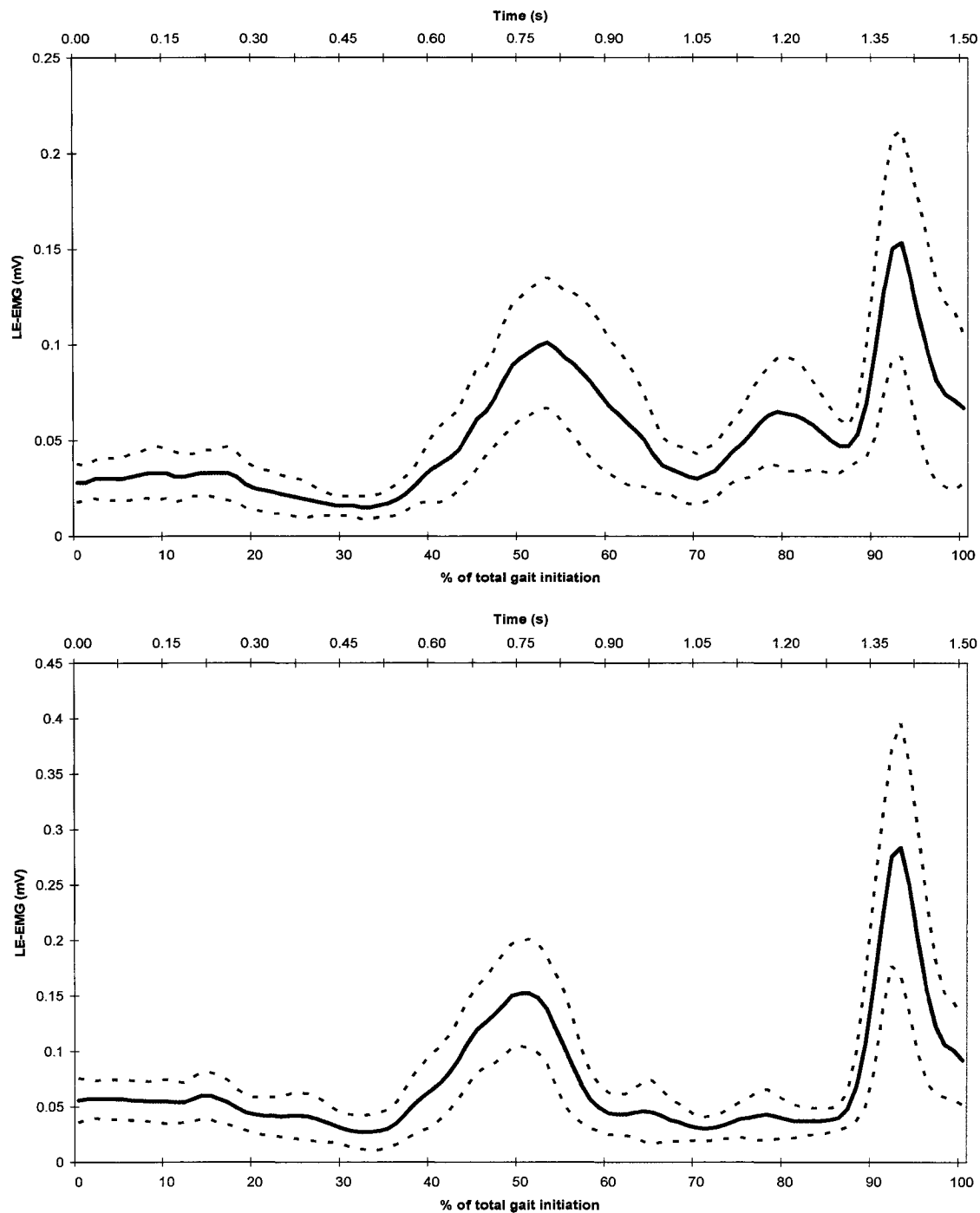


Figure 4.1: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) side erector spinae from 10 trials for subject eight. Time normalized linear envelope EMG throughout gait initiation.

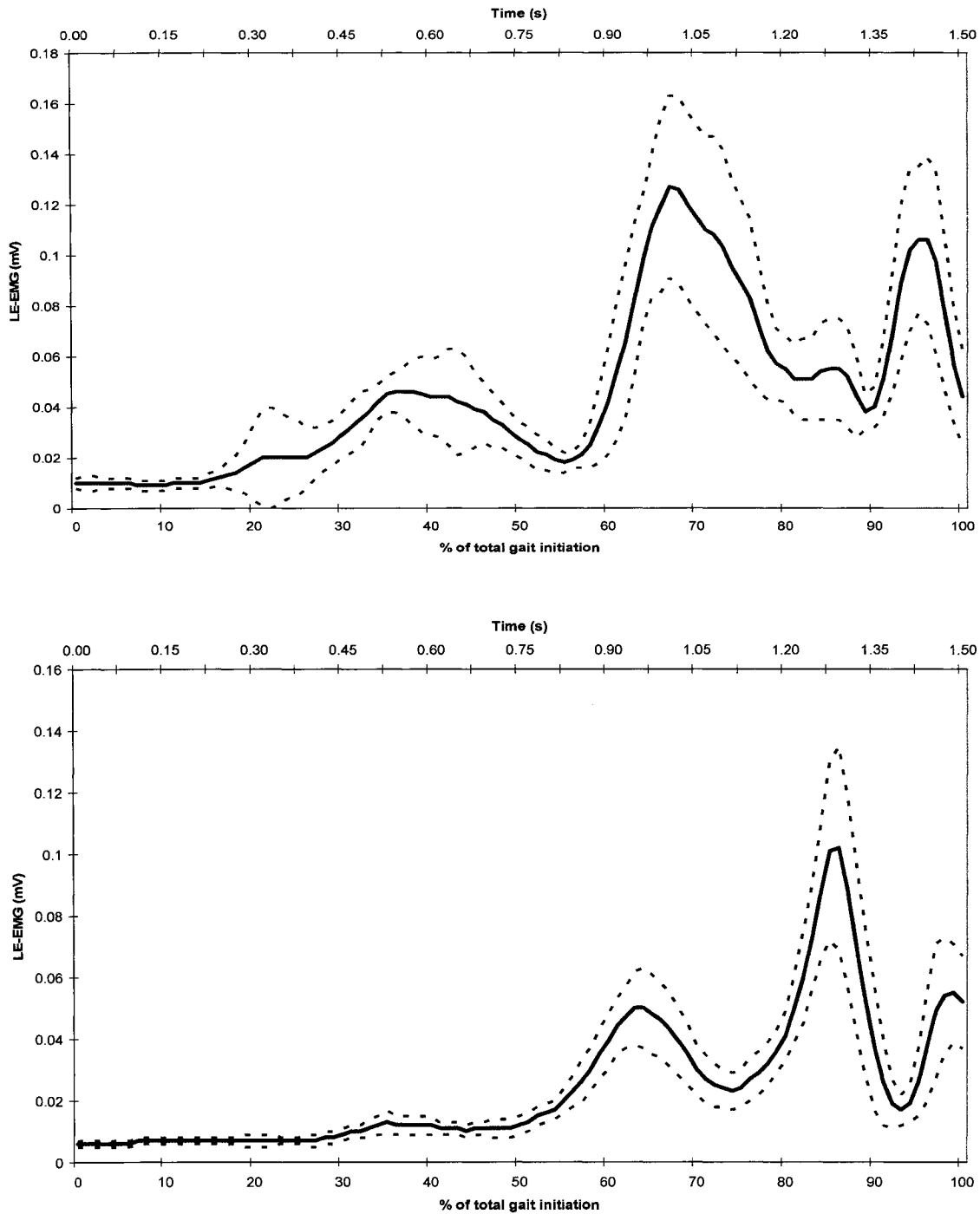


Figure 4.2: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) leg tensor fasciae latae from 10 trials for subject eight. Time normalized linear envelope EMG throughout gait initiation.

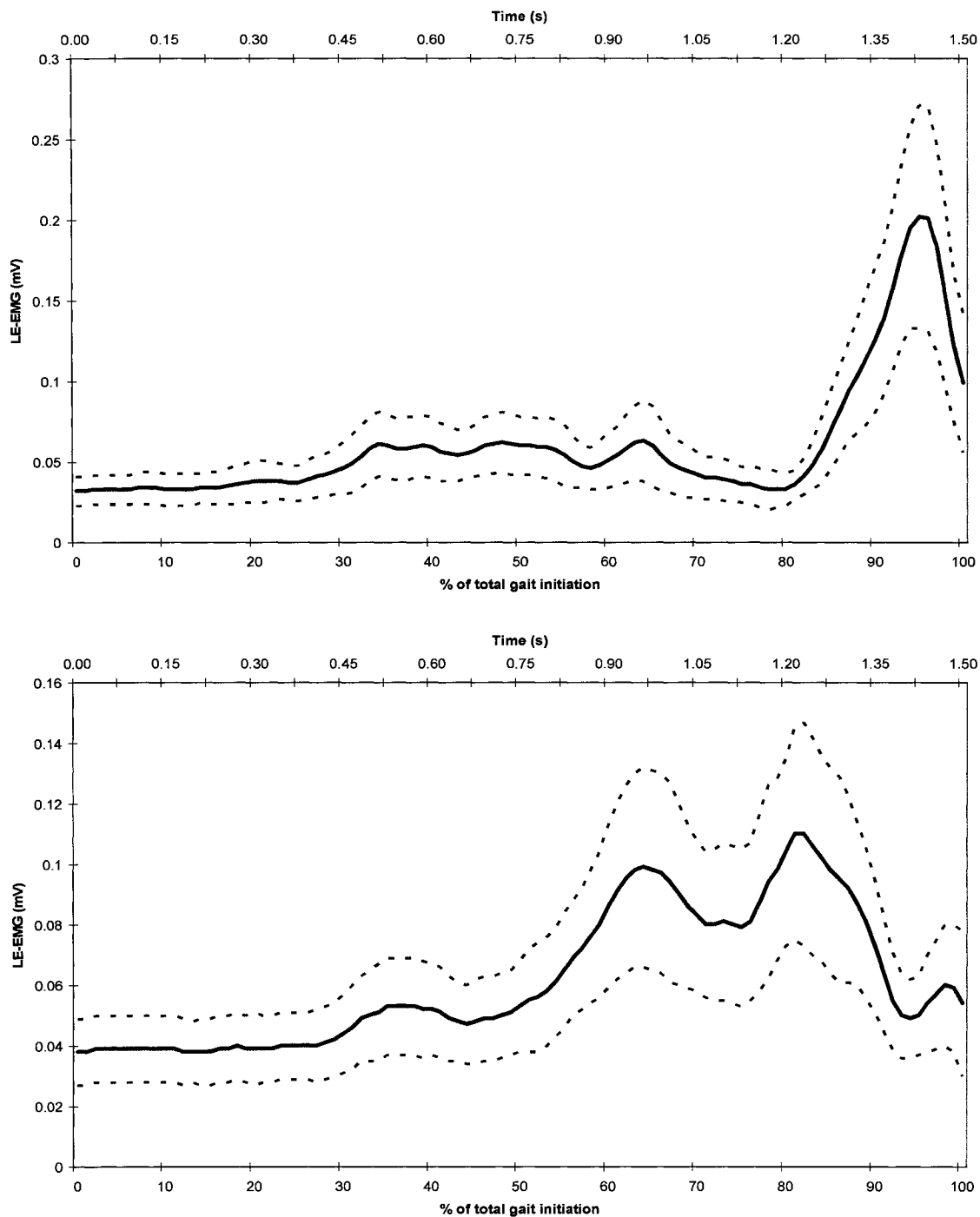


Figure 4.3: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) leg adductor magnus from 10 trials for subject eight. Time normalized linear envelope EMG throughout gait initiation.

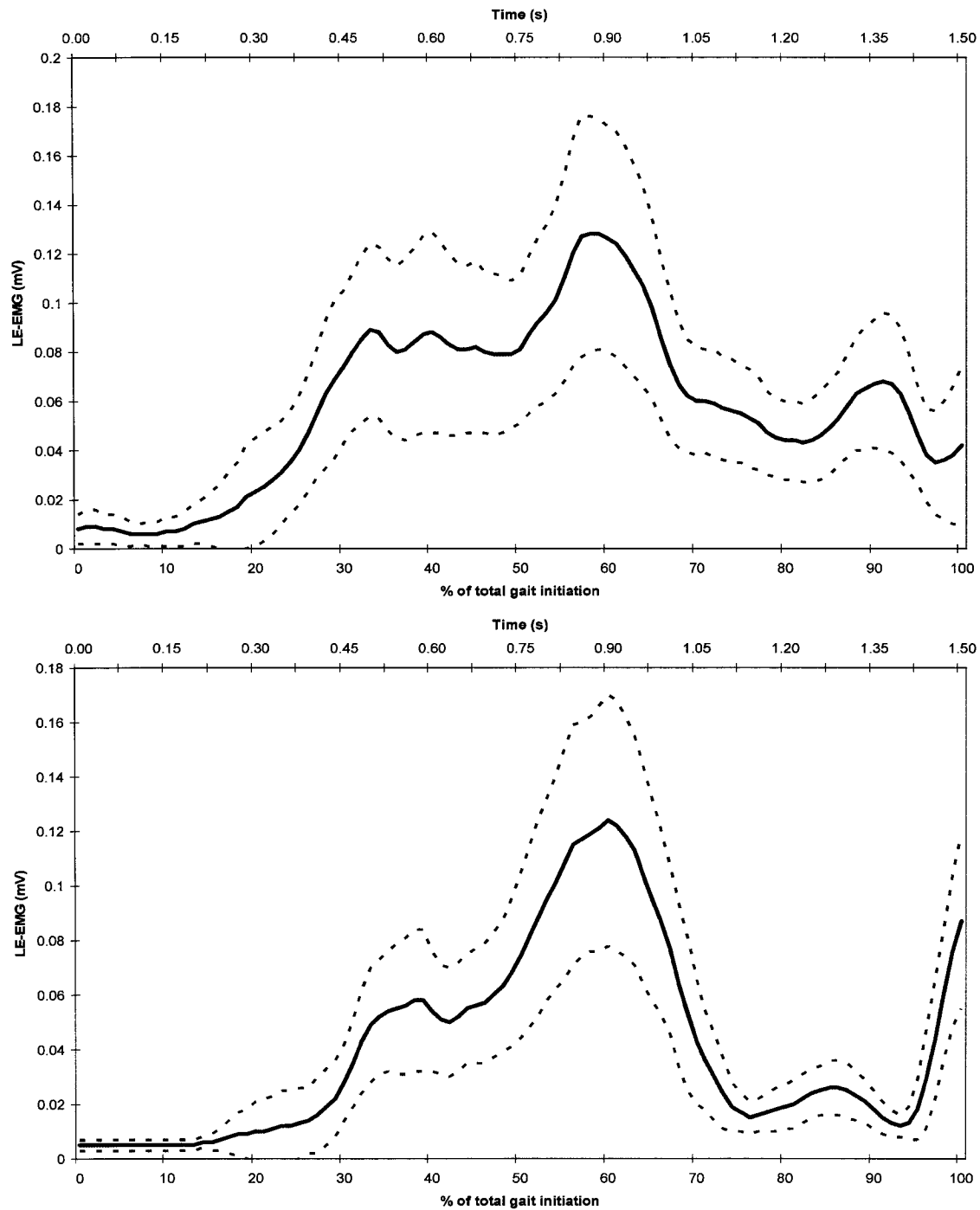


Figure 4.4: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) leg tibialis anterior from 10 trials for subject eight. Time normalized linear envelope EMG throughout gait initiation.

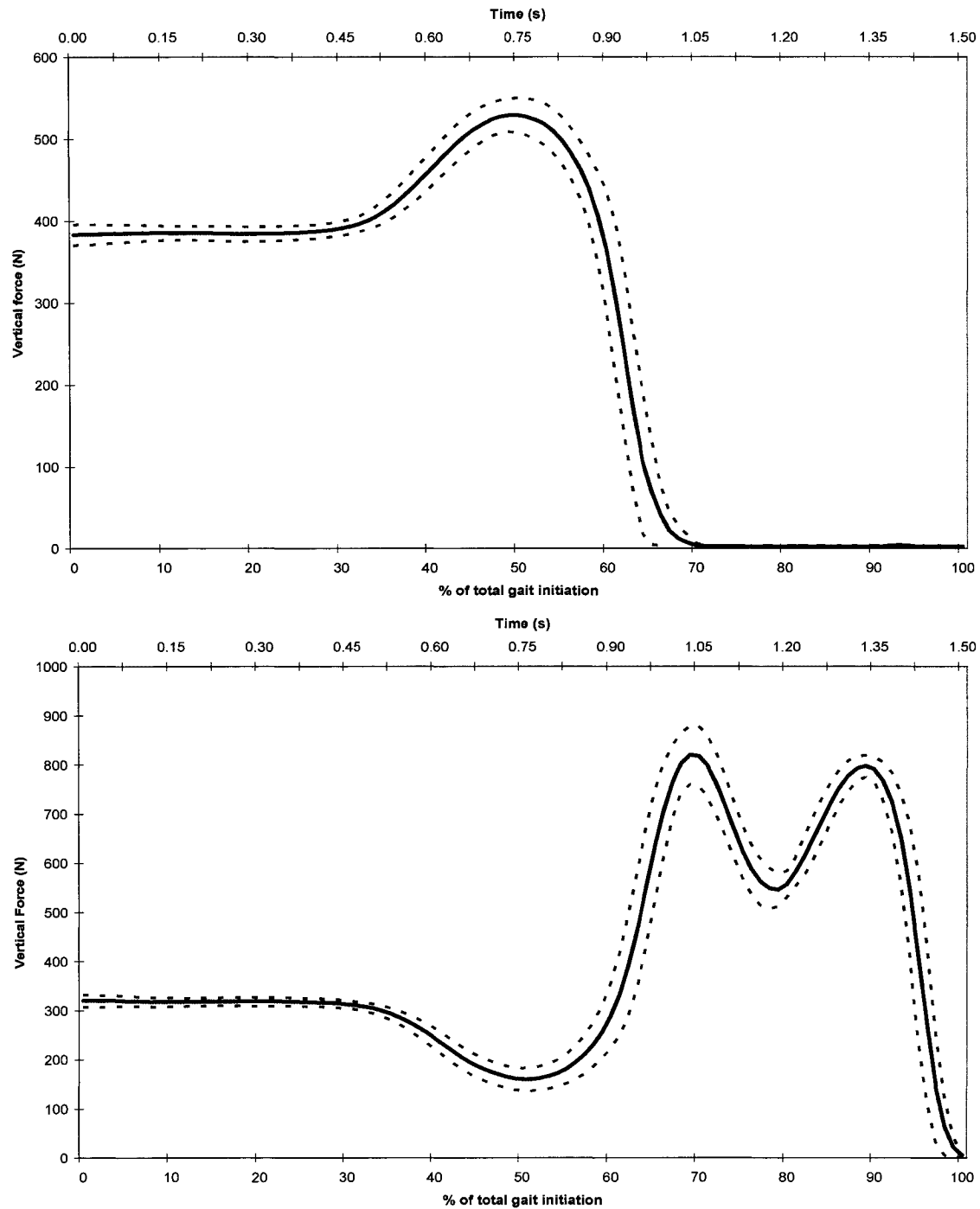


Figure 4.5: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) limb vertical ground reaction force from 10 trials for subject eight throughout gait initiation.

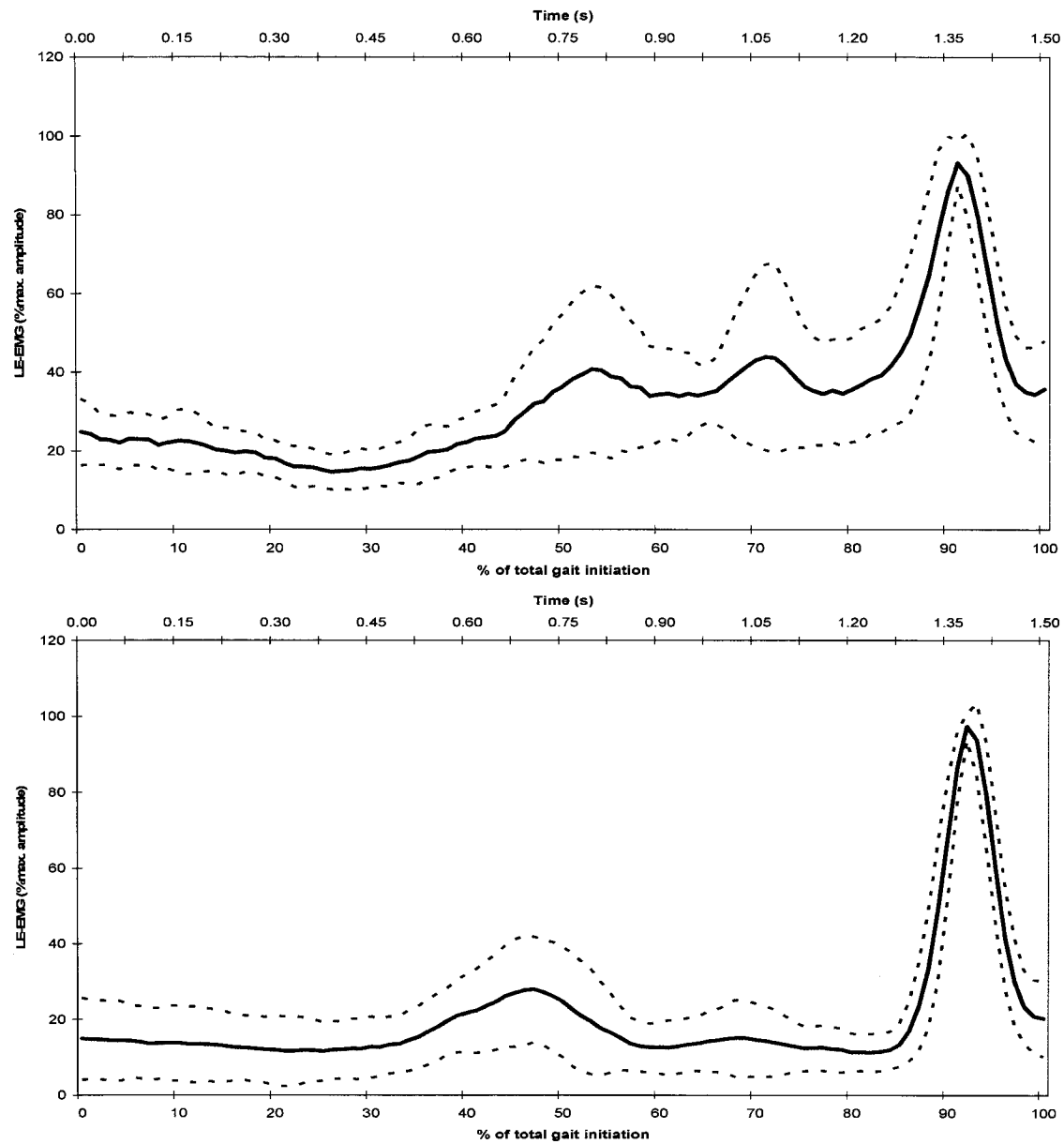


Figure 4.6: Grand ensemble LE-EMGs (\pm SD) for lead (top) and trail (bottom) erector spinae throughout gait initiation.

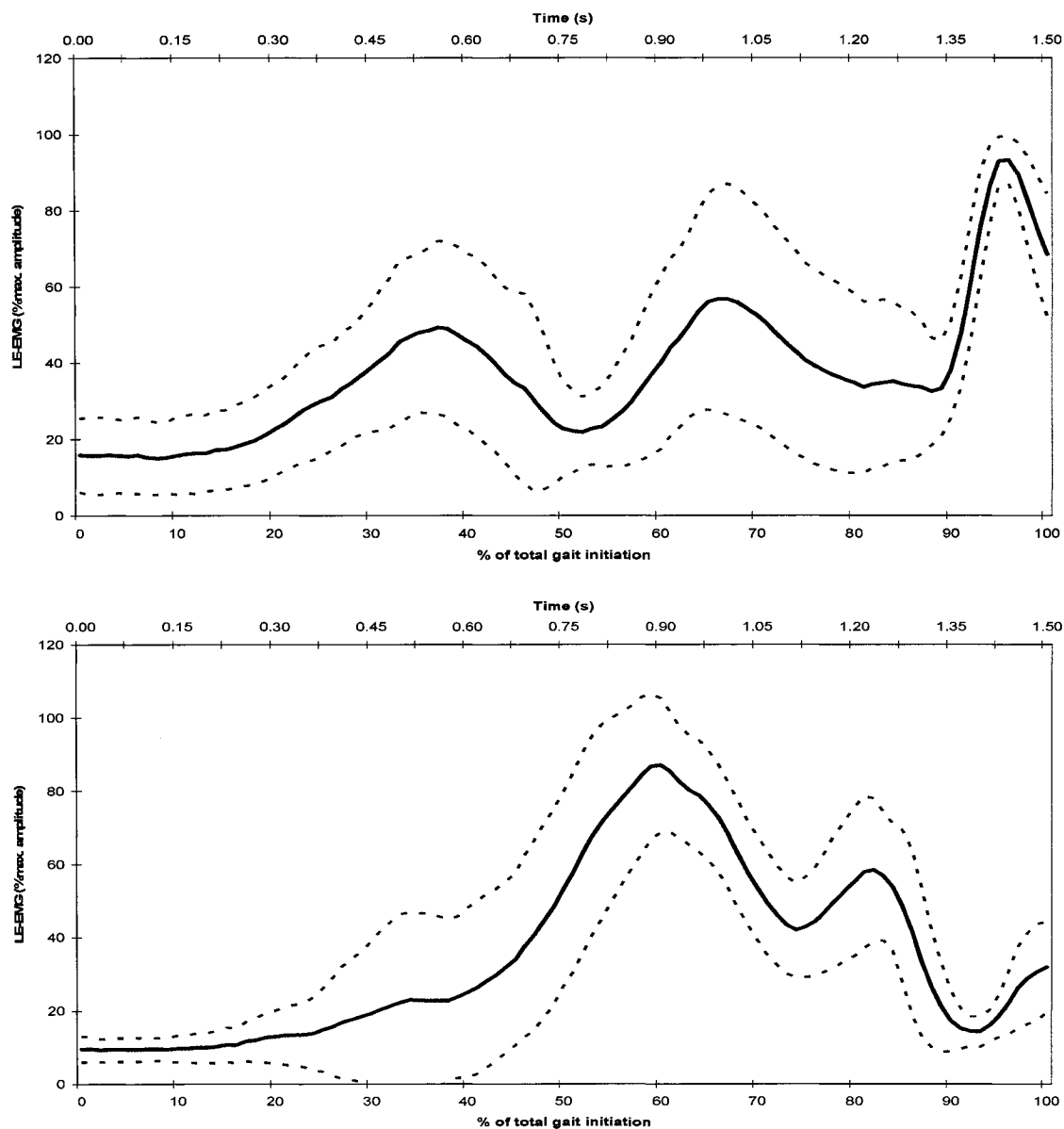


Figure 4.7: Grand ensemble LE-EMGs (\pm SD) for lead (top) and trail (bottom) tensor fasciae latae throughout gait initiation.

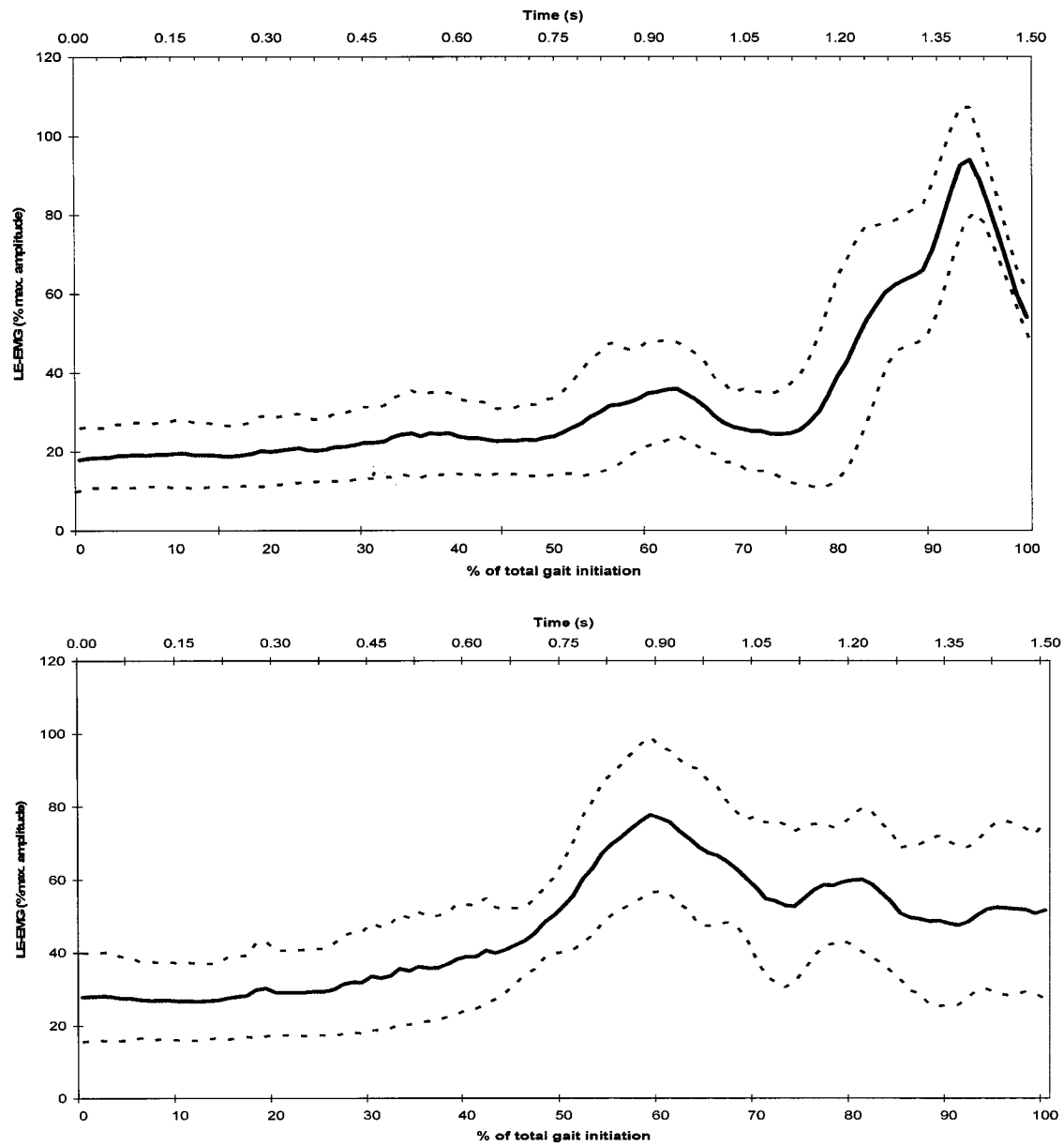


Figure 4.8: Grand ensemble LE-EMGs (\pm SD) for lead (top) and trail (bottom) adductor magnus throughout gait initiation.

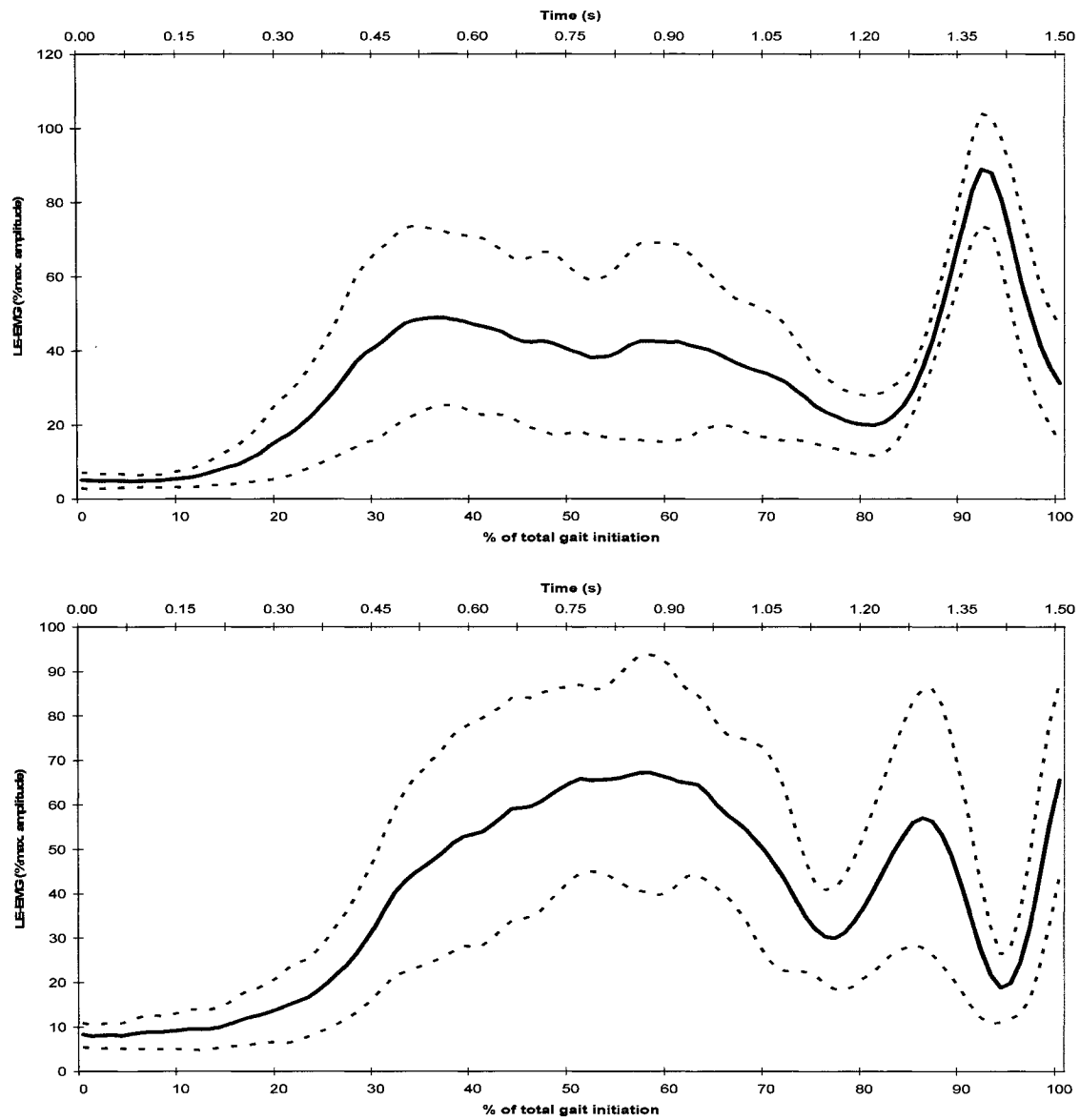


Figure 4.9: Grand ensemble LE-EMGs (\pm SD) for lead (top) and trail (bottom) tibialis anterior throughout gait initiation.

The muscle onset times from the grand ensemble averaged curves are given in Table 4.3. Similarities are seen between the patterns of muscle activity found from the ensemble-averaged data and those found from the grand ensemble averaged data. However, there were some inconsistencies in the middle of the gait initiation cycle. From the grand ensemble averaged data the lead leg adductor magnus was the fifth muscle to become active, followed by the trail leg adductor magnus, whereas the opposite was found from the ensemble-averaged data (Tables 4.3 and 4.1, respectively).

Table 4.3: Muscle onset times of the grand ensemble averaged linear envelope EMGs

<u>Muscle</u>	<u>Onset timing</u>
L-ES	44%
T-ES	35%
L-TFL	15%
T-TFL	17%
L-ADD	19%
T-ADD	23%
L-TA	13%
T-TA	16%

Note: Muscle onset timings are all expressed as a percentage of total gait initiation (0-100%). L-ES & T-ES are the lead leg and trail leg erector spinae; L-TFL & T-TFL are the lead leg and trail leg tensor fasciae latae; L-ADD & T-ADD are the lead leg and trail leg adductor magnus; L-TA & T-TA are the lead leg and trail leg tibialis anterior.

CHAPTER V

DISCUSSION

This study was performed to determine the order of lower limb and trunk muscle recruitment from quiet stance to dynamic gait and, in turn, determine the role of these muscles as well as the role of the lead leg and trail leg throughout the gait initiation process. The need for such data was evident given the varying procedures used for data collection and analysis undertaken in previous research.

From the ensemble-averaged data, the lead leg tibialis anterior was consistently the first active muscle in all but two of the subjects (Table 4.1). In one subject the trail leg tibialis anterior fired first; in the other, the trail leg tensor fasciae latae activated first (Table 4.2). The reason for this inconsistency may be due to the fact that these two subjects already had a greater weight distribution on their lead leg and, hence, when the go command was given there was less of a need to transfer weight onto the lead limb. Another possible explanation is that the subjects may have been anticipating the command, which would influence the activity of these muscles both before and at the onset of gait.

It is not surprising that the first muscle to be activated was the tibialis anterior, as the start of this muscle activity has been identified by several authors as being the primary part of the definition of the onset of gait initiation (Mann *et al.*, 1979; Brunt *et al.*, 1991; Crenna & Frigo, 1991; Elble *et al.*, 1994; Mickelborough *et al.*, 2004). The initial bilateral inhibition of the gastrocnemius/soleus activity, followed closely by a contraction of the tibialis anterior, has been identified as being responsible for the posterolateral shift of the COP towards the lead leg as well as for ankle dorsiflexion

(Cook *et al.*, 1973; Cook & Cozzens 1976; Mann *et al.*, 1979; Elble *et al.*, 1994; Elble *et al.*, 1996).

Although trail leg tibialis anterior onset has been shown by Brunt *et al.*, (1991) to come before that of the lead leg, the present data and those of Mann *et al.*, (1979), Crenna & Frigo (1991) and Mickelborough *et al.*, (2004) did not show this trend. In fact, the findings of both Mann *et al.*, (1979) and Mickelborough *et al.*, (2004) are in agreement with those of this study. Differences in detection criterion used by Crenna & Frigo (1991) and Brunt *et al.*, (1991) may explain some of the discrepancies found between these and the present study.

In 77.8% of the subjects the lead tibialis anterior muscle, once activated, remained active throughout the entire gait initiation cycle. In two subjects the lead leg tibialis anterior became inactive for a very short period and then activated again prior to lead leg toe-off. This sequence is similar to the findings of Brunt *et al.*, (1991) and Mickelborough *et al.*, (2004), in which the lead limb tibialis anterior activity continued until just before lead limb toe-off. In agreement with the two subjects from the present study Mann *et al.*, (1976) reported that the lead limb muscle activity ceased slightly before lead leg toe-off. It has been shown that the ankle dorsiflexor muscles are involved in controlling the sagittal motion of the foot to allow for safe foot placement during the early stages of stance, as well as a sufficient clearance of the toes over the ground towards the beginning of toe-off to prevent tripping (Gefen, 2001). It has also been suggested that the combined effect of the initial inhibition of the gastrocnemius/soleus activity and the bilateral burst of tibialis anterior activity is to produce an external dorsiflexion moment at the ankles

tending to rotate the body forwards over the feet and move the COM anteriorly (Mickelborough *et al.*, 2004).

The second muscle to become active, in 88.8% of the subjects, was the lead leg tensor fasciae latae (Table 4.2). This finding is similar to that found by Carlsöö (1966) and Mann *et al.*, (1979) where the tensor fasciae latae was inhibited on the trail leg and activated on the lead leg at the onset of gait initiation. Herman *et al.*, (1973) and Elble *et al.*, (1996) also reported early hip abductor activity in the lead limb during gait initiation. Changes in the vertical ground reaction force below the lead leg was observed. The action of the hip abductors may have aided to laterally stabilize the pelvis and prevent pelvic drop (Hughes, Hsu & Matava, 2002). This is supported by Winter *et al.*, (1993) and Winter *et al.*, (1996) who suggested that the hip abductors contribute to the control of frontal plane motion of the COM and the lateral loading/unloading mechanism during the stance phase.

In five of the subjects, the lead leg tensor fasciae latae remained active throughout the entire gait initiation cycle, whereas in one subject it became inactive just before lead leg toe-off and then active again later near trail leg toe-off. In two subjects a third burst of muscle activity was observed. In these subjects, the muscle also became inactive just before lead leg toe-off but then became active again towards the end of lead leg toe-off. This was followed by a slight period of inactivity before trail leg toe-off and then a third burst of muscle activity occurred near trail leg toe-off. Since the tensor fasciae latae muscle is also used to flex the hip when lifting the lead leg for toe-off, the second burst of activity could be in preparation for this event. The third burst could be explained by the fact that the lead leg must begin to bear weight when the trail leg prepares for toe-off,

thus the lead leg tensor fasciae latae would provide stability for the lead leg (Carlsöö, 1966). Similarly, both the hip adductors and abductors contribute to limb stability (Kirker *et al.*, 2000).

In 55.6% of the subjects the third muscle to be activated was the trail limb tibialis anterior (Table 4.2). As mentioned earlier, other studies have indicated that this muscle, in combination with the lead leg tibialis anterior and the bilateral inhibition of the gastrocnemius/soleus activity, have been proposed to help to initially direct the COP initially posterolaterally towards the lead leg while accelerating the COM forward and toward the trail leg, followed by the movement of the COP across to the trail leg side (Mann *et al.*, 1979; Brenière & Do, 1986; Crenna & Frigo 1991). In all of the subjects within the present study, this muscle became active at approximately the time when the vertical ground reaction force begins to increase beneath the lead leg. Since the COP was not analysed in this study it is difficult to determine whether the function of this muscle was simply to aid in the initial lateral shift towards the lead leg or to aid in both the posterior and lateral shift. It would seem more plausible that the two tibialis anterior muscles aid in the posterior movement of the COP whereas the lead leg tensor fasciae latae aids in the lateral shift, as this muscle has been identified as contributing to the lateral loading/unloading mechanism during stance phase (Winter *et al.*, 1993; Winter *et al.*, 1996).

In 77.8% of the subjects, the trail leg tibialis anterior remained active after its initial activation. In one of the subjects, the trail leg tibialis anterior became inactive when the weight began to shift onto the lead leg and became active shortly thereafter, however, the initial amplitude of the muscle activation was very minimal and therefore

this activity may not have been significant. It is therefore plausible that the true muscle activation did not occur until the second burst of activity. In the other subject, the trail leg tibialis anterior became inactive near trail leg-toe off and then became active again shortly thereafter at trail leg toe-off. This is similar to results reported by Mann *et al.*, (1976) where the trail limb tibialis anterior remained active until slightly after lead leg toe-off and did not become active again until somewhat after trail leg toe-off. It is at this time when the ankle must dorsiflex to create sufficient clearance of the toes over the ground to prevent tripping (Gefen, 2001).

The fourth and fifth muscles to become active were not as apparent, as only 44.4% of the subjects' trail leg tensor fasciae latae became active fourth, while in 33.3% of the subjects it was the trail leg adductor magnus (Table 4.2). The mean onset times expressed as a percentage of total gait initiation cycle for these two muscles, shown in Table 4.1, suggest that both the leg trail tensor fasciae latae and the trail leg adductor magnus were activated almost simultaneously (26.7% and 27.2%, respectively). It appears that the fifth muscle to become active was the trail adductor magnus; however, once again, only 44.4% of the subjects demonstrated this muscle activity. This muscle, once active, remained active in all of the subjects.

The initial amplitude of the trail leg tensor fasciae latae and trail leg adductor magnus activity was very small, and it was not until later that they become highly active. This increase in amplitude coincides with toe-off of the lead leg. It is not surprising that the activity in these two muscles would increase at this time, as the trail leg must accept the entire weight of the body when lead leg toe-off occurs. As hip abductors and adductors, they would both assist in limb support and pelvic stability (Carlsöö, 1966;

Mann *et al.*, 1979). In all of the subjects, once the trail leg tensor fasciae latae became active it remained active until trail leg toe-off.

The sixth muscle to become active was not easy to determine, as only 33.3% of the subjects' lead leg adductor magnus was activated, with 22.2% of the subjects' trail leg tensor fasciae latae firing, while in another 22.2% of the subjects' trail leg adductor magnus was the next to become active. It is not clear why the lead leg adductor magnus would become active at this point. It would seem more logical that it would have become active either simultaneously with the lead leg tensor fasciae latae or soon after, as they both have the same function of hip flexion and hip joint stabilization. The large standard deviation found in Table 4.1 indicates that there was considerable variability among subjects for this muscle. Additionally, the large amounts of fat tissue overlying this muscle and slight differences in electrode placement could have affected the muscle reading.

The final two muscles to become active are the erector spinae on the trail limb side followed by the erector spinae on the lead limb side (Table 4.1). In 55.6% of the subjects the trail side erector spinae was the seventh muscle to become active, while in 33.3% of the subjects it was the lead side erector spinae. The large standard deviation found in Table 4.1 for this muscle indicates that there was considerable between subject variability, however, this may be due to two outliers. The lead side erector spinae on the other hand had a smaller standard deviation and in 66.7% of the subjects this muscle was the last muscle to become active.

In seven of the subjects the burst of activity in the trail side erector spinae was associated with an increase in the ground reaction force beneath the lead leg. At this

moment, the trail side erector spinae may aid in preventing the individual from falling over to the right by laterally flexing the vertebral column, thus recovering stability (White & McNair, 2002). The burst of activity in the lead side erector spinae was associated with an increase in the ground reaction force beneath the trail leg and a subsequent decrease beneath the lead leg during its preparation for toe-off. The lead side erector spinae may have assisted in the control of the forward fall and may also be preventing the individual from collapsing to the left and thus bringing the COM back into alignment under the support base. It has been suggested that this muscle plays a role in decreasing the overall vertical displacement of the body, and thus lead to a smoother trajectory for the COM during the stride cycle (White & McNair, 2002).

In six of the subjects the trail side erector spinae remained active until body weight was primarily shifted onto the trail leg and then became inactive for a short period of time. The second burst occurred at approximately trail leg toe-off at which point lead leg heel-strike occurred. Interestingly, another function of the trunk muscles is shock absorption at foot-strike (White & McNair, 2002). In the remaining subjects the muscle remained active until trail leg toe-off. The lead side erector spinae remained active until trail leg toe-off in all but two subjects, in which there was a second burst occurring slightly before trail leg-toe off. Shiavi (1990) considered this activity to be related to weight transference between limbs and to the reversal in direction of pelvic and thoracic rotation. He suggested that these muscles contract bilaterally to balance the torso and pelvis in preparation for swing phase.

The order at which the muscles became active from the grand ensemble-averaged data, shown in Table 4.3, show similarities to those found from the individual subject

ensemble averages. Differences, however, arise in the order of the fifth and sixth muscles to become. From the ensemble-averaged data there was uncertainty as to which muscle became active fifth and sixth. It was unclear as to whether it was the lead leg adductor magnus, or the trail leg adductor magnus. From the grand ensemble averaged data the fourth muscle to become active was the trail leg tensor fasciae latae, followed by activity in the lead leg adductor magnus, and then the trail leg adductor magnus. The seventh and eighth muscles to become active found from the grand ensemble averaged data match those found in the ensemble-averaged data.

The vertical reaction forces in this study are similar to that determined in previous works by Brunt *et al.*, (1991) and Patchay & Gahery (2003), where there was an initial increase in the vertical reaction force under the lead leg and a subsequent decrease in the trail leg, followed by a decrease in the vertical reaction forces under the lead leg in preparation for toe-off and an increase in the trail leg vertical reaction force in preparation for weight bearing. This initial loading of the lead leg increased the thrust against the ground and consequently increases the vertical ground reaction force thus aiding the lifting of the limb (Tokuno *et al.*, 2003).

Across subjects and across muscles, there is a wide range of peak amplitudes recorded for each subject's ensemble average EMG curves. Some of the amplitude differences could be due to muscle bulk, fat tissue overlying the muscle, minor differences in the electrode placement, poor skin resistance (Winter & Yack, 1987) and the speed at which the subjects initiated gait (Cook & Cozzens, 1976; Crenna & Frigo, 1991).

Overall, the initial hypothesis corresponded with the present data. The major differences were the time at which the erector spinae muscles became active. It was hypothesized that they would occur after the opposite hip abductor and adductor muscles to produce counter-flexion and lateral flexion moment of the trunk. Perhaps the failure of the erector spinae muscles to become active at this time may have been obscured by the process of ensemble averaging. The other main difference occurred in the order in which the lead adductor magnus became active. It was hypothesized that the adductor magnus would co-contraction to stabilize the hip joint against the action of the hip abductors of the same leg and would therefore become active either simultaneously with the lead leg tensor fasciae latae or shortly thereafter. This was, however, found to be true for the trail leg. Since only 33.3% of the subjects activated their lead leg adductor magnus sixth whereas in 22.2% of the subjects it was found that the lead leg adductor magnus either became active third or fourth, this would suggest that perhaps there were errors in surrounding the determination of the lead leg adductor magnus activity or the earlier detection of activity may have been obscured by the process of ensemble averaging.

CHAPTER VI

CONCLUSIONS AND RECOMMENDATIONS

The current study provides a baseline of normal gait initiation muscle activity against which to compare that of patients with gait initiation and balance difficulties. The results give an indication of the patterns of activity for normal adults during gait initiation. Knowledge of the muscle activation patterns before lead leg toe-off may lead to improved rehabilitation treatment strategies to reduce the risk of falling and to aid patients encountering difficulties in executing their first step.

On the basis of this study, initiation of gait began with the activation of the lead leg tibialis anterior which was followed closely by the activation of the lead leg tensor fasciae latae. The trail limb tibialis anterior was the next muscle to become activate. During this time there was a slight increase in the vertical ground reaction force below the lead leg with a simultaneous decrease below the trail leg. Therefore it is believed that these aforementioned muscles play a role in the shifting of the weight towards the lead leg. In addition, it has been suggested that the lead leg tensor fasciae latae functions to stabilize the hip and leg. The vertical ground reaction force slowly begins to increase below the trail leg and simultaneously decreases below the lead limb. This is in preparation for lead leg toe-off. The next muscle to become active was the trail leg tensor fasciae latae, which was followed closely by the muscle activation of the trail leg adductor magnus. It was suggested that these two muscles also provide stability for the hip and leg.

The next muscle to become active was the lead leg adductor magnus. Exactly why this muscle became active at this point in the gait initiation process is not completely

understood. It could be preventing lead leg hip abduction during trail leg toe-off. Lastly the erector spinae on the trail side became active followed by the erector spinae on the lead side. It is believed that these muscles aid in the generation and control of motion between the trunk and pelvis.

Future studies should include the synchronization of the path of the combined COP, COM, and vertical ground reaction forces with the EMG of several muscles to give further insight into the functional role of those muscles during gait initiation. Additionally, a detailed inverse dynamic analysis combined with a mathematical model, could be used to provide information about individual muscle forces. Furthermore, a study addressing the effects of initiating gait with the contralateral limb should be carried out to determine if there are similar strategies used. Finally, a study using participants suffering from gait initiation difficulties should be performed to determine the specificity of our results.

REFERENCES

- Brenière, Y., Do, M.C. & Sanchez, J. (1981). A biomechanical study of the gait initiation process. *Journal Français de Biophysique & Médecine Nucléaire*, 5, 197-205.
- Brenière, Y. & Do, M.C. (1986). When and how does steady state gait movement induced from upright posture begin? *Journal of Biomechanics*, 19, 1035-1040.
- Brenière, Y., Do, M.C. & Bouisset, S. (1987). Are dynamic phenomena prior to stepping essential to walking? *Journal of Motor Behavior*, 19, 62-76.
- Brunt D., Lafferty, M.J., Mckeon, A., Goode, B., Mulhausen, C. & Polk, P. (1991). Invariant characteristics of gait initiation. *American Journal of Physical Medicine & Rehabilitation*, 70, 206-212.
- Brunt, D., Vander Linden, D.W. & Behrman, A.L. (1995). The relation between limb loading and control parameters of gait initiation in persons with stroke. *Archives of Physical Medicine and Rehabilitation*, 76, 627-634.
- Brunt, D., Liu, S., Trimble, M., Bauer, J. & Short, M. (1999). Principles underlying the organization of movement initiation from quiet stance. *Gait and Posture*, 10, 121-128.
- Burleigh, A.L., Horak, F.B. & Malouin, F. (1994). Modification of postural responses and step initiation: evidence for goal-directed postural interactions. *Journal of Neurophysiology*, 72, 2892-2902.
- Carlsöö, S. (1966). The initiation of walking. *Acta Anatomica*, 65, 1-9.
- Chang, H. & Krebs, D.E. (1999). Dynamic balance control in elders: gait initiation assessment as a screening tool. *Archives of Physical Medicine and Rehabilitation*, 80, 490-494.
- Couillandre, A., Breniere, Y. & Maton, B. (2000). Is human gait initiation program affected by a reduction of the postural basis? *Neuroscience Letters*, 285, 150-154.
- Crenna, P., Frigo, C., Giovannini, P. & Piccolo, I. (1990). The initiation of gait in Parkinson's disease. In *Motor Disturbances*, ed. Berardelli, A., 161-173. Orlando: Academic Press.
- Crenna, P. & Frigo, C. (1991). A motor programme for the initiation of forward-oriented movements in humans. *Journal of Physiology*, 437, 635-653.
- DeLuca, C.J. (2002). Surface Electromyography: detection and recording. DelSys Inc.

- Dietrich, G., Breniere, Y. & Do, M.C. (1994). Organization of local anticipatory movements in single step initiation. *Human Movement Science*, 13, 195-210.
- Ditz, V. & Colombo, G. (1998). Influence of body load on the gait pattern in Parkinson's disease. *Movement Disorder*, 13, 255-261.
- Elble, R.J., Moody, C., Leffler, K. & Sinha, R. (1994). The initiation of normal walking. *Movement Disorder*, 9, 139-146.
- Elble, R.J., Cousins, R., Leffler, K. & Hughes, L. (1996). Gait initiation by patients with lower-half parkinsonism. *Brain*, 119, 1705-1716.
- Gantchev, N., Viallet, F., Aurenty, R. & Massion, J. (1996). Impairment of posturo-kinetic co-ordination during initiation of forward oriented stepping movements in parkinsonian patients. *Electroencephalography and Clinical Neurophysiology*, 101, 110-120.
- Gefen, A. (2001). Simulations of foot stability during gait characteristic of ankle dorsiflexor weakness in the elderly. *IEEE Transactions on Neural Systems and Rehabilitation Engineering*, 9(4), 333-337.
- Gélat, T. & Breniere, Y. (2000). Adaptation of the gait initiation process for stepping on to a new level using a single step. *Experimental Brain Research*, 133, 538-546.
- Halliday, S.E., Winter, D.A., Frank, J.S., Patla, A.E. and Prince, F. (1998). The initiation of gait in young, elderly, and Parkinson's disease subjects. *Gait and Posture*, 8, 8-14.
- Herman, R., Cook, T., Cozzens, B. & Freedman, W. (1973). Control of postural reactions in man: the initiation of gait. In *Control of Posture and Locomotion*, ed: Stein, R.B., Pearson, K.G., Smith, R.S., Redford, J.B., 363-388, New York.: Plenum Press.
- Herman, R., Wirta, R., Bampton, S. & Finley, F.R. (1976). Human solutions for locomotion: single limb analysis. In *Neural Control of Locomotion*, ed: Herman R.M., Grillner, S., Stein, P.S.G., Stuart, D.G., 13-49, New York: Plenum Press.
- Hughes, P., Hsu, J. & Matava. (2002). Hip anatomy and biomechanics in the athlete. *Sports Medicine and Arthroscopy Review*, 10, 103-114.
- Jian, Y., Winter, D.A., Ishac, M.G. & Gilchrist, L. (1993). Trajectory of the body COG and COP during initiation and termination of gait. *Gait and Posture*, 1, 9-22.
- Knutson, L.M. & Soderberg, G.L. (1995). EMG: use and interpretation in gait. In *Gait Analysis Theory and Application*, ed. Craik, R.L., & Oatis, C.A., 307-325. New-York: Mosby-Year Book, Inc.

- Ludewig, P.M. & Cook, T.M. (2000). Alterations in shoulder kinematics and associated muscle activity in people with symptoms of shoulder impingement. *Physical Therapy*, 80, 276-291.
- Mann, R.A., Hagy, J.L., White, V. & Liddell, D. (1979). The initiation of gait. *The Journal of Bone and Joint Surgery*, 61-A, 232-239.
- Mickelborough, J., Linden, M.L., Tallis, R.C. & Ennos, A.R. (2004). Muscle activity during gait initiation in normal elderly people. *Gait and Posture*, 19, 50-57.
- Nolan, L. & Kerrigan, D.C. (2003). Keep on your toes: gait initiation from toe-standing. *Journal of Biomechanics*, 36, 393-401.
- Patchay, S., Gahery, Y. and Serratrice, G. (1997). Gait initiation and impairments of ground reaction forces as illustrated in old age by 'La marche a petits pas'. *Neuroscience Letters*, 236, 143-146.
- Patchay, S., Gahery, Y. & Serratrice, G. (2002). Early postural adjustments associated with gait initiation and age-related walking difficulties. *Movement Disorders*, 17, 317-326.
- Patchay, S. & Gahery, Y. (2003). Effect of asymmetrical limb loading on early postural adjustments associated with gait initiation in young healthy adults. *Gait and Posture*, 18, 85-94.
- Polcyn, A.F., Lipsitz, L.A., Kerrigan, D.C. & Collins, J.J. (1998). Age-related changes in the initiation of gait: degradation of central mechanisms for momentum generation. *Archives of Physical Medicine and Rehabilitation*, 79, 1582-1589.
- Robertson, D.G.E. (2005). Biomech Motion Analysis System. Available online: <http://www.health.uottawa.ca/biomech/csb/software/biomech.htm>.
- Robertson, D.G.E & Dowling, J.J. (2003). Design and responses of Butterworth and critically damped digital filters. *Journal of Electromyography and Kinesiology*, 13, 569-573.
- Herman, R., Wirta, R., Bampton, S. & Finley, F.R. (1976). Human solutions for locomotion: single limb analysis. In *Neural Control of Locomotion*, ed: Herman R.M., Grillner, S., Stein, P.S.G., Stuart, D.G., 13-49, New York: Plenum Press.
- Shiavi, R. (1990). Electromyographic patterns in normal adult locomotion. In : *Gait in Rehabilitation*, ed: Smidt, G.L., 97-119, New York : Churchill Livingstone.
- Tokuno, C.D., Sanderson, D.J., Inglis, T. & Chua, R. (2003). Postural and movement adaptations by individuals with unilateral below-knee amputation during gait initiation. *Gait and Posture*, 18, 158-169.

- Vaugoyeau, M., Viallet, F., Mesure, S. & Massion, J. (2003). Coordination of axial rotation and step execution: deficits in Parkinson's disease. *Gait and Posture*, 18, 150-157.
- White, S. & McNair, P. (2002). Abdominal and erector spinae muscle activity during gait: the use of cluster analysis to identify patterns of activity. *Clinical Biomechanics*, 17, 177-184.
- Winter, D.A. (1995). Human balance and posture control during standing and walking. *Gait and Posture*, 3, 193-214.
- Winter, D.A. (2005). *Biomechanics and Motor Control of Human Movement*. New Jersey: John Wiley & Sons.
- Winter, D.A., Fuglevand, A.J. & Archer, S.E. (1994). Crosstalk in surface EMG: theoretical and practical estimates. *Journal of Electromyography and Kinesiology*, 4, 15-26.
- Winter, D.A., Prince, F., Frank, J.S., Powell, C., & Zabjek, K.F. (1996). Theory Regarding A/P and M/L balance in quiet stance. *Journal of Neurophysiology*, 75, 2334-2343.
- Winter, D.A. & Yack, H.J. (1987). EMG profiles during normal human walking: stride-to-stride and inter-subject variability. *Electroencephalography and Clinical Neurophysiology*, 67, 402-411.

APPENDIX A

Muscle Activation Patterns of Eight Muscles from Eight Subjects

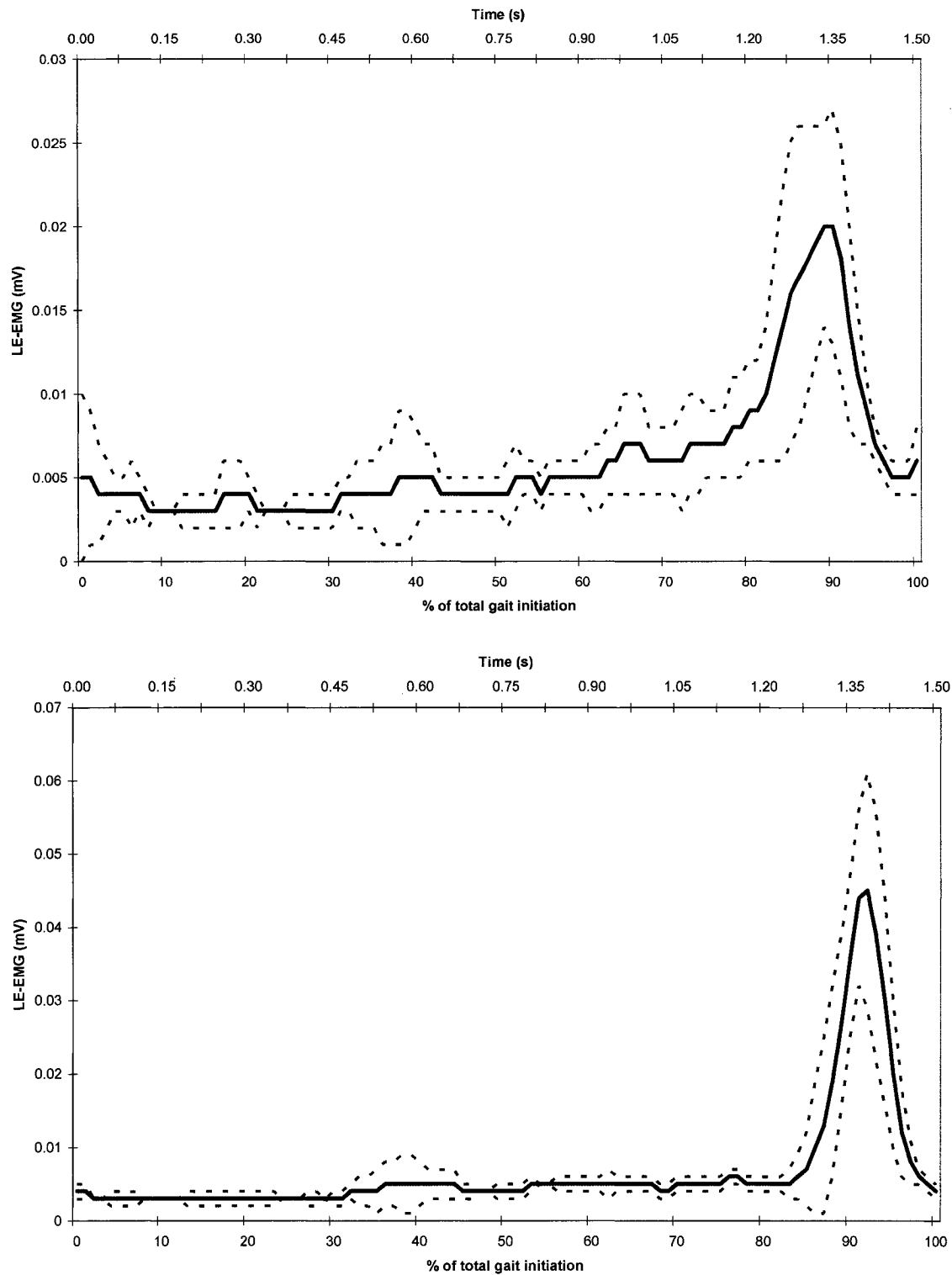


Figure A.1: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) side erector spinae from 10 trials for subject one. Time normalized linear envelope EMG throughout gait initiation.

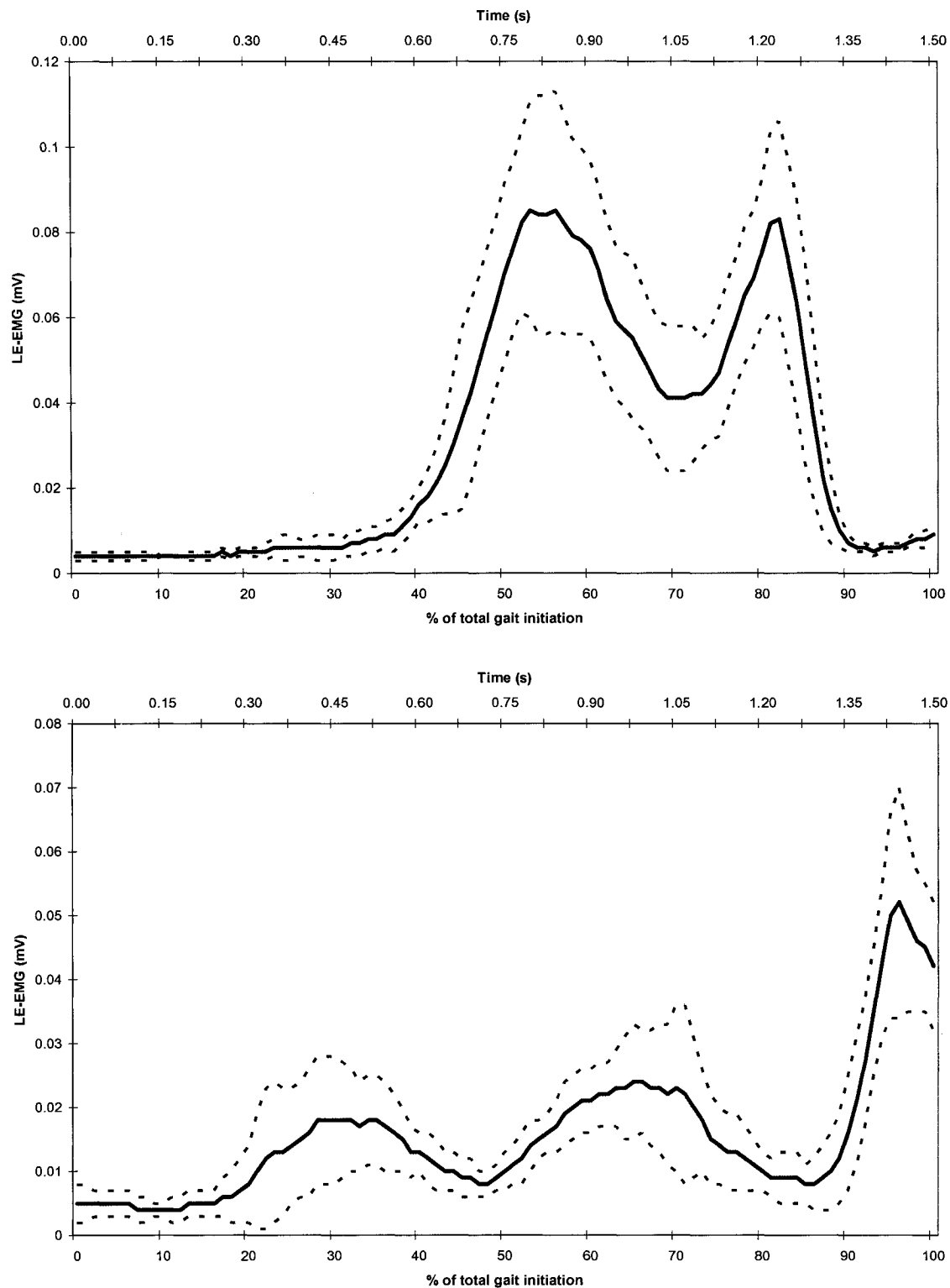


Figure A.2: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) leg tensor fasciae latae from 10 trials for subject one. Time normalized linear envelope EMG throughout gait initiation.

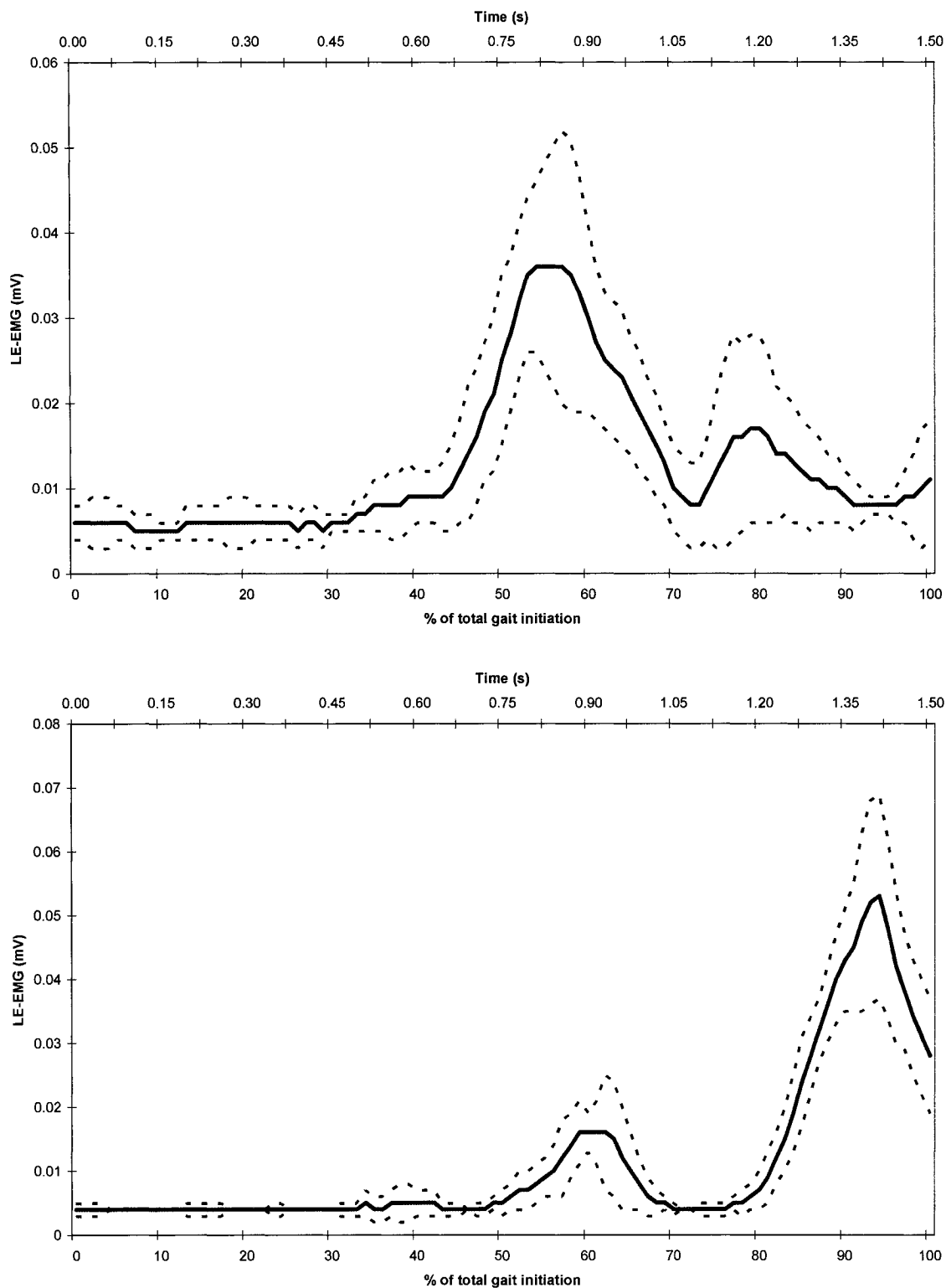


Figure A.3: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) leg adductor magnus from 10 trials for subject one. Time normalized linear envelope EMG throughout gait initiation.

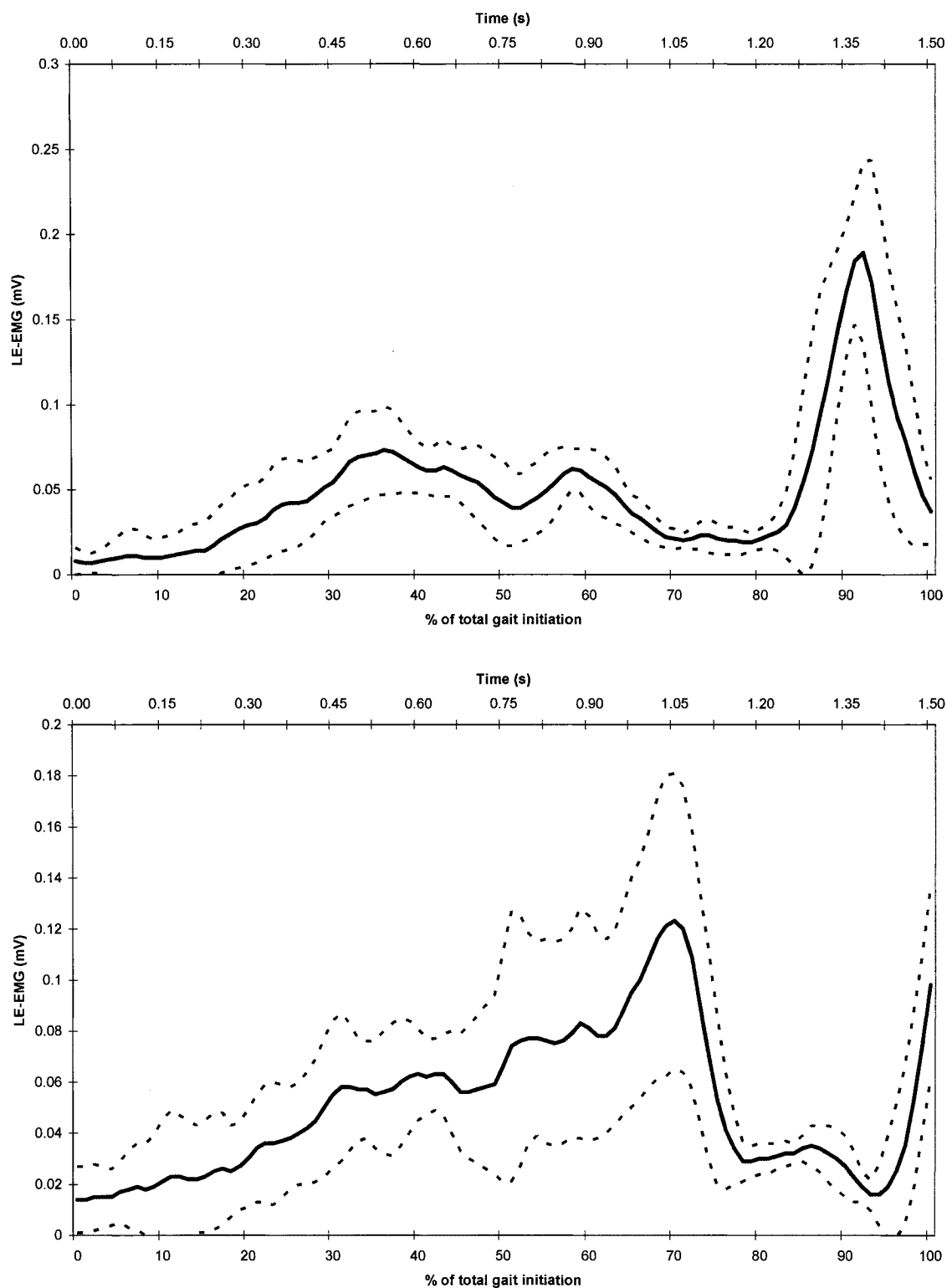


Figure A.4: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) leg tibialis anterior from 10 trials for subject one. Time normalized linear envelope EMG throughout gait initiation.

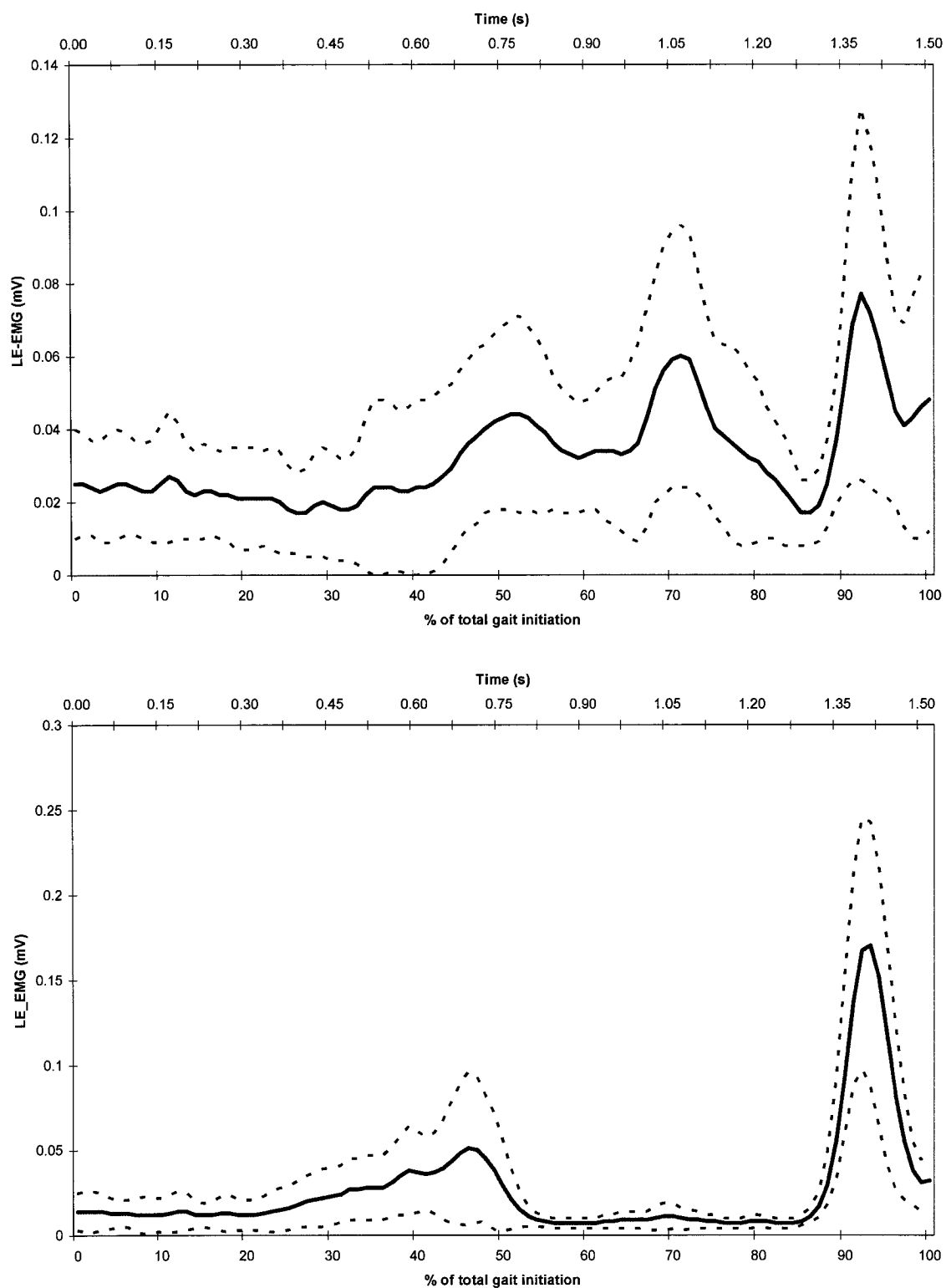


Figure A.5: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) side erector spinae from 10 trials for subject two. Time normalized linear envelope EMG throughout gait initiation.

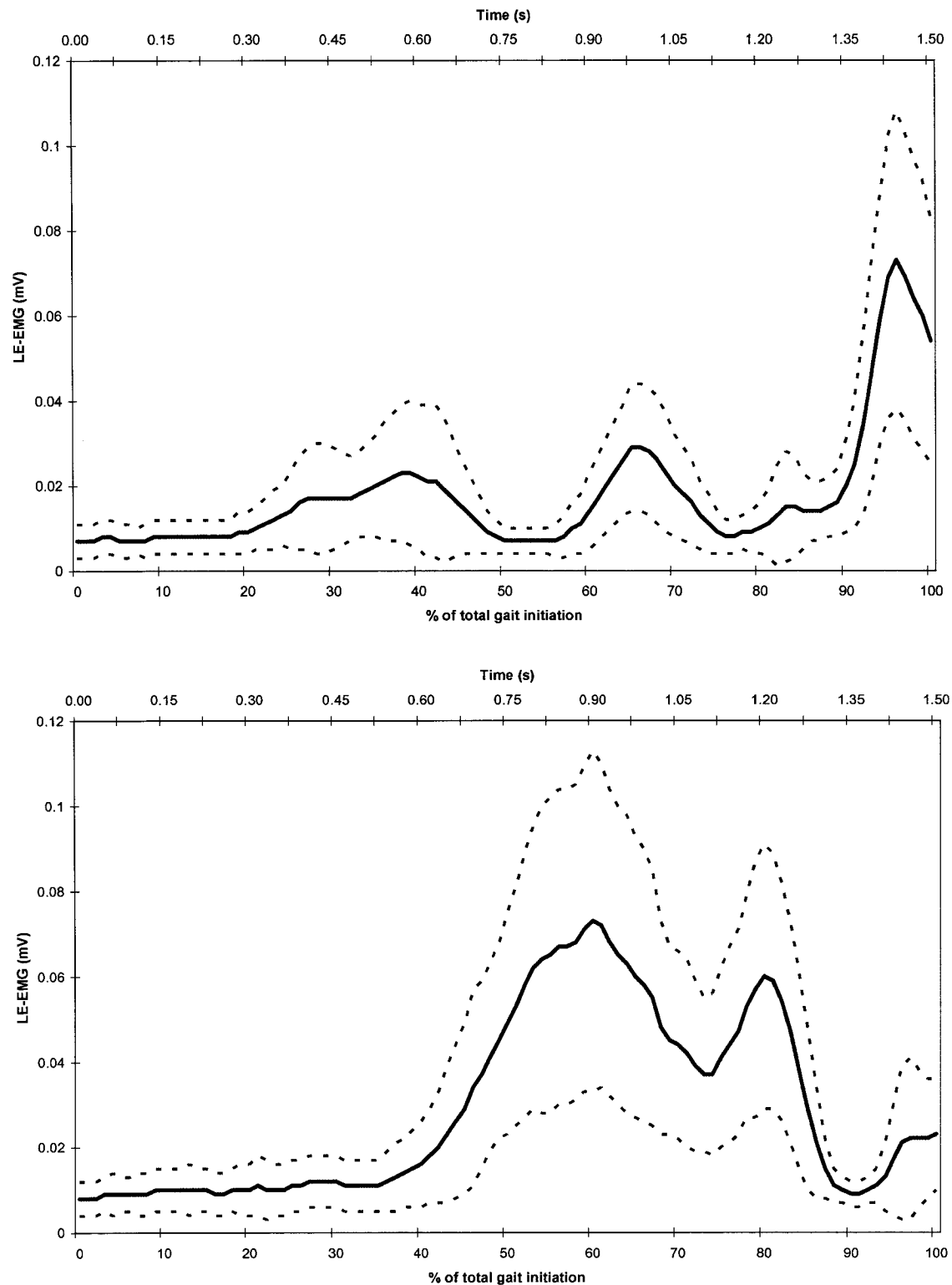


Figure A.6: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) leg tensor fasciae latae from 10 trials for subject two. Time normalized linear envelope EMG throughout gait initiation.

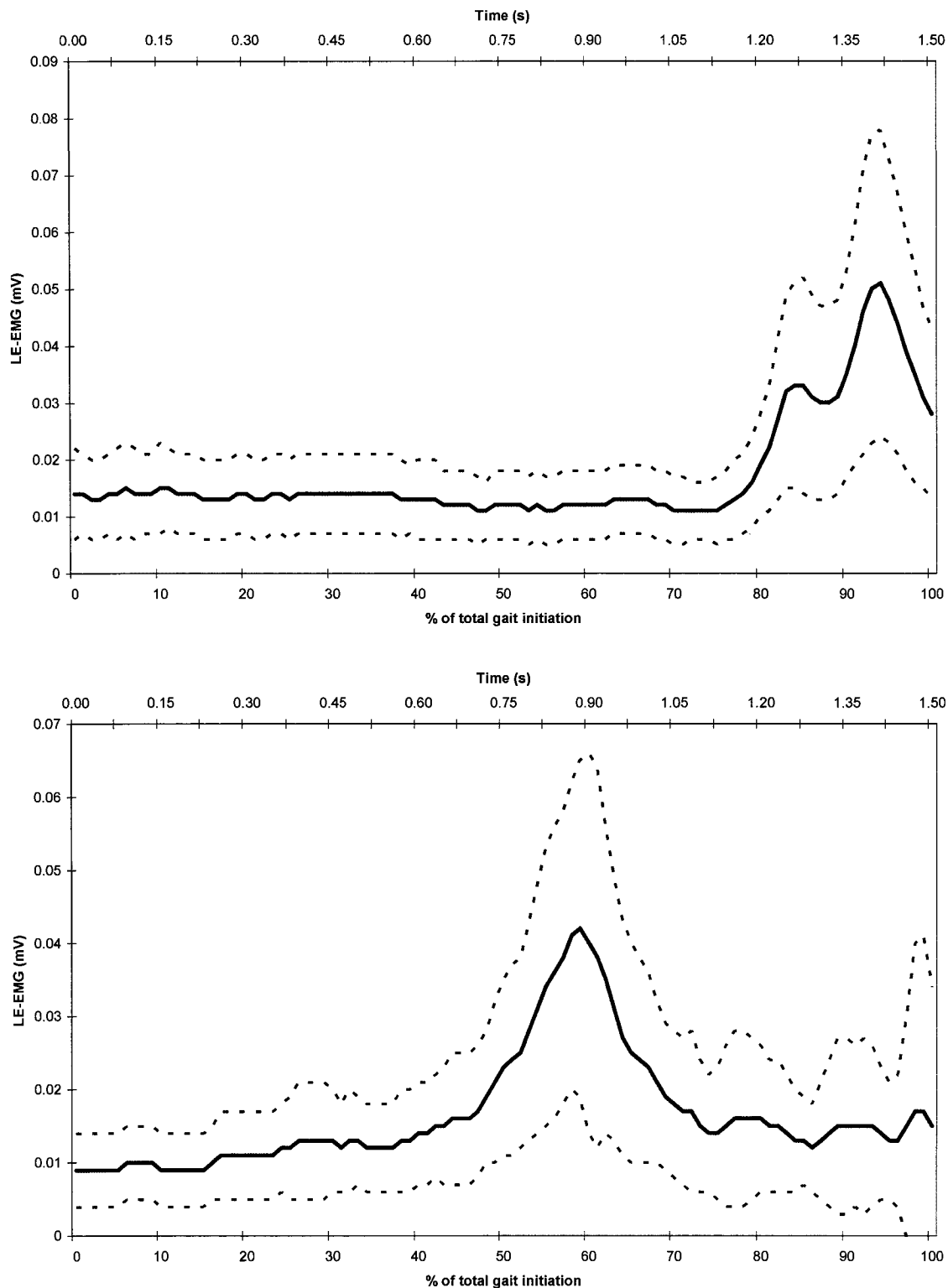


Figure A.7: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) leg adductor magnus from 10 trials for subject two. Time normalized linear envelope EMG throughout gait initiation.

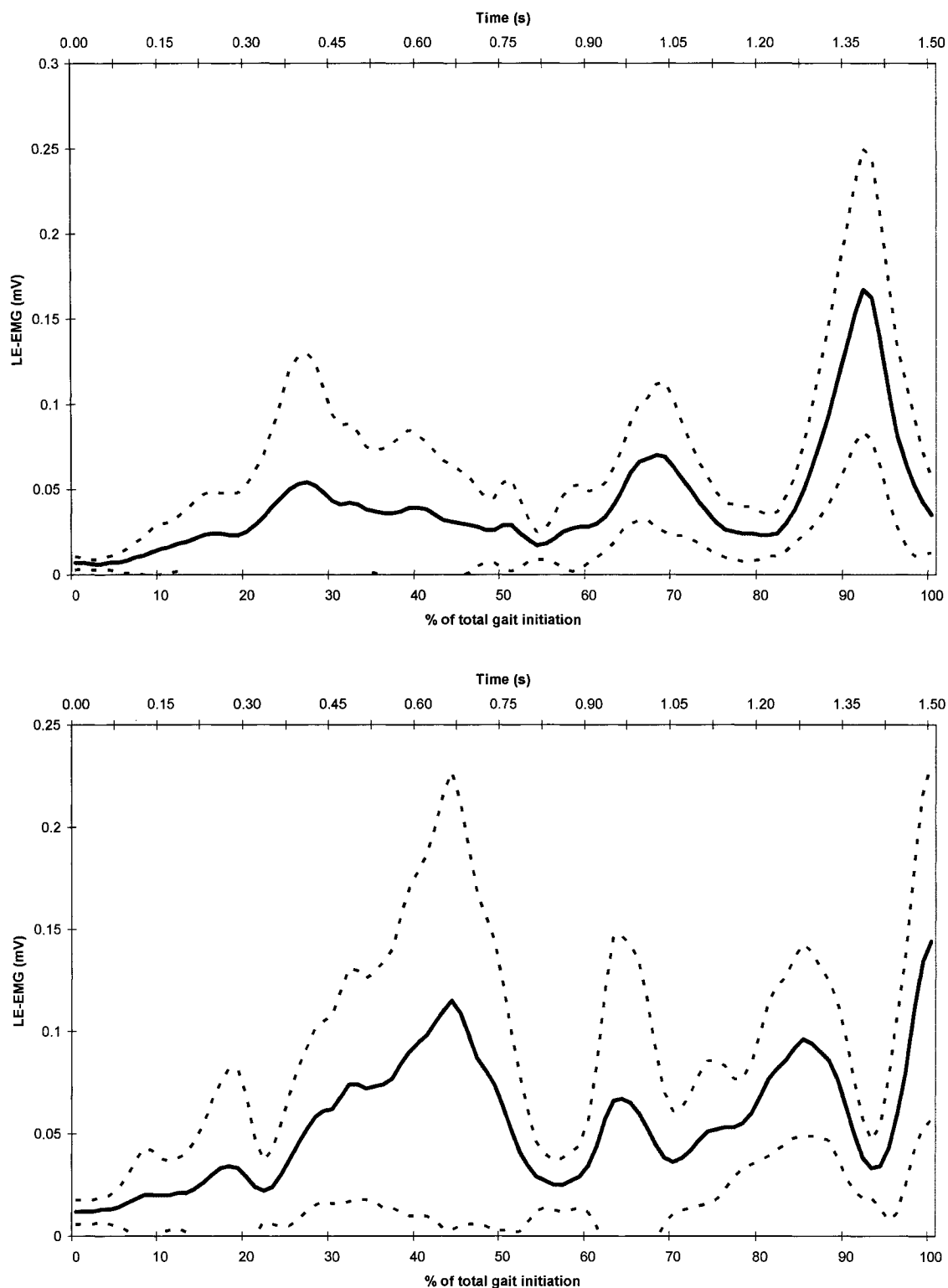


Figure A.8: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) leg tibialis anterior from 10 trials for subject two. Time normalized linear envelope EMG throughout gait initiation.

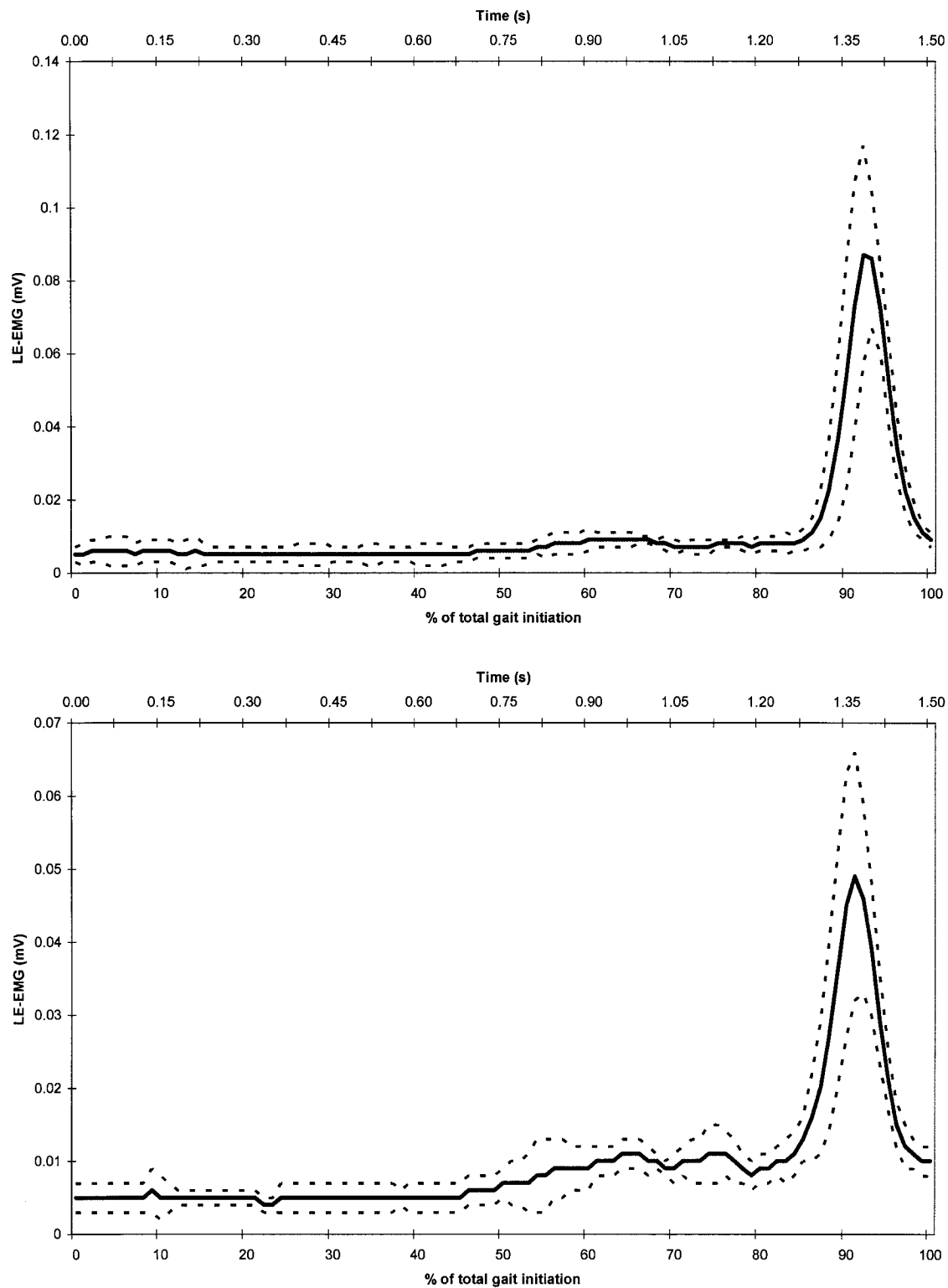


Figure A.9: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) side erector spinae from 10 trials for subject three. Time normalized linear envelope EMG throughout gait initiation.

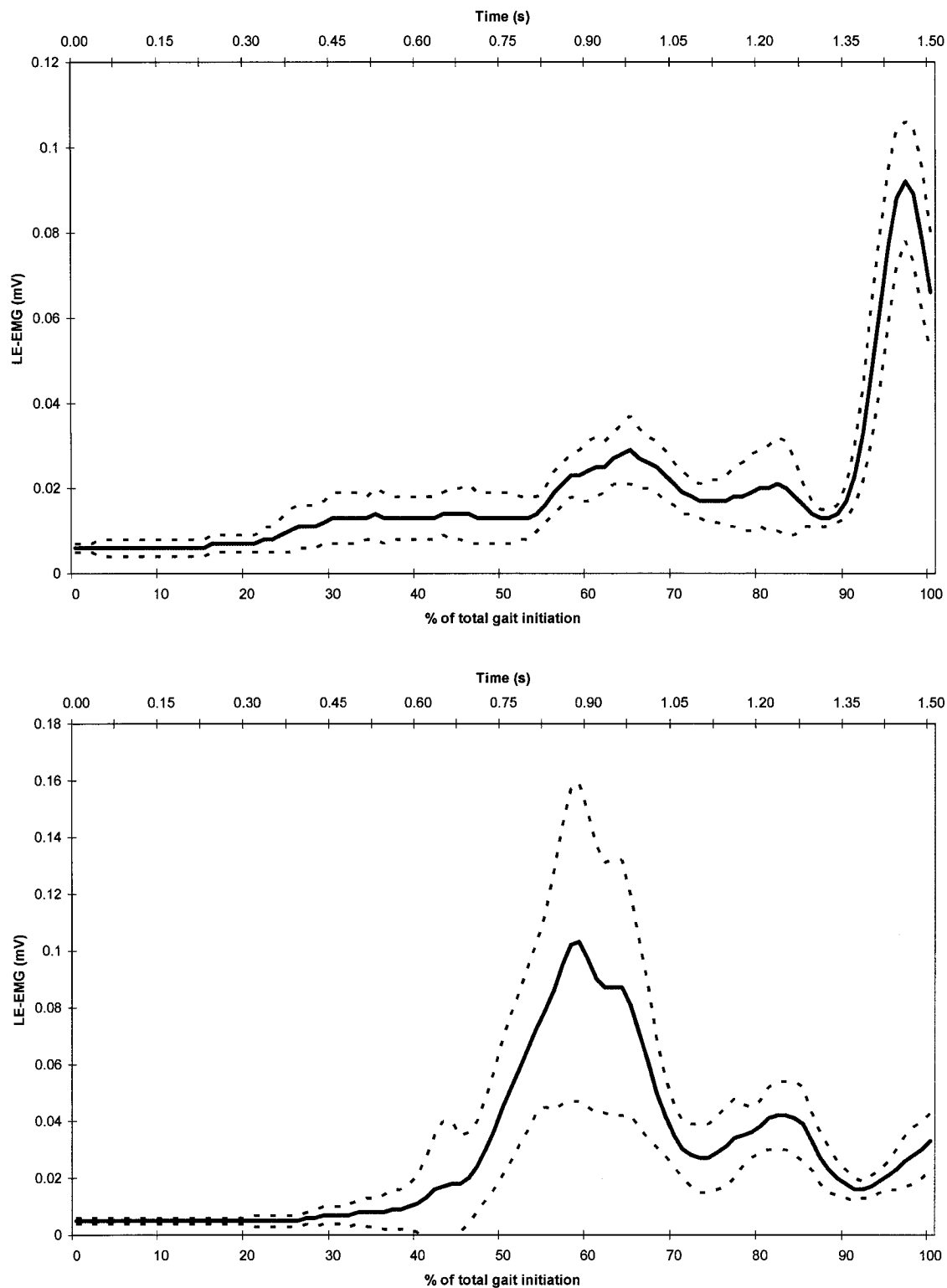


Figure A.10: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) leg tensor fasciae latae from 10 trials for subject three. Time normalized linear envelope EMG throughout gait initiation.

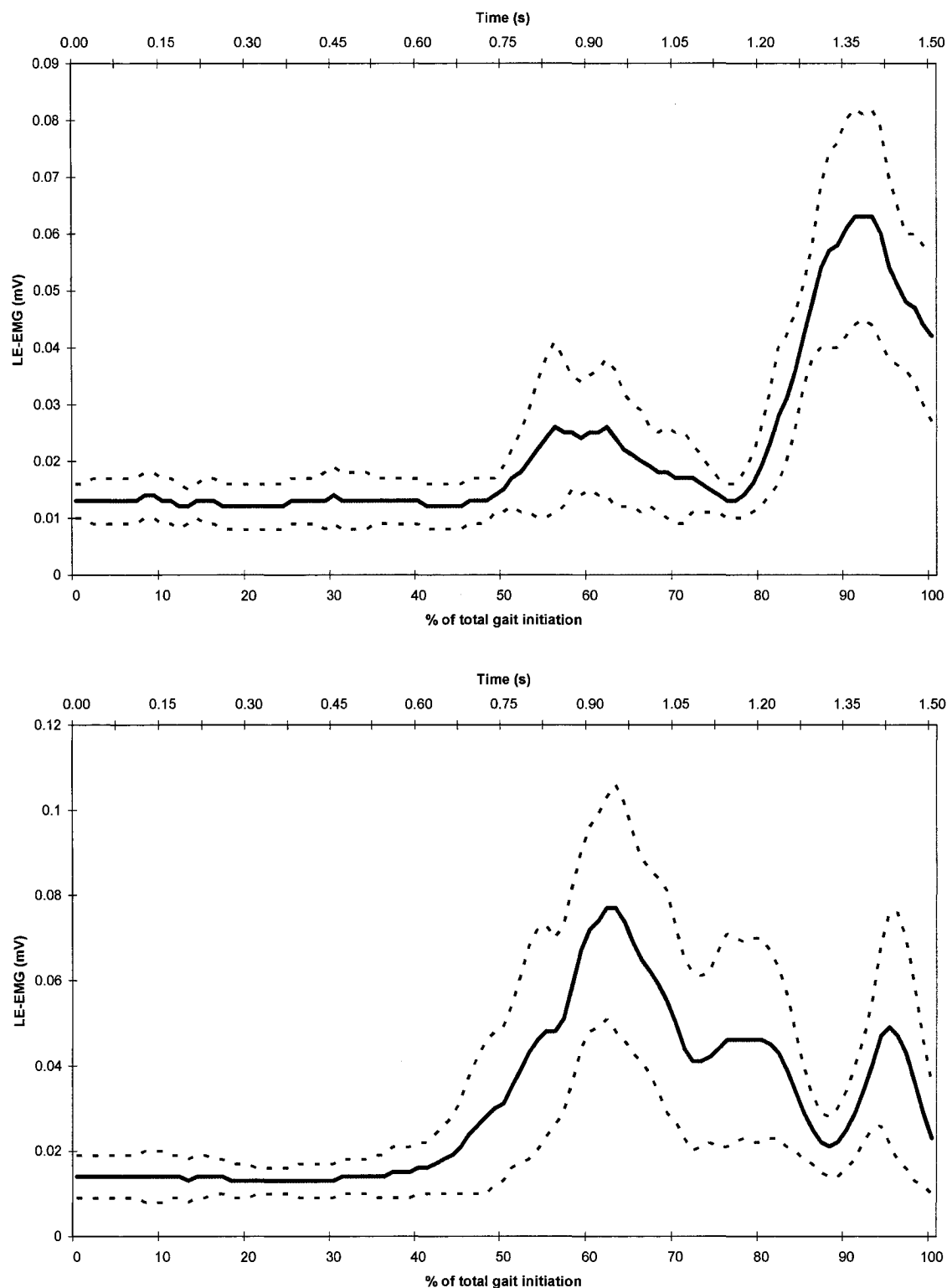


Figure A.11: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) leg adductor magnus from 10 trials for subject three. Time normalized linear envelope EMG throughout gait initiation.

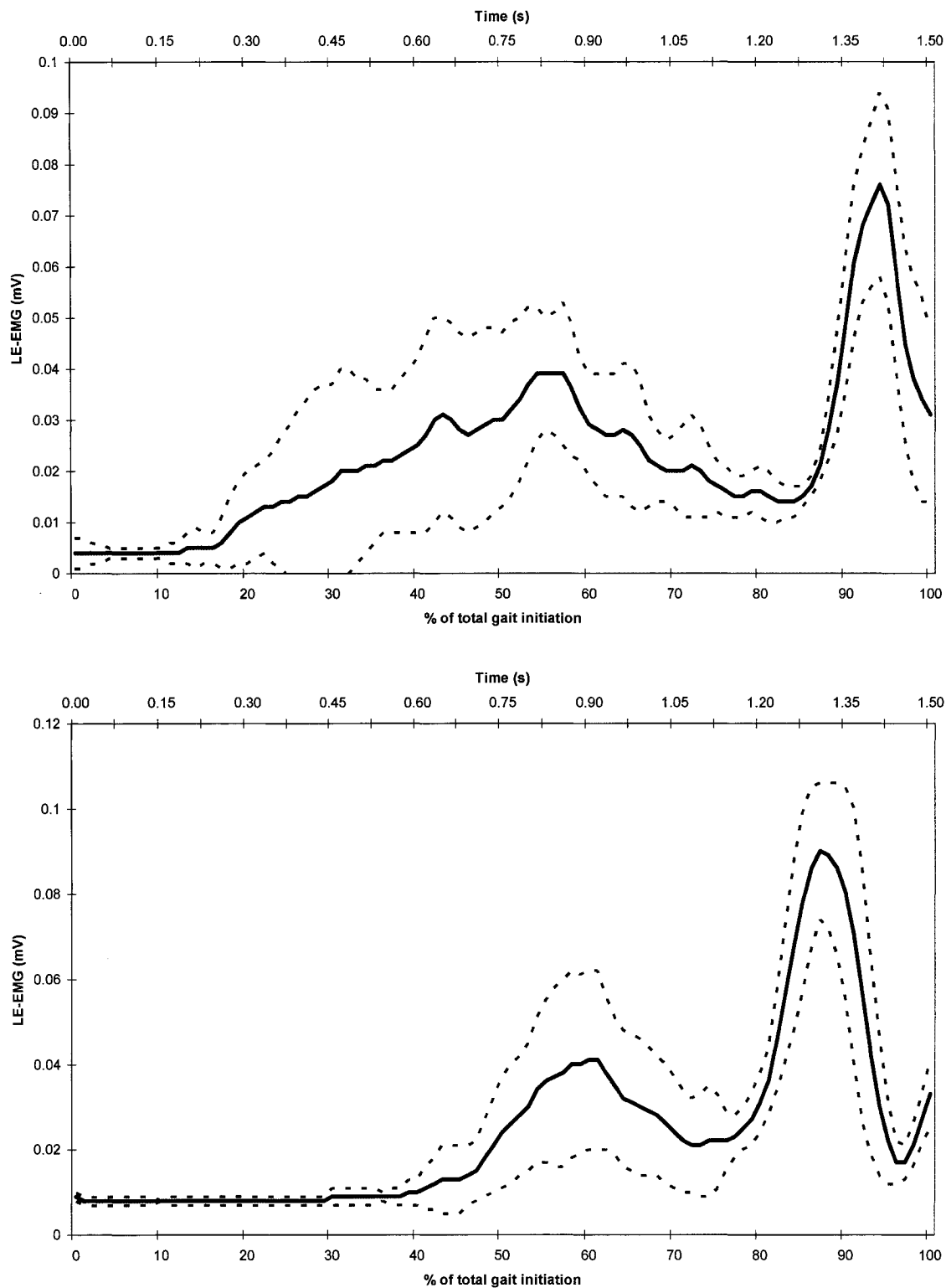


Figure A.12: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) leg tibialis anterior from 10 trials for subject three. Time normalized linear envelope EMG throughout gait initiation.

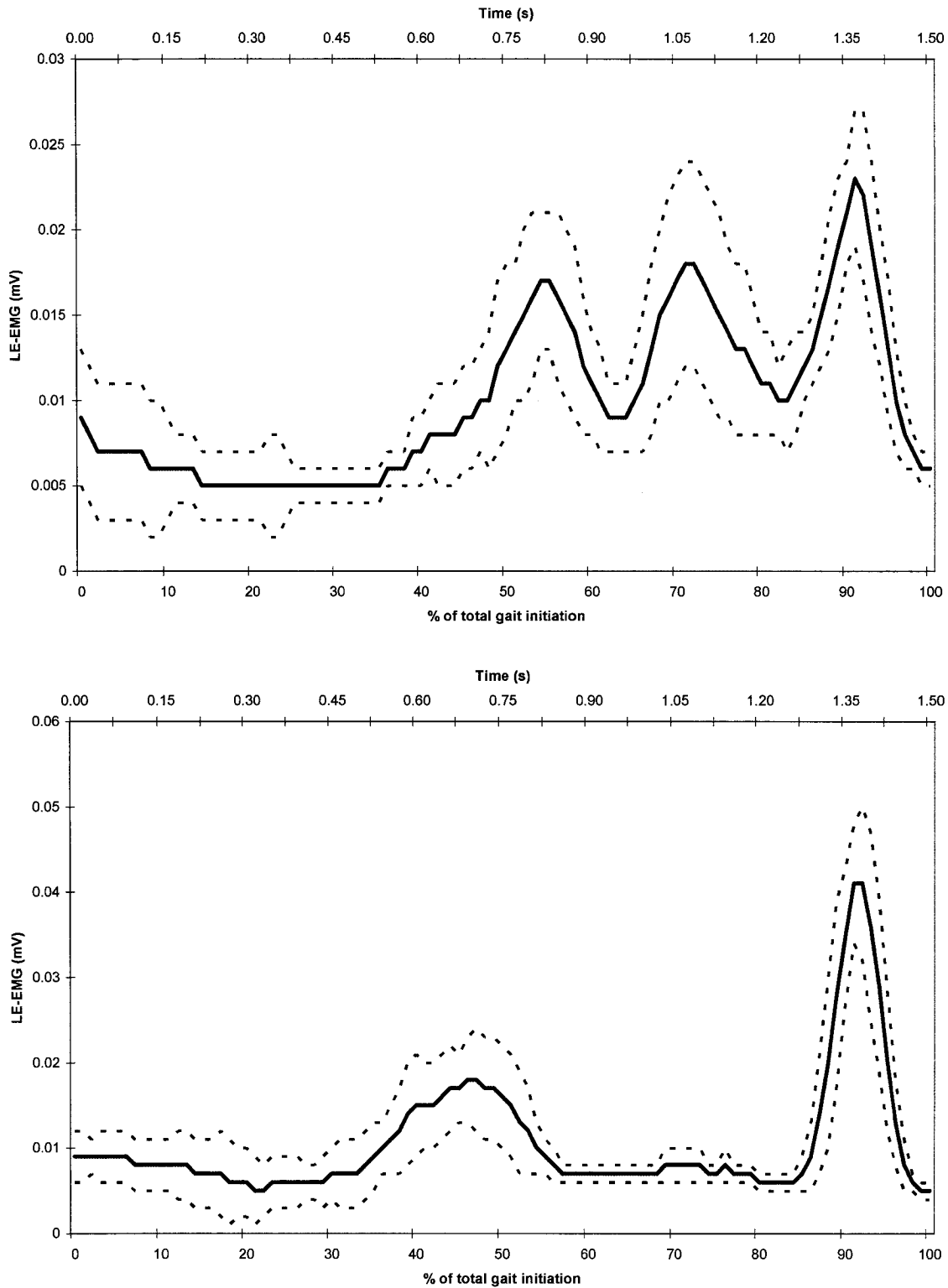


Figure A.13: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) side erector spinae from 10 trials for subject four. Time normalized linear envelope EMG throughout gait initiation.

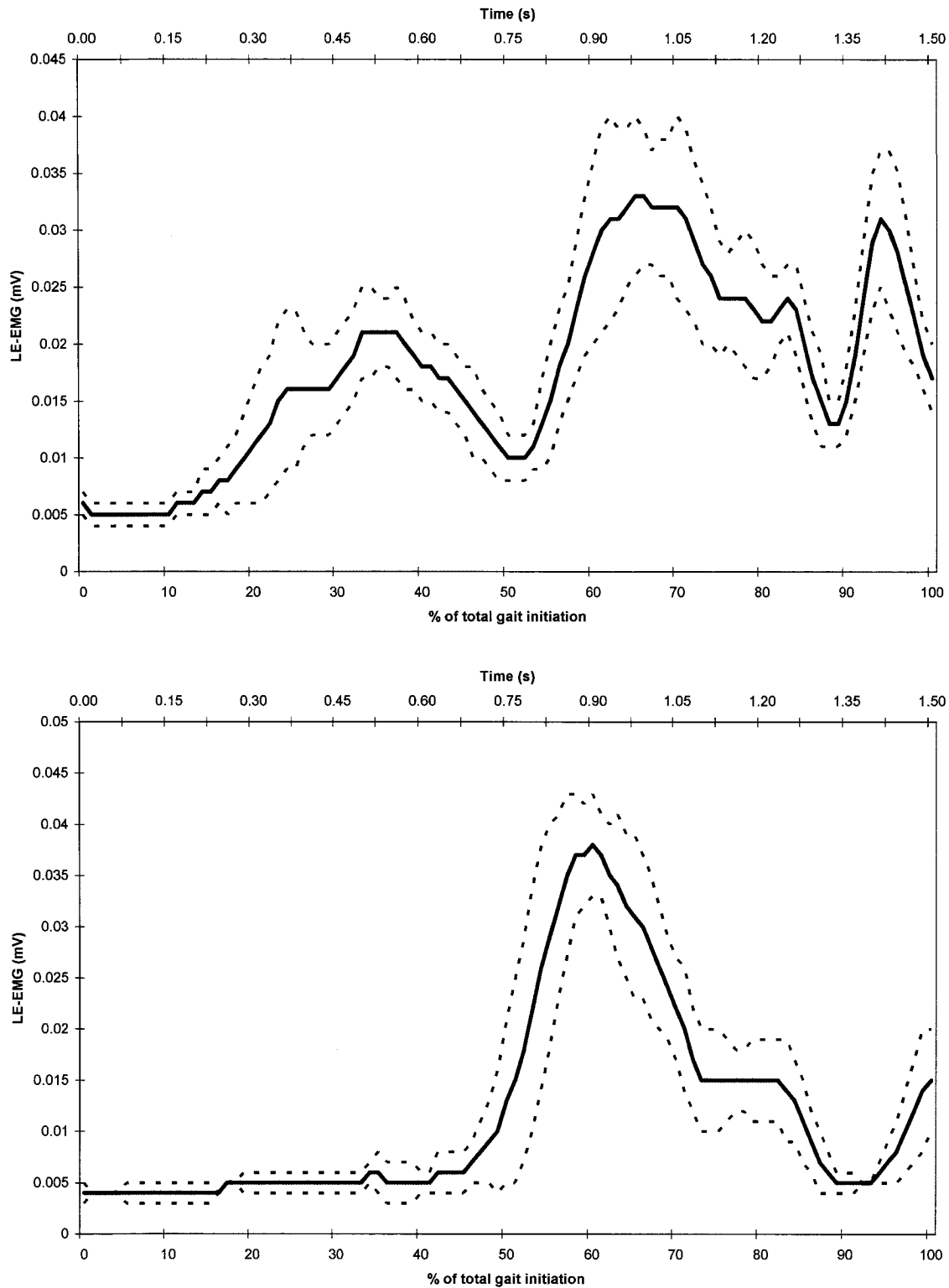


Figure A.14: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) leg tensor fasciae latae from 10 trials for subject four. Time normalized linear envelope EMG throughout gait initiation.

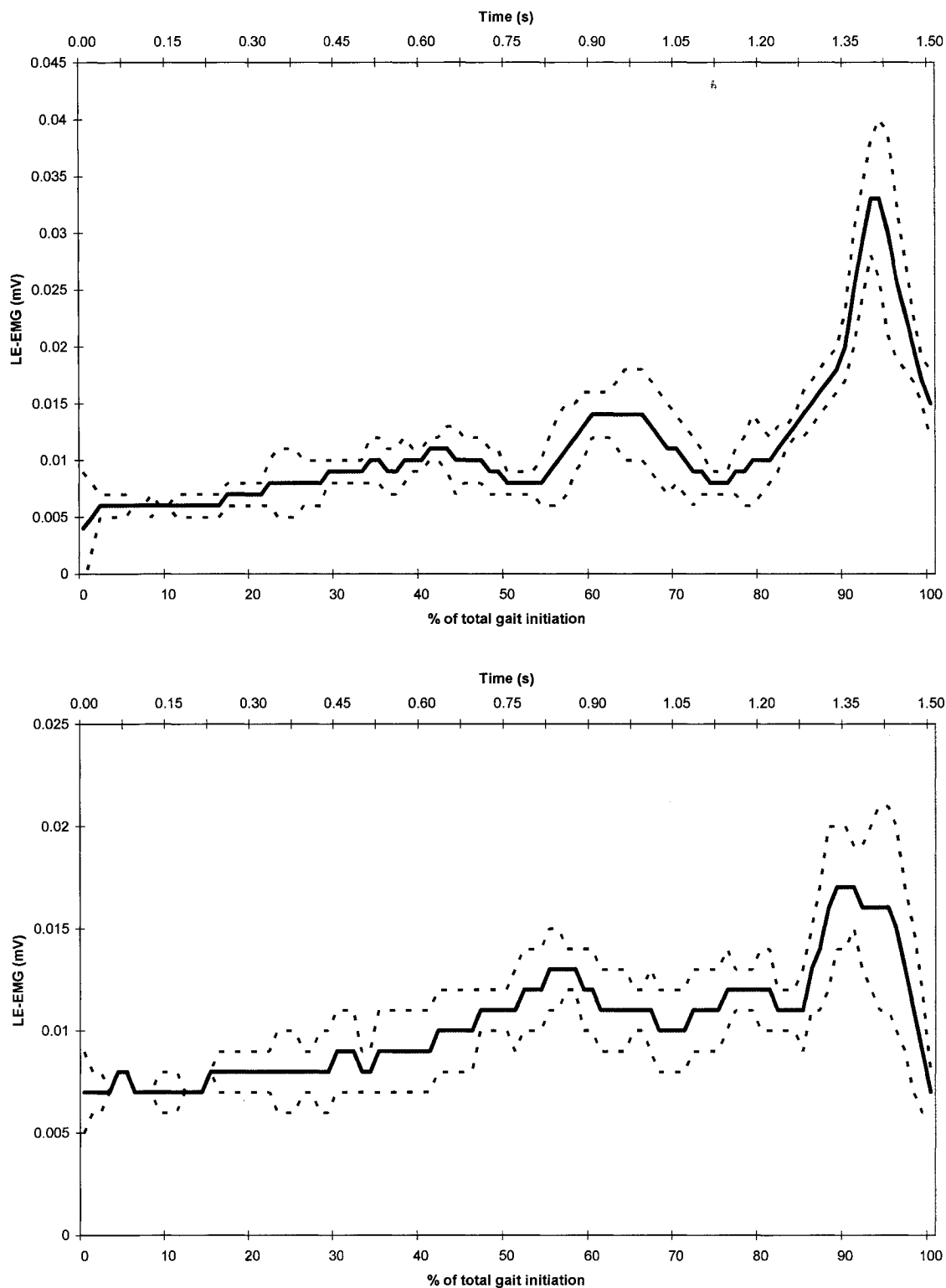


Figure A.15: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) leg adductor magnus from 10 trials for subject four. Time normalized linear envelope EMG throughout gait initiation.

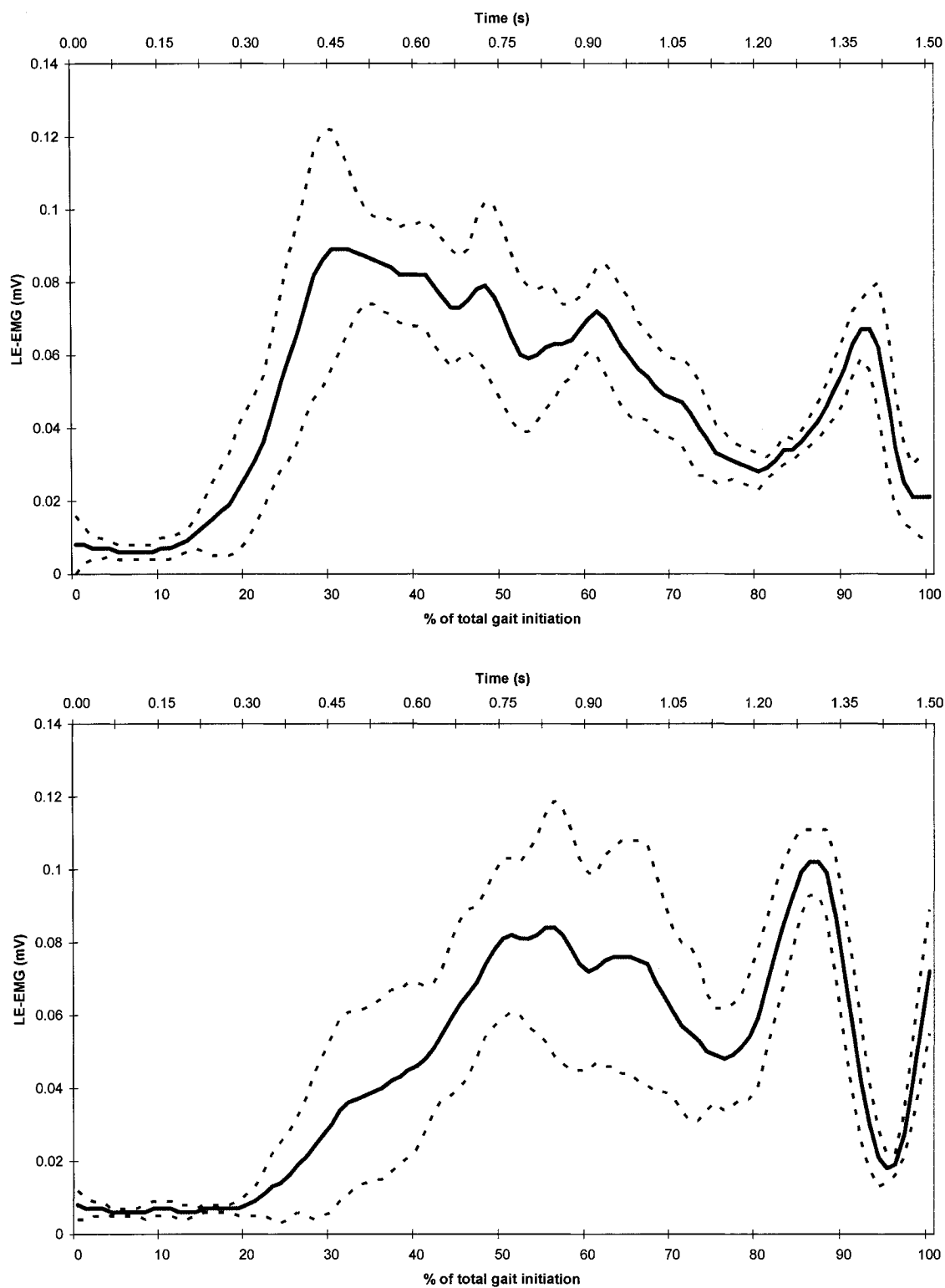


Figure A.16: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) leg tibialis anterior from 10 trials for subject four. Time normalized linear envelope EMG throughout gait initiation.

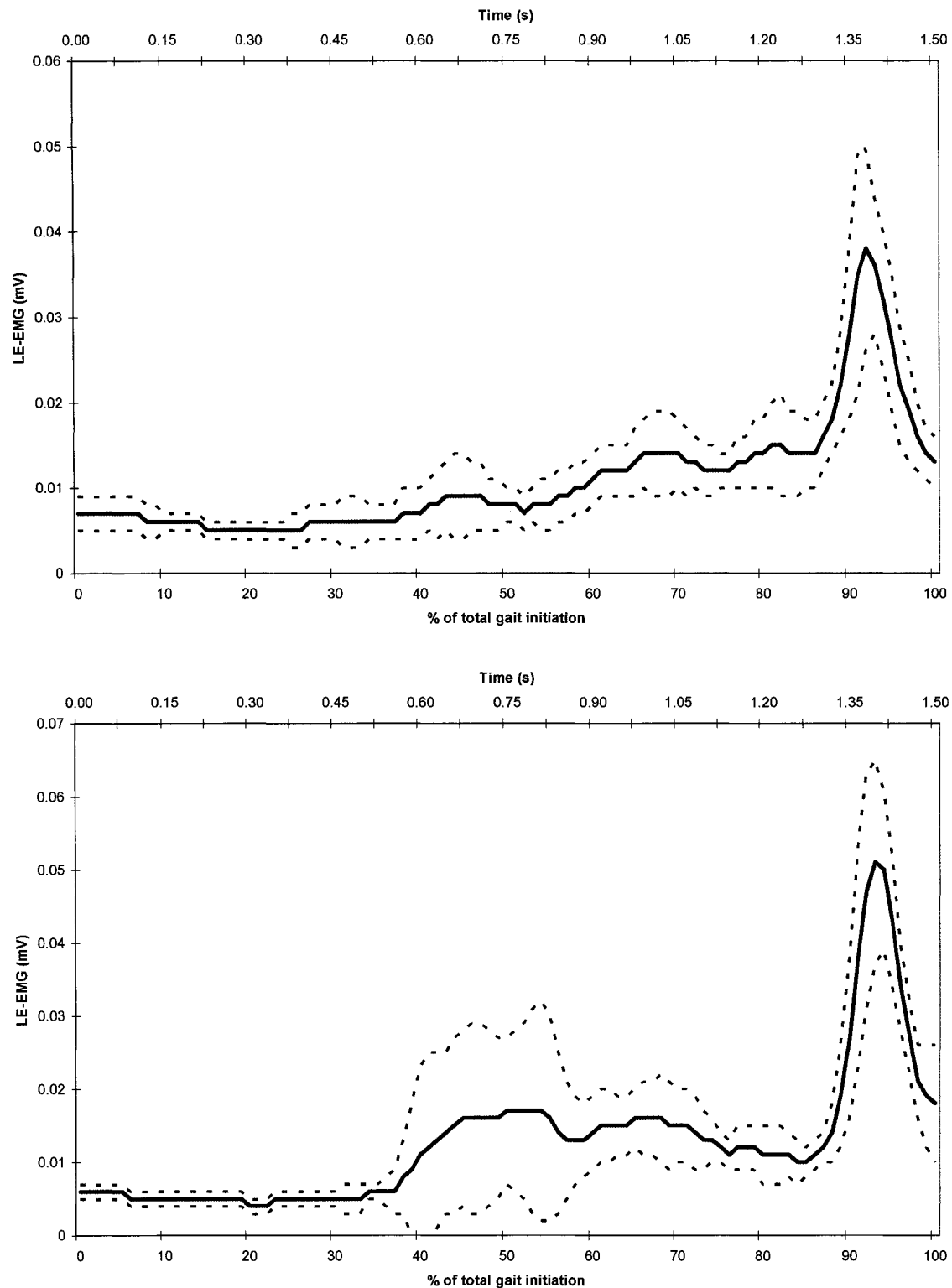


Figure A.17: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) side erector spinae from 10 trials for subject five. Time normalized linear envelope EMG throughout gait initiation.

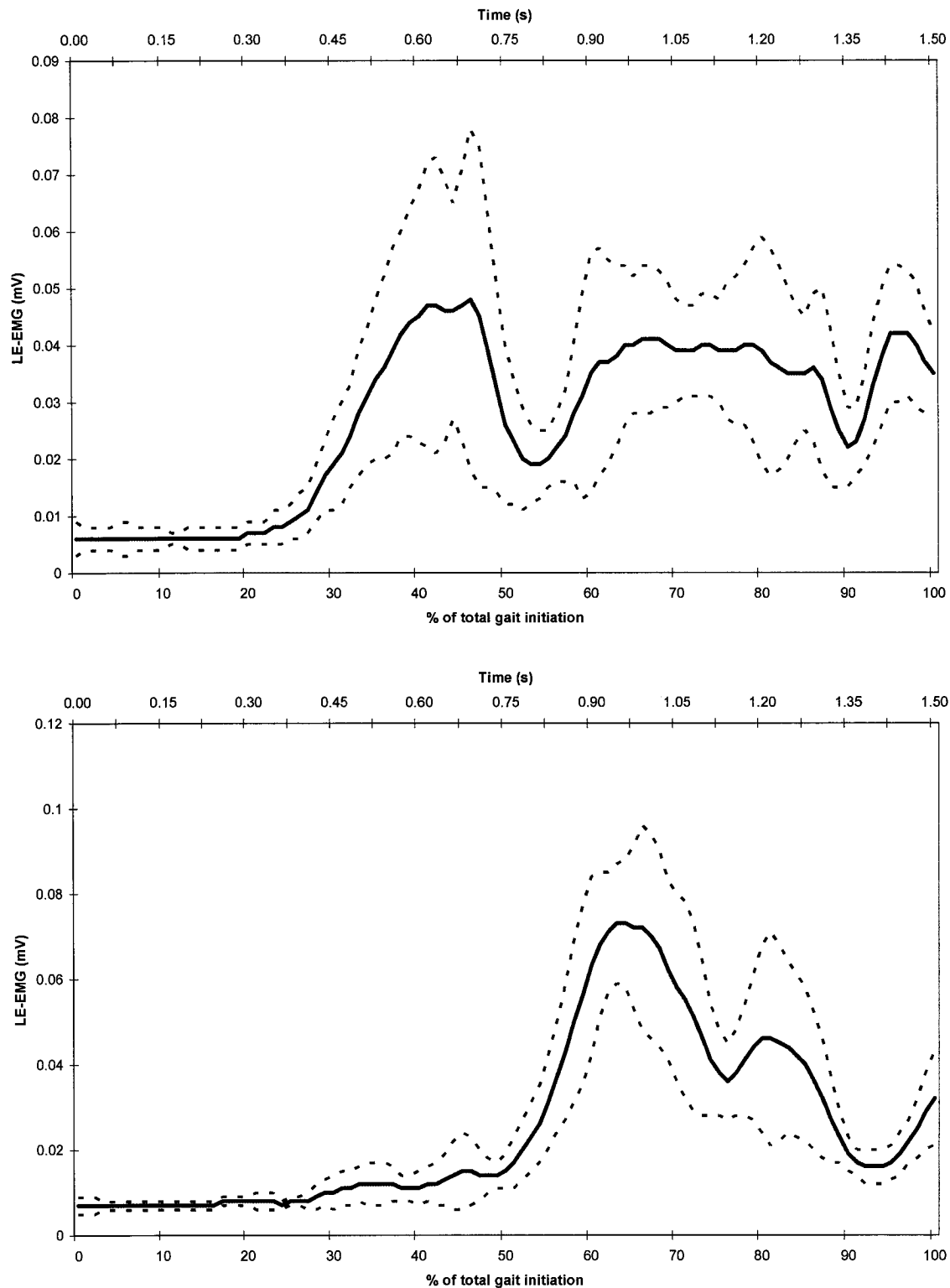


Figure A.18: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) leg tensor fasciae latae from 10 trials for subject five. Time normalized linear envelope EMG throughout gait initiation.

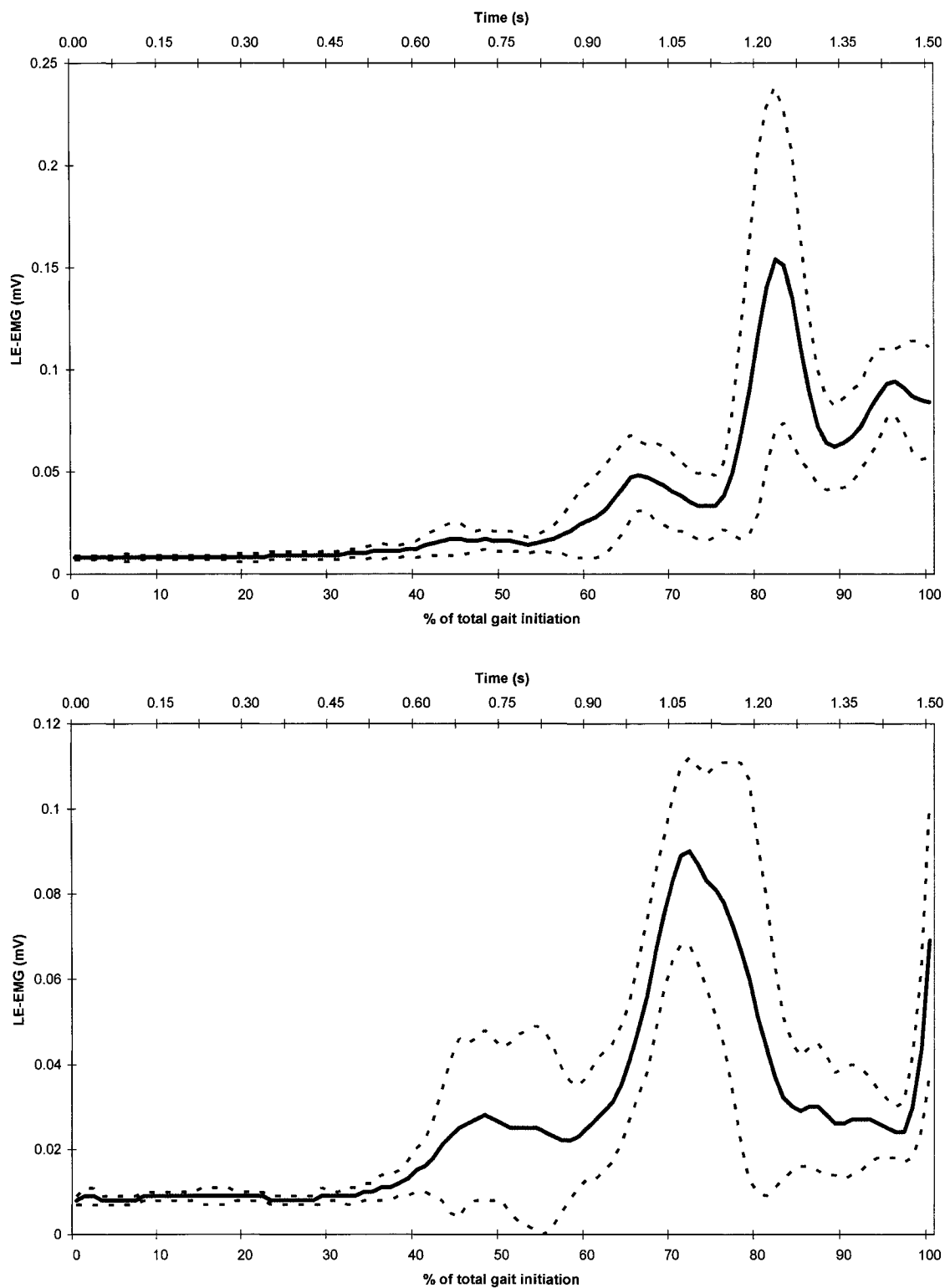


Figure A.19: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) leg adductor magnus from 10 trials for subject five. Time normalized linear envelope EMG throughout gait initiation.

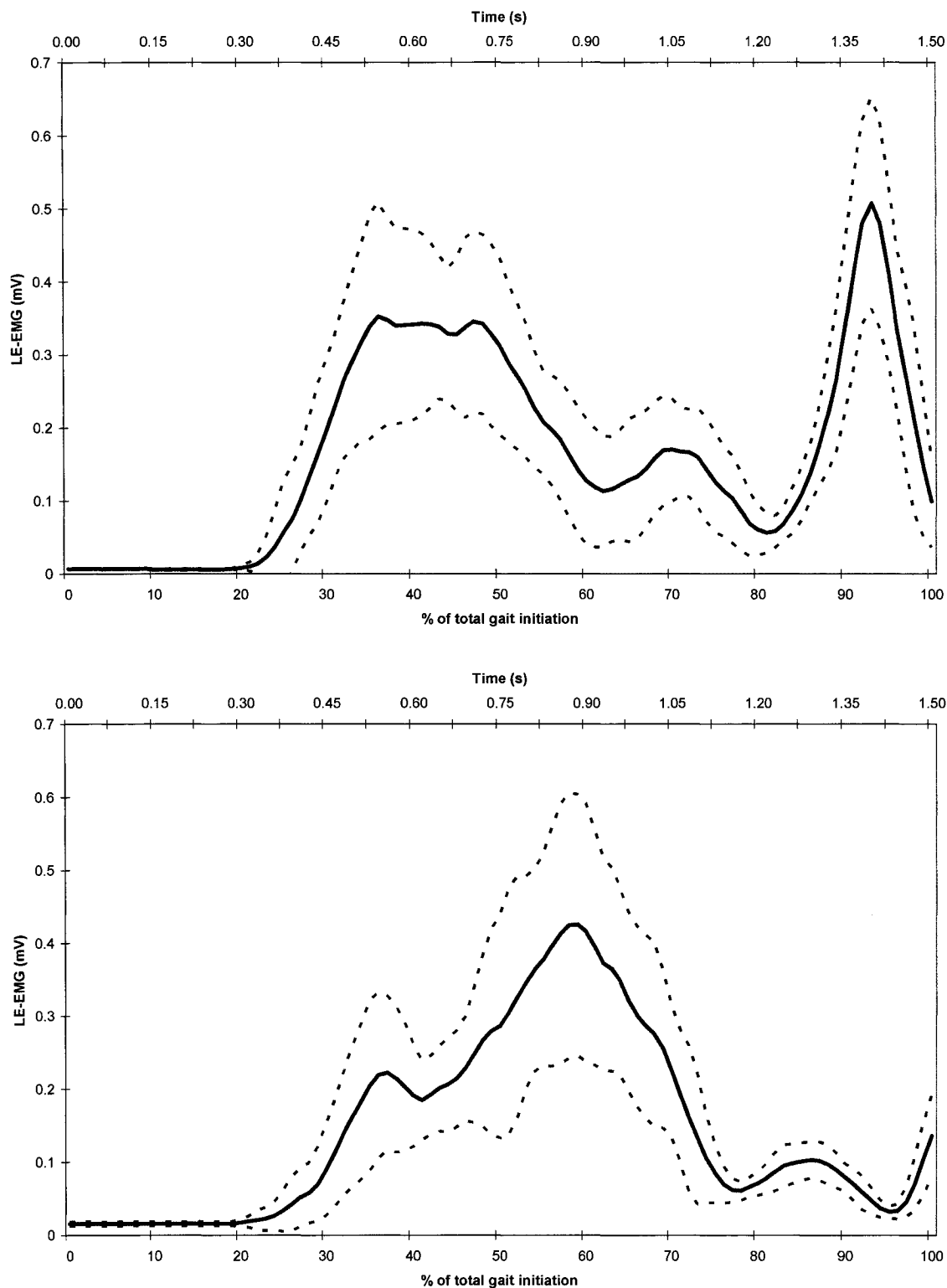


Figure A.20: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) leg tibialis anterior from 10 trials for subject five. Time normalized linear envelope EMG throughout gait initiation.

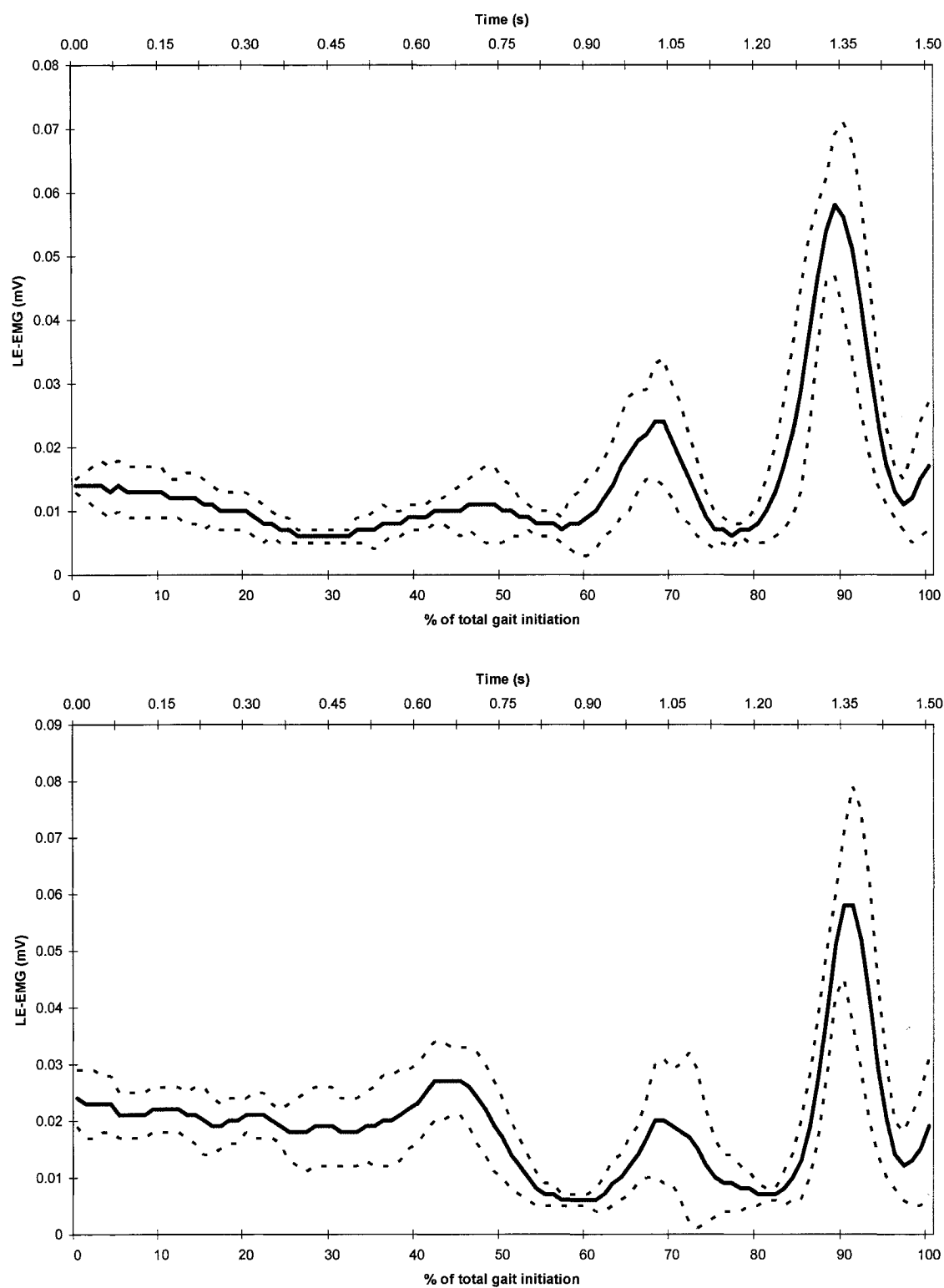


Figure A.21: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) side erector spinae from 10 trials for subject six. Time normalized linear envelope EMG throughout gait initiation.

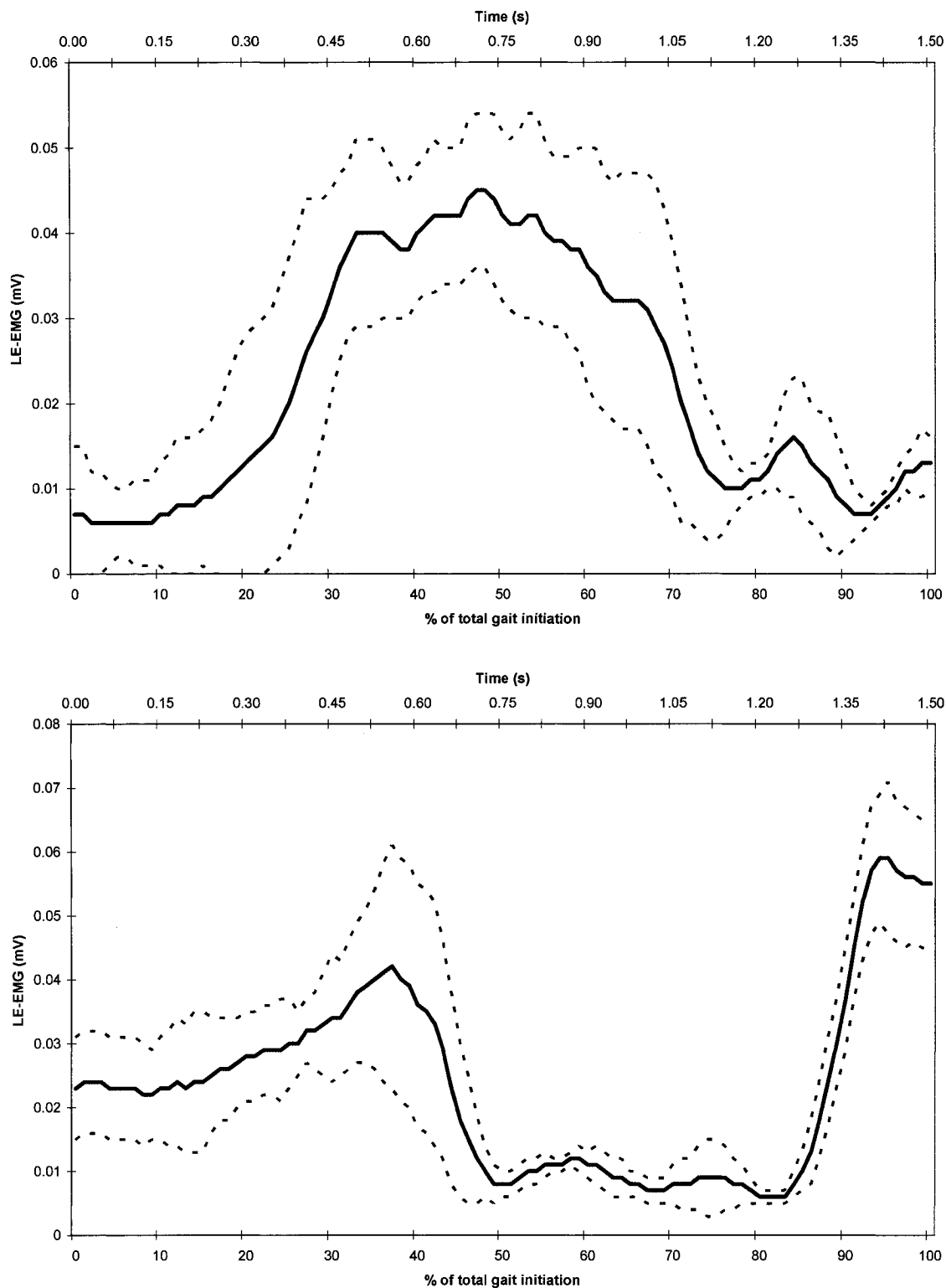


Figure A.22: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) leg tensor fasciae latae from 10 trials for subject six. Time normalized linear envelope EMG throughout gait initiation.

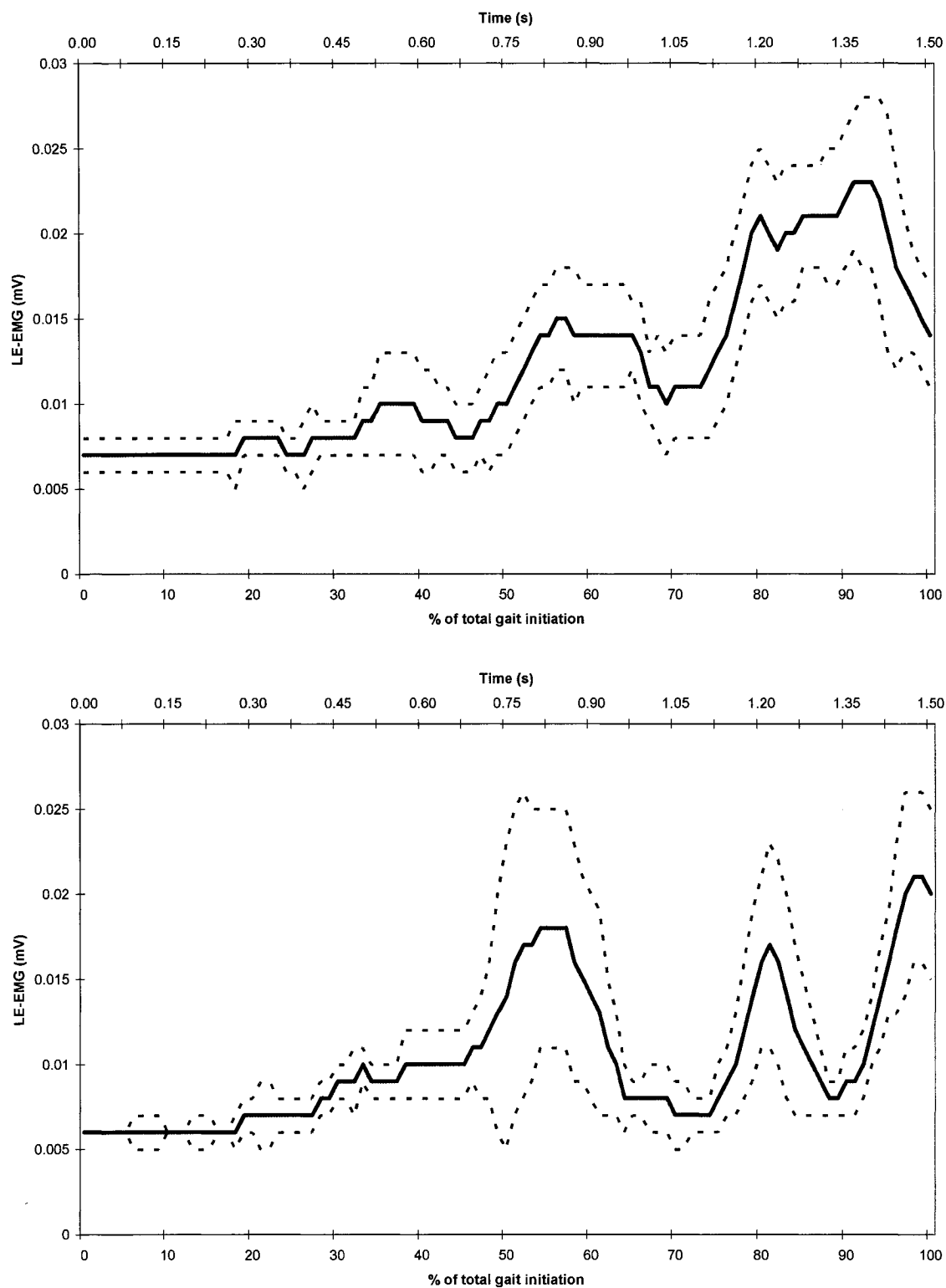


Figure A.23: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) leg adductor magnus from 10 trials for subject six. Time normalized linear envelope EMG throughout gait initiation.

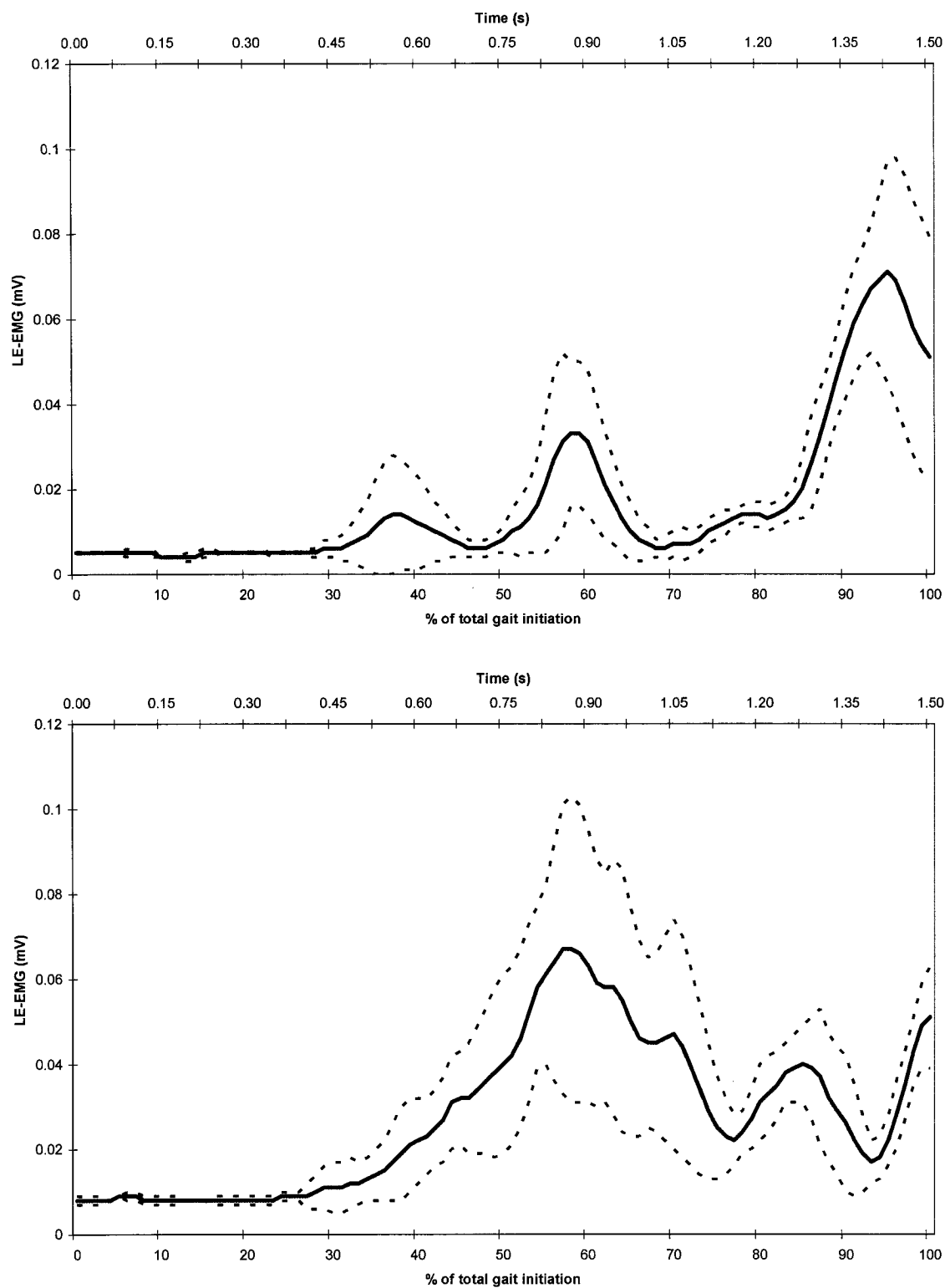


Figure A.24: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) leg tibialis anterior from 10 trials for subject six. Time normalized linear envelope EMG throughout gait initiation.

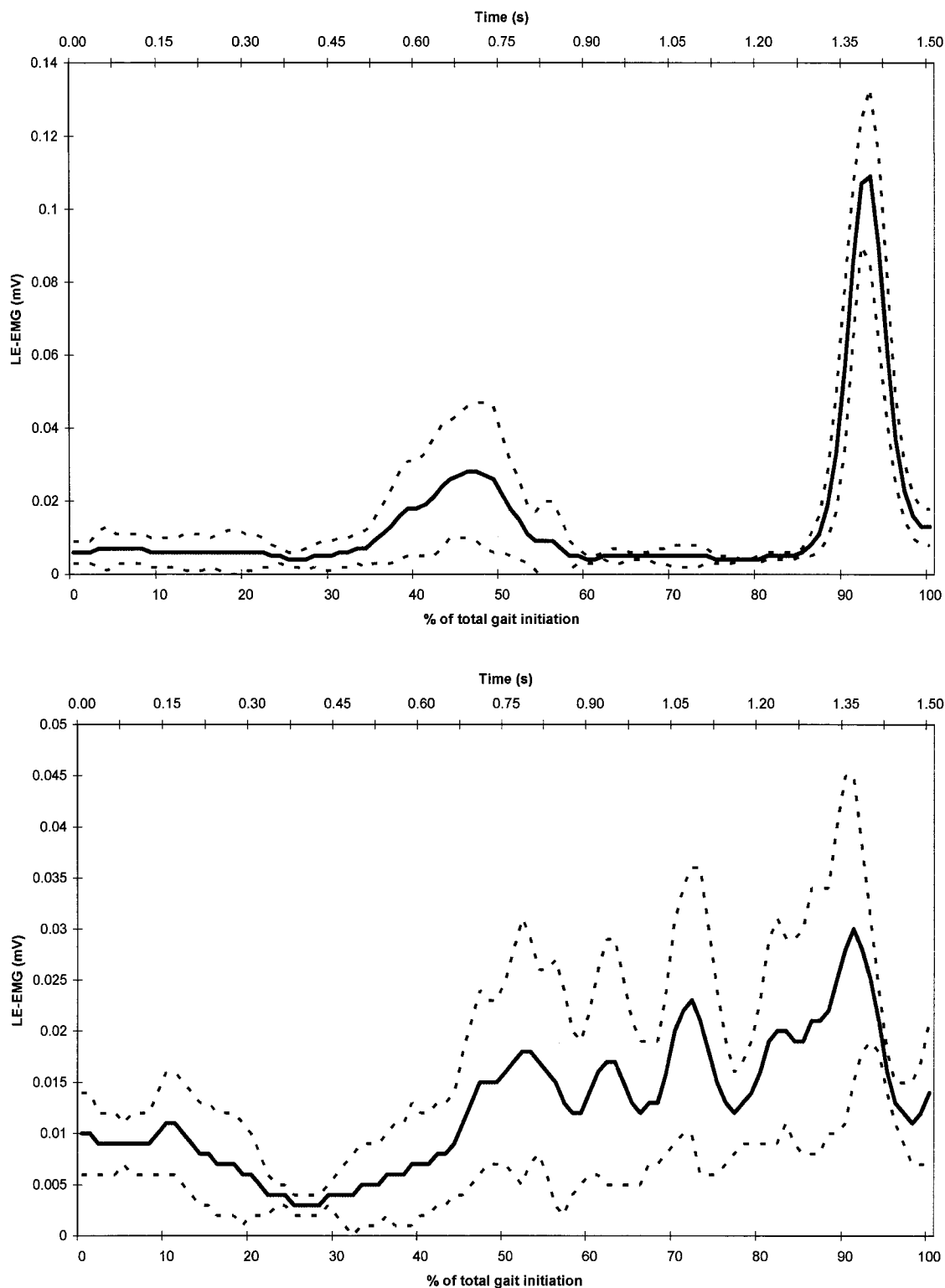


Figure A.25: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) side erector spinae from 10 trials for subject seven. Time normalized linear envelope EMG throughout gait initiation.

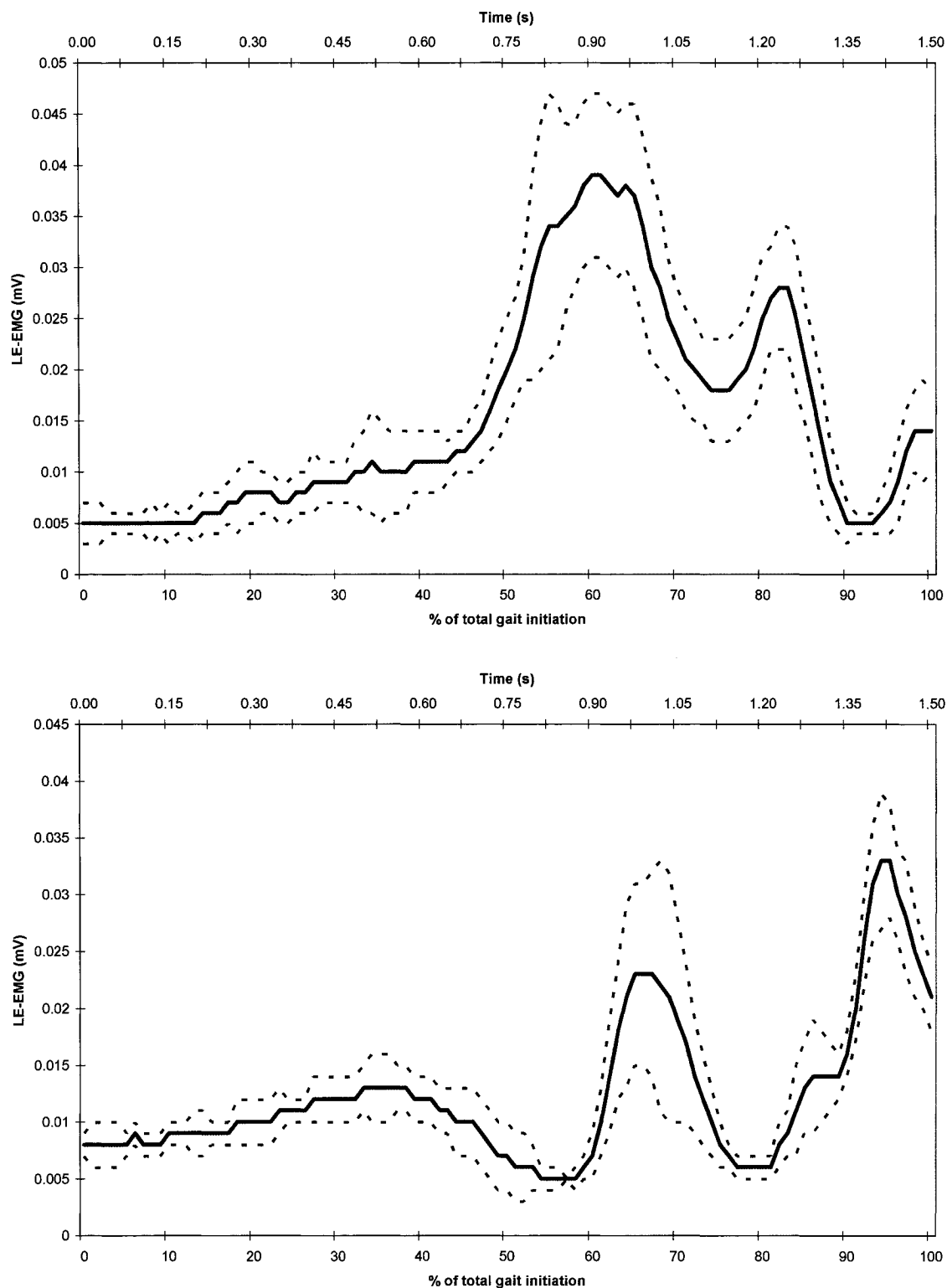


Figure A.26: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) leg tensor fasciae latae from 10 trials for subject seven. Time normalized linear envelope EMG throughout gait initiation.

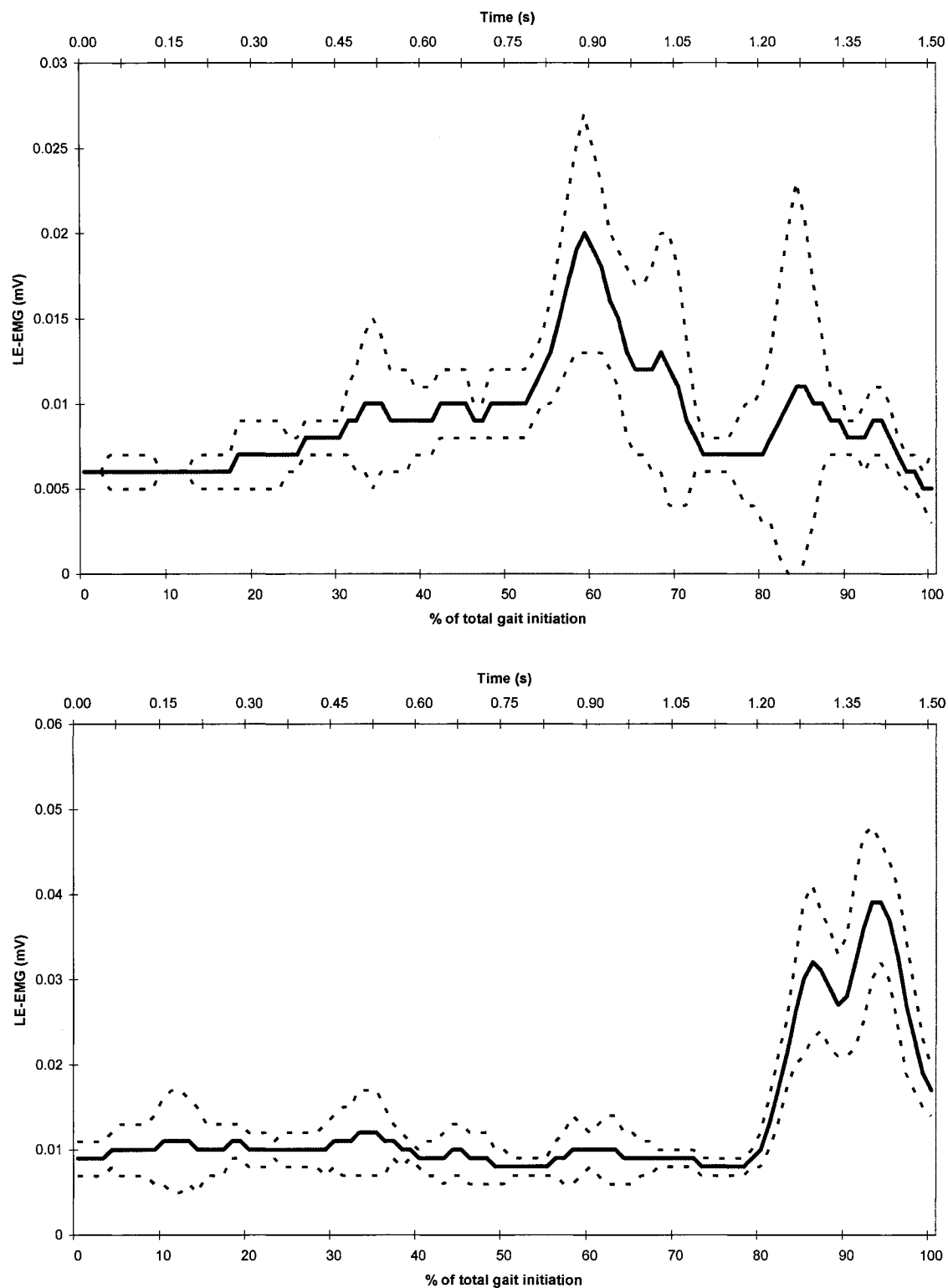


Figure A.27: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) leg adductor magnus from 10 trials for subject seven. Time normalized linear envelope EMG throughout gait initiation.

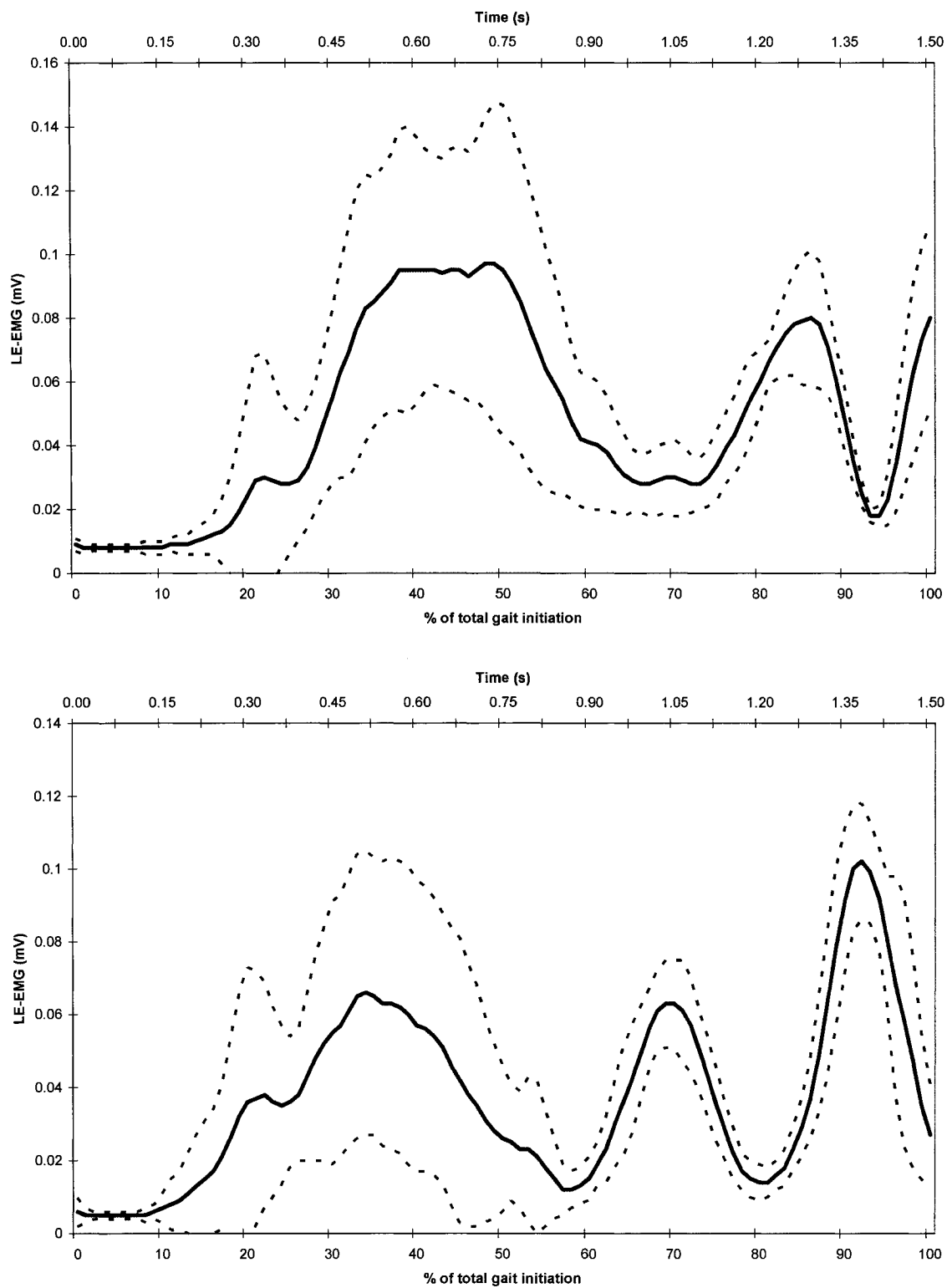


Figure A.28: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) leg tibialis anterior from 10 trials for subject seven. Time normalized linear envelope EMG throughout gait initiation.

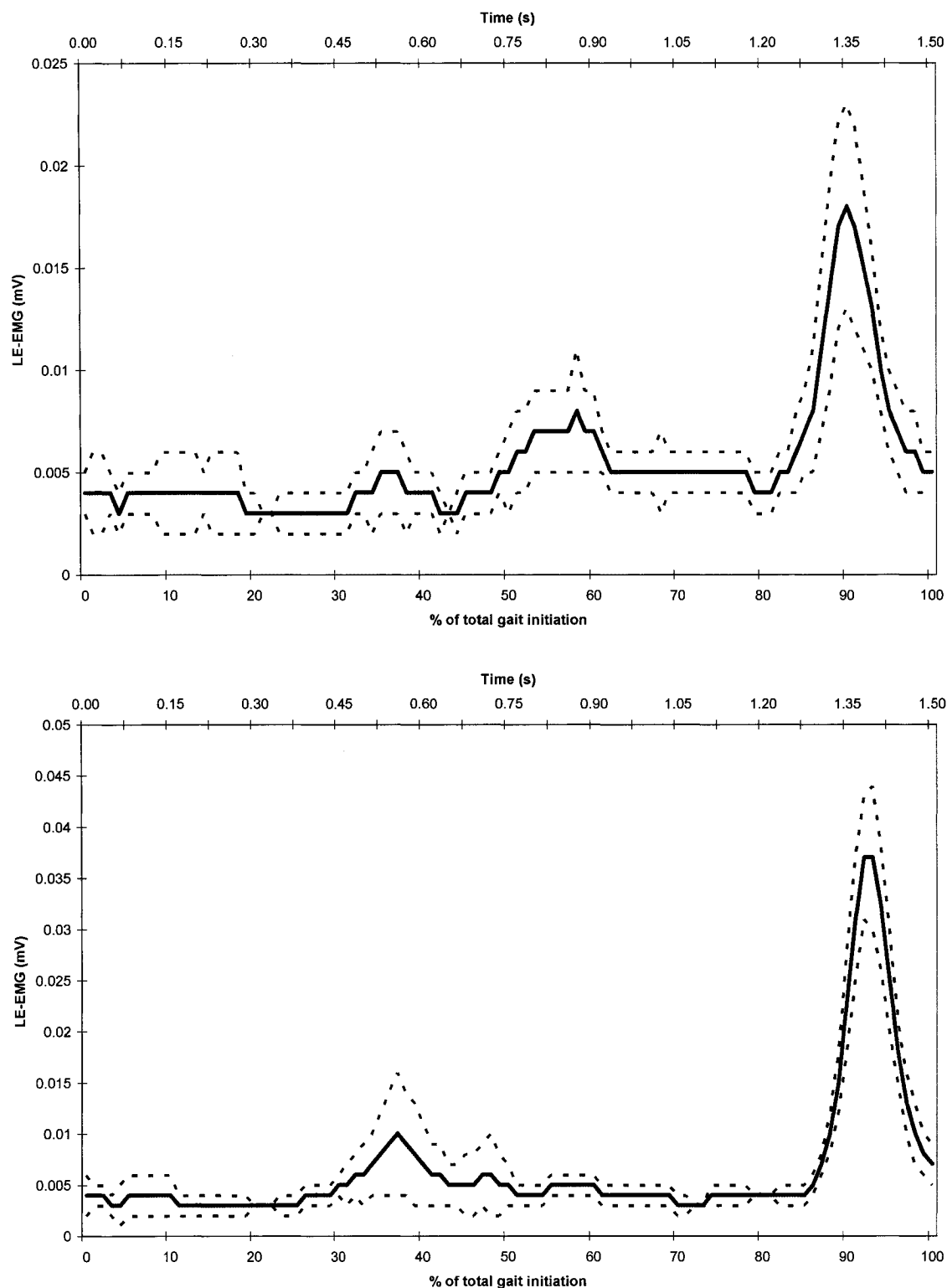


Figure A.29: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) side erector spinae from 10 trials for subject nine. Time normalized linear envelope EMG throughout gait initiation.

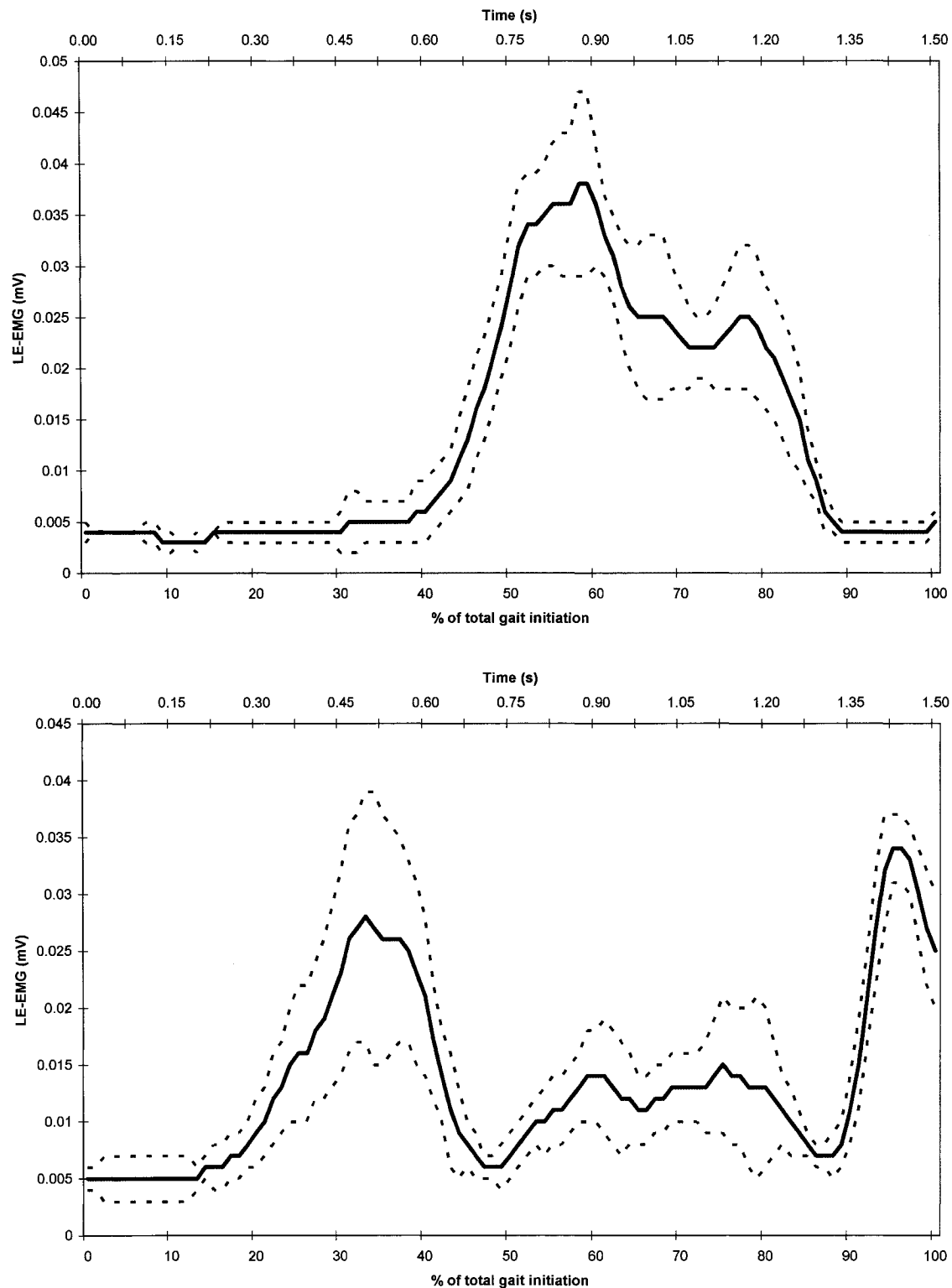


Figure A.30: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) leg tensor fasciae latae from 10 trials for subject nine. Time normalized linear envelope EMG throughout gait initiation.

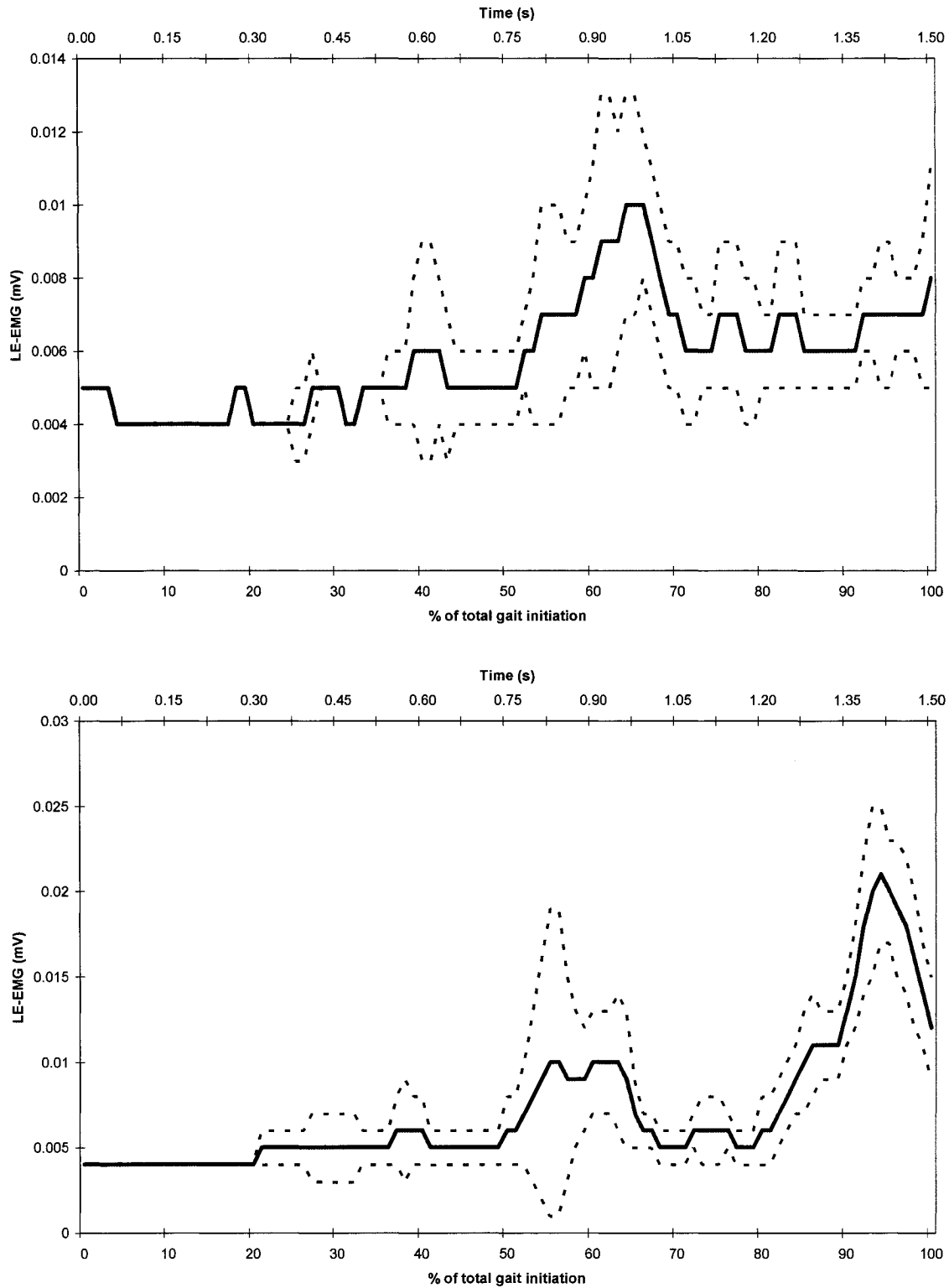


Figure A.31: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) leg adductor magnus from 10 trials for subject nine. Time normalized linear envelope EMG throughout gait initiation.

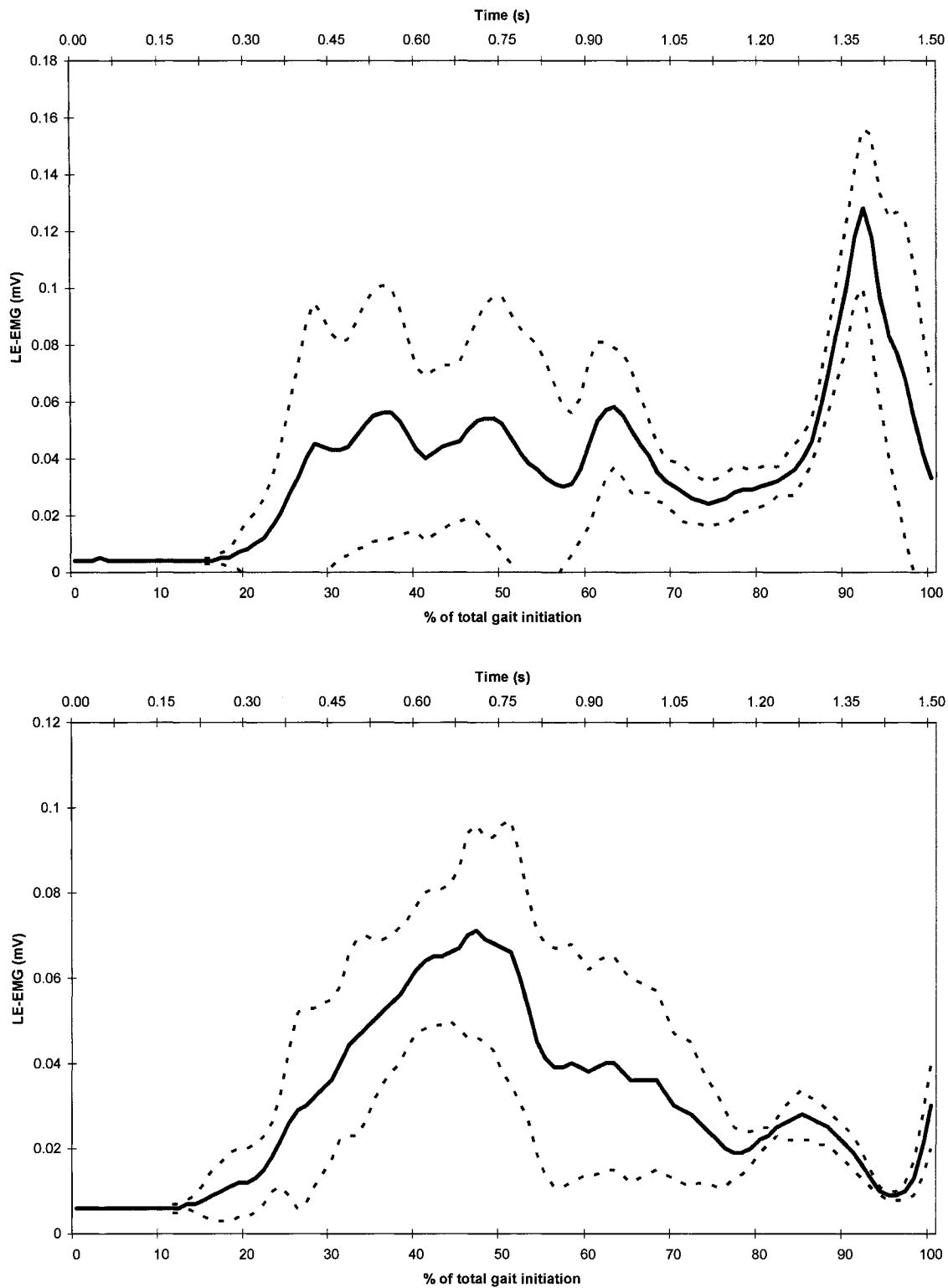


Figure A.32: Ensemble mean (\pm SD) of the lead (top) and trail (bottom) leg tibialis anterior from 10 trials for subject nine. Time normalized linear envelope EMG throughout gait initiation.