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The effect of FES of the tibial nerve on physiological activation of leg muscles during gait☆

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ABSTRACT

The effects of surface functional electrical stimulation (FES) of the tibial nerve of healthy subjects were evaluated. The FES was applied at three different times during gait: early, mid and late stances. The purpose of this work is to understand the effect of unilateral stimulation on the bilateral activation patterns of leg muscles, because FES is used in practice to improve gait, while associated neuromuscular change is not often measured. The experimental protocol presented here will be transferred to stroke subjects, who could benefit from improved push-off during gait. Results show that FES of the tibial nerve changes the offset timing of the tibialis anterior muscle on the stimulated side and the on- and offset timings of the tibialis anterior muscle of the leg contralateral to stimulation. Additionally, activity levels of the semitendinosus ipsilateral and tibialis anterior contralateral to the stimulated leg significantly decreased, with respect to the non-stimulated condition. For the semitendinosus, this was a difference of 6–7 μV, with $p < 0.05$. For the tibialis anterior, this was a difference of 7–15 μV, with a significance of $p = 0.00$, respectively.

This information is important for future applications of stimulation as it means that stimulation not only affects the stimulated muscle but also the physiological motor control by the CNS.

1. Introduction

Functional electrical stimulation (FES) involves electrical stimulation of a muscle or nerve to provide functional improvement. Applications of FES include restoration of upper limb functions such as reaching and grasping and lower limb functions such as standing, balance, posture and gait.

Reports show that the Physiological Cost Index (PCI) and walking speed improve in response to stimulation of the tibialis anterior muscle/peroneus nerve to minimise drop foot [1,2]. Studies also show that activities of daily living, quality of life and range of motion are improved due to use of FES [3]. Additionally, physiological activity, measured using electromyography (EMG) of the upper limbs is improved in response to upper limb FES [3]. However, to our knowledge, no studies to date have investigated the effect on neuromuscular control of leg muscles, while applying FES during gait. The most similar research is the measurement of reflexes and joint kinematics, following randomised, non-functional electrical stimulation [4–6] of lower leg cutaneous nerves during gait.

It is necessary to study the effect of FES in healthy and patient populations, in order to fully understand the consequences of functional stimulation [3], not only on the stimulated muscles, but also on the neuromuscular control of other muscle groups.

Results from EMG studies [7–14] show that calf muscle activation and as a consequence, push-off during gait are adversely affected as a result of a stroke. Muscle activation patterns of both sides are affected and muscles from both sides change during the recovery period [13–16]. Research shows that the plantarflexors provide a major contribution to push-off [17] and swing initiation [18]. Reinforcing this, Bajd et al. found a 40% increase in force from rest to push-off, as well as a decrease in the time duration of push-off during FES of the plantarflexors of SCI subjects. Bajd et al. found that FES of ankle plantarflexors causes the heel to rise, in

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preparation for swing, providing forward and upward propulsion to the swinging leg; as well as knee flexion, which is important for effective shortening of the swinging leg. They concluded that stimulation of calf muscles alone can provoke swing [18]. This was also confirmed by Ichie and Munih [19], who added that stimulation elicits the flexion withdrawal response, through the activation of afferent fibres. This implies that FES of the calf muscles of stroke subjects could improve push-off and alter the activation patterns not only of the affected side, but also the non-paretic side.

We previously reported that FES of the tibial nerve affects the activation patterns of the gastrocnemius medialis (GM) of healthy subjects during gait [20]. Direct and/or reflexive motor responses were generated in this muscle. It was expected that the stimulation would decrease the amplitude and possibly duration of physiological activation bursts of the GM. However, this was not the case. The reason may be attributed to the presence of antidromic stimulation, which would block the physiological activation of the gastrocnemius during the stimulation burst. As FES of the tibial nerve affects the response of the GM, the stimulation interacts with the physiological motor system. As such, it can be expected that FES of the tibial nerve will, directly or indirectly, influence activation patterns of other leg muscles.

The aim of the research presented here was to determine if muscle activation patterns of upper and lower leg muscles and angular velocity of both legs change due to the unilateral application of FES to the tibial nerve during gait in healthy subjects. This study has been performed in preparation for similar testing in stroke subjects who could benefit from improved push-off during gait.

2. Methodology

Data was collected from six healthy subjects, four males and two females, median age 24, ranging between 22 and 29 years old. The subjects had no history of neurological disorders. Each subject signed an informed consent form, which was approved of by the Medical Ethical Committee, of the Roessingh, Rehabilitation Centre, as part of a study for FES of stroke subjects.

A detailed methodology has already been presented [20], the following is a summary of this protocol, with relevant additions where needed. The results presented are from the same group of subjects and protocol.

EMG data was recorded, using the ambulant set-up of the Porti-5 from TMSI, Enschede, NL at a frequency of 2048 Hz, and high pass filtered at 5 Hz during gait. Muscles measured included the gastrocnemius medialis (ipsilateral: iGM, contralateral: cGM), tibialis anterior (iTA, cTA), semitendinosus (iST, cST) and rectus femoris (iRF, cRF). EMG electrodes were applied according to SENIAM guidelines [21].

Kinematic data was recorded from the thigh, shank and foot of the side ipsilateral to stimulation, as well as the contralateral shank, using MT9 inertial sensors from Xsens Technologies, Enschede, The Netherlands. The MT9 on the thigh and shanks were fixed to in-house, custom-made Perspex strips. The strips were positioned on the lateral side of each segment. On the shanks this was between the lateral tibial condyle and lateral malleolus and on the thigh, between the greater trochanter and the lateral femoral condyle. The MT9 of the foot was placed on the bridge of the foot. Each inertial sensor contains three uniaxial gyroscopes, accelerometers and magnetometers. Gyroscopes measure angular velocity. Sample rate was 100 Hz. In this study, only angular velocity recorded from the gyroscopes of sagittal plane movement is reported. The ipsilateral shank sensor was connected directly to a biphasic stimulator to allow control of stimulation. The control principle has been previously detailed [20,22]. When shank angular velocity changed direction, at the beginning of stance, the angular velocity signal was integrated, resulting in angle change since this instant. Stimulation began at a preset shank angle change.

Synchronisation between kinematic and EMG data was possible using a custom-built Labview program from which the researcher triggered synchronisation pulses during recording at the beginning and end of each trial. A device worn by the subject received this signal via Bluetooth, then serially transmitted the pulses simultaneously to both the EMG system and the xbusmaster.

An EMG electrode was utilised as the stimulation electrode. The optimal stimulation site for tibial nerve stimulation, at the popliteal fossa, was determined using a hand held stimulation probe. The anode was fixed to the lower leg, under the gastrocnemii. Stimulation was applied at 50 Hz. Burst duration was 300 ms, consistent with the work of Bajd et al. [18].

The experimental procedure was divided into stimulation and non-stimulation blocks, as shown in Fig. 1. To prevent the influence of the order of stimulation timing on the results, timing was randomised between the subjects.

During gait trials, subjects walked continuously for 3 min around the gait lab at a self-determined pace while data was recorded. Following the first non-stimulation trial, subjects stood with the foot of the stimulated leg on a force plate, mimicking posture at push-off. Stimulation amplitude was increased until the stimulated leg was forcefully pushed forwards and upwards from the force plate.

Mid-stimulation (Sm) started approximately half-way through the stance phase; we assumed that this angle coincided with normal contraction time of the gastrocnemii. Early stimulation (Se) was applied 10° before and late stimulation (Sl) was applied 10° after Sm. Sl may have continued into the swing phase, depending on the Sm angle. Statistical analysis revealed that the non-stimulation (NS) conditions did not produce significantly different results, thus NS denotes results from all non-stimulation trials, which have been combined, mainly for statistical purposes.

2.1. Data processing

The EMG data was processed offline in Matlab (The MathWorks, MA, USA). Stimulated trials underwent stimulation artefact removal [23]. A 50 Hz high pass filter was applied to artefact-free signals to remove any remaining filter response to the stimulus artefacts. The EMG was further processed using two approaches. One was to determine the EMG activity between the stimulation pulses. The other utilised a standardised burst detection method, based on the approximated generalised likelihood ratio (AGLR)

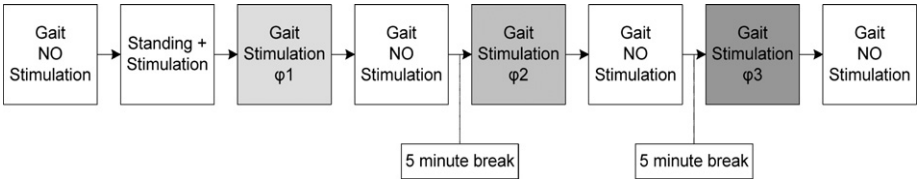

Fig. 1. Block diagram of the experimental protocol. φ1, φ2 and φ3 represent a different change in angle since heel strike. The order of the three stimulation conditions (early, mid and late) was randomly chosen per subject.

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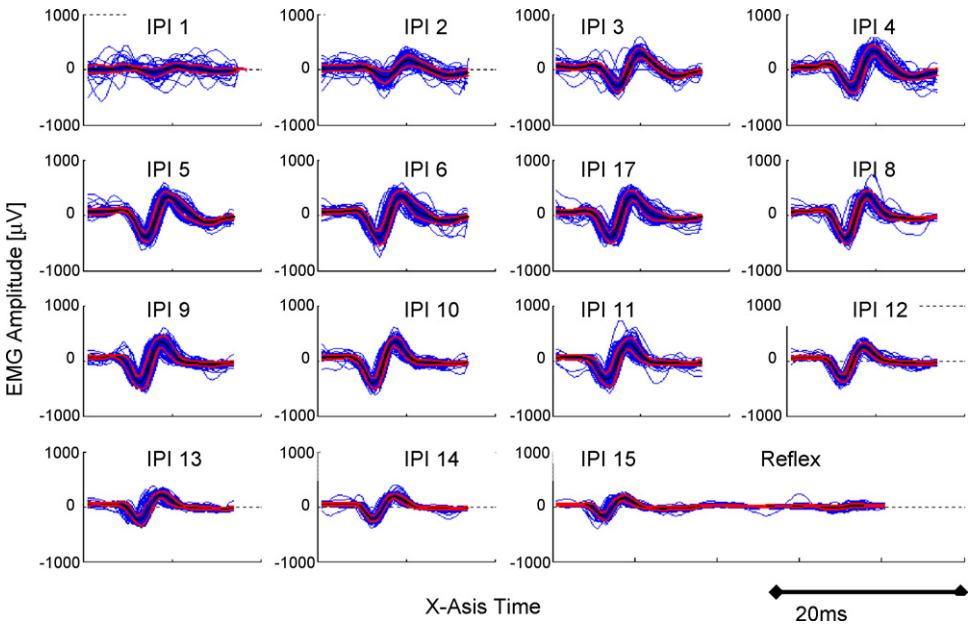

Fig. 2. An example of synchronised activity measured in the interval between stimulation pulses of the iTA, in response to FES of the tibial nerve during Se. The signal after the first pulse does not show synchronised activity in this example. The responses measured in the interval between stimulation pulses during successive pulses reveal clear waveforms between 8 and 15 ms; the timing of which may indicate a reflex.

principle [24]. The AGLR generated on and off times of the physiological EMG burst; normalised to percentage gait cycle and smooth rectified EMG (low-pass filtered to 25 Hz) enabling the determination of the magnitude of bursts [24]. Analysis of the response measured in the interval between stimulation pulses of all muscles was carried out, to investigate the direct effect of stimulation during the stimulation burst. This analysis involved the following processing sequence: for each stimulation trial of each subject, the ensemble average of all interpulse interval signals following corresponding stimuli of the bursts were calculated. The minimum number of steps per person, for each stimulation condition was 55. A root mean squared value (RMS) of each averaged signal was calculated. The RMS signals were then normalised against the RMS value found over all gait cycles for that muscle during the previous non-stimulated trial. The resulting values are thus dimensionless.

Segment angular velocities, recorded utilising the rate gyroscopes, were used to assess the impact of stimulation on kinematics. For segmental angular velocity to be used, each sensor underwent a sensor to segment calibration before the gait trials began. Calibration trials involved rotation of the segments with attached sensors in the sagittal plane, to facilitate a coordinate transformation procedure in Matlab [25]. The time course of the angular velocity signal was normalised to percentage gait cycle. The start of a gait cycle was determined using the impact response of the accelerometer of the foot sensor.

2.2. Statistical analysis

In addition to the mixed model statistical analysis previously detailed [20], mixed model analysis was also performed on the maximum angular velocities in the sagittal plane and timing of these maxima, for each segment measured with the inertial sensors. The minimum number of steps analysed, therefore over which a median value was taken, was 55. This minimum value represents simply the number of heel strikes that could be reconstructed, from the 3 min walk. The level of significance was $p < 0.05$. Mixed Model Analysis works on the same principle as ANOVA, but handles missing samples more effectively.

3. Results

3.1. Interpulse interval responses

Across each stimulation burst, 15 pulses were delivered. The response measured in the interval between stimulation pulses, and up to 50 ms after the final pulse of each burst was investigated for each muscle. In some muscles, synchronised responses to the stimulation pulses were generated. Fig. 2 is an example of synchronised activity observed in the interval between stimulation pulses of the iTA.

Synchronised responses were observed on all muscles of the stimulated leg, although less frequently on the iRF. On the contralateral side, the cRF revealed synchronous activity in two subjects, during each stimulation condition. The only other muscle on the contralateral leg to show this activity was the semitendinosus. However, this occurred only in one subject, during one stimulation condition. Fig. 2 shows that the response to the first pulse is not synchronous; therefore, as expected, a motor response was not generated in this muscle. However, the successive responses are clearly synchronous. The interval between each stimulation pulse is approximately 20 ms, the peak of the response measured following the second stimulation pulse occurs at around 10 ms, which is around 30 ms after the first stimulation pulse (SP 1). Therefore the peak, observed after the second stimulation pulse may be a reflex, in response to SP 1, etc. No synchronised activity is observed after SP 1, in Fig. 2; this shows that no motor response was generated in response to this first stimulation pulse. This indicates that we did not stimulate the peroneal nerve. However, there is no signal 35 ms after SP 15, where the final reflex is expected, if the signals observed between the stimulation pulses are purely reflexive.

Fig. 3 shows box plots of the mean, normalised RMS values calculated between each stimulation pulse for the iGM, iTA and iST. The values for the stimulated muscle are excessively larger than the other values. Note that the scale of the *y*-axis of the iGM is ten times larger than for the other two muscles. Fig. 3 highlights that responses measured in the interval between stimulation pulses of the iTA and iGM are different, in terms of the size and

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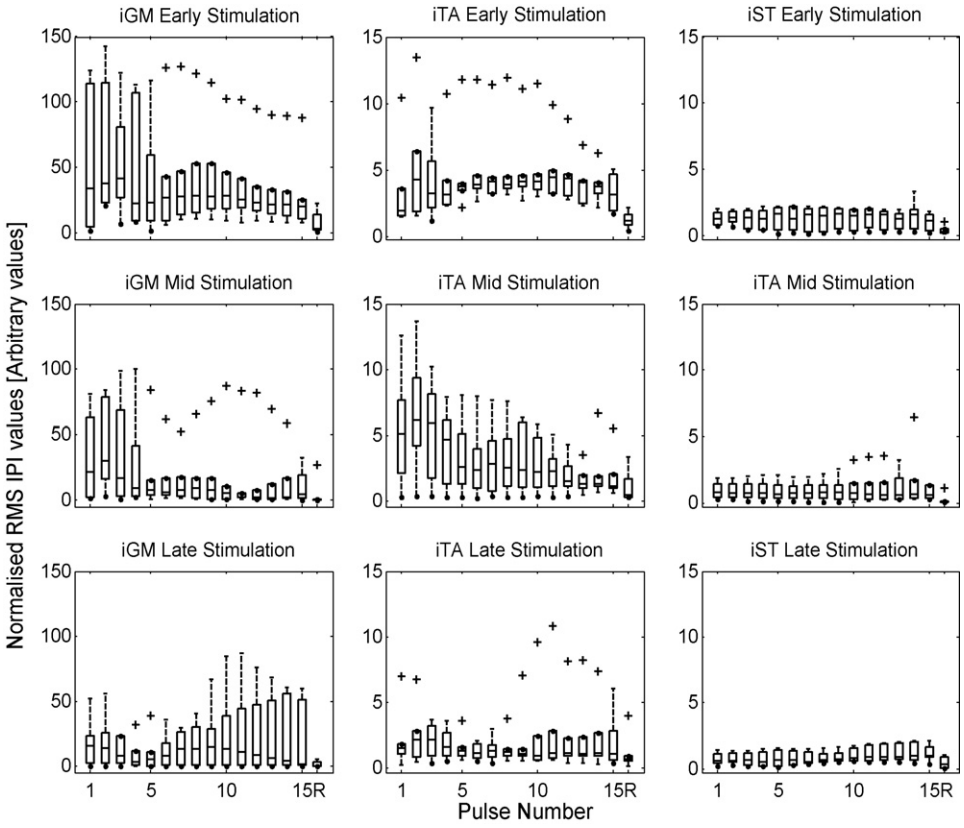

Fig. 3. Relative normalised responses measured between stimulation pulses RMS values of muscles, which revealed synchronous activity in response to FES of the tibial nerve. From left to right these are the iGM (stimulated muscle), the iTA, and the iST. Note the different scales used, to highlight the differences in magnitude. For iGM, the range is 0–150 and for iTA and iST 0–15.

variation of size across the stimulation burst. This confirms that the iTA responses are not measurements of crosstalk.

Apart from the responses observed in the iGM (details discussed in Monaghan et al. [20]), the magnitudes of responses measured from iTA were always larger than in other muscles. In the iTA, this magnitude increased between the first and second pulses for every stimulation condition, as Fig. 3 shows. Approximately 35 ms after the final pulse, an additional reflexive response is expected. In Fig. 3, "R" indicates the amplitude of the signal at this time. To prove our hypothesis that the synchronised pulses measured in the intervals between stimulation pulses are only reflexive, in response to two stimulations pulses prior to a given interval, the amplitude of this "R" is expected to be of comparable amplitude to the others in the plot. This was not the case, according to Fig. 3. Furthermore, although no synchronised responses were seen following SP 1 of Fig. 2, no visible reflex peak was observed after the final stimulation pulse either; the reason for this is observation unclear.

The synchronised activity observed in the iST in response to the stimulation were very low in value as demonstrated on Fig. 3. In Fig. 3, the outliers are represented by plus signs.

3.2. Physiological on- and offset timings

Fig. 4 shows an example of the results from the AGLR burst detection program, which was used to process the EMG bursts. Results consisted of median on and off times for each muscle per subject, per stimulation condition, normalised to percentage gait cycle [20,24]. Each column represents a given stimulation condition, NS, Se, Sm, and Sl, from left to right. The bars under each muscle burst represent the median on- to offset times of the muscles. The timing of stimulation is shown in the bottom rows, represented by the black bar. The faint grey bars protruding from the black bars represent the 25 and 75% of respective timing.

Statistical analysis revealed that the stimulation of the tibial nerve during gait affects the physiological activation timing of muscles from the same and contralateral leg muscles. These muscles were the iGM and iTA_2 (second burst of iTA) and cTA_1 and cTA_2 (first and second bursts of the cTA). The significant effects on the iGM have already been reported [20]. Table 1 summarises the remaining effects of the stimulation on the individual muscles, where a significant change was observed.

No significant effects were observed on the onset times of the ipsilateral muscles. Offset times of the iTA and cTA were statistically significantly affected by stimulation. Although statistically significant, these changes were relatively small, with the exception of the delay in iTA offset time. This is due to activity, in the between stimulation pulses, giving the impression that the muscle continued activity throughout stance.

Table 1
Significant effects of stimulation on burst timing of leg muscles.

	iTA_2 off	cTA_1 off	cTA_2 on	cTA_2 off
Overall (p)	0.013	0.01	0.015	0.009
NS–Se (Δ%)	21%	5%	1%	–
NS–Sm (Δ%)	–	–	–	–
NS–Sl (Δ%)	–	–	2%	3%
Se–Sm (Δ%)	21%	−7%	−2%	–
Se–Sl (Δ%)	22%	−5%	–	–
Sm–Sl (Δ%)	–	–	3%	–

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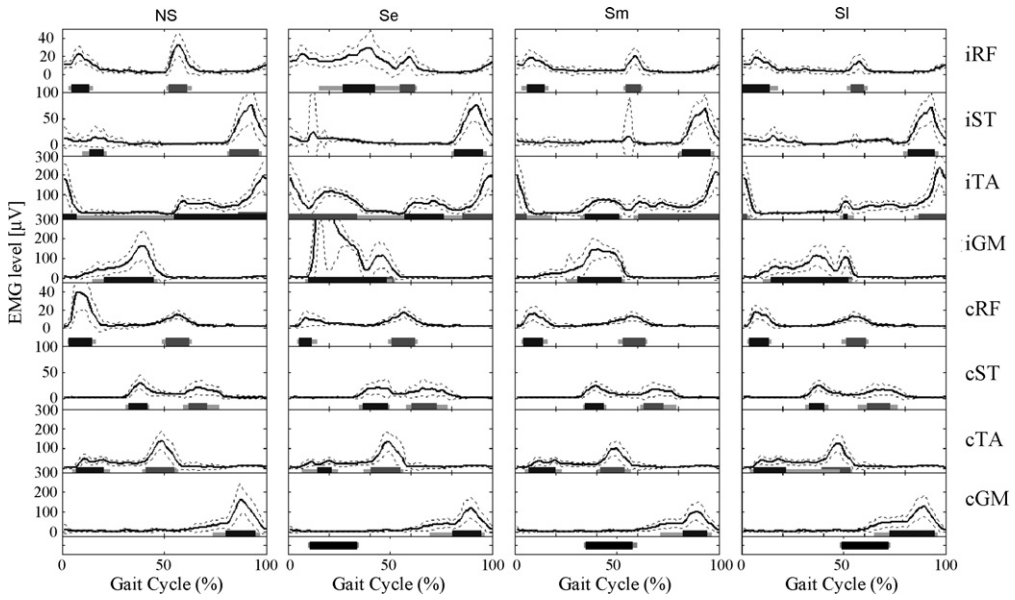

Fig. 4. Muscle activation patterns for Subject 5. Each column represents a stimulation condition, from left to right, NS, Se, Sm, Sl. Each row represents a muscle, from top to bottom: iRF, iST, iTA, iGM, cRF, cST, cTA, cGM. Solid bars represent muscle activation on. Grey bars represent the 25 and 75% of the timing. The bottom row, solid black bar highlights where stimulation occurred in the gait cycle.

3.3. Activity level

The activation level of each burst was determined utilising the AGLR method. Mixed model analysis revealed that the electrical stimulation caused a statistically significant change to the burst amplitude of three muscles, the iGM, the iST and the cTA.

From these results, it is clear that the cTA was the affected the most by stimulation. This was mainly during Sl or Sm. Se did not cause any significant changes to the activity level of the cTA, even though the cTA was physiologically active, and in the swing phase of gait at the time of stimulation.

3.4. Kinematic changes

Statistical analysis of angular velocity revealed that the most clearly defined kinematic changes occurred at the foot, during stance in response to Se; causing a large increase in angular velocity.

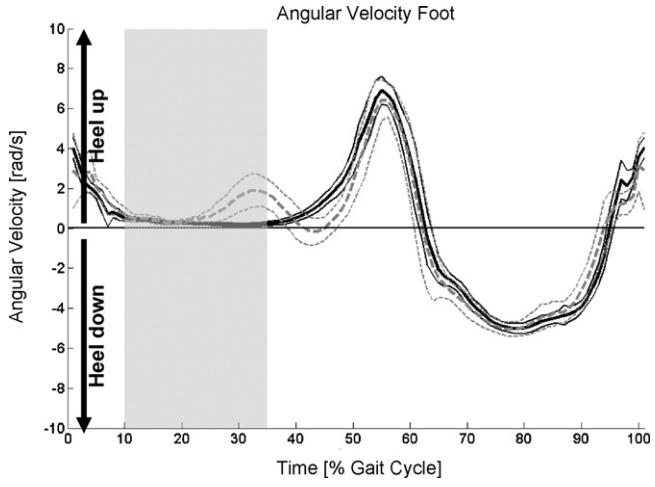

Fig. 5. Mean angular velocity of the foot, of Subject 1, in the sagittal plane during gait. Solid black line: NS. Grey dashed line: Se. Thin lines at each side of the heavy lines represent the standard deviations of the signals. The grey box indicates where stimulation was active.

Table 2
Significant effects of stimulation on muscle amplitude of activation.

Muscle/effect	
iST Decreased	cTA Decreased
Overall significance $p < 0.05$	 $p = 0.000$
Pairwise comparisons NS–Sm = 6 μV: $p = 0.05$ NS–Sl = 7 μV: $p = 0.03$	 NS–Sm = 15 μV: $p = 0.000$ NS–Sl = 7 μV: $p = 0.04$ Sm–Se = −16 μV: $p = 0.000$ Sm–Sl = −9 μV: $p = 0.03$

This has been highlighted in Fig. 5. The overall statistical significance of this change was $p = 0.001$. This peak, occurring during each stance phase is the heel rising prematurely during stance in response to Se, at 2.3 rad/s ± 0.6 compared to no stimulation, 1.2 rad/s ± 0.4. This occurrence highlights the strength of the stimulation, as the whole body weight was on the foot. However, this foot lift did not facilitate functional push-off since it was too early for the push-off peak.

The only other change was that the timing of peak of angular velocity of the shank during swing occurred significantly earlier. This trend was due to Se compared to NS ($p = 0.04$) and Sm ($p = 0.05$).

4. Discussion

Stimulation of the tibial nerve of healthy subjects influences not only the stimulated muscle, but the muscle activation patterns of muscles from both legs (Tables 1 and 2). Synchronised activity was observed in the iTA and marginally in the iST. On- and offset times of the cTA and the offset time of the iTA were significantly altered. EMG levels of the cTA and iST were significantly decreased, in response to Sm and Sl. A significant increase of angular velocity of the foot during stance was observed, during Se. Stimulation did not affect the angular velocity of the larger segments, such as the shank or thigh of the same leg, nor the contralateral shank.

Synchronised responses were observed following individual stimulus pulses, mainly in leg muscles ipsilateral to stimulation.

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Results from other studies showed that reflexive changes occurred in upper leg muscles and in muscles contralateral to stimulation [4–6,17]. We found synchronised responses in the cRF, in only two subjects, and in the cST of one subject during one stimulation condition. The magnitudes of these responses were very small; therefore we cannot rule out that they may have been remainders of the removed stimulus artefacts.

Apart from the iGM, clear synchronised responses were also visible in the iTA. Fig. 2 shows that no synchronised activity occurred after the first stimulation pulse of Se, for this subject, but did occur in response to subsequent stimulation pulses. Had a direct motor response been induced, it would have been measured after the first stimulation pulse. This was not the case. The synchronised activity appeared in response to the second stimulation pulse. This implies that a reflex response to the first stimulation pulse was visible in the interval between stimulation pulses 2 and 3; and not a direct motor response to stimulation. If the synchronised activity measured at the iTA was a motor response to the stimulation, this suggests that the peroneal nerve, which innervates the tibialis anterior muscle was stimulated, along with the tibial nerve, as we measured synchronised activity in the iGM [20]. Direct stimulation of the peroneal nerve did not occur in the example provided in Fig. 2, as no motor response is present after the first stimulation pulse. However, due to the close proximity of the tibial and peroneal nerve, the possibility that the peroneal nerve was unintentionally stimulated in other cases cannot be ruled out.

The possibility that the synchronous activity is crosstalk is not likely, because the amplitudes and patterns of amplitudes are very different, as shown in Fig. 3. It should be noted that the RMS of the response to stimulation is related to the RMS of the physiological activity to normalise the values and compare the influence of stimulation across all subjects. RMS of physiological activity was calculated because it is not possible to find the peak-to-peak value of this stochastic signal; RMS of the evoked response was found in order to relate this to the RMS of the physiological response. As described in our previous work [20], an RMS value of 1, does not mean that the activation was at the same level. The synchronised EMG of the response to stimulation is a linear summation of activation from many motor units. However, during physiological activation, RMS EMG is proportional to the square root of the amount of independently activated motor units.

Previous studies reveal that bilateral reflex responses were generated in response to cutaneous stimulation [4–6,17]. We did not observe these reflexive changes, as the lack of bilateral synchronised signals showed. This may be attributed to different stimulation conditions used [4–6,17] as the impact of stimulation depends on stimulation parameters [5,6]. In our study, 300 ms bursts of 15 pulses, at 50 Hz were applied. Zehr et al. [6] applied three to six pulses at 200 Hz to the sural and tibial nerves, Tax et al. [5] gave five pulses at 200 Hz. Berger and Quintern [26] applied a single pulse to the tibial nerve during gait. Due to the differing parameters, it is difficult to compare the activity levels achieved during each stimulation study. The other studies mentioned randomised stimulation time between gait cycles, whereas we applied a stimulation burst at the same predictable time for each stimulation condition tested. Because of this, adaptation of the physiological activation patterns may have occurred. Thus a centrally controlled, non-reflexive response may have been involved, particularly on the cTA, where synchronous activity was not detected, but a change in burst timing or amplitude was reported.

Random stimulation of the tibial nerve at the foot [4–6,17], is effectively an electrical perturbation. Tax et al. [5] reported that the reflexive response to perturbation in the cat serves to minimise stumbling and ensure that balance and cadence is maintained. Based on other studies, relating the similar nature of cat and human reflexes, it is possible that human reactions serve the same purpose as those of the cat.

The stimulation condition Sm applied during our experiments was not expected to cause strong deflections from an optimal gait pattern. Our aim was to support push-off; therefore, particularly with Sm, push-off characteristics were expected to have appeared more pronounced. This was not the case, with Sm; we have previously attributed this to the occurrence of antidromic activation [20], colliding with the physiological activation at push-off. This collision would block physiological activity and prevent a net increase in muscle activity. Blockage due to antidromic firing would be less prevalent in early stance, where the unwanted heel rise was observed, because physiological calf muscle activity was still low; not reaching maximum activity level, until terminal stance.

The FES caused shorter bursts and decreased amplitude of the cTA. Tax et al. [5] stated that the response to stimulation is not necessarily related to the phase that the stimulated limb is in, but is dependent on the phase of the leg in which the responses occur. They found that the cTA of healthy subjects showed a suppression of activity in response to stimulation during contralateral end swing [5]. The decreased activity levels that we observed, may also be attributed to inhibition of activity, as the changes occurred during Sm and Sl; corresponding to contralateral terminal swing and early stance. During this time, the cTA is actively decelerating the limb, in preparation for heel strike, and stance, while the side ipsilateral to stimulation enters into push-off. Therefore these subjects experienced both shorter bursts and decreased amplitude of the cTA due to sensory input from antidromic firing or from cutaneous activation, indicating active triceps surae. This caused the cTA to adapt its activation pattern. Sensing that the opposite limb was ready for push-off, it had to terminate activation earlier than normal. It would be expected that as a consequence, the triceps surae would onset earlier in order to provide balance and posture control during stance. However, this is more the role of the soleus [27], which was not measured in these experiments. This would be an interesting addition for a future study.

The stimulation applied caused significant changes to the foot during early stance, actually causing the ankle to lift, with full body weight. We found less kinematic effects of stimulation on the larger segments. Zehr et al. [6] also noted that their cutaneous perturbation had a larger effect on the kinematics of more distal limb segments. It was additionally unexpected that during the stimulation time that we considered to be optimal, no significant changes occurred to the kinematics. However, as we described [20] this could be due to collisions of physiological activation with antidromic firing. With no net change in activation, no change in the mechanical effect of the stimulated muscle can be expected.

It was expected that the results would provide a more clear indication of the effects of FES on the tibial nerve of healthy subjects. It would be beneficial to continue these tests on a larger healthy population, as well as to incorporate other measurements such as changes in kinetics, to provide more information about the change to push-off, including ankle moment, and power, however this was beyond the scope of the results presented here. The present study shows that the stimulation interacts with the CNS, yielding modified control of the on- and offset times of muscles at both sides. This is important for future applications of stimulation, for patient groups who can benefit from this, as it means that stimulation not only affects the stimulated muscle but also the physiological motor control by the CNS. For optimal utilisation of FES for rehabilitation purposes, better understanding of the physiological effects of FES is required.

The aim of the present research was to investigate how FES of the tibial nerve affects the muscle activation patterns of upper and lower leg muscles in healthy subjects, with the intention of

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applying the technique, in the future, to improve push-off of the post-stroke population who show a clear lack of this function during gait [7–12]. The results show that changes occur in healthy subject gait. In particular, unwanted early heel rise occurred in response to Se. Although the Sm did not induce the exaggerated push-off movements we had expected in these healthy subjects, we hypothesise that tibial nerve stimulation will benefit those with low calf muscle activation [7–12]. The antidromic activation, which we believe blocked physiological activation in this healthy group [20] is not expected in subjects with low activation, as this did not manifest during Se when the calf muscle activity was relatively low. Furthermore, Bajd et al. [18,28,29] were able to induce push-off in SCI subjects, and the drop foot stimulator has proven to be successful in carefully selected subject groups since 1960s [2,3,30].

The next step is to repeat the experiments performed in this study on the stroke population, with a primary goal of restoring lost push-off function in these subjects as well as to investigate changes in muscle activation patterns. As well as decreased amplitude of muscle activation, stroke subjects also exhibit co-contractions [13], and extra bursts [14] of the muscles on the non-paretic side. Therefore, further to the goals stated, it is hypothesised that FES of the paretic calf muscles will reduce the need for compensatory activity of the non-paretic side.

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Conflict of interest statement

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