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Corresponding Author:	Andrei Leonardo Perez Olivera London South Bank University London, UNITED KINGDOM
First Author:	Andrei Leonardo Perez Olivera
Order of Authors:	Andrei Leonardo Perez Olivera
	Matthew C. Solan
	Kiros Karamanidis
	Katya N. Mileva
	Darren C. James
Abstract:	M. abductor hallucis (AbH) is the strongest intrinsic foot muscle and its dysfunction underlies various foot disorders. Attempts to strengthen the muscle by voluntary exercises are constrained by its complex morphology and oblique mechanical action, which leads to an inability even in asymptomatic individuals to fully activate AbH. This study investigated the extent and magnitude of this inability whilst also providing preliminary evidence for the virtue of targeted sub-maximum neuromuscular electrical stimulation (NMES) as a countermeasure for an AbH activation deficit. The voluntary activation ratio (VAR) was assessed via the twitch interpolation technique in the left AbH of 13 healthy participants during maximum voluntary 1st metatarsophalangeal joint flexion-abduction contractions (MVC). Participants were grouped ("able" or "unable") based on their ability to fully activate AbH (VAR ≥0.9). 7s-NMES trains (20Hz) were then delivered to AbH with current intensity increasing from 150% to 300% motor threshold (MT) in 25% increments. Perceived comfort was recorded (10cm-visual analogue scale; VAS). Only 3 participants were able to activate AbH to its full capacity (able, mean(range) VAR: 0.93 (0.91-0.95), n=3; unable: 0.69 (0.36-0.83), n=10). However, the maximum absolute forces produced during the graded sub-maximum direct-muscle NMES protocol were comparable between groups implying that the peripheral contractility of AbH is intact irrespective of the inability of individuals to voluntary activate AbH to its full capacity. These findings demonstrate that direct-muscle NMES overcomes the prevailing inability for high voluntary AbH activation and therefore offers the potential to strengthen the healthy foot and restore function in the pathological foot.

A voluntary activation deficit in *m.* abductor hallucis exists in asymptomatic feet.

Andrei L. Pérez Olivera[1], Matthew C. Solan[2], Kiros Karamanidis[1], Katya N. Mileva[1], Darren C. James[1]

[1] Sport and Exercise Science Research Centre, School of Applied Sciences, London South Bank University, UK.

[2] Department of Trauma and Orthopaedic Surgery, Royal Surrey County Hospital, Guildford, Surrey GU2 5XX, UK.

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Corresponding Author
Andrei Leonardo Pérez Olivera, PhD Candidate
Sport and Exercise Science Research Centre
School of Applied Sciences, London South Bank University
103 Borough Road, London SE1 0AA, UK
Telephone: +44 (0)2078157910
E-mail: perezola@lsbu.ac.uk

Abstract

M. abductor hallucis (AbH) is the strongest intrinsic foot muscle and its dysfunction underlies various foot disorders. Attempts to strengthen the muscle by voluntary exercises are constrained by its complex morphology and oblique mechanical action, which leads to an inability even in asymptomatic individuals to fully activate AbH. This study investigated the extent and magnitude of this inability whilst also providing preliminary evidence for the virtue of targeted sub-maximum neuromuscular electrical stimulation (NMES) as a countermeasure for an AbH activation deficit. The voluntary activation ratio (VAR) was assessed via the twitch interpolation technique in the left AbH of 13 healthy participants during maximum voluntary 1st metatarsophalangeal joint flexion-abduction contractions (MVC). Participants were grouped (*"able"* or "*unable"*) based on their ability to fully activate AbH (VAR ≥0.9). 7s-NMES trains (20Hz) were then delivered to AbH with current intensity increasing from 150% to 300% motor threshold (MT) in 25% increments. Perceived comfort was recorded (10cm-visual analogue scale; VAS). Only 3 participants were able to activate AbH to its full capacity (*able*, mean(range) VAR: 0.93 (0.91-0.95), *n*=3; *unable*: 0.69 (0.36-0.83), *n*=10). However, the maximum absolute forces produced during the graded sub-maximum direct-muscle NMES protocol were comparable between groups implying that the peripheral contractility of AbH is intact irrespective of the inability of individuals to voluntary activate AbH to its full capacity. These findings demonstrate that direct-muscle NMES overcomes the prevailing inability for high voluntary AbH activation and therefore offers the potential to strengthen the healthy foot and restore function in the pathological foot.

Introduction

M. abductor hallucis (AbH) has the largest physiological cross-sectional area of all intrinsic foot muscles (Kura et al., 1997; Tosovic et al., 2012) and is thus the main force generating muscle in the foot. It flexes and abducts the 1st metatarsal phalangeal joint (1MPJ) and this oblique mechanical action is functionally relevant in stiffening 1MPJ for postural control (Fiolkowski et al., 2003; Kelly et al., 2012; Kelly et al., 2015) and forward progression during gait (Farris et al., 2019; Kelly et al., 2015). In doing so AbH also acts as dynamic controller of the medial longitudinal arch (Kelly et al., 2014; Kelly et al., 2015; Kirby, 2017; Wong, 2007) and a conduit for the reciprocal transfer of energy between the ankle, the forefoot and the ground (Farris et al., 2019). AbH dysfunction underlies a number of common foot deformities (Arinci Incel et al., 2003; Latey et al., 2018; Zhang et al., 2019) leading to diminished functional capacities in a large number of individuals; most notably in sufferers of Hallux Valgus - an insidious forefoot deformity that affects ~20% of adults aged 18 to 65 and ~35% over the age of 65 (Nix et al., 2010). The consequence is an impaired gait pattern with potential for developing other musculoskeletal disorders (Shih et al., 2014), as well as postural instability, which in the elderly increases the likelihood of falling (Menz and Lord, 2005). Training regimes focussed on strengthening AbH are therefore sought for prevention and early stage treatment for this condition.

However, targeted strengthening of AbH via voluntary exercises is difficult. Despite the potential efficacies of gross foot manoeuvres such as the short-foot (Jung et al., 2011; Mulligan and Cook, 2013) and toe flexor (Goldmann et al., 2013; Jung et al., 2011; Yamauchi and Koyama, 2019a) exercises for strengthening the intrinsic foot musculature, there is typically a greater reliance on the extrinsic foot muscles during these movements (McKeon et al., 2015; Yamauchi and Koyama, 2019b). Consequently, AbH activation has been shown to contribute with half of its maximal capacity (Yamauchi and Koyama, 2019b). The toes spread exercise, on the other hand, requires more of AbH activation and has an evidence base for targeted strengthening the muscle (Kim et al., 2013; Kim et al., 2015). Notwithstanding the efficacy of the exercise, the oblique mechanical action of AbH means that voluntary activation of the muscle is challenging for many healthy people (Arinci Incel et al., 2003; Boon and Harper, 2003). An inability to voluntary activate AbH partially or completely implies a potential insufficiency in neural activation and force generating capacity of the muscle and so strengthening AbH is equivocally important to these asymptomatic individuals as it is to pathological cohorts.

The complex morphology of AbH may explain a common neuromuscular deficit in asymptomatic feet. The muscle has distinct architectural fibre arrangements (unipennate, bipennate and multipennate) along its length, which have been shown to vary between individuals (Tosovic et al., 2012). Importantly, a feature of muscles with broad origins and distinct segments, such as AbH, is the selective recruitment of motoneurons to fine-tune a movement and control the differentiated lines of force within the differing segments (functional differentiation) (Paton and Brown, 1994; Tosovic et al., 2012). AbH typically exhibits a multipennate arrangement at the proximal segment of the muscle (Tosovic et al., 2012), which will then be characterised by distinct populations of motor units to perform 1st metatarsal phalangeal joint (1MPJ) flexion and abduction independently of each other. Thus, activating AbH to its full capacity would require the synchronous recruitment of both pools of motor units. Hence, in individuals who do not actively train (consciously or subconsciously) the mechanical action of AbH, this synchronous recruitment and full activation of the muscle may not be achievable. Indeed, Arinci Incel et al. (2003) reported full interference patterns of motor unit potentials from synergistic muscle activation to AbH in 70% (*n*=14) of their cohort during voluntary abduction of the Hallux, whereas Boon and Harper (2003) noted that 19% of their participants were in fact unable to voluntarily activate AbH. The prevalence of an inability to fully activate AbH therefore seems to be common; however, the extent of the activation deficit has yet to be experimentally quantified.

The interpolated twitch technique is an approach used for quantifying the neural drive to a target muscle and thus the completeness of a voluntary muscle activation (Allen et al., 1995; Behm et al., 1996; Taylor, 2009). Despite concerns over its validity (see Horstman, 2009), it continues to be extensively employed in neuromuscular research using accepted methodologies that address the limiting factors of the technique (Herbert and Gandevia, 1999; Gandevia, 2001; Bampouras et al., 2006; de Haan et al., 2009). Simply put, it consists of electrically stimulating a nerve trunk or axonal terminal branches (i.e. at or near the motor point) during a maximal voluntary contraction and an increase in force elicited by the superimposed stimulation highlights a deficit in voluntary activation. Quantification of the deficit is commonly expressed as a ratio (VAR) of the force evoked by a stimulus delivered during MVC to the force elicited from an identical stimulus delivered at rest.

The aim of the present study therefore was to use the twitch interpolation technique to establish the prevalence and magnitude of a voluntary activation deficit in AbH of asymptomatic feet. A secondary aim of the study was to provide preliminary evidence that targeted sub-maximum neuromuscular electrical stimulation (NMES) of AbH can overcome the voluntary activation deficit and elicit equivocal force to individuals who are capable of complete voluntary AbH activation. Thus, the study hypotheses were: i) a high prevalence exists for incomplete voluntary activation of AbH in healthy individuals; and ii) direct muscle NMES will evoke comparable 1MPJ flexion-abduction force in participants with and without a voluntary AbH activation deficit. The implication of this secondary hypothesis is that NMES may be used as strengthening modality for restoring function in the healthy foot as well as offsetting weakness in the pathological foot.

Methods

Participants

Thirteen healthy volunteers (10M/3F, mean ± standard error of the mean [SEM]: 25.2 ± 1.7 years; 74.0 ± 3.5 kg; 1.7 ± 0.0 m) signed a written informed consent to participate in this study that received prior local ethical approval (SAS1807) and complied with the Declaration of Helsinki (2013). Prior to participation, all volunteers completed a health screen questionnaire and reported good health and absence of lower extremity injuries, underlying pathologies and neurological problems.

Experimental procedures

Study design

Participants visited the laboratory on three separate occasions: for familiarisation and two main testing sessions. The familiarisation session served to acclimate participants to all experimental procedures and to optimise the delivery of NMES to AbH (see below for protocol). In the first main session, the optimisation procedures were repeated for verification purposes, then participants' ability to maximally activate AbH voluntarily was quantified using the interpolated twitch technique. During the second main session and following verification of AbH motor point location and motor threshold (MT), the force evoked by 7s trains of NMES delivered to AbH at different sub-maximum_stimulus intensities was recorded.

In each visit, participants were seated in a custom-made apparatus with their left foot securely fixed at the ankle and forefoot and positioned in 35º plantar flexion with respect to foot flat (Goldmann and Brüggemann, 2012; Olivera et al., 2020) (Figure 1A). The Hallux was covered with a polymer gel support and secured to a force transducer by a semi-rigid thermoplastic cable that encapsulated the proximal phalanx immediately distal to 1MPJ and mounted to the experimental apparatus above the foot in 10° dorsal flexion (Olivera et al., 2020) (Figure 1B). During the familiarisation sessions and the first main trial a uni-axial force transducer (1000Hz sampling frequency, range: 0-250 N; RDP Electronics Ltd, UK), calibrated for measuring low forces and mounted to the experimental apparatus above the foot was used to record the voluntary and interpolated twitch forces (Figure 1). In the second main trial, a tri-axial force transducer (1000Hz sampling frequency, range: 0-50N; Applied Measurements Ltd, UK) was used to account for the abduction force elicited from direct-muscle NMES. The force data was collected through an A/D convertor (1401power, Cambridge Electronic Design Ltd., UK) and imported into Spike2 software (v7.12, CED Ltd., UK) for analysis.

Procedures for NMES optimisation

The procedures for optimisation of the direct-muscle NMES delivery involved AbH motor point location and MT determination, which ensured the effectiveness of, and participant adherence to, the experimental protocol (Gobbo et al., 2014). The navicular tuberosity was used as the origin to a 7 x 4cm matrix drawn over the skin overlying AbH (James et al., 2013; James et al., 2018; Olivera et al., 2020) (Figure 1B). A single square-wave (1ms) pulse was delivered systematically over each point of the matrix at 10mA current intensity using a constant-current stimulator (DS7A, Digitimer, UK) and a custom-made pen-type cathode with the anode positioned over 1MPJ (Figure 1B). The stimulation point at which the current evoked the largest twitch force was identified as the AbH motor point. Then, trains of 5 x 1ms pulses were delivered to this location at 20Hz pulse frequency (Olivera et al., 2020) (Figure 1C) and increasing current, starting at 0.5mA with increments of 0.5mA. AbH MT represented the lowest current, which evoked a twitch force that exceeded the baseline force level by 2 standard deviations (James et al., 2018).

To identify the stimulus intensity capable of recruiting the full range of AbH muscle force, a twitch force recruitment curve in response to single square-wave (1ms) pulses delivered to the motor point at increasing current intensities was constructed (Figure 2A). The stimulation started at 1mA current intensity with 1mA increments until saturation of the evoked twitch force amplitude was reached. Finally, the recorded current at this point was multiplied by 130% to ensure supramaximal stimulation intensity (James et al., 2018) for the interpolated twitch technique delivery (Allen et al., 1995).

Voluntary activation testing

Participants attempted 3 x ~5s 1MPJ flexion MVCs separated by 5 minutes rest. In each, participants concomitantly attempted abduction of the Hallux in order to fully engage AbH contraction. Upon reaching the force plateau, a supramaximal (130%) 1ms 100Hz doublet stimulus (Behm et al., 1996; Oskouei et al., 2003) was delivered over the motor point of AbH (Figure 2B). The additionally evoked twitch force represents the engagement of motor units that have not been activated through voluntary command and therefore are excited by the interpolated stimulus (Allen et al., 1995; Behm et al., 1996; Taylor, 2009). Participants were instructed to maintain maximal effort until instructed to relax, following which, a second supramaximal twitch (same stimulus parameters) was evoked 1-2s into rest (Figure 2B) to account for possible potentiation of neural drive to the muscle during voluntary contractions (Allen et al., 1995). Visual feedback and appropriate encouragement were provided as well as demonstration, instruction and practice trials prior to recording the MVCs (Gandevia, 2001).

Sub-maximum evoked (NMES) AbH force testing

7s NMES trains of 1ms pulses were delivered to AbH at 20Hz pulse frequency with increasing current intensity starting at 150% MT with 25% MT increments up to 300% MT (Figure 2C). One minute rest was given between each train to avoid cumulative fatigue. Participant's perceived discomfort was quantified for each NMES intensity with a 10cm visual analogue scale (VAS), where 0 represents 'no discomfort' and 10 represents 'maximal discomfort' (Maffiuletti et al., 2014).

Data analysis

The voluntary activation ratio (VAR) for AbH during each MVC was calculated using the following equation (Allen et al., 1995):

$$VAR = 1 - \left(\frac{interpolated\ twitch\ amplitude - MVC\ force}{resting\ twitch\ amplitude}\right)$$

where the *interpolated twitch amplitude* is the extra force evoked from AbH in response to the supramaximal doublet stimulus during MVC and *MVC force* is the maximal force measured prior stimulus onset (Figure 2B). The highest VAR and corresponding MVC force achieved out of the 3 attempts from each participant was considered for analysis. Participants were deemed "*able*" to fully activate AbH if their VAR was ≥0.9 (e.g., 90% of full capacity) (Herbert and Gandevia, 1996). Correspondingly, those with a VAR that was <0.9 comprised the “unable” group.

The maximum force (N) evoked during each of the 7s NMES trains (Figure 2C) was entered for analysis. Then the mean (±SEM) NMES current (mA) required at each stimulus intensity was plotted against the respective maximum evoked force (N) and VAS score to assess the relationship between the stimulus intensity, force production and participants’ discomfort, respectively.

In addition, the maximum evoked force (N) in the *able* group (participants with a VAR ≥0.9) was normalized (%) to the MVC force from the contraction that produced the highest VAR and then plotted against the respective NMES stimulus intensity (150% to 300% MT) to assess the relative magnitude of the sub-maximum NMES delivery.

Results

The interpolated twitch technique identified that only 3 participants (23%) in the cohort were able to activate AbH ≥90% (i.e. VAR ≥0.9) of its full capacity (mean (range) VAR: 0.93 (0.91 – 0.95); Figure 3A). The average (range) VAR of the remaining 10 participants (*unable*) was 0.69 (0.36 – 0.83). Despite this difference between the groups, their average (range) MVC force was comparable (*able*: 34.8 (29.8 – 41.2)N vs *unable*: 31.8 (17.6 – 75.4)N) but with a larger MVC force range.

Despite not being able to activate AbH to its full capacity, the *unable* group produced comparable NMES-evoked forces to the *able* group during the graded sub-maximum NMES protocol (Figure 3B). This was achieved at lower current intensities (Figure 3B), but with a slightly higher pain score (Figure 3C). For example, stimulation at 200% MT generated an average force of 11.8 (2.0 – 27.8)N in the *unable* group and of 11.2 (7.0 – 18.5)N in the *able* group, rated with VAS scores of 4.5 (1 – 6.5) compared to 3.3 (2 – 5), respectively. The current intensity at 200% MT was equivalent to 6.5 (2.4 – 16.4)mA in the *unable* group compared to 7.9 (6.0 – 10.8)mA) in the *able* group. The relative magnitude of the evoked force in the *able* group via the sub-maximum NMES protocol ranged on average from 14% MVC at 150% MT to 54% MVC at 300% MT (Table 1, Figure 3D).

Discussion

The aim of this study was to quantify the prevalence of voluntary activation deficit in AbH within the asymptomatic foot. Using the twitch interpolation technique and a cohort of healthy individuals this study demonstrated that: (i) only few (23%) of the participants were able to activate AbH to near full capacity as ascertained by achieving a high voluntary activation ratio (≥0.9) during a maximum 1MPJ flexion-abduction contraction; and ii) targeted sub-maximum NMES protocol is applicable to evoke forces from AbH independently on the individual's ability for voluntary activation; and at the intensities investigated, causes low-to-medium discomfort level. These two findings support the experimental hypothesis posed.

The finding that the majority of participants, or 77% of our cohort, were unable to voluntary activate AbH to at least 90% (i.e. VAR ≥0.9) of its maximal force generating capacity was not surprising. This finding is consistent with previous studies reporting, indirectly, a partial (Arinci Incel et al., 2003) or complete inability (Boon and Harper, 2003) to voluntary activate AbH. The cause of this inability is uncertain but it is reasonable to suspect that an insufficiency in neural drive to the muscle, due to its morphological variability and specificity, is responsible for being unable to activate AbH close to its full capacity. Given its architecture and correspondingly its distinct populations of motor units (Paton and Brown, 1994; Tosovic et al., 2012), it could be that synchronous activation of these pools is unachievable for many individuals since they may not necessarily possess the innate motor coordination that would otherwise be developed with active (conscious or subconscious) training of the intrinsic musculature. Thus, our instruction to participants during data collection was to perform Hallux abduction concomitant to maximum 1MPJ flexion by encouraging them to displace a small ball of plasticine, placed on the dorsal aspect of 1MPJ, away from the digits during the MVC. A number of participants could not achieve this voluntarily; therefore, we can speculate that the supraspinal drive of these individuals was insufficient or inefficient to innervate and synchronise the abduction motor unit pools for a full activation of AbH.

Despite the inability to perform the contraction as instructed, the *unable* participants still exerted a comparable MVC to the *able* participants (31.8N vs 34.8N, respectively). Since VAR confirmed that AbH activation capacity had not been reached in the *unable* group, it is likely that they performed the instructed movement with greater activation of the prime Hallux flexor intrinsic (i.e. flexor hallucis brevis; FHB) and extrinsic (i.e. flexor hallucis longus; FHL) muscles (Arinci Incel et al., 2003; Bruening et al., 2019; Gooding et al., 2016; Yamauchi and Koyama, 2019b). Indeed, the upper force range recorded during the MVC trials (75.4N) suggests this was the case as this value is comparable to the lower bound maximum force generating capacity of AbH and FHB combined (Kurihara et al., 2014). With this said, we cannot be certain that there is no functionally relevant contribution of synergist activation of these toe flexor muscles on the resultant MVC force recorded in the *able* participants. The influence of FHL was minimised in this study by placing the ankle in 35º plantar flexion (Goldmann and Brüggemann, 2012), but negating the activation of FHB during a 1MPJ flexion-abduction MVC is difficult. Therefore, we acknowledge that the relative magnitude (%MVC) of the evoked force from these participants during the graded sub-maximum NMES protocol (Figure 3D) may be overestimated. This implies that to achieve a certain percentage of the true AbH MVC will in fact require less current intensity than reported here.

Direct-muscle NMES evoked comparable (absolute) forces in all participants independently on their ability to voluntarily activate AbH to full capacity (Figure 3B); and this was achieved with a stimulation intensity causing relatively low discomfort. This finding implies that the peripheral contractility of AbH is intact irrespective of the inability to voluntary contract AbH to its full capacity; therefore, it might be possible to increase the voluntary activation capacity of the muscle with targeted NMES exposure.

Training muscle via evoked contractions is different to voluntary activation of muscle because motor unit discharge patterns are ostensibly non-selective, spatially fixed and temporally synchronised to stimulation frequency (Bickel et al., 2011). Despite this, previous research has consistently recommended the use of NMES for muscle strengthening benefits at intensities which evoke muscle contractions of ≥20% MVC (Alon and Smith, 2005; Maddocks et al., 2016; Maffiuletti et al., 2019; Talbot et al., 2003). Indeed, NMES training of the quadriceps at low stimulus intensities, but above the recommended training level (~30-60% MVC), proved sufficient to induce beneficial adaptations in muscle morphology and concomitant strength gains (Natsume et al., 2018), whereas NMES training at 5-10% MVC (in the same muscle) was not (Natsume et al., 2015). In the present study, the NMES current intensity at which all *able* participants exceeded the 20% MVC threshold was 200% MT (mean: 31% MVC; range: 21-45% MVC; Figure 3D) and this was achieved with low-to-mild discomfort (Figure 3C). Our data show that training AbH at/around the minimum suggested threshold for strength gains (20% MVC; Alon and Smith, 2005; Maddocks et al., 2016; Maffiuletti et al., 2019; Talbot et al., 2003) may not only be effective, but is also tolerable, which suggests a potential clinical utility for this approach to alleviate or combat common foot pathologies. In addition, targeting the motor point of AbH optimises the delivery of NMES by requiring less intensity to evoke a contraction (Gobbo et al., 2014), which along with using its MT for stimulus intensity selection should minimise the recruitment of deeper motor units belonging to surrounding muscles that occurs at high intensities (Bickel et al., 2011).

Finally, the interpolated twitch technique is not without its limitations, and these should be considered when interpreting the present findings. Firstly, it has been noted to be less sensitive at high levels of muscle activation (Herbert and Gandevia, 1999) and that it relies on the assumption of full activation capacity at a VAR of 100% (de Haan et al., 2009). It has also been shown to be sensitive to changes of joint-angle configuration (Bampouras et al., 2006) and it may also under-report voluntary activation due to antidromic collision with the efferent (voluntary) output following the interpolated twitch stimulus (Gandevia, 2001). However, some of these neurophysiological complexities can be overcome. Specifically, the second consecutive pulse of a supramaximal doublet stimulus maximises force production due to the recruitment of the motor units which may have been in the refractory period following the first pulse (Belanger and Comas, 1981; Herbert and Gandevia, 1999). This, in turn, overcomes the antidromic phenomena and variability in force found in single pulses (Oskouei et al., 2003). Additionally, the sensitivity of the technique to joint-angle configuration was overcome by positioning the 1MPJ in 10° of dorsal flexion, which we have previously shown to be optimal for AbH force production (Olivera et al., 2020). Therefore, assuming that a VAR ~1 (i.e. ~100%) represents full activation capacity, we believe the experimental protocol reported here represents a robust method to quantify insufficiencies in AbH activation.

In conclusion, the findings of the present study have shown that a large prevalence of (healthy) participants who are unable to activate AbH to its full capacity, which is most likely due to the complex muscle morphology and/or a neural activation deficit. Despite this, targeted NMES applied directly to AbH at well-tolerable intensities can alleviate these deficits and evoke comparable forces in all individuals, which has implications for both improving function in the healthy foot and offsetting weakness in the pathological foot.

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Table 1. Mean (range) NMES-evoked force delivered at increasing stimulus intensities and expressed as a percentage of MVC in *able* participants (VAR ≥0.9; *n*=3).

	NMES stimulus intensity relative to AbH motor threshold						
	150%	**175%**	**200%**	**225%**	**250%**	**275%**	**300%**
% MVC	14 (4-32)	26 (17-44)	31 (21-45)	38 (26-46)	43 (24-57)	49 (31-66)	54 (38-72)

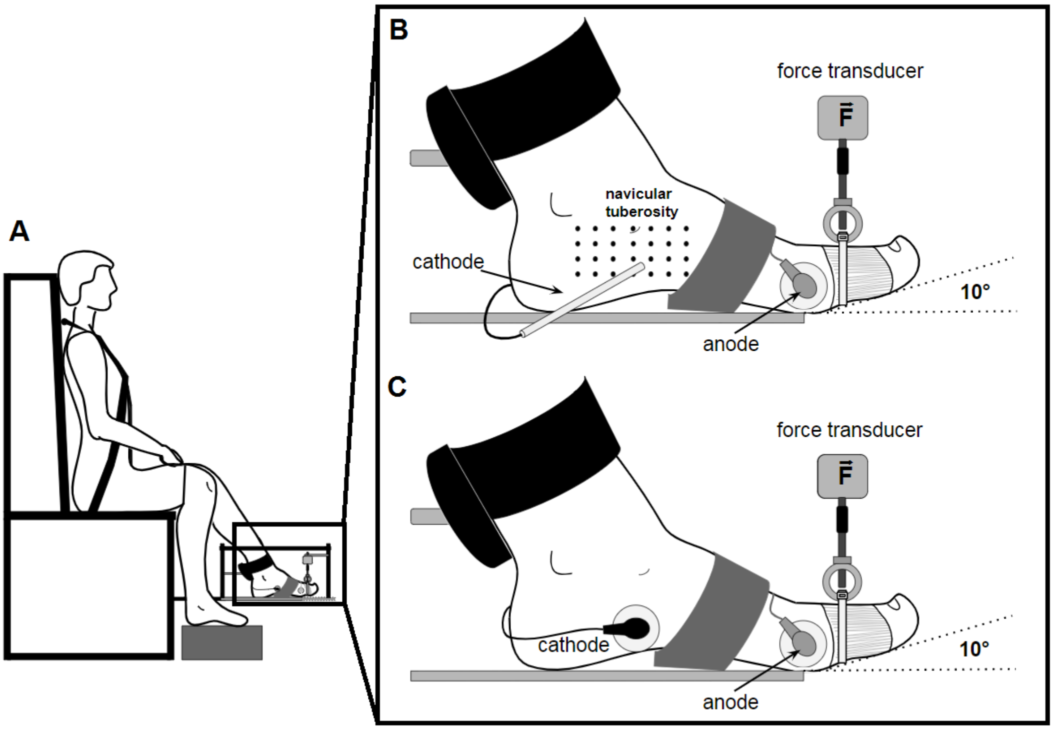


Figure 1. Experimental set-up and foot-hallux arrangement: A) Participant seating position on the custom-built apparatus with the left foot fixed to the foot platform and the ankle positioned at 35º plantarflexion; B) Sagittal view of the experimental foot with the Hallux suspended from the force transducer (tri-axial or uni-axial) in 10º 1MPJ dorsal flexion. A 7 x 4 cm matrix drawn over the skin overlying AbH, with the navicular tuberosity used as an origin point, serves as a map for motor point location; C) Electrode positioning for motor threshold determination and NMES delivery to AbH: cathode over the motor point and anode over 1MPJ.

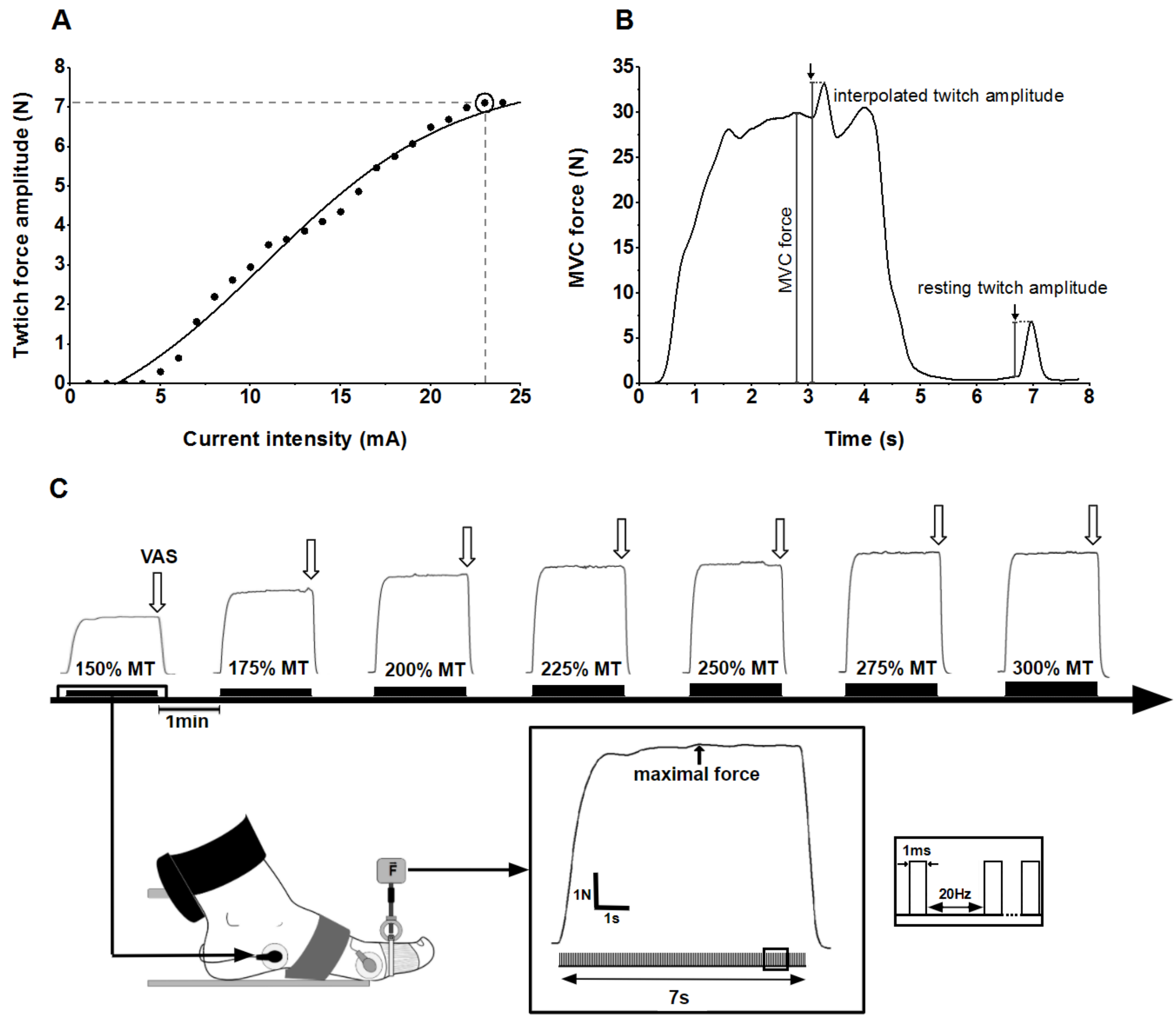


Figure 2. Examples of the stimulation protocols used in the study and the respective evoked AbH forces: A) Twitch force recruitment curve constructed by gradually increasing stimulus strength to identify the current intensity (mA; vertical dashed line) corresponding to maximal twitch force (circled); B) Twitch interpolation evoked by 1ms 100Hz paired pulse stimulation delivered during and following a maximum voluntary 1MPJ flexion contraction with abduction of the Hallux. The arrows indicate the stimulation time points and the vertical lines mark the interpolated, resting and MVC force amplitudes used to calculate the voluntary activation ratio; C) NMES-evoked force from 7s 20Hz trains of 1ms pulses delivered to AbH motor point with stimulus intensities increasing from 150% to 300% of motor threshold (MT). At each intensity, maximum AbH evoked force was recorded (filled vertical arrow) as well as the level of perceived discomfort (VAS; open vertical arrows).

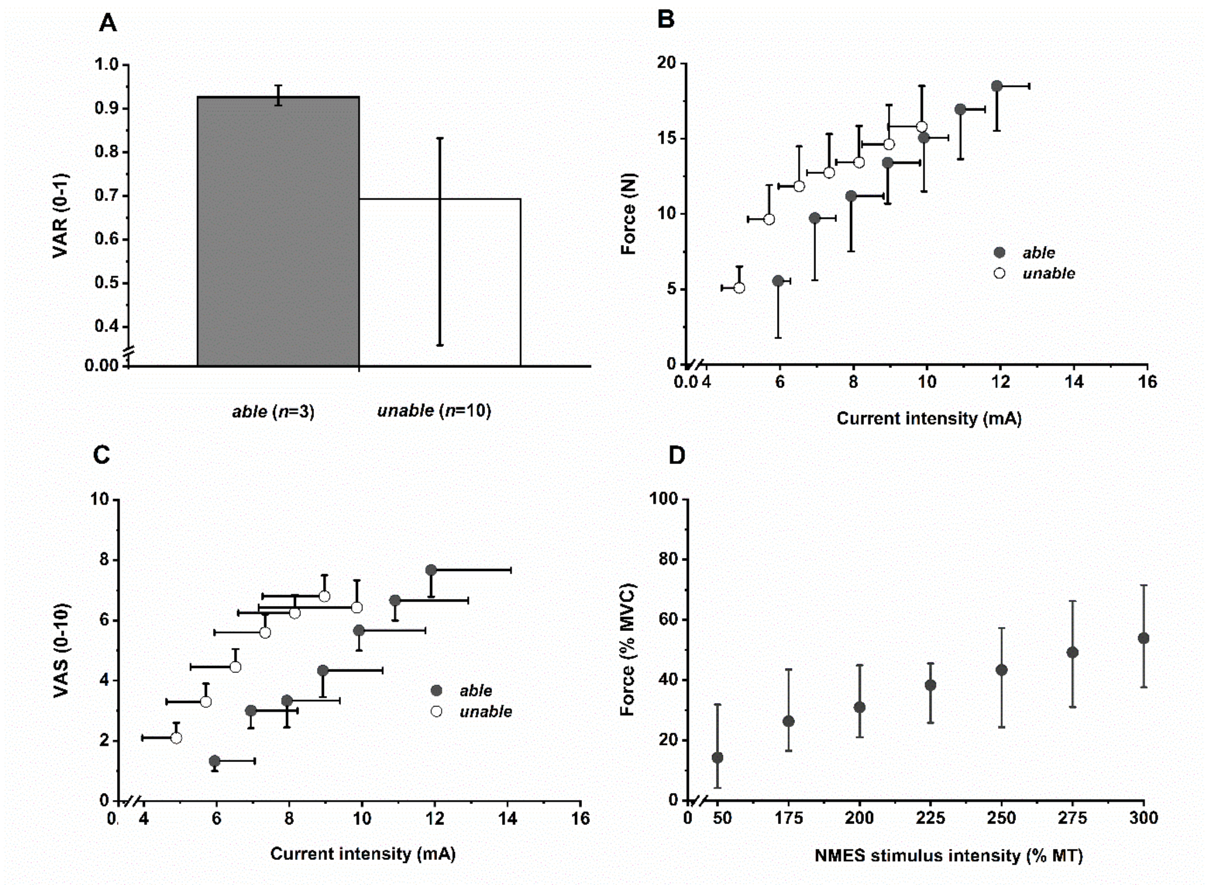
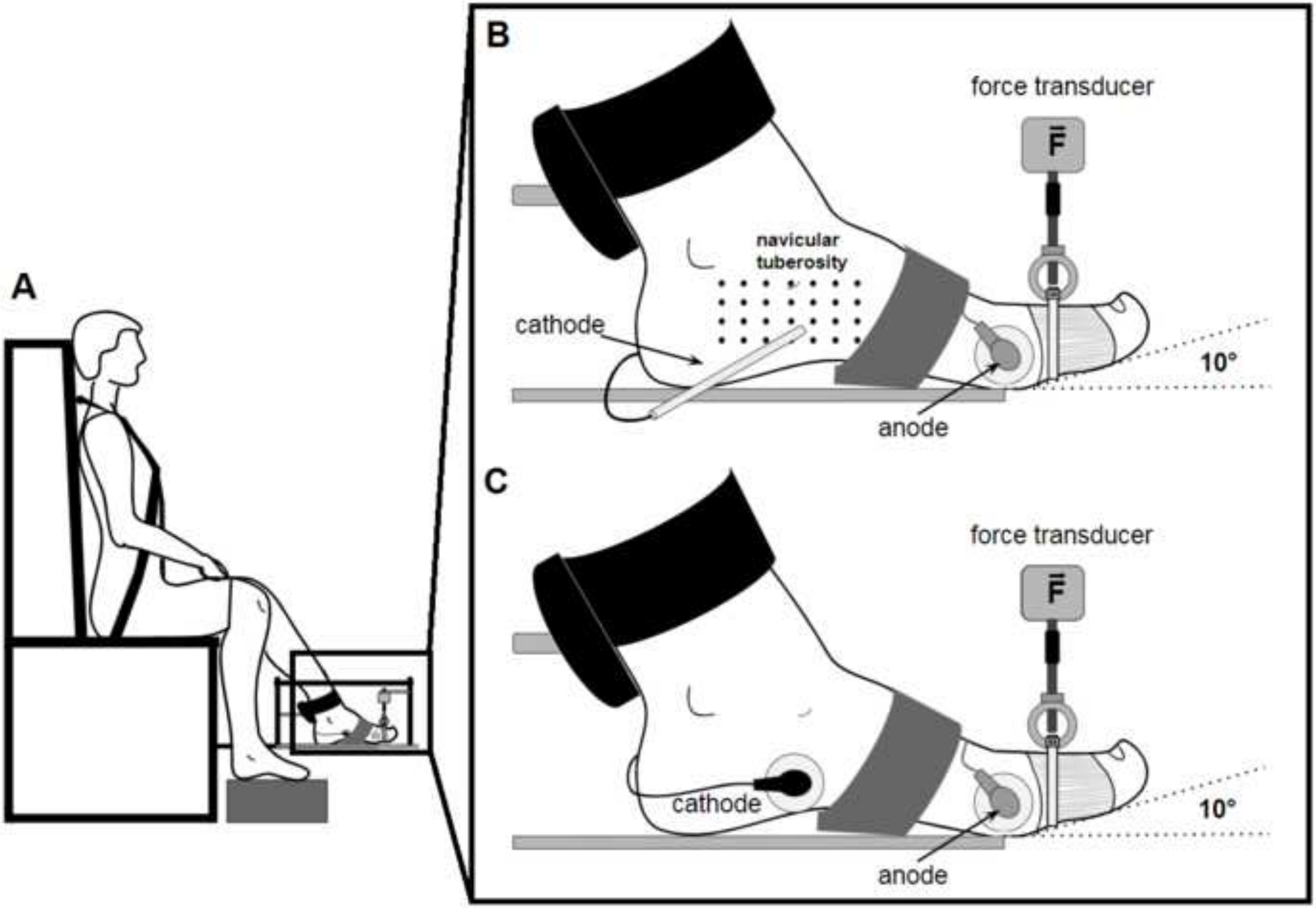


Figure 3. A) Mean (range) voluntary activation ratio (VAR) in the tested population of healthy individuals (n=13); B) Mean (±SEM) NMES-evoked force (N) plotted against the mean (±SEM) current intensity (mA) in *able* (filled circles) and *unable* participants (*n*=10; unfilled circles); C) Mean (±SEM) perceived level of discomfort (visual analogue scale; VAS) plotted against the mean (±SEM) current intensity (mA) in *able* and *unable* participants; D) Mean (range) %MVC plotted against NMES stimulus intensity in the *able* group (*n*=3).

Figure 1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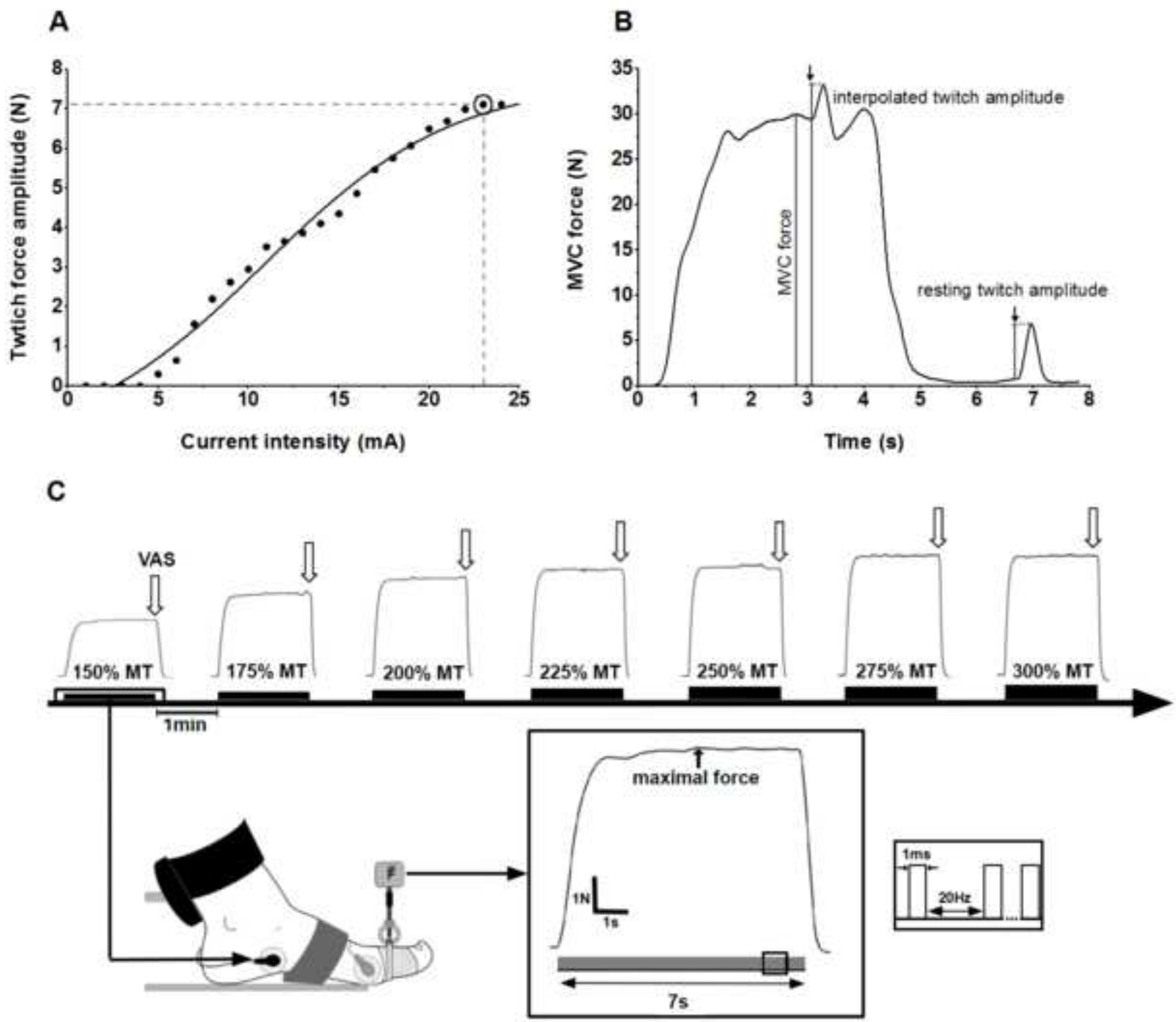

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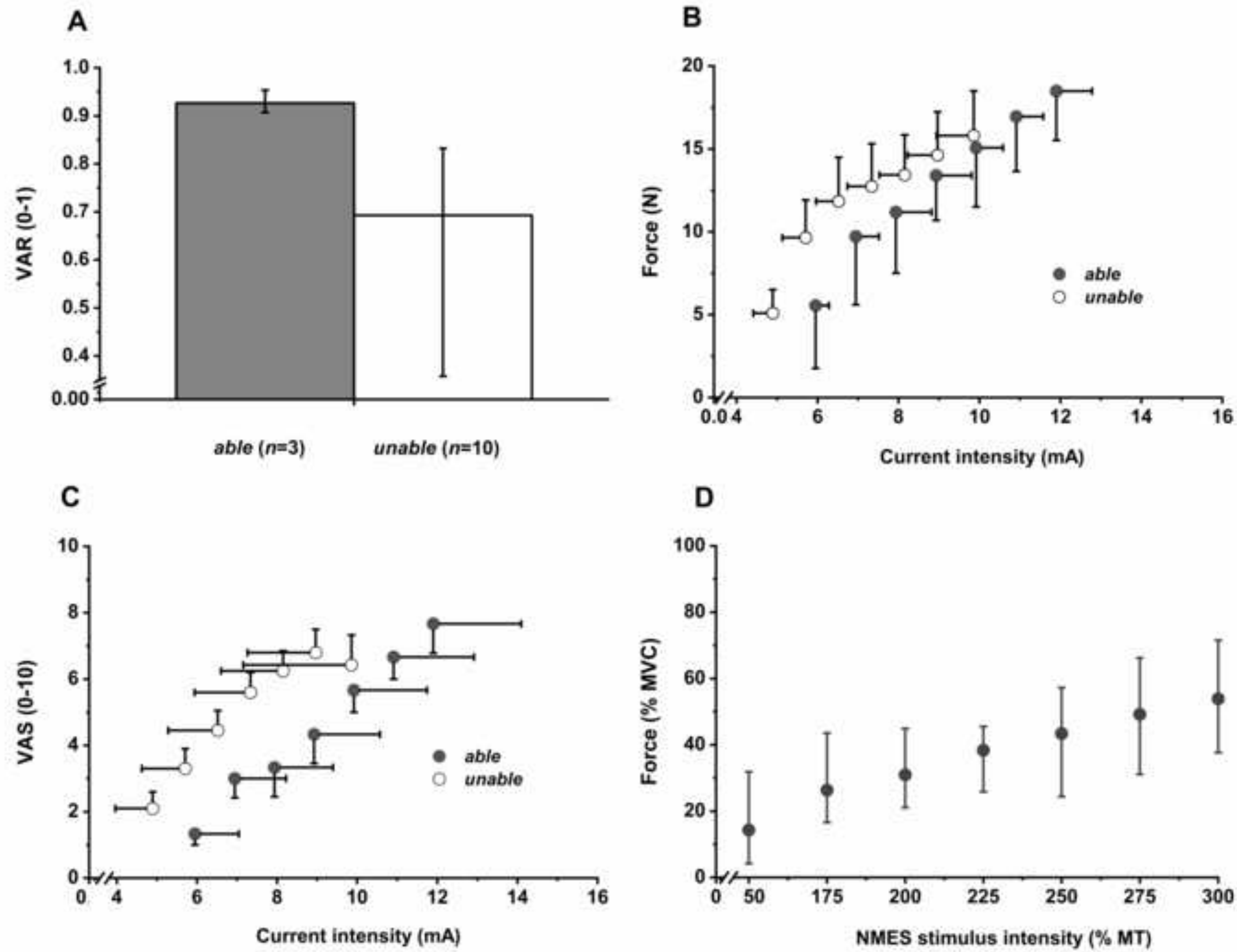

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Author Declaration

We wish to confirm that there are no known conflicts of interest associated with this publication and there has been no significant financial support for this work that could have influenced its outcome.

We confirm that the manuscript has been read and approved by all named authors and that there are no other persons who satisfied the criteria for authorship but are not listed. We further confirm that the order of authors listed in the manuscript has been approved by all of us.

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We further confirm that any aspect of the work covered in this manuscript that has involved either experimental animals or human participants has been conducted with the ethical approval of all relevant bodies and that such approvals are acknowledged within the manuscript.

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The corresponding author (on behalf of my fellow authors)

Andrei Leonardo Pérez Olivera
Sport and Exercise Science Research Centre
School of Applied Sciences
London South Bank University, UK