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SHORT COMMUNICATION

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# ANATOMICAL LANDMARKS FOR TIBIAL NERVE MOTOR BRANCHES IN THE MANAGEMENT OF SPASTIC EQUINOVARUS FOOT AFTER STROKE: AN ULTRASONOGRAPHIC STUDY

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***Objective:*** **To identify the anatomical landmarks of tibial motor nerve branches to the gastrocnemii, soleus and tibialis posterior muscles for selective motor nerve blocks in the management of spastic equinovarus foot.**

***Design:*** **Observational study.**

***Patients:*** **Twenty-five chronic stroke patients with spastic equinovarus foot.**

***Methods:*** **Motor nerve branches to the gastrocnemii, soleus and tibialis posterior muscles were tracked in the affected leg, using ultrasonography, and located in the space (vertical, horizontal and deep) according to the position of the fibular head (proximal/distal) and a virtual line from the middle of the popliteal fossa to the Achilles tendon insertion (medial/lateral).**

***Results:*** **Mean coordinates for the gastrocnemius medialis motor branch were: 1.5 cm (standard deviation (SD) 2.7) vertical (proximal), 1.7 cm (SD 1.3) horizontal (medial), 1.1 cm (SD 0.4) deep; for the gastrocnemius lateralis motor branch: 0.9 cm (SD 2.2) vertical (proximal), 1.8 cm (SD 1.7) horizontal (lateral), 1.0 cm (SD 0.3) deep; for the soleus motor branch: 1.4 cm (SD 1.1) vertical (distal), 1.6 cm (SD 0.7) horizontal (lateral), 2.8 cm (SD 0.7) deep; and for the tibialis posterior motor branch: 4.3 cm (SD 1.5) vertical (distal), 1.9 cm (SD 0.9) horizontal (lateral), 4.2 cm (SD 0.8) deep.**

***Conclusion:*** **These findings may help in the identification of tibial motor nerve branches to the gastrocnemii, soleus and tibialis posterior muscles for selective motor nerve blocks in the management of spastic equinovarus foot.**

*Key words:* equinus deformity; muscle spasticity; rehabilitation; ultrasonography.

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## LAY ABSTRACT

This study aimed to identify the motor nerve branches to the main calf muscles, in order to assist in the management of spastic foot. Twenty-five chronic stroke patients with spastic foot were evaluated with ultrasonography. The nerve branches to the gastrocnemii, soleus and tibialis posterior muscles were located in space (vertical, horizontal and deep), based on the position of the fibular head (proximal/distal) and a posterior line in the middle of the leg (medial/lateral). The coordinates for the gastrocnemius medialis motor branch were: 1.5 cm proximal, 1.7 cm medial, 1.1 cm deep; for the gastrocnemius lateralis motor branch: 0.9 cm proximal, 1.8 cm lateral, 1.0 cm deep; for the soleus motor branch: 1.4 cm distal, 1.6 cm lateral, 2.8 cm (SD 0.7) deep; and for the tibialis posterior motor branch: 4.3 cm distal, 1.9 cm lateral, 4.2 cm deep. These findings may help in the management of spastic foot.

Spastic muscle overactivity is a positive feature of upper motor neurone syndrome, which may lead to multiple patterns of motor dysfunction affecting the upper (adducted shoulder with internal rotation; flexed elbow; pronated forearm; flexed wrist; flexed fingers; thumb-in-palm; clenched fist) and/or lower (adducted thigh; flexed knee; extended knee; plantar flexed foot/ankle; equinovarus foot; striatal toe; flexed toes) limbs (1–3). Patients with spastic muscle overactivity usually need clinical interventions, such as drugs, physical therapy or other rehabilitation procedures in combination (4, 5).

Spastic equinovarus foot is the pattern most commonly treated in patients with stroke (3). It has 4 main causes: calf muscles (soleus, gastrocnemii, tibialis posterior, flexor digitorum and flexor hallucis longus muscles) spastic overactivity; calf muscles contracture/shortening leading to fixed deformity; drop-foot during the swing phase of gait due to muscle weakness (e.g. tibialis anterior, extensor digitorum and hallucis); imbalance between the tibialis anterior and peroneus (brevis and longus) muscles leading to hindfoot varus in the swing phase of gait (6).

Nerve blocks involve the injection of medications near to peripheral nerves in order to obtain a (short- or long-term) reduction in, or abolition of, conduction. Selective neural blockade technique is commonly based on the use of a disposable needle for conduction anaesthesia (delivering electrical stimulation), positio-

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ned according to surface anatomical landmarks (6–8). The medication is usually injected when the needle tip is in close contact with the targeted motor nerve corresponding to a (clinically evident) muscular contraction of selected muscles seen at ≤ 1.0 mA intensity and 100 μs duration of stimulation (7). Electromyography (EMG) may also be used to help target the appropriate motor nerve branch by monitoring the H-reflex (8).

Diagnostic nerve block (DNB) allows spastic muscle overactivity to be differentiated from contracture, and the respective role of different muscles in spastic overactivity patterns to be determined (6, 7). DNB consists of injecting a small dose of local anaesthetic near to the nerve (motor branches) innervating spastic muscles in order to temporarily suppress their overactivity (6). DNB may lead to a decrease in spastic muscle overactivity and clonus disappearance in the selected muscles within a few minutes. The duration of DNB relies mainly on the type of local anaesthetic injected (for example lidocaine is shorter lasting than bupivacaine or ropivacaine) (9). On the other hand, therapeutic nerve block (TNB) for managing spastic muscle overactivity consists of perineural injection of phenol (in concentrations between 5% and 7%) or alcohol (in concentrations between 45% and 100%) to obtain neurolysis (7).

Spastic muscle overactivity may lead to the development of changes in the muscle over time (i.e. contracture, atrophy, loss of sarcomeres, accumulation of intramuscular connective tissue, increased fat content, degenerative changes at the myotendinous junction) (10). These local anatomical modifications lead to a mismatch between surface landmarks and the actual position of the spastic muscles (11–14). Therefore, the use of ultrasonography (US) has gained importance in improving botulinum toxin injections for managing some of the most frequent spastic muscle overactivity patterns, such as equinovarus foot (13–15). On this basis, it is plausible that the soft-tissue contracture process due to spastic paresis may also alter the anatomical position of some other key structures for managing spastic muscle overactivity, such as motor nerve branches. Thus, the main aim of this study was to identify, by means of US, the anatomical landmarks of tibial motor nerve branches to the gastrocnemii, soleus and tibialis posterior muscles for selective motor nerve blocks in the management of spastic equinovarus foot due to chronic stroke.

## METHODS

This was a single-centre observational study. Inclusion criteria were as follows: age > 18 years; spastic equinovarus foot consequent to first-ever unilateral ischaemic or haemorrhagic stroke (documented by a computerized tomography scan or magnetic resonance imaging; subarachnoid haemorrhage excluded); calf muscles spastic muscle overactivity grade of at least 1 on the Modified Ashworth Scale (MAS) (16); at least 6 months since stroke onset; no botulinum toxin injection into the affected leg calf muscles in the 5 months before recruitment. Exclusion criteria were as follows: participation in other trials; fixed contractures (tone grade of 4 on the MAS) or bony deformities at the affected lower limb; previous treatment of spastic equinovarus foot with neurolytic or surgical procedures; other neurological or orthopaedic conditions involving the affected lower limb. All participants were outpatients scheduled to receive selective DNB of tibial motor nerve branches to the gastrocnemii, soleus and tibialis posterior muscles. Written informed consent for participation in the study was obtained from all patients. The study was carried out accordance with the Declaration of Helsinki and was approved by the local ethics committee.

### *Ultrasonographic evaluation*

Patients remained in the prone position with their legs outstretched during the procedure. All patients underwent real-time B-mode US, performed using a MyLab 70 XVision system (Esaote SpA, Genoa, Italy) interfaced with a linear probe (scanning frequency 15–18 MHz). The examination technique consisted of locating the tibial nerve at the terminal division of the sciatic nerve (in the lower third of the thigh) and distally tracking its motor nerve branches to the gastrocnemii, soleus and tibialis posterior muscles. This, so-called "elevator technique", enables assessment of nerve shape, echogenicity, thickness and its relation with surrounding tissues (e.g. skin, muscles or vessels) (17). The correct identification of tibial motor nerve branches to the gastrocnemii, soleus and tibialis posterior muscles was checked by means of needle (70-mm, echogenic-coated, 22-gauge needle with length graduation) electrical stimulation (1 Hz frequency and 100 μs duration). The tibial nerve motor branches were located in the space (vertical, horizontal and deep) according to the position of the fibular head (upper end) and a virtual line extending from the middle of the popliteal fossa to the Achilles tendon insertion (8). In addition, the spastic calf muscle echo intensity was graded semiquantitatively according to the Heckmatt scale (grade 1: normative; grade 2: increase in muscle echo intensity while bone echo is still distinct; grade 3: marked increase in muscle echo intensity and reduced bone echo; grade 4: very high muscle echo intensity and complete loss of bone echo) (18).

### *Clinical evaluation*

Patients remained in the supine position with their knees extended during the evaluation. The spastic ankle passive range of motion (PROM) was measured using a handheld goniometer. The sensitivity of the measurement was set at 5°. The dorsiflexion angle was defined as positive and the plantar flexion angle as negative, taking 0° as the neutral position of the joint (18). The MAS is a 6-point scale grading the resistance of a relaxed limb to rapid passive stretch (0 = no increase in muscle tone; 1 = slight increase in muscle tone at the end of the range of motion; 1+ = slight increase in muscle tone through less than half of the range of motion; 2 = more marked increase in muscle tone through most of the range of motion; 3 = considerable increase in muscle tone; 4 = joint is rigid)(16). For statistical purposes, a score of 1 was considered as 1, and a score of 1+ was considered as 2, and so on, up to a score of 4, which was considered as 5 (19). The MAS was used to evaluate spastic calf muscles tone. Also, the Tardieu scale (TS) was used to evaluate spastic calf muscles tone according to the TS grade, which measured the gain of the muscle reaction to fast stretch in dorsiflexion (0: no resistance throughout passive movement; 1: slight resistance throughout passive movement; 2: clear catch at a precise angle,

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**Table I.** Demographic and clinical features of patients

| Patients' features | |
|---|---|
| Age, years, mean (SD) | 69.1 (7.9) |
| Sex, male/female, *n* | 17/8 |
| Time since stroke onset, years, mean (SD) | 5.5 (3.4) |
| Affected lower limb trochanter length, cm, mean (SD) | 78.1 (5.6) |
| Affected ankle dorsiflexion PROM, °, mean (SD) | –5.4 (5.8) |
| Calf muscles spasticity | |
| MAS (0–5), median (IQR) | 3.0 (2.0; 4.0) |
| TS grade (0–4), median (IQR) | 2.0 (2.0; 3.0) |
| TS angle, °, mean (SD) | 8.4 (5.5) |
| Calf muscles echo intensity (Heckmatt grade 1–4) | |
| Gastrocnemius medialis, median (IQR) | 2.0 (2.0; 3.0) |
| Gastrocnemius lateralis, median (IQR) | 2.0 (2.0; 2.5) |
| Soleus, median (IQR) | 2.0 (2.0; 3.0) |
| Tibialis posterior, median (IQR) | 2.0 (1.5; 3.0) |

SD: standard deviation; n: number; PROM: passive range of motion; MAS: Modified Ashworth scale; TS: Tardieu scale; IQR: interquartile range.

interruption of the passive movement, followed by release; 3: fatigable clonus occurring at a precise angle; 4: unfatigable clonus occurring at a precise angle), and the TS angle, which measured the difference between the angle of catch-and release/clonus at fast stretch in dorsiflexion and the ankle PROM (18).

*Statistical analysis*

Statistical analysis was performed using the Statistical Package for Social Science for Macintosh, version 20.0 (SPSS Inc, Chicago, IL, USA). Descriptive statistics were used to define the tibial nerve motor branches location in the space. Spearman's rank correlation test was used to assess the association between anatomical landmarks of the tibial nerve motor branches and other US and clinical features of patients. The alpha level for significance was set at $p<0.05$.

## RESULTS

A total of 25 chronic stroke patients were recruited from among 78 consecutive outpatients. The enrolment period was from March to June 2017. The patients' demographic and clinical features are shown in Table I.

The mean coordinates for the gastrocnemius medialis motor branch were 1.5 cm (standard deviation (SD) 2.7) vertical (proximal to the fibular head), 1.7 cm (SD 1.3) horizontal (medial to the virtual line extending from the middle of popliteal fossa to the Achilles tendon insertion), and 1.1 cm (SD 0.4) deep (distance from the skin). The mean coordinates for the gastrocnemius lateralis motor branch were 0.9 cm (SD 2.2) vertical (proximal to the fibular head), 1.8 cm (SD 1.7) horizontal (lateral to the virtual line extending from the middle of popliteal fossa to the Achilles tendon insertion), and 1.0 cm (SD 0.3) deep (distance from the skin). The mean coordinates for the soleus motor branch were 1.4 cm (SD 1.1) vertical (distal to the fibular head), 1.6 cm (SD 0.7) horizontal (lateral to the virtual line extending from the middle of popliteal fossa to the Achilles tendon insertion), and 2.8 cm (SD 0.7) deep (distance from the skin). The mean coordinates for the tibialis posterior motor branch were 4.3 cm (SD 1.5) vertical (distal to the fibular head), 1.9 cm (SD 0.9) horizontal (lateral to the virtual line extending from the middle of popliteal fossa to the Achilles tendon insertion), and 4.2 cm (SD 0.8) deep (distance from the skin). US images of tibial motor nerve branches to the gastrocnemii, soleus and tibialis posterior muscles are shown in Fig. 1.

Table II shows the results of the correlation between anatomical landmarks of the tibial nerve motor branches and other US and clinical features (Spearman's rank correlation test).

## DISCUSSION

For patients with spastic equinovarus foot, DNB of the tibial nerve and its motor branches is mandatory to determine the causes of the muscle overactivity pattern and to define its management appropriately (6, 8). In particular, DNB of the tibial nerve main trunk (mixed

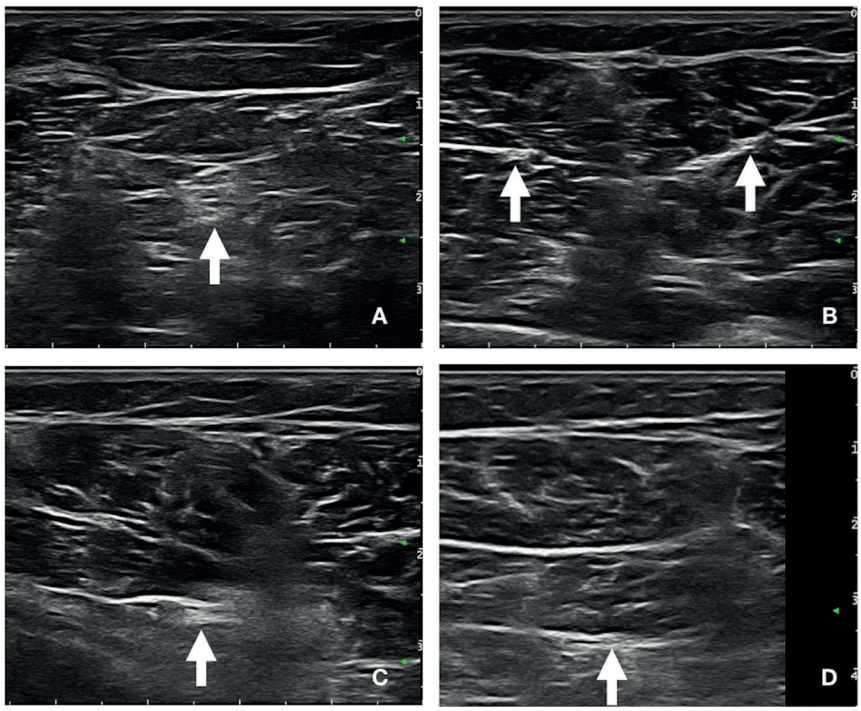


**Fig. 1.** Ultrasound images of: (A) the tibial nerve trunk, and its motor branches to (B) the gastrocnemii muscle, (C) soleus muscle, and (D) tibialis posterior muscle.

**Table II.** Correlation between anatomical landmarks of the tibial nerve motor branches and other ultrasound (US) and clinical features (Spearman's r)

| | GM motor branch coordinates | | | GL motor branch coordinates | | | Soleus motor branch coordinates | | | Tibialis posterior motor branch coordinates | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Parameter | Vertical | Horizontal | Deep | Vertical | Horizontal | Deep | Vertical | Horizontal | Deep | Vertical | Horizontal | Deep |
| Time since onset | −0.388 | −0.141 | −0.062 | −0.327 | −0.190 | 0.123 | 0.141 | 0.204 | 0.068 | 0.142 | 0.279 | 0.107 |
| Affected lower limb length | 0.486* | 0.112 | −0.073 | 0.404* | 0.036 | 0.289 | 0.072 | 0.382 | 0.240 | 0.143 | 0.210 | 0.147 |
| Affected ankle PROM | 0.046 | 0.132 | 0.014 | −0.161 | −0.024 | 0.186 | −0.227 | 0.177 | −0.010 | −0.007 | 0.055 | −0.210 |
| Calf muscles spasticity | | | | | | | | | | | | |
| Modified Ashworth scale | 0.401* | −0.018 | 0.488* | 0.298 | −0.054 | 0.250 | 0.459* | −0.111 | −0.207 | 0.162 | 0.113 | 0.080 |
| Tardieu scale grade | 0.145 | −0.168 | 0.534* | 0.212 | 0.127 | 0.284 | −0.140 | −0.292 | −0.344 | −0.207 | −0.213 | 0.019 |
| Tardieu scale angle | 0.097 | −0.149 | 0.342 | −0.033 | −0.082 | 0.077 | 0.056 | 0.046 | −0.281 | −0.223 | −0.075 | −0.278 |
| Spastic muscle echo intensity | | | | | | | | | | | | |
| GM | −0.020 | 0.110 | 0.701* | | | | | | | | | |
| GL | | | | −0.039 | −0.033 | 0.253 | | | | | | |
| Soleus | | | | | | | −0.170 | −0.049* | 0.020 | | | |
| Tibialis posterior | | | | | | | | | | −0.065 | −0.255 | −0.027 |

*Significant correlation ($p < 0.05$).
GM: gastrocnemius medialis; GL: gastrocnemius lateralis; PROM: passive range of motion.

sensorimotor nerve block) is used to differentiate spastic muscle overactivity from contracture by inducing a non-selective decrease in spastic overactivity of the calf muscles (6). On the other hand, to determine the respective role of different calf muscles (e.g. the soleus, gastrocnemii and tibialis posterior muscles) in spastic equinovarus pattern, a selective DNB of the tibial motor nerve branches is needed (6, 8). From a therapeutic perspective, selective nerve blocks of the tibial nerve motor branches may be performed with neurolytic agents (i.e. phenol or alcohol) in order to provide a prolonged (but not permanent) reduction in calf muscle tone in patients with spastic equinovarus foot (6, 7).

Consistent with US evaluation of the affected leg performed on a sample of 25 adult chronic stroke patients with spastic equinovarus foot, this study located in space (vertical, horizontal and deep) the anatomical landmarks of tibial motor nerve branches to the gastrocnemii, soleus and tibialis posterior muscles according to the fibular head (upper end) position (proximal/distal) and a virtual line from the middle of popliteal fossa to the Achilles tendon insertion (medial/lateral). The location of the soleus and tibialis posterior motor nerve branches has been determined previously by Deltombe et al. in 12 stroke patients with spastic equinovarus, using computed tomography scanning (8). Although the findings of the current study are based on the same references and are in line with those of Deltombe, the current study has 2 further strengths. First, patients were evaluated by means of US, an imaging technique commonly used in daily practice, which allows nerve blocks to be performed not only by targeting the location of the motor nerve branches, but also by guiding the injection of anaesthetics or chemical denervating agents (7). On this basis, the use of US may solve some difficulties related to the (possible) discrepancies between anatomical landmarks and the "actual" nerve location due to the specific anatomy of each patient (e.g. femoral and tibial bone rotation) (8). Furthermore, US allows to (simultaneously) view the target nerve, needle and spreading of injection agent. US-guided nerve blocks have been reported to enable reduction of the injected volume by delivering the medication (e.g. local anaesthetic or neurolytic agent) precisely to the target nerve, as well as reducing the risk of injury to important adjacent structures, such as blood vessels (20). This should be taken into account, in particular for patients on anti-coagulant therapy. Secondly, the tibial motor nerve branches to the gastrocnemius medialis and lateralis were located, which are predominantly involved in 12.5% of patients with spastic equinovarus foot (21). The location of motor nerve branches to the soleus and gastrocnemii muscles have also been defined previously with the aim of facilitating neural blockade procedures by Sook Kim et al. in 22 adult cadavers using anatomical dissection (22). However, the findings of the current study are not comparable with those of Sook Kim because of the different population (chronic stroke patients with spastic equinovarus vs adult cadavers) and anatomical references considered (Sook Kim's results are based on femur epicondyles and malleoli position) (22).

Spastic muscle overactivity may affect limb anatomy, causing the disruption of normal muscle architecture (13, 18). In particular, muscle fibrosis may lead to atrophy and reduction in muscle volume (13). On this basis, one might assume that the development of changes in surrounding muscles due to spastic paresis would relate to anatomical location of the nerve branches. Interestingly, the findings of the current study do not appear to be in line with this hypothesis. Indeed, this study failed to observe a clear association between anatomical landmarks of the tibial nerve motor branches and the US/clinical features recorded during evaluation (see Table II). This was probably because patients showed few changes in anatomy of the spastic calf muscles, as quantified using the Heckmatt scale (see Table I).

This study has some limitations. First, the sample size was small. Secondly, the study did not compare the



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anatomical landmarks of tibial motor nerve branches to the gastrocnemii, soleus and tibialis posterior muscles between legs (i.e. affected vs. healthy) within the same individual. This was because the main aim of this study was not to evaluate modifications in anatomical landmarks due to spastic muscle overactivity, but to provide information, from a clinical practice perspective, for selective motor nerve blocks in the management of spastic equinovarus foot due to chronic stroke. Thirdly, we did not perform US evaluation of other nerve branches (e.g. the tibialis posterior nerve main trunk or the motor nerve branches to the flexor digitorum longus and flexor hallucis longus muscles) that might be a target for selective blocks in the management of spastic equinovarus foot. Fourthly, no treatment (i.e. selective nerve block with anaesthetics or therapeutic nerve block with neurolytic agents) was given.

In conclusion, US may be useful to localize motor nerve branches to the gastrocnemii, soleus and tibialis posterior muscles for evaluating and treating their spastic overactivity using neural blockade procedures. In daily practice, US should be coupled with needle electrical stimulation in order to maximize precise identification of the tibialis posterior motor nerve branches and the safety of nerve blocks by overcoming possible difficulties due to the specific anatomy of each patient. For clinicians without access to US, the anatomical landmarks proposed in this study may represent a useful guide for identification of tibial nerve motor branches by means of other injection techniques, such as needle electrical stimulation. To further validate these findings, larger scale studies are required, taking into account the limitations reported above.

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