

CLINICAL SPASTICITY ASSESSMENT USING THE MODIFIED TARDIEU SCALE DOES NOT REFLECT JOINT ANGULAR VELOCITY OR RANGE OF MOTION DURING WALKING: ASSESSMENT TOOL IMPLICATIONS

Megan BANKY, PhD^{1,2}, Ross A. Clark, PhD², Benjamin F. MENTIPLAY, PhD³, John H. OLVER, MBBS⁴ and Gavin WILLIAMS, PhD^{1,5}

From the ¹Department of Physiotherapy, Epworth HealthCare, Melbourne, ²School of Health and Sport Sciences, University of the Sunshine Coast, Sunshine Coast, ³La Trobe Sport and Exercise Medicine Research Centre, La Trobe University, ⁴Epworth Monash Rehabilitation Medicine Unit and ⁵Department of Physiotherapy, University of Melbourne, Melbourne, Australia

Objective: Spasticity assessment is often used to guide treatment decision-making. Assessment tool limitations may influence the conflicting evidence surrounding the relationship between spasticity and walking. This study investigated whether testing speeds and joint angles during a Modified Tardieu assessment matched lower-limb angular velocity and range of motion during walking.

Design: Observational study.

Subjects: Thirty-five adults with a neurological condition and 34 assessors.

Methods: The Modified Tardieu Scale was completed. Joint angles and peak testing speed during V3 (fast) trials were compared with the same variables during walking in healthy people, at 0.40–0.59, 0.60–0.79 and 1.40–1.60 m/s. The proportion of trials in which the testing speed, start angle, and angle of muscle reaction matched the relevant joint angles and angular velocity during walking were analysed.

Results: The Modified Tardieu Scale was completed faster than the angular velocities seen during walking in 88.7% (0.40–0.59 m/s), 78.9% (0.60–0.79 m/s) and 56.2% (1.40–1.60 m/s) of trials. When compared with the normative dataset, 4.2%, 9.5% and 13.7% of the trials met all criteria for each respective walking speed.

Conclusion: When applied according to the standardized procedure and compared with joint angular velocity during walking, clinicians performed the Modified Tardieu Scale too quickly.

Key words: muscle spasticity; patient outcome assessment; rehabilitation; brain injuries; gait; walking.

Accepted Nov 24, 2020; Epub ahead of print Dec 7, 2020

J Rehabil Med 2021; 53: jrm00137

Correspondence address: Dr Megan Banky, PhD, Physiotherapy Department, Epworth Rehabilitation, 29 Erin Street, Richmond VIC 3121, Australia. E-mail: megan.banky@epworth.org.au

Spasticity is a common impairment following neurological injury (1–5). The effective assessment and management of spasticity receives significant attention due to the detrimental effects it has on patient outcomes, carer burden, and quality of life (6, 7). Current spasticity guidelines recommend that only spasticity impacting function should receive intervention (8).

LAY ABSTRACT

Spasticity is an abnormal increase in muscle tightness, which is common following neurological injury. Spasticity has been shown to have a profound impact on an individual's independence and quality of life. The main goal reported by patients in this population is to return to independent, normal walking. Yet, despite this there is a lack of consensus regarding the relationship between spasticity and walking outcomes. This may be due to a disconnect between clinical, bed-based assessment methods and how spasticity manifests during walking. This study aimed to establish how well a routine clinical assessment (the Modified Tardieu Scale) matched the speed and range of joint movement during walking. The findings suggest that, currently, clinicians performed the assessment too fast, which may lead to "false-positive" assessment findings. This may result in the identification and treatment of spasticity that is not impacting walking, leading to sub-optimal patient outcomes and significant healthcare wastage.

As such, the role of a clinical assessment is to identify the presence of spasticity and decipher whether the spasticity warrants intervention, such as botulinum toxin-A (BoNT-A).

Walking limitations are the most significant self-perceived, functional problem reported by individuals following neurological injury (9). A primary rehabilitation goal is often to improve walking independence, quality, speed, and endurance (10–12). In relation to spasticity, the clinician's role is to identify whether spasticity is present, and subsequently determine if a patient's walking may benefit from spasticity-related interventions.

A definition of spasticity published by Pandyan et al. (13); "disordered sensori-motor control, resulting from an upper motor neurone lesion, presenting as intermittent or sustained involuntary activation of muscles", encompasses all the positive features of upper motor neurone syndrome (UMNS) under an umbrella term of spasticity. This terminology defines spasticity as a broader sensori-motor phenomenon (13), when compared with the more constrained, velocity-dependent definition published by Lance (14); "a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon

jerk, resulting from hyper-excitability of the stretch reflexes, as one component of the UMNS" (14). For the purpose of this study, Lance's definition has been used to define spasticity, since, with this definition, spasticity can be assessed as an individual construct. The Modified Tardieu Scale (MTS) is an often-recommended spasticity assessment, aligning with Lance's (14) definition of spasticity (15–17). The MTS classifies the response of a relaxed muscle to a fast, passive stretch (V3). The assessment protocol involves a clinician moving the joint "as fast as possible" through its full range of motion (ROM) without specifying or measuring the speed of completion. The MTS is applied according to this standardized protocol regardless of the functional status or goals of the patient. For example, a household ambulator walking at ≤ 0.30 m/s is assessed at the same speed as a community ambulator walking at ≥ 0.80 m/s, whose muscles and joints are moving much faster when walking (18). This "one-size-fits" all approach does not take into account the variability in joint ROM and angular velocity (or speed of lower-limb movement) with changes in walking speed (19). As such, the MTS may not sensitively discriminate individuals who have spasticity impacting their walking.

While it is well established that interventions, such as BoNT-A, reduce spasticity at an impairment-based level, current treatment modalities for spasticity do not necessarily lead to improved walking outcomes (20–22). This may be because standardized protocols for scales such as the MTS do not reflect joint movement during walking (23–25). For example, if clinicians test at a speed that is slower than the joint angular velocity seen during walking, they may fail to identify spasticity that is affecting walking (i.e. false-negative). Conversely, if clinicians test at a speed that exceeds the joint angular velocity seen during walking, they may identify spasticity that is not impacting walking (i.e. false-positive).

Matching the joint angles and testing speed of the MTS to the ROM and angular velocity seen during walking may assist in identifying patients who have spasticity impacting functional performance, leading to treatment decisions that optimize patient outcomes and healthcare resources. This study aimed to compare the joint start angle, angle of muscle reaction, and testing speed during a standardized MTS assessment of 4 major muscle groups of the lower-limb, collected in people with neurological conditions, with joint ROM and angular velocity in a healthy population walking at a range of speeds.

METHODS

This study was approved by the Epworth HealthCare (681-15) and University of Sunshine Coast Human Research Ethics

Committees (S/17/1011). All participants provided written informed consent. The funders played no role in the conduct or reporting of this study.

Participants

Two groups were recruited to participate in this study. All sample sizes were determined in consultation with a biostatistician. While they were not based on formal power calculations, they exceeded the sample sizes of similar studies examining the properties of the MTS (26, 27).

Group 1: Participants with a neurological condition. A convenience sample of 35 adults (≥ 18 years of age) attending physiotherapy for walking limitations related to an acquired neurological injury diagnosis, with at least 2 confirming features of upper motor neurone syndrome (radiological or clinical), were recruited. Participants were excluded if unable to provide consent or if they were unable to have the MTS completed on their affected lower-limb. Participants attended a 1.5-h assessment session and were assessed by 3 different assessors.

Group 2: Assessors experienced in spasticity assessment. Thirty-four physiotherapists, rehabilitation physicians, and rehabilitation registrars with ≥ 3 years experience in neurological clinical practice and who regularly assessed and treated spasticity were recruited from a range of healthcare networks. Each assessor attended an assessment session lasting 4–6 h and were asked to complete the assessment protocol on 2–4 of the participants. A large group of assessors was recruited to ensure that the data were reflective of the application of the MTS in clinical practice.

Data collection

All participants with a neurological condition were asked to complete a self-selected 10-m walk test with shoes on. Orthoses were permitted, where required.

The MTS was completed on the participant's more affected gastrocnemius, soleus, hamstrings at 90° hip flexion, and quadriceps, using standardized testing positions (15, 27). The hamstrings were also assessed at 40° hip flexion to more closely reflect the position of the hip joint during the swing phase of the gait cycle (19, 28). This modified position was included because the standardized position (in 90° hip flexion) inherently does not reflect joint position during walking, due to excessive hip flexion. Three V1 (slow) and V3 (fast) trials were performed for each muscle group by 3 of the assessors on each participant, totalling 90 trials per participant (i.e. 3 slow and 3 fast movements per assessor for each of the 5 muscle groups tested). Three assessors were used per patient to allow a greater representation of everyday clinical practice, as well as allowing for inter- and intra-rater variability analysis, reported elsewhere (29). Only V3 trials were analysed in this study. Assessor order was randomized; however, the order of muscle groups tested remained consistent to avoid excessive re-positioning of the participant. Assessors were blinded to each other's performance.

Participants were asked to remain relaxed throughout the assessment. The following instructions were provided to the assessors:

- V1: move the joint slowly through its full ROM.
- V3: move the joint through its full ROM as fast as possible. Assessors were instructed to stop at the "point of muscle reaction" during V3.

The Optitrack (NaturalPoint, Inc., Corvallis, OR, USA) 3-dimensional motion analysis (3DMA) system and customized

Table I. Participant position and anatomical landmarks used for marker placement

Trial	Starting position	End position	Marker placement
Gastrocnemius	Supine; Hip/knee neutral Ankle maximum PF	Supine; Hip/knee neutral Ankle maximum DF	Medial femoral condyle, medial malleolus, medial calcaneus, first metatarsal head
Soleus	Supine; Hip/knee at 90° flexion Ankle maximum PF	Supine; Hip/knee at 90° flexion Ankle maximum DF	Medial femoral condyle, medial malleolus, medial calcaneus, first metatarsal head
Hamstrings at 40° flexion	Supine; Hip flexed to 40° Knee maximum flexion	Supine; Hip flexed to 40° Knee maximum extension	Adductor muscle belly, medial femoral condyle, medial malleolus
Hamstrings at 90° flexion	Supine; Hip flexed to 90° Knee maximum flexion	Supine; Hip flexed to 90° Knee maximum extension	Adductor muscle belly, medial femoral condyle, medial malleolus
Quadriceps	Prone; Hip neutral Knee maximum extension	Prone; Hip neutral Knee maximum flexion	Greater trochanter, lateral femoral condyle, lateral malleolus

Markers used for calculation of joint range of motion and angular velocity (i.e. testing speed).
PF: plantarflexion; DF: dorsiflexion.

LabVIEW 2014 program (National Instruments, Austin, TX, USA) used to assess each trial have been described previously in detail (30). The 13-camera system was used to record the movement of reflective markers placed on specific anatomical landmarks of the lower-limb. Table I outlines the participant position and marker placement used for each muscle. This enabled the peak testing speed, start angle, and angle of muscle reaction to be calculated, exported into a database and used for analysis.

Data analysis

Three parameters of the V3 assessment were analysed and compared with joint angles and angular velocity during walking: (i) testing speed, (ii) joint start position, and (iii) angle of muscle reaction. Three walking speeds were selected for comparison and the reference ranges for each of the testing parameters were calculated for each of these walking speeds. These reference values were taken from a normative dataset in a healthy control cohort when walking in 0.20 m/s increments (19). The minimum available reference walking speed of 0.40–0.59 m/s and the

maximum available reference walking speed 1.40–1.60 m/s were chosen to reflect the entire spectrum of patients, ranging from minimally ambulant to well recovered. A third reference walking speed, of 0.60–0.79 m/s, was selected as it matched the median walking speed of our participants and reflects the walking speed required to achieve “limited community mobility” following neurological injury (18). The sub-sections below outline the exact statistical methods applied for each of the 3 parameters (testing speed, joint start position and angle of muscle reaction). Each individual test was compared with the reference dataset, using Microsoft Excel 2016 (Microsoft, Redmond, WA, USA), based on the relevant walking speed for each muscle.

Testing speed. For each muscle group, a reference range for testing speed was determined based on the anticipated peak angular velocity of the relevant joint at the stage of the gait cycle where the muscle is lengthening, and therefore a spastic response may be elicited (Table II). For gastrocnemius and

Table II. Joint angular velocity and joint range of motion (ROM) normative reference values for each muscle group and walking speed

Muscle group	Phase of gait cycle	Walking speed, m/s	Mean peak joint angular velocity (°/s) (SD 1.96)	Start angle	Joint ROM
GAS (swing)	Ankle DF Swing	0.40–0.59	100 (48–153)	16° PF	16° PF–12° DF
		0.60–0.79	130 (52–208)	26° PF	26° PF–11° DF
		1.40–1.60	214 (147–281)	29° PF	29° PF–11° DF
GAS (stance)	Ankle DF Stance	0.40–0.59	44 (29–59)	10° PF	10° PF–20° DF
		0.60–0.79	54 (36–71)	11° PF	11° PF–20° DF
		1.40–1.60	93 (54–131)	16° PF	16° PF–16° DF
SOL (swing)	Ankle DF Swing	0.40–0.59	100 (48–153)	16° PF	16° PF–12° DF
		0.60–0.79	130 (52–208)	26° PF	26° PF–11° DF
		1.40–1.60	214 (147–281)	29° PF	29° PF–11° DF
SOL (stance)	Ankle DF Stance	0.40–0.59	44 (29–59)	10° PF	10° PF–20° DF
		0.60–0.79	54 (36–71)	11° PF	11° PF–20° DF
		1.40–1.60	93 (54–131)	16° PF	16° PF–16° DF
HAM 40° HF	KE Swing	0.40–0.59	209 (130–288)	62° KF	62° KF–2° KE
		0.60–0.79	258 (174–342)	66° KF	66° KF–3° KE
		1.40–1.60	384 (294–474)	66° KF	66° KF–1° KF
HAM 90° HF	KE Swing	0.40–0.59	209 (130–288)	62° KF	62° KF–2° KE
		0.60–0.79	258 (174–342)	66° KF	66° KF–3° KE
		1.40–1.60	384 (294–474)	66° KF	66° KF–1° KF
QUAD	KF Swing	0.40–0.59	219 (181–257)	2° KE	2° KE–62° KF
		0.60–0.79	244 (193–295)	3° KE	3° KE to 66° KF
		1.40–1.60	384 (294–474)	1° KF	1° KF to 66° KF

Data in this table relating to joint angular velocity and joint angles have been taken from Mentiplay et al. (19) for healthy participants walking at each reference speed (0.40–0.59, 0.60–0.79 and 1.40–1.60 m/s). The phase of the gait cycle selected for each muscle group represents where the muscle group is lengthening and therefore spasticity is likely to be elicited.

GAS: gastrocnemius; SOL: soleus; HAM: hamstrings; QUAD: quadriceps; DF: dorsiflexion; PF: plantarflexion; KE: knee extension; KF: knee flexion; ROM: range of motion.

soleus these values were taken during the gait cycle when the ankle is dorsiflexing in stance phase (Fig. 1.1A) and swing phase (Fig. 1.1B); for the quadriceps the reference value was taken during late stance/early swing phase when the knee moves into its most flexed position (Fig. 1.2C); and for the hamstrings the value was taken at terminal swing when the knee transitions from flexion to extension (Fig. 1.2D). A trial was deemed to reflect joint angular velocity during walking if the peak testing speed fell within the mean (standard deviation (SD) 1.96°/s) of the reference value. Based on these values, presented in Table II, each trial was categorized as either: slower than the reference range, within the reference range, or faster than the reference range (for each of the walking speeds). Further analysis of the testing speed was completed for each muscle group by computing the absolute difference between each trial and the mean joint angular velocity in Table II, and calculating the median and quartile values of the distribution of scores derived from this.

Start angle. Similarly, previous literature was used to ascertain the mean joint angles during each relevant stage of the gait cycle when walking at the 3 reference speeds (19). Table II outlines the joint angles representing the normative ROM for each muscle group (mean SD 1.96°) to the extremes of the movement). Example traces for the walking speed of 0.60–0.79 m/s are shown in Fig. 1.

For the start angle to reflect joint position during walking, the starting position of the joint needed to allow for the test to include the entire ROM listed in Table II. For example, if walking at 1.40–1.60 m/s, for gastrocnemius/soleus (swing) the ankle needed to start at $\geq 29^\circ$ ankle plantarflexion, for

gastrocnemius/soleus (stance) the ankle needed to start at $\geq 16^\circ$ plantarflexion, for hamstrings trials the knee needed to start at $\geq 66^\circ$ knee flexion, and for quadriceps trials the knee needed to start at $\leq 1^\circ$ knee flexion. This was to ensure that a spastic reaction occurring within the relevant joint ROM was not missed secondary to an inadequate start angle.

Angle of muscle reaction. Only trials in which a muscle reaction was present (Tardieu score 2–4) were analysed in this stage. Where a muscle reaction was not present (Tardieu score 0–1), the angle of muscle reaction was not analysed. In order to meet this criterion, the angle of muscle reaction needed to occur between the start and end angle, which are listed in Table II. For example, when walking at 1.40–1.60 m/s the angle of muscle reaction for quadriceps trials needed to occur between 1 – 66° knee flexion to be deemed relevant to walking.

Finally, the number of trials which met all 3 criteria: testing speed, start angle, and angle of muscle reaction, were calculated to determine how well the overall assessment of each muscle group using the MTS reflects lower-limb angular velocity and joint angles during walking at the 3 different speeds.

RESULTS

A total of 35 participants with a neurological condition were recruited to the study (stroke=15; traumatic brain injury=13; neurosurgical=4; multiple sclerosis=2; cerebro-vasculitis=1). The median age of

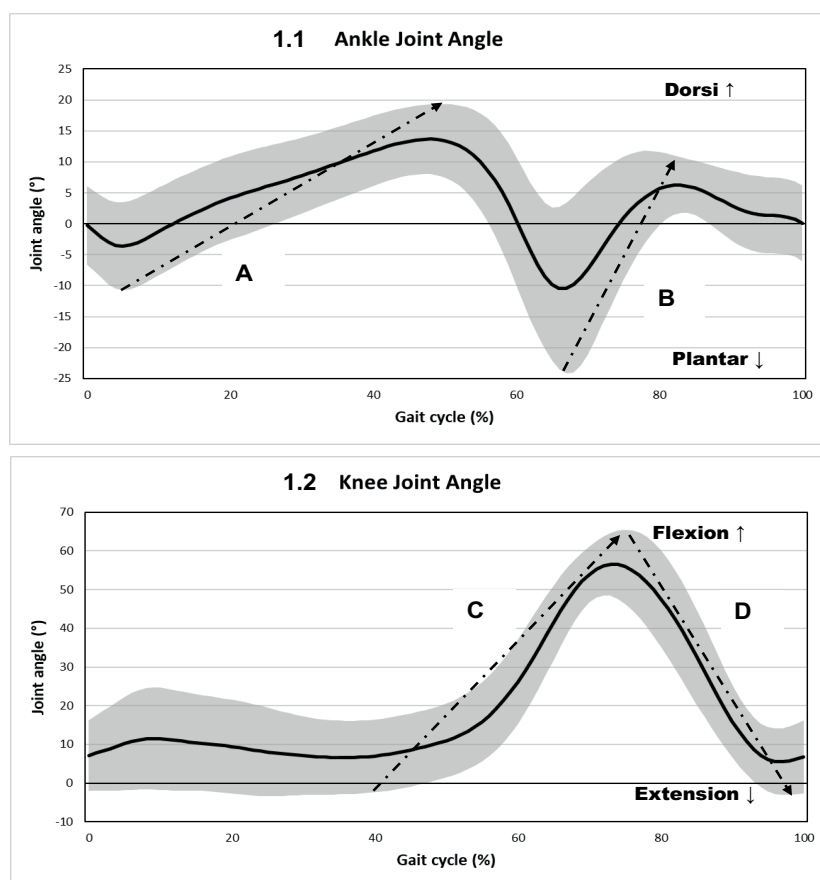


Fig. 1. Ankle and knee joint reference angles (0.60–0.79 m/s). Ankle (Fig. 1.1) and knee (Fig. 1.2) joint reference angles (standard deviation (SD) 1.96) to the extreme of the relevant movement adapted from Mentiplay et al. (19) from healthy participants walking at 0.60–0.79 m/s. (A) Gastrocnemius/soleus (stance phase); (B) Gastrocnemius/soleus (swing phase); (C) Quadriceps; (D) Hamstrings at 40° and 90° hip flexion.

Table III. Testing speed: proportion of trials slower than, within, and faster than the joint angular velocity for the 3 reference walking speeds

Muscle group	Mean peak testing speed, °/s	Walking speed, m/s	Mean peak joint angular velocity (SD 1.96) ^a	Slower than range, n (%)	Within range, n (%)	Faster than range, n (%)	Median (IQR) difference, °/s	Min-max absolute difference, °/s
GAS (swing) n = 307	348	0.40–0.59	100 (48–153)	0 (0)	12 (4)	295 (96)	245 (161–326)	2–628
		0.60–0.79	130 (52–208)	0 (0)	39 (13)	268 (87)	215 (131–296)	4–598
		1.40–1.60	214 (147–281)	10 (3)	83 (27)	214 (70)	131 (62–212)	0–514
GAS (stance) n = 307	348	0.40–0.59	44 (29–59)	0 (0)	0 (0)	307 (100)	301 (217–382)	58–684
		0.60–0.79	54 (36–71)	0 (0)	0 (0)	307 (100)	291 (207–372)	48–674
		1.40–1.60	93 (54–131)	0 (0)	6 (1)	301 (99)	252 (168–333)	9–255
SOL (swing) n = 313	404	0.40–0.59	100 (48–153)	0 (0)	8 (3)	305 (97)	293 (211–399)	1–305
		0.60–0.79	130 (52–208)	0 (0)	19 (6)	294 (94)	264 (181–369)	2–637
		1.40–1.60	214 (147–281)	7 (2)	41 (13)	265 (85)	179 (102–285)	2–553
SOL (stance) n = 313	404	0.40–0.59	44 (29–59)	0 (0)	0 (0)	313 (100)	349 (267–455)	39–723
		0.60–0.79	54 (36–71)	0 (0)	0 (0)	313 (100)	340 (257–445)	29–713
		1.40–1.60	93 (54–131)	0 (0)	5 (2)	308 (98)	300 (218–406)	6–674
HAM 40° HF n = 314	320	0.40–0.59	209 (130–288)	3 (1)	112 (36)	199 (63)	114 (61–172)	1–461
		0.60–0.79	258 (174–342)	16 (5)	168 (54)	130 (41)	78 (38–128)	3–412
		1.40–1.60	384 (294–474)	123 (39)	179 (57)	12 (4)	78 (36–129)	1–290
HAM 90° HF n = 313	351	0.40–0.59	209 (130–288)	2 (1)	68 (22)	243 (77)	143 (85–198)	0–427
		0.60–0.79	258 (174–342)	5 (2)	138 (44)	170 (54)	95 (47–150)	0–378
		1.40–1.60	384 (294–474)	78 (25)	212 (68)	23 (7)	60 (28–104)	0–288
QUAD n = 313	349	0.40–0.59	219 (181–257)	7 (2)	34 (11)	272 (87)	136 (80–182)	0–344
		0.60–0.79	244 (193–295)	10 (3)	66 (21)	237 (76)	111 (58–157)	1–320
		1.40–1.60	384 (294–474)	70 (22)	140 (45)	103 (33)	52 (28–93)	1–235

^aThe value in brackets is the mean (100) \pm 1.96SD. I.e. 100 is the mean; 48 is the mean minus 196 SD; 153 is the mean + 1.96 SD.

GAS: gastrocnemius; SOL: soleus; HAM: hamstrings; HF: hip flexion; QUAD: quadriceps; n: number of trials; IQR: interquartile range.

Example interpretation of table: for gastrocnemius, swing phase (top row) 307 trials were analysed. The mean peak testing speed across these trials was 348°/s. For a trial to represent joint angular velocity at each of the 3 walking speeds it needed to fall within 1.96 SD of the mean peak angular velocity, i.e. 48–153°/s for 0.40–0.59 m/s. None of the 307 trials were completed slower than 48°/s, 12 (4%) were completed between 48 and 153°/s and therefore matched joint angular velocity, and 295 (96%) were completed faster than 153°/s. The median absolute difference between each of the 307 trials and the 100°/s mean reference speed was 245°/s and the 25th and 75th percentiles (or IQR) were 161°/s and 326°/s, respectively. The minimum absolute difference between a single trial and the 100°/s mean reference speed was 2°/s and the maximum difference between a single trial and the mean reference speed was 628°/sec.

participants was 55.0 years (interquartile range (IQR) 41.5–60.0), median time since diagnosis 23 months (IQR 8.5–91.5), and 22 (62.8%) were male. The median self-selected walking speed for the 35 participants was 0.72 m/s (IQR 0.30–1.11). Nine participants wore an ankle-foot orthosis to complete the 10-m walk test.

Thirty-four assessors were recruited, including rehabilitation physiotherapists ($n=26$), rehabilitation physicians ($n=5$), acute physiotherapists ($n=2$), and a rehabilitation registrar ($n=1$). The assessors had a median of 10.0 years' clinical experience (IQR 7.1–20.0 years).

Testing speed

Overall, testing was completed faster than the angular velocities seen during walking, with 88.7% (0.40–0.59 m/s), 78.9% (0.60–0.79 m/s), and 56.2% (1.40–1.60 m/s) of trials being completed faster than the respective reference values for each walking speed (Table III). The ankle joint demonstrated the poorest results, with 70–100% of all gastrocnemius and soleus trials being completed faster than the speed required to replicate ankle dorsiflexion angular velocity during walking, at all 3 reference walking speeds. The hamstrings at 40° hip flexion demonstrated the highest proportion of trials (36–54%), where testing speed matched lower-limb angular velocity (i.e. within range), for the joint

angular velocity of the 2 slower walking speeds (0.40–0.59 m/s and 0.60–0.79 m/s). The hamstrings at 90° hip flexion demonstrated the highest proportion of trials (68%), where testing speed matched lower-limb angular velocity for the fastest walking speed (1.40–1.60 m/s).

The distribution of scores derived from the absolute difference between each trial and the reference value further highlight the mismatch between testing speed and lower-limb joint angular velocity, especially: (i) at the ankle joint; and (ii) compared with slower walking speeds. The testing speed of the MTS did replicate joint angular velocity at the fastest walking speed (1.40–1.60 m/s) to a greater extent than the 2 slower walking speeds, especially at the knee joint. This is highlighted in Fig. 2, which depicts the percentage of trials that were: (i) slower than; (ii) within; and (iii) faster than the reference angular velocity range for each muscle group, compared with each of the 3 walking speeds.

Start angle

The proportion of trials in which the MTS start angle enabled the entire joint ROM seen during walking to be assessed was excellent for hamstrings at 90° hip flexion, gastrocnemius and soleus (stance phase), when matched and compared with all 3 walking speeds (Table IV). For gastrocnemius and soleus (swing phase), the results were excellent when compared with the ankle

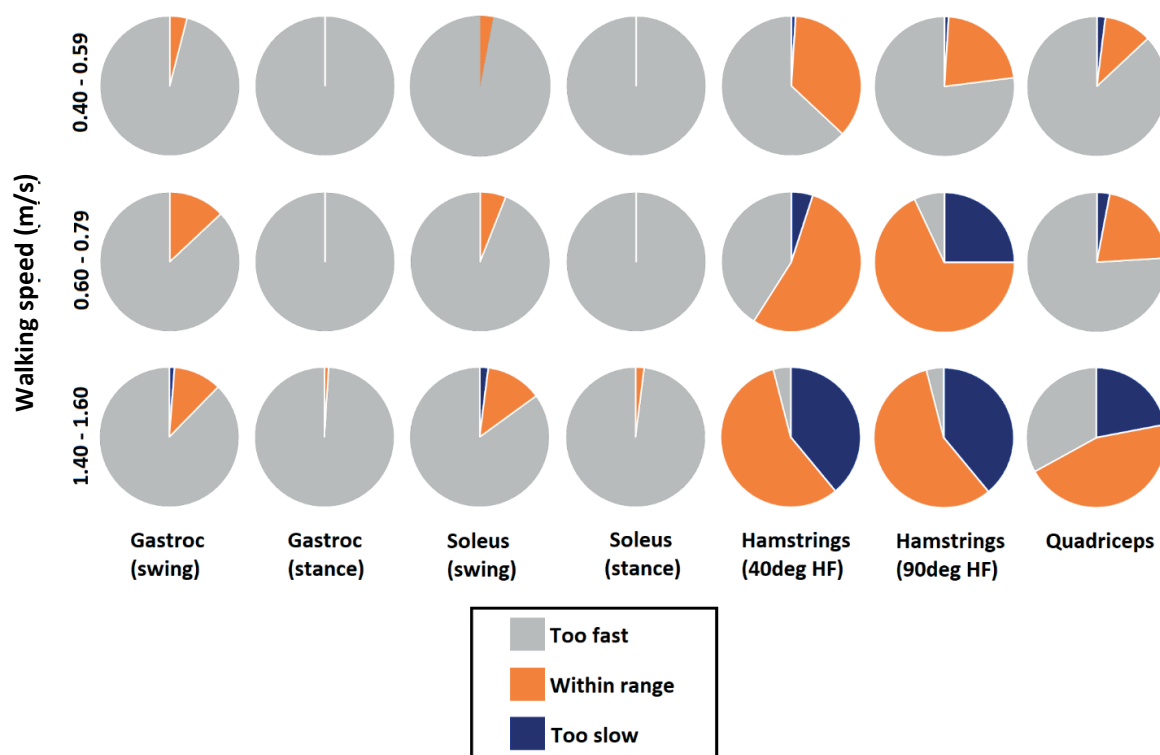


Fig. 2. Percentage of trials that were slower than (dark blue), within (orange), and faster (light grey) than the reference range for each muscle group and walking speed reference. Gastroc: gastrocnemius; HF: hip flexion.

joint ROM at the slowest walking speed (0.40–0.59 m/s) as the ankle only plantarflexed to a maximum of 16°. The results were less promising when compared with

joint ROM at the 2 faster walking speeds, where 26° and 29° plantarflexion were required, respectively. This demonstrated that, as walking speed increases, so too

Table IV. Joint start angle and angle of muscle reaction: proportion of trials within the range of motion for the 3 reference walking speeds

Muscle group	Mean start angle, °	Walking speed, m/s	Reference ROM	Correct start angle, n (%)	TS ≥ 2, n (%)	Mean angle of muscle reaction, °	Muscle reaction within reference ROM, n (%)
GAS (swing) n = 307	29° PF	0.40–0.59	16° PF–12° DF	286 (93)	237 (77)	8° PF	176/237 (74)
		0.60–0.79	26° PF–11° DF	195 (64)			227/237 (96)
		1.40–1.60	29° PF–11° DF	162 (53)			232/237 (98)
GAS (stance) n = 307	29° PF	0.40–0.59	10° PF–20° DF	297 (97)	237 (77)	8° PF	142/237 (18)
		0.60–0.79	11° PF–20° DF	297 (97)			148/237 (62)
		1.40–1.60	10° PF–16° DF	286 (93)			176/237 (74)
SOL (swing) n = 313	32° PF	0.40–0.59	16° PF–12° DF	302 (96)	211 (67)	8° PF	171/211 (81)
		0.60–0.79	26° PF–11° DF	230 (73)			203/211 (96)
		1.40–1.60	29° PF–11° DF	193 (62)			207/211 (98)
SOL (stance) n = 313	32° PF	0.40–0.59	10° PF–20° DF	313 (100)	211 (67)	8° PF	120/211 (57)
		0.60–0.79	11° PF–20° DF	313 (100)			131/211 (62)
		1.40–1.60	16° PF–16° DF	302 (96)			171/211 (52)
HAM 40° HF n = 314	54° KF	0.40–0.59	62° KF–2° KE	89 (28)	97 (31)	8° KF	78/97 (80)
		0.60–0.79	66° KF–3° KE	70 (22)			79/97 (81)
		1.40–1.60	66° KF–1° KE	70 (22)			67/97 (69)
HAM 90° HF n = 313	97° KF	0.40–0.59	62° KF–2° KE	308 (98)	236 (75)	31° KF	213/236 (90)
		0.60–0.79	66° KF–3° KE	300 (96)			219/236 (93)
		1.40–1.60	66° KF–1° KE	300 (96)			217/236 (92)
QUAD n = 313	10° KF	0.40–0.59	2° KE–62° KF	1 (0)	67 (21)	109° KF	0/67 (0)
		0.60–0.79	3° KE–66° KF	1 (0.3)			0/67 (0)
		1.40–1.60	1° KE–66° KF	16 (5)			0/67 (0)

ROM: range of motion; TS: Tardieu score; GAS: gastrocnemius; SOL: soleus; HAM: hamstrings; HF: hip flexion; QUAD: quadriceps; n: number of trials; PF: plantarflexion; DF: dorsiflexion; KF: knee flexion; KE: knee extension.

Example interpretation of table: for gastrocnemius swing phase (top row), 307 trials were analysed. For the start angle to include the entire joint ROM seen during walking at 0.40–0.59 m/s, the starting position needed to exceed the first value of the reference ROM (i.e. ≥16° PF). For an angle of muscle reaction to fall within the joint ROM seen during walking it needed to occur within the reference ROM (i.e. between 16° PF and 12° DF). The mean start angle across the 307 trials was 29° PF. When compared with the joint ROM seen when walking at 0.40–0.59 m/s, 286 (93%) of the 307 trials had a start angle ≥16° PF. Spasticity was present in 237/307 (77%) of trials, as indicated by a TS ≥ 2. The mean angle of muscle reaction across these 237 trials was 8° PF. The muscle reaction fell between 16° PF and 12° DF in 176/237 (74%) of these trials.

does ankle plantarflexion ROM, and the MTS may need to be adjusted accordingly to include the entire ROM relevant to walking at the required speed. The results for the quadriceps were poor, with the knee often starting in excessive flexion (mean starting position 10°). This was seen to a lesser extent for hamstrings at 40°, where the knee joint position frequently commenced in less than the 62–66° of flexion required, depending on the walking speed (mean starting position 54°).

Angle of muscle reaction

Table IV highlights the proportion of trials (Tardieu score 2–4) whereby the angle of muscle reaction fell within the joint ROM during walking for each muscle group, at each of the 3 walking speeds. Over 90% of the spastic muscle reactions fell within the relevant joint ROM at 0.60–0.79 m/s and 1.40–1.60 m/s for the gastrocnemius and soleus (swing phase), and the hamstrings at 90° hip flexion, for all 3 reference speeds. The quadriceps demonstrated poor results, with the mean angle of muscle reaction occurring at 109°, and no trials demonstrating a muscle reaction within the joint ROM seen during walking for any of the 3 walking speeds.

Combined testing speed, joint start angle, and angle of muscle reaction

Of the 2,180 trials analysed, 1,296 had a spastic muscle reaction. When compared with the normative joint angles and angular velocities when walking at 0.40–0.59 m/s, only 54/1,296 (4.2%) of trials met all criteria (Table V). When compared with the walking

speed of 0.60–0.79 m/s, 123/1,296 (9.5%) of trials met all criteria, and when compared with the fastest walking speed of 1.40–1.60 m/s, 178/1,296 (13.7%) of trials met all criteria. The best, yet still modest, result was for hamstrings at 90° hip flexion, where 19–61% of trials met all criteria, with results more promising when compared with the fastest walking speed.

DISCUSSION

Overall, the V3 testing speed, when completed in accordance with the standardized MTS protocol poorly reflected the lower-limb joint angular velocities seen when walking at a slow (0.40–0.59 m/s), medium (0.60–0.79 m/s), or fast (1.40–1.60 m/s) walking speed. Testing speeds were typically faster than the relevant joint angular velocity during walking. This was most apparent at the ankle joint when assessing the calf complex (gastrocnemius and soleus), and was evident when comparing the speed of assessment with ankle dorsiflexion angular velocity (swing and stance phase), at all 3 walking speeds. This may be problematic, as an assessment that identifies spasticity at a speed that exceeds the joint angular velocity during walking may produce an apparent “false-positive” finding, whereby spasticity is present, but is not impacting walking. Treatment of spasticity that is not impacting function, does not align with international guidelines (8), and may result in healthcare wastage. This is of particular significance for the calf complex, as it has the highest prevalence of lower-limb spasticity and it is the lower-limb muscle group most frequently injected with BoNT-A (31). Given the importance of the calf complex for walking (32, 33), and the perceived importance of walking within this patient population, it seems imperative that clinical assessment methods are further standardized to more closely align with muscle function and joint biomechanics during walking.

When the V3 testing speed was compared with joint angular velocity during walking for the hamstring and quadriceps muscles, there was greater variability seen between the 3 walking speeds. Overall, the testing speed best matched joint angular velocity during fast walking (1.40–1.60 m/s) with 57%, 68%, and 45% of trials falling within the relevant reference speed for the hamstrings at 40° hip flexion, hamstrings at 90° hip flexion, and quadriceps, respectively, at this walking speed. This compared with 36%, 22%, and 11% of trials for these muscle groups when compared with the relevant joint angular velocity during walking at the slower walking speed of 0.40–0.59 m/s. This highlights the potential importance of matching V3 testing speed to an individual's walking speed to assist in discriminating patients who have spasticity impacting

Table V. Proportion of trials that met all criteria for each walking speed

Muscle	Walking speed, m/s	Tardieu score ≥ 2	Valid for all criterion, n (%)
GAS (swing) $n = 307$	0.40–0.59	237	0/237 (0)
	0.60–0.79		0/237 (0)
	1.40–1.60		0/237 (0)
GAS (stance) $n = 307$	0.40–0.59	237	0/237 (0)
	0.60–0.79		0/237 (0)
	1.40–1.60		2/237 (10)
SOL (swing) $n = 313$	0.40–0.59	211	3/211 (1)
	0.60–0.79		12/211 (6)
	1.40–1.60		13/211 (6)
SOL (stance) $n = 313$	0.40–0.59	211	0/211 (0)
	0.60–0.79		0/211 (0)
	1.40–1.60		2/211 (1)
HAM 40° HF $n = 314$	0.40–0.59	97	6/97 (6)
	0.60–0.79		11/97 (11)
	1.40–1.60		17/97 (18)
HAM 90° HF $n = 313$	0.40–0.59	236	45/236 (19)
	0.60–0.79		100/236 (42)
	1.40–1.60		144/236 (61)
QUAD $n = 313$	0.40–0.59	67	0/67 (0)
	0.60–0.79		0/67 (0)
	1.40–1.60		0/67 (0)

GAS: gastrocnemius; SOL: soleus; HAM: hamstrings; HF: hip flexion; QUAD: quadriceps.

their walking from those who have spasticity that is not impacting their walking. This would align with the international consensus statements, which recommend that spasticity intervention should only be provided when impacting active or passive function (8).

The quadriceps had no trials whereby the angle of muscle reaction fell within the normal knee flexion ROM seen during the swing phase of the gait cycle. The mean angle of muscle reaction occurred at 109° of knee flexion, while the knee is only required to flex to approximately 60–65° in swing phase. This highlights the risk of assessment findings potentially leading to incorrect treatment decisions. For example, BoNT-A to the quadriceps is a common treatment for a stiff-legged gait pattern, yet our data suggests that a spastic muscle reaction most commonly occurs at >100° knee flexion. As such, quadriceps spasticity may not be a primary contributing factor and, while it is possible that BoNT-A will result in a reduction in spasticity at an impairment-based level, the patients stiff-legged gait pattern may not resolve following the intervention. This may be due to the spastic reaction occurring well beyond the 62–66° of knee flexion seen during walking. This example highlights that the presence of spasticity may not always occur in a range that will impede walking. This may distract from other important factors contributing to stiff-legged gait, such as reduced ankle plantarflexion and hip flexion during late-stance to early-swing phase (34). Consideration of where in the range the angle of muscle reaction occurs and how this relates to the patient's functional performance and goals may assist in deciphering the patients who are likely to benefit from spasticity intervention, ultimately optimizing patient outcomes.

The assessment of hamstrings completed at 90° hip flexion demonstrated the greatest, yet still modest, results overall, with the largest proportion of trials meeting all criteria when matched to each of the walking speeds. However, the standardized testing position of the hamstrings is with the hip in 90° flexion (15), which does not reflect the normal joint position during walking, where the hip joint only reaches 30–40° of hip flexion during swing phase (19, 35). Therefore, an assessment at 40° hip flexion was included in the testing protocol, and this variation to the testing position had several important implications for the results. By reducing the angle of hip flexion from 90° to 40°, the proportion of hamstring trials in which spasticity was evident (Tardieu score ≥ 2) reduced from 75% (236/313) to 31% (97/314). While the results for this assessment are restricted to the angle at the knee joint itself, when determining whether hamstring spasticity is impacting walking performance and requiring treatment to improve walking, the position of the hip joint may need to be

carefully considered. The results in the standardized position (i.e. 90°) may be overinflated by a physiological restriction in passive muscle length when the hamstrings are placed on maximal stretch across both the hip and knee joint. Previous studies investigating normal hamstring length by placing the hip in 90° flexion and moving the knee from flexion to extension found that the normal knee flexion deficit was $31 \pm 7.5^\circ$ (36), and 35–45° (37). These results are comparable to the mean knee flexion angle (31°) identified during the V3 component of the hamstrings at 90° hip flexion assessment. As such, it may be possible that, at 90° hip flexion, trials were deemed to have a spastic response, and, in some cases, this may have been due a physiological restriction in muscle length.

The findings of this study further highlight the disconnect that exists between clinical assessment methods of spasticity and functional activities, such as walking (23, 25, 38). This study quantified the disparity that was suggested in a previous systematic review in relation to the standardized application of spasticity assessment tools and how they reflect walking biomechanics (ROM and angular velocity) (38). This highlights the potential lack of ecological validity (or functional relevance) of current scales of spasticity. It also justifies the need for further studies to be completed investigating whether a spasticity assessment using a testing speed matched to an individual's lower-limb joint angular velocity during walking impacts the relationship between spasticity assessment findings and walking performance following neurological injury. This may assist in clarifying the relationship between the treatment of focal spasticity and walking outcomes.

Study limitations

This study provided valuable insights into the relevance of the MTS for walking when applied according to the standardized protocol. However, the results of the present study are not generalizable to other activities, such as sit-to-stand or stair ascent/descent, where joint biomechanics differ significantly. Furthermore, the V3 component of the MTS assessment was only compared with lower-limb biomechanics when walking at 3 speeds (0.40–0.59 m/s, 0.60–0.79 m/s, and 1.40–1.60 m/s). The MTS could not be compared with walking speeds <0.40 m/s, as this normative data was not available (19). However, the 3 walking speeds selected were deemed appropriate, as these 3 speeds represent those who are minimally ambulant through to well recovered, as well as the median walking speed of the recruited cohort.

Joint angles and velocities during the spasticity assessment were compared with the gait biomechanics in a healthy cohort with a normal walking pattern (19). Often patients with a neurological condition present

with different compensatory patterns to maximize their walking speed and independence (39, 40). For example, an older person with a stroke may walk at a normal walking speed (e.g. 1.20 m/s), but with an associated abnormality, such as a stiff-legged walking pattern. In this instance, the knee joint would not be flexing at a “normal” angular velocity despite walking at a “normal” speed. However, due to the variability in these gait patterns and a primary goal of patients often being to return to “normal” walking (10–12), it was deemed most appropriate to compare the participants with a healthy cohort walking at a slower, more relevant speed.

This study highlighted several limitations in regard to the current application of the MTS in relation to walking. It has been suggested that measuring spasticity during active movements is likely to be more informative and have a greater relevance to function than current “passive” bedside assessment methods (24). This is due to the inability of bedside measurements to replicate the stretching of a partially active muscle, which is present during functional tasks, such as walking, sit to stand, or reach and grasp. However, an active movement-based assessment of spasticity is likely to be difficult to implement in a clinical setting, where both financial and time constraints exist, and clinicians are expected to assess a patient and decipher an effective management plan promptly. Future research, comparing the similarity of findings of an upright, partially active spasticity assessment (that is inclusive of differing sensori-motor input), with the MTS assessment matched to an individual’s lower-limb joint angles and angular velocities, would be of value. This would assist in ascertaining whether the global sensory input when in an upright walking position results in different spasticity assessment findings, when compared with the current, standardized MTS testing protocol. The implementation of the MTS performed in a manner that matches the lower-limb joint angles and angular velocities during walking using low-cost, validated technologies that are able to provide real-time feedback, such as a smartphone (30), may improve the accuracy of clinical assessment and optimize treatment decision-making.

Finally, only the construct of velocity-dependent spasticity, as aligned with Lance’s definition (14), was examined in this study. The examination of other positive features of upper motor neurone syndrome, such as hypertonia, dystonia or co-contraction, was beyond the scope of the current paper. As such, conclusions cannot be drawn regarding the implications of these other positive features of the UMNS on walking speed. Further research is required to examine whether the assessment of these impairments accurately reflects muscle function during walking.

ACKNOWLEDGEMENTS

This research was supported by grants awarded to Megan Banky from the Royal Automobile Club of Victoria, Epworth Research Institute, and Physiotherapy Research Fund. Megan Banky also received a part-time University of Sunshine Coast and Australian Government Award Scholarship to facilitate the completion of this study. Dr Ross Clark is supported by a National Health and Medical Research Council R.D. Wright Biomedical Fellowship (Number: 1090415).

The authors have no conflicts of interest to declare.

REFERENCES

1. Sommerfeld DK, Eek EU, Svensson AK, Holmqvist LW, von Arbin MH. Spasticity after stroke: its occurrence and association with motor impairments and activity limitations. *Stroke* 2004; 35: 134–139.
2. Thibaut A, Chatelle C, Ziegler E, Bruno MA, Laureys S, Gosseries O. Spasticity after stroke: physiology, assessment and treatment. *Brain Inj* 2013; 27: 1093–1105.
3. Urban PP, Wolf T, Uebele M, Marx JJ, Vogt T, Stoeter P, et al. Occurrence and clinical predictors of spasticity after ischemic stroke. *Stroke* 2010; 41: 2016–2020.
4. Rizzo MA, Hadjimichael OC, Preiningerova J, Vollmer TL. Prevalence and treatment of spasticity reported by multiple sclerosis patients. *Mult Scler* 2004; 10: 589–595.
5. Williams G, Banky M, Olver J. Distribution of lower limb spasticity does not influence mobility outcome following traumatic brain injury: An observational study. *J Head Trauma Rehabil* 2015; 30: E49–E57.
6. Esquenazi A. The human and economic burden of post-stroke spasticity and muscle overactivity. *J Sci Commun* 2011; 18: 607–614.
7. Lundstrom E, Smits A, Borg J, Terent A. Four-fold increase in direct costs of stroke survivors with spasticity compared with stroke survivors without spasticity: the first year after the event. *Stroke* 2010; 41: 319–324.
8. Royal College of Physicians BSORM, The Chartered Society of Physiotherapy, Association of Chartered Physiotherapists in Neurology and the Royal College of Occupational Therapists. Spasticity in adults: management using botulinum toxin. National Guidelines. London: Royal College of Physicians; 2018.
9. van Bennekom C, Jelles F, Lankhorst GJ, Kuik DJ. Value of measuring perceived problems in a stroke population. *Clin Rehabil* 1996; 10: 288–294.
10. Combs SA, Van Puymbroeck M, Altenburger PA, Miller KK, Dierks TA, Schmid AA. Is walking faster or walking farther more important to persons with chronic stroke? *Disabil Rehabil* 2013; 35: 860–867.
11. Bohannon RW, Horton MG, Wikholm JB. Importance of four variables of walking to patients with stroke. *Int J Rehabil Res* 1991; 14: 246–250.
12. Lord SE, McPherson K, McNaughton HK, Rochester L, Weatherall M. Community ambulation after stroke: how important and obtainable is it and what measures appear predictive? *Arch Phys Med Rehabil* 2004; 85: 234–239.
13. Pandyan AD, Gregoric M, Barnes MP, Wood D, Van Wijck F, Burridge J, et al. Spasticity: clinical perceptions, neurological realities and meaningful measurement. *Disabil Rehabil* 2005; 27: 2–6.
14. Lance JW. Symposium synopsis. In: Feldman RG YR, Koella WP, editors. *Spasticity: Disordered control*. Chicago: Yearbook Medical; 1980: p. 485–494.
15. Boyd RN, Graham KH. Objective measurement of clinical findings in the use of botulinum toxin type A for the

- management of children with cerebral palsy. *Eur J Neurol* 1999; 6: S23–S35.
16. Patrick E, Ada L. The Tardieu Scale differentiates contracture from spasticity whereas the Ashworth Scale is confounded by it. *Clin Rehabil* 2006; 20: 173–181.
 17. Haugh AB, Pandyan AD, Johnson GR. A systematic review of the Tardieu Scale for the measurement of spasticity. *Disabil Rehabil* 2006; 28: 899–907.
 18. Perry J, Garrett M, Gronley JK, Mulroy SJ. Classification of walking handicap in the stroke population. *Stroke* 1995; 26: 982–989.
 19. Mentiplay BF, Banky M, Clark RA, Kahn MB, Williams G. Lower limb angular velocity during walking at various speeds. *Gait Posture* 2018; 65: 190–196.
 20. Cofré Lizama LE, Khan F, Galea MP. Beyond speed: gait changes after botulinum toxin injections in chronic stroke survivors (a systematic review). *Gait Posture* 2019; 70: 389–396.
 21. Gupta AD, Chu WH, Howell S, Chakraborty S, Koblar S, Visvanathan R, et al. A systematic review: efficacy of botulinum toxin in walking and quality of life in post-stroke lower limb spasticity. *Systematic Reviews* 2018; 7: 1–9.
 22. Kaji R, Osako Y, Suyama K, Maeda T, Uechi Y, Iwasaki M. Botulinum toxin type A in post-stroke lower limb spasticity: a multicenter, double-blind, placebo-controlled trial. *J Neurol* 2010; 257: 1330–1337.
 23. Dietz V, Sinkjaer T. Spastic movement disorder: impaired reflex function and altered muscle mechanics. *Lancet Neurol* 2007; 6: 725–733.
 24. Burridge JH, Wood DE, Hermens HJ, Voerman GE, Johnson GR, Van Wijck F, et al. Theoretical and methodological considerations in the measurement of spasticity. *Disabil Rehabil* 2005; 27: 69–80.
 25. Platz T, Eickhof C, Nuyens G, Vuadens P. Clinical scales for the assessment of spasticity, associated phenomena, and function: a systematic review of the literature. *Disabil Rehabil* 2005; 27: 7–18.
 26. Mehrholz J, Wagner K, Meissner D, Grundmann K, Zange C, Koch R, et al. Reliability of the Modified Tardieu Scale and the Modified Ashworth Scale in adult patients with severe brain injury: a comparison study. *Clin Rehabil* 2005; 19: 751–759.
 27. Ben-Shabat E, Palit M, Fini NA, Brooks CT, Winter A, Holland AE. Intra- and interrater reliability of the Modified Tardieu Scale for the assessment of lower limb spasticity in adults with neurologic injuries. *Arch Phys Med Rehabil* 2013; 94: 2494–2501.
 28. Winter DA. Biomechanical motor patterns in normal walking. *J Mot Behav* 1983; 15: 302–330.
 29. Banky M, Clark RA, Pua YH, Mentiplay BF, Olver JH, Williams G. Inter- and intra-rater variability of testing velocity when assessing lower limb spasticity. *J Rehabil Med* 2019; 51: 54–60.
 30. Banky M, Clark RA, Mentiplay BF, Olver JH, Kahn MB, Williams G. Toward accurate clinical spasticity assessment: validation of movement speed and joint angle assessments using Smartphones and camera tracking. *Arch Phys Med Rehabil* 2019; 100: 1482–1491.
 31. Nalysnyk L, Papapetropoulos S, Rotella P, Simeone JC, Alter KE, Esquenazi A. OnabotulinumtoxinA muscle injection patterns in adult spasticity: a systematic literature review. *BMC Neurol* 2013; 13: 1–11.
 32. Neptune RR, Sasaki K, Kautz SA. The effect of walking speed on muscle function and mechanical energetics. *Gait Posture* 2008; 28: 135–143.
 33. Requião LF, Nadeau S, Milot MH, Gravel D, Bourbonnais D, Gagnon D. Quantification of level of effort at the plantarflexors and hip extensors and flexor muscles in healthy subjects walking at different cadences. *J Electromyogr Kinesiol* 2005; 15: 393–405.
 34. Esquenazi A. Evaluation and management of spastic gait in patients with traumatic brain injury. *J Head Trauma Rehabil* 2004; 19: 109–118.
 35. Umberger BR, Martin PE. Mechanical power and efficiency of level walking with different stride rates. *J Exp Biol* 2007; 210: 3255–3265.
 36. Gajdosik RL, Rieck MA, Sullivan DK, Wightman SE. Comparison of four clinical tests for assessing hamstring muscle length. *J Orthop Sports Phys Ther* 1993; 18: 614–618.
 37. Gnat R, Kuszewski M, Koczar R, Dziewońska A. Reliability of the passive knee flexion and extension tests in healthy subjects. *J Manipulative Physiol Ther* 2010; 33: 659–665.
 38. Banky M, Ryan HK, Clark R, Olver J, Williams G. Do clinical tests of spasticity accurately reflect muscle function during walking: a systematic review. *Brain Inj* 2017; 31: 440–455.
 39. Nadeau S, Gravel D, Arsenault AB, Bourbonnais D. Plantarflexor weakness as a limiting factor of gait speed in stroke subjects and the compensating role of hip flexors. *Clin Biomech* 1999; 14: 125–135.
 40. Williams G, Morris ME, Schache A, McCrory PR. People preferentially increase hip joint power generation to walk faster following traumatic brain injury. *Neurorehabil Neural Repair* 2010; 24: 550–558.