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Association between Selective Motor Control of the Lower Limb and Gait Performance in Chronic Stroke Survivors: A Cross-Sectional Study

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ABSTRACT:

Background: Impaired walking is among the most disabling consequences of stroke. Loss of selective motor control (SMC), the ability to move one joint without unwanted synergistic movement at others, may contribute to this impairment, but its link with clinically measured gait performance in the chronic phase is poorly defined.

Objective: To examine the association between lower-limb SMC and gait performance in people with chronic stroke.

Methods: In this hospital-based cross-sectional study, 101 adults aged 45–70 years, at least six months after a first unilateral stroke and able to walk 10 M independently, were recruited by convenience sampling. SMC of the paretic limb was rated with the Selective Control Assessment of the Lower Extremity (SCALE; 0–10). Gait speed (10-Meter Walk Test, 10MWT), functional mobility (Timed Up and Go, TUG) and walking endurance (6-Minute Walk Test, 6MWT) were measured. Associations were tested with Spearman correlation and multiple linear regression adjusted for age, time since stroke, spasticity and assistive-device use.

Results: The mean SCALE score was 5.4 ± 2.4 . Higher SMC correlated with faster gait speed ($\rho = 0.64$), longer 6MWT distance ($\rho = 0.53$) and shorter TUG time ($\rho = -0.70$; all $p < 0.001$). After adjustment, SCALE remained independently associated with all three outcomes (standardised $\beta = 0.70, 0.60$ and -0.67 , respectively; $p < 0.001$), explaining 32–51% of outcome variance. Participants with high SMC walked more than twice as fast as those with low SMC.

Conclusion: Lower-limb SMC is positively and independently associated with gait speed, endurance and functional mobility in chronic stroke. A simple bedside rating of SMC may help identify survivors whose walking limitations are driven by impaired movement selectivity and guide targeted rehabilitation. Longitudinal and interventional studies are needed to test causality.

INTRODUCTION

Stroke remains one of the leading causes of death and long-term disability across the world, with more than twelve million new cases recorded in 2019 alone [1]. The burden is growing in India, where an ageing population and a rising prevalence of vascular risk factors have increased the number of survivors who need prolonged rehabilitation [2]. Advances in acute care have improved survival, yet many people leave hospital with persistent motor deficits. Most regain some ability to walk without hands-on help, but the walking that returns is often slow, effortful and unreliable outdoors, which restricts participation at home and in the community [3,4]. Because independent walking is among the goals that survivors prioritise most, understanding what limits it in the chronic phase is a clinical necessity.

Hemiparetic gait is characterised by reduced speed, shortened stance on the paretic side, asymmetric stepping and greater variability from one stride to the next [5–9]. These deviations are usually attributed to weakness, spasticity and sensory loss, and rehabilitation has therefore concentrated on strengthening and tone management [10,11]. Paretic muscle torque does relate to locomotor performance [12], but the relationship is far from complete. Determinants of walking differ with the severity of the deficit, and a considerable share of the variance in walking ability is left unexplained by strength alone [13]. Some survivors recover substantial strength yet continue to walk inefficiently, which suggests that the quality of motor command, and not only its magnitude, shapes functional gait [14].

Selective motor control is the capacity to activate a chosen muscle group or joint without recruiting other muscles in obligatory patterns. After stroke, damage to the corticospinal tract and associated descending pathways limits this fractionation, and movement collapses into stereotyped flexor and extensor synergies [15–17]. Studies of the paretic arm first characterised abnormal torque coupling [16,18], and subsequent work documented analogous coupling in the leg, in which attempted hip flexion draws in knee flexion and ankle dorsiflexion, while extension of one joint forces extension of its neighbours [19–21]. Such coupling is directly relevant to walking, because foot clearance in swing and controlled loading in stance require joints to move independently and in a precisely timed order [22]. Walking depends on spinal rhythm-generating circuits that are continuously shaped by supraspinal drive [23], and reduced descending control has been linked to merged muscle modules and a simplified repertoire of muscle coordination, which in turn predicts poorer locomotor performance [24,25].

Despite this mechanistic evidence, clinical measurement of SMC in adults with stroke has lagged behind. Common assessments emphasise strength, tone or global motor scores such as the Fugl-Meyer scale [26], which combine reflex activity, synergy-dominated movement and isolated movement in a single total. The Selective Control Assessment of the Lower Extremity (SCALE) was designed to isolate the specific ability to perform voluntary single-joint movements at the hip, knee, ankle, subtalar joint and toes, and it can be administered at the bedside in a few minutes [27,28]. Although SCALE was developed for children with spastic cerebral palsy, it has been adopted for other upper motor neuron conditions, including stroke, where its relationship with functional walking merits systematic study.

Gait performance can be captured with simple tests that reflect different domains of walking. Comfortable gait speed is regarded as a summary indicator of functional status and predicts home and community walking activity [29–31]. The Timed Up and Go test (TUG) integrates rising, turning and transitional movements and is sensitive to balance and lower-limb impairment in chronic stroke [32,33]. The 6-Minute Walk Test (6MWT) reflects submaximal walking capacity, which depends on both neuromuscular efficiency and cardiovascular fitness [34,35]. Studies that tie locomotor outcomes to motor control have mainly depended on instrumented gait analysis, electromyography or force-plate measures [36–38], which are rarely available in routine practice. Evidence linking a quick clinical rating of SMC to a battery of practical walking tests is therefore limited.

We therefore examined whether lower-limb SMC, rated with SCALE, is associated with gait speed, functional mobility and walking endurance in people with chronic stroke attending a tertiary rehabilitation service. We hypothesised that better SMC would be positively associated with walking performance on all three measures, independent of age, time since stroke, spasticity and the use of an assistive device.

METHODS

Study design and setting

This analytical cross-sectional study was carried out in the outpatient and inpatient neurorehabilitation units of the Department of Physiotherapy at Swamy vivekanandha Medical College Hospital and Research Institute, Tiruchengode Namakkal District,

Tamilnadu. The research was conducted from March 2026 to July 2026. Reporting follows the STROBE recommendations for observational studies [39]. The institutional ethics committee approved the protocol at Madhav University, Pindwara (Sirohi) and all participants gave written informed consent after the purpose and procedures had been explained in their preferred language.

Participants and sample size

Consecutive eligible patients were approached and enrolled by convenience sampling. Eligible participants were aged 45 to 70 years; had a first-ever unilateral ischaemic or haemorrhagic stroke confirmed by neuroimaging; were at least six months post-stroke; could walk at least 10 m independently, with or without a walking aid or orthosis; and could understand and follow verbal instructions. Exclusion criteria were recurrent stroke or bilateral involvement; severe cognitive impairment or aphasia that compromised comprehension; other neurological disorders affecting gait, such as Parkinson's disease or spinal cord injury; severe lower-limb musculoskeletal disorders; uncorrected visual or vestibular impairment; and unstable cardiovascular disease.

The sample size was estimated for a bivariate correlation, assuming a smaller-than-expected true coefficient of 0.30 (a moderate effect by conventional benchmarks [40]), a two-sided α of 0.05 and 80% power. This required 85 participants, and allowing for about 15% incomplete or unusable assessments gave a target of 100. A total of 101 participants were recruited and all completed every assessment.

Assessment procedure

Demographic and clinical data (age, sex, stroke type, paretic side, time since stroke and use of an assistive device) were taken from hospital records and interview. Ankle plantarflexor spasticity of the paretic limb was graded with the Modified Ashworth Scale [41]. All assessments were completed in a single session, in a fixed order. SMC was assessed first, while participants were rested, so that fatigue from walking could not contaminate the rating. The gait tests followed after a rest, and a further rest of at least five minutes separated successive tests. One physiotherapist trained in SCALE scoring rated SMC, and a second physiotherapist, who had no knowledge of the SCALE score, recorded the gait measures. Participants wore their usual footwear and used their customary walking aid throughout. Each gait test was preceded by a practice trial, and a therapist walked beside the participant with a gait belt available to prevent falls. Heart rate and perceived exertion were monitored, and testing stopped if symptoms occurred.

Selective motor control

SMC of the paretic lower limb was rated with SCALE [27]. Participants sit or lie in a standardised position and are asked to perform isolated, reciprocal movements at the hip (flexion–extension), knee (flexion–extension), ankle (dorsiflexion–plantarflexion), subtalar joint (inversion–eversion) and toes (flexion–extension). Each joint is scored 2 if movement is isolated and fully controlled, 1 if it is impaired by mirror movement, substitution or incomplete range, and 0 if no isolated movement is possible. The five joint scores are summed to give a total of 0 to 10, where higher values indicate better selectivity. Because the tool was validated mainly in children with cerebral palsy [27,28], the same examiner, scoring rules and positioning were applied to every participant.

Gait performance

Gait speed was measured with the 10-Meter Walk Test (10MWT). Participants walked at a comfortable self-selected pace along a 14 m walkway, with 2 m allowed for acceleration and 2 m for deceleration, and the time over the central 10 m was recorded with a stopwatch. The mean of three trials was converted to metres per second. Self-selected speed is a reliable measure in people with hemiparesis [42–44]. Participants were also grouped into the speed-based categories of household (<0.4 m/s), limited community (0.4 – 0.8 m/s) and community (>0.8 m/s) ambulation [29].

Functional mobility was assessed with the TUG [32]. Participants stood from a standard armchair, walked 3 m at a comfortable pace, turned, returned and sat down, and the elapsed time in seconds was recorded. The mean of two trials was analysed, with shorter times indicating better mobility. The test is reliable and related to lower-limb impairment in chronic stroke [33]. Walking endurance was assessed with the 6MWT [34]. Participants walked back and forth along a marked 30 m corridor for six minutes, were told they could slow down or rest as needed, and received standardised encouragement. The total distance in metres was recorded. The test has good reliability and is related to peak oxygen uptake in stroke [35].

Statistical analysis

Continuous variables are summarised as mean $\pm$ standard deviation, or median and interquartile range when skewed, and categorical variables as counts and percentages. Normality was examined with the Shapiro–Wilk test. Because SCALE is an ordinal score and TUG times were positively skewed, Spearman’s rank correlation coefficient (ρ) was used as the primary measure of association, with 95% confidence intervals obtained through the Fisher transformation. Coefficients were interpreted as negligible (<0.30), low ($0.30–0.50$), moderate ($0.50–0.70$), high ($0.70–0.90$) or very high (>0.90) [45]. Because shorter TUG times indicate better performance, an inverse correlation with SCALE was expected and is interpreted as a positive functional association. Multiple linear regression estimated the association of SCALE with each outcome, unadjusted and adjusted for age, log-transformed time since stroke, Modified Ashworth grade and assistive-device use; adjusted models are reported as standardised coefficients (β). For descriptive comparison, participants were divided into low (1–4), moderate (5–7) and high (8–10) SMC groups, which were compared with the Kruskal–Wallis test. A two-sided p value below 0.05 was considered significant.

RESULTS

Participant characteristics

All 101 participants completed the assessments. Their mean age was 58.5 ± 6.6 years, 63 (62.4%) were men, and the median time since stroke was 21 months (interquartile range 14–29). Seventy participants (69.3%) had an ischaemic stroke, and the paretic side was left in 59 (58.4%). Thirty-nine participants (38.6%) walked with an assistive device, and most had mild to moderate ankle plantarflexor spasticity (Table 1). The mean SCALE score was 5.4 ± 2.4 (median 5, interquartile range 4–7), covering almost the whole range of the scale. Mean comfortable gait speed was 0.56 ± 0.26 m/s; 29 participants (28.7%) were household ambulators, 54 (53.5%) were limited community ambulators and 18 (17.8%) were community ambulators.

Table 1. Demographic and clinical characteristics of participants (N = 101)

Characteristic	Value
Age, years, mean $\pm$ SD	58.5 ± 6.6
Sex, male / female, n (%)	63 (62.4) / 38 (37.6)
Stroke type, ischaemic / haemorrhagic, n (%)	70 (69.3) / 31 (30.7)
Paretic side, left / right, n (%)	59 (58.4) / 42 (41.6)
Time since stroke, months, median (IQR)	21 (14–29)
Assistive device use, yes / no, n (%)	39 (38.6) / 62 (61.4)
Modified Ashworth grade (ankle plantarflexors), 0 / 1–1+ / 2 / 3, n	16 / 46 / 33 / 6
SCALE score (0–10), mean $\pm$ SD; median (IQR)	5.4 ± 2.4 ; 5 (4–7)
Walking category, household / limited community / community, n (%)	29 (28.7) / 54 (53.5) / 18 (17.8)

SD, standard deviation; IQR, interquartile range; SCALE, Selective Control Assessment of the Lower Extremity.

Association between SMC and gait performance

SCALE scores correlated significantly with all three gait outcomes (Table 2). The association was moderate with gait speed ($\rho = 0.64$) and with 6MWT distance ($\rho = 0.53$), and high with TUG time ($\rho = -0.70$), the negative sign indicating that better SMC accompanied faster completion of the task. Figure 1 shows the relationship with gait speed, which rose in a near-linear fashion across the SCALE range. Figure 2 shows the corresponding relationships for walking endurance and functional mobility. The gait outcomes were themselves only moderately interrelated (10MWT with 6MWT, $\rho = 0.43$; 10MWT with TUG, $\rho = -0.47$), so SMC was linked with distinct aspects of walking and not merely with one underlying speed factor.

Table 2. Gait performance and its correlation with SCALE score

Gait outcome	Mean $\pm$ SD	Spearman ρ (95% CI)	p value
10MWT gait speed (m/s)	0.56 $\pm$ 0.26	0.64 (0.50 to 0.75)	<0.001
6MWT distance (m)	199.5 $\pm$ 99.3	0.53 (0.36 to 0.66)	<0.001
TUG time (s)	30.0 $\pm$ 11.9	-0.70 (-0.79 to -0.57)	<0.001

10MWT, 10-Meter Walk Test; 6MWT, 6-Minute Walk Test; TUG, Timed Up and Go; CI, confidence interval. The negative coefficient for TUG indicates that higher SCALE scores accompany shorter (better) times.

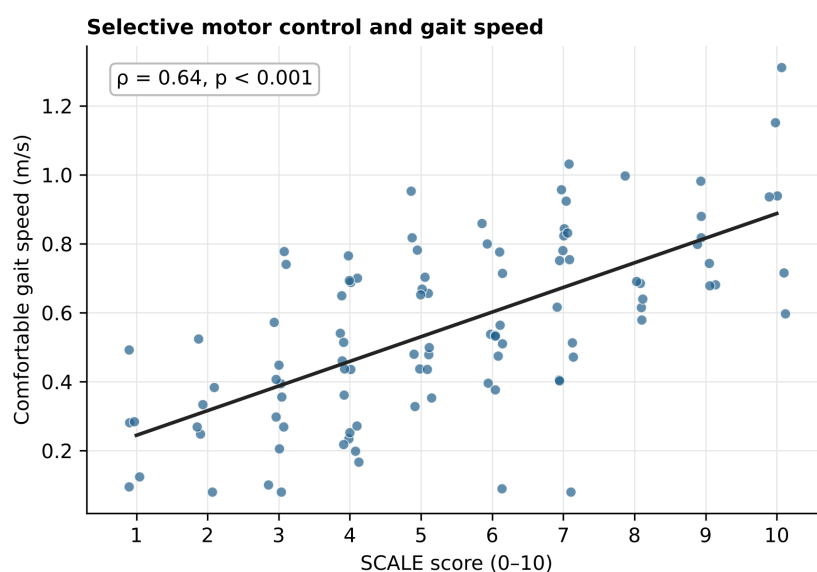


Figure 1. Relationship between SCALE score and comfortable gait speed. Points are individual participants (horizontal jitter added for visibility); the line is the least-squares fit.

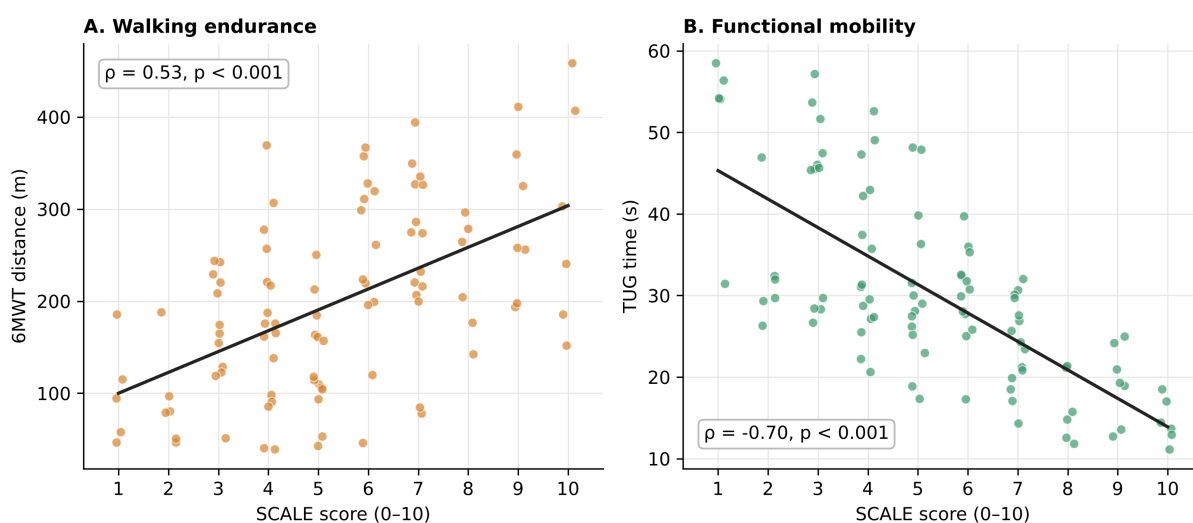


Figure 2. Relationship between SCALE score and (A) 6-Minute Walk Test distance and (B) Timed Up and Go time. Lines are least-squares fits.

Independent contribution of SMC

In unadjusted regression, each additional SCALE point was associated with 0.071 m/s faster gait speed (95% CI 0.055 to 0.088), a 22.6 m longer 6MWT distance (15.7 to 29.6) and a 3.5 s shorter TUG time (−4.2 to −2.8) (Table 3). Adjustment for age, time since stroke, spasticity and assistive-device use scarcely changed these estimates. In the adjusted models SCALE remained a strong independent correlate of gait speed ($\beta = 0.70$), 6MWT distance ($\beta = 0.60$) and TUG time ($\beta = -0.67$), all with $p < 0.001$. The models accounted for 46%, 32% and 51% of the variance in gait speed, 6MWT distance and TUG time, respectively.

Table 3. Linear regression of gait outcomes on SCALE score

Outcome	Unadjusted B per SCALE point (95% CI)	Unadjusted R ²	Adjusted β (95% CI)	Adjusted model R ²	p value
Gait speed (m/s)	0.071 (0.055 to 0.088)	0.42	0.70 (0.53 to 0.87)	0.46	<0.001
6MWT distance (m)	22.6 (15.7 to 29.6)	0.30	0.60 (0.41 to 0.78)	0.32	<0.001
TUG time (s)	−3.49 (−4.20 to −2.78)	0.49	−0.67 (−0.83 to −0.51)	0.51	<0.001

B, unstandardised coefficient; β , standardised coefficient. Adjusted models include age, log time since stroke, Modified Ashworth grade and assistive-device use; the p value refers to the SCALE term.

Gait performance across SMC categories

Of the 101 participants, 40 had low, 42 moderate and 19 high SMC. Performance improved stepwise across these groups on every measure (Figure 3). Mean gait speed was 0.38 ± 0.20 , 0.61 ± 0.22 and 0.81 ± 0.20 m/s; mean 6MWT distance was 153 ± 80 , 213 ± 99 and 269 ± 91 m; and mean TUG time was 38.1 ± 11.9 , 28.2 ± 7.6 and 16.8 ± 4.3 s, respectively (Kruskal–Wallis $p < 0.001$ for each outcome). Participants with high SMC therefore walked more than twice as fast as those with low SMC and completed the TUG in less than half the time.

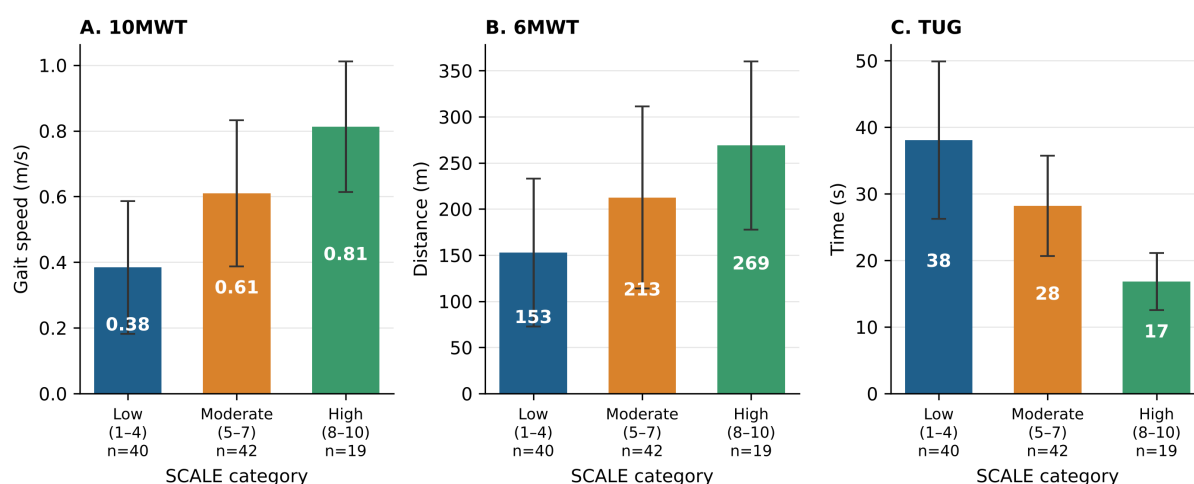


Figure 3. Gait outcomes by SCALE category: (A) 10-Meter Walk Test speed, (B) 6-Minute Walk Test distance and (C) Timed Up and Go time. Bars show mean $\pm$ SD.

DISCUSSION

This study set out to establish whether the ability to move the paretic leg selectively is related to how well people walk years after a stroke. In 101 chronic survivors, a bedside rating of SMC showed moderate to high correlations with gait speed, endurance and functional mobility, and these associations persisted after accounting for age, time since onset, spasticity and use

of a walking aid. The findings support the view that walking after stroke depends on the organisation of motor output across joints as well as on the force that muscles can generate.

The most plausible explanation lies in the neural basis of both constructs. Damage to descending corticospinal and related pathways reduces the capacity to fractionate movement, so that voluntary effort is channelled into obligatory synergies [16,17,19]. During walking, the leg must switch rapidly between patterns that are incompatible with a flexion–extension synergy, for example flexing the hip and knee while dorsiflexing the ankle to clear the foot, then extending all three joints to accept weight. Abnormal coupling of joint torques in the paretic limb has been demonstrated during gait itself [21], and individuals who cannot dissociate joints are forced to rely on compensations such as circumduction, hip hiking or vaulting [5,6]. These strategies lower step efficiency and slow progression. Reduced coordination complexity of muscle activation has also been shown to predict poorer walking performance [24,25], which is consistent with the dose–response pattern we observed across SCALE categories.

The strength of the association with gait speed is in line with earlier work showing that sensorimotor impairments explain a meaningful part of walking performance in the chronic phase [46], and that muscle torque alone does not tell the full story [12,13]. Our adjusted estimate that each SCALE point corresponds to roughly 0.07 m/s has practical meaning. Changes of about 0.10 m/s are regarded as meaningful in people with stroke and other conditions [31,47,48], so a gain of one to two SCALE points would be expected to accompany a perceptible change in speed. Moving from low to high SMC categories corresponded to a shift from household towards community ambulation by the speed classification of Perry and colleagues [29], which is the transition most relevant to participation in everyday life [4,30].

The strongest correlation was with the TUG. This test demands more than straight-line walking: sit-to-stand transfers, turning, deceleration and re-seating require rapid, well-timed changes in limb loading and joint alignment, and these are the movements in which an inability to dissociate hip, knee and ankle actions should be most costly. The TUG has previously been linked to paretic lower-limb impairment and locomotor capacity in chronic stroke [33], and impaired coordination may add the postural instability and reliance on the non-paretic side that raise fall risk. Our data cannot separate coordination from balance, and both probably contribute, but the result suggests that a SMC deficit is one identifiable source of poor functional mobility.

The association with the 6MWT was the weakest of the three, though still moderate and independent. This is expected, since endurance depends on aerobic capacity, deconditioning, motivation and cardiovascular health as well as on neuromotor control [34,35]. Even so, the finding is compatible with studies showing that hemiparetic walking carries a higher energy cost and lower mechanical efficiency than normal walking [49]. Co-contraction and ill-timed muscle activity in individuals with poor selectivity may increase the energy needed per metre and hasten fatigue, whereas more selective activation may allow the paretic limb to contribute propulsion more effectively [36,37]. The distance gained per SCALE point (about 23 m) falls within the range described as a small meaningful change in walking distance [48], although stroke-specific thresholds are needed to interpret it.

Several clinical implications follow. First, a quick SCALE rating could complement strength and tone testing when profiling a survivor with unexplained slow walking. A person with good strength but low SMC would be expected to benefit more from approaches that retrain movement selectivity, such as task-specific, high-repetition and motor learning-based gait training with feedback, than from strengthening alone [50–52]. Current guidelines recommend intensive, task-oriented locomotor training in the chronic phase [53,54], and our results suggest that SMC could be used to stratify who is likely to respond; responders and non-responders to locomotor training have been shown to differ in their baseline walking characteristics [38]. Second, SMC may serve as an additional treatment target and outcome in trials, because a measure that tracks the underlying impairment may be more sensitive to the effect of neuro-rehabilitation than speed alone [11,55].

Some limitations should be acknowledged. The cross-sectional design precludes causal inference, and it is equally plausible that people who walk more develop better motor control through activity. Recruitment from a single centre by convenience sampling limits generalisability, and our sample did not include people with severe walking disability, so the relationship may differ at the lower end of function. SCALE was developed in children with cerebral palsy [27,28], its scoring is based on observation and therefore subjective, and its psychometric properties in adult stroke have not been fully established; inter-rater reliability was not assessed in our sample. We could not account for all potential confounders, including proprioception, paretic muscle strength, fear of falling, cognition, cardiorespiratory fitness and the effects of previous therapy, and residual confounding is possible. The 10MWT and TUG were timed manually, and clinical tests cannot resolve the kinematic or electromyographic

mechanisms underlying the associations. Finally, the minimal detectable change of these tests varies with spasticity and impairment severity [56], which should be considered when interpreting individual scores.

Future work should follow survivors longitudinally to test whether change in SMC precedes change in walking, combine SCALE with kinematics, electromyography and muscle synergy analysis, define stroke-specific reliability and clinically important differences for SCALE, and evaluate interventions that target selectivity directly in randomised designs.

CONCLUSION

In adults with chronic stroke, better selective motor control of the paretic lower limb was associated with faster walking, shorter Timed Up and Go times and greater walking endurance, independent of age, time since stroke, spasticity and assistive-device use. Selective motor control appears to be a clinically relevant correlate of functional ambulation that is easily measured and not captured by strength testing alone. Assessing it routinely, and addressing it through task-specific motor learning approaches, may help therapists individualise gait rehabilitation. Longitudinal and interventional studies are required to confirm causality and to establish whether improving selectivity translates into better walking.

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