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Acute fatigue induced changes the passive and active mechanical properties of the gastrocnemius medialis in individuals with chronic stroke and healthy adults

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Abstract

Purpose: The consequences and underlying mechanisms of neuromuscular fatigue in the plantar flexors in a pathological population such as stroke patients remain poorly understood and have been very little investigated. The aim of this study was to (1) characterise the mechanical properties of the plantar flexors before, immediately after and at a later stage following an isometric fatigue protocol, and (2) compare this response with that of a healthy population.

Methods: Thirty volunteers took part in a fatigue protocol consisting of 60 isometric contractions, using an ergometer. Torque, shear modulus, stiffness and sEMG were measured under active and passive conditions before (P1), immediately after (P2) and after a 3-minute rest period (P3).

Results: Under active conditions, both groups exhibited a similar response pattern, with a decrease in torque, shear modulus, RFD and sEMG amplitude immediately following the protocol (P2), followed by incomplete recovery after a 3-minute rest period (P3). In the passive condition, although the shear modulus gradually decreased from P1 to P3 in both groups, overall stiffness did not respond in the same way. The stroke group showed no significant change in stiffness at any measurement time point, whilst the control group's stiffness gradually increased.

Conclusion: Fatigue led to mechanical changes in both groups, but with distinct local and global responses. The paretic muscle retains a fatigue response that is relatively comparable to that of a healthy muscle. Thus, the paretic muscle remains capable of relaxing under load despite its chronically elevated stiffness, which could be utilised to achieve clinical functional benefits.

Keywords : plantar flexors ; ankle joint ; Shear waves elastography ; stiffness ; neuromuscular fatigue ; muscle

Abbreviations

BMI : Body-Mass Index
CG : Control Group
EMG : Electromyography
GL : Gastrocnemius Lateralis
GM : Gastrocnemius Medialis
MVC : Maximal Voluntary Contraction
P1 : Pre-fatigue Phase
P2 : Post-fatigue Phase
P3 : Recovery Phase
PF : Plantar Flexor
RFD : Rate Force Development
RMS : Root Mean Square
ROM : Range Of Motion
SD : Standard Deviation
sEMG : Surface Electromyography
SG : Stroke Group
SOL : Soleus
SWE : Shear Wave Elastography
SWV : Shear Wave Velocity
TA : Tibialis Anterior

1. Introduction

Stroke is a leading cause of long-term disability, resulting from central and peripheral neurological damage that impairs postural balance, gait and functional independence (Zhao et al. 2015; Nozoe et al. 2024; Tiedt et al. 2026). Functional rehabilitation, primarily based on strengthening and stretching exercises, aims to restore muscle force and range of motion in order to limit further motor decline (Belghith et al. 2024; Ng et al. 2025). It must be emphasized that, neuromuscular fatigue is reported in up to 70% of patients, depending on the population studied, the time elapsed since stroke, and the diagnostic criteria used (Stein et al. 1996; Staub and Bogousslavsky 2001; Barbour and Mead 2012). Thus, fatigue constitutes a distinct and often underrecognized barrier to both rehabilitation and quality of life in this population.

Neuromuscular fatigue is classically defined as a reversible decline in the capacity to generate or sustain the required force output during physical exertion (Bigland-Ritchie and Woods 1984; Enoka and Duchateau 2008). It arises from both central mechanisms, involving supraspinal and spinal levels, and peripheral mechanisms, extending from the neuromuscular junction to the contractile apparatus itself. In the post-stroke context, muscle tissue undergoes substantial remodeling characterized by changes in fiber type composition, sarcomere organization, and connective tissue content, which are likely to alter the fatigue profile of the paretic limb (Gracies 2005a). Although the mechanical properties of paretic muscle at rest have been extensively investigated (Lee et al. 2019; Jalal et al. 2020; Garcia-Bernal et al. 2021; Huang et al. 2022; García-Bernal et al. 2022), how these properties evolve under fatiguing conditions remains largely unexplored. Because muscle architecture and neural constraints influence both force generation and passive mechanical behaviour, the structural remodelling that occurs after stroke may also modify the properties of paretic muscle to fatiguing exercise. Yet characterizing the mechanical response of paretic muscle to fatigue could provide critical insight for tailoring rehabilitation protocols and support the development of novel, fatigue-informed therapeutic strategies.

Force generation is governed by the mechanical properties of muscle tissue, which emerge from the combined contributions of active and passive structural elements (Hill 1938; Lieber et al. 2025). Active force originates from actin-myosin cross-bridge cycling, whereas passive resistance is primarily determined by the elastic properties of the extracellular matrix, in particular its collagen content (Chao and Sun 2020). The mechanical properties of both active and passive components can be non-invasively assessed in vivo using shear wave elastography (SWE), a technique that quantifies the shear modulus, a mechanical parameter directly related

to tissue stiffness (Eby et al. 2013). In a previous investigation conducted in a young, healthy population, we demonstrated that a fatiguing isometric exercise protocol induced substantial alterations in both the active and passive mechanical properties of the gastrocnemius medialis (GM). Specifically, the fatigue protocol reduced the active shear modulus of the GM by 38% and the passive shear modulus by 34% at 20° of dorsiflexion, with these localized alterations accompanied by a concomitant 25% decrease in musculotendinous stiffness (Vincent et al. 2024). Given that spastic myopathy following stroke is itself characterized by marked alterations in muscle architecture, fiber composition, and extracellular matrix properties (Azzollini et al. 2021; Qi et al. 2023), it remains unclear whether the mechanical and physiological responses to fatigue are exacerbated, attenuated, or otherwise altered in this already-compromised tissue. Neuromuscular fatigue may also, at least in part, underlie the self-reported fatigue commonly described by post-stroke patients, which constitutes a major obstacle to engagement and progress throughout the rehabilitation process (Aali et al. 2020; Alghamdi et al. 2021). A better understanding of the underlying physiological and biomechanical mechanisms therefore appears essential to inform and optimize rehabilitation strategies in this population.

The aims of this study were to (1) assess, using SWE, the adaptations in the passive an active mechanical properties of the GM induced by maximal isometric protocol in individuals with chronic stroke; and (2) to determine whether the extent of these adaptations differed between the paretic limb of the patients and that of healthy control subjects, in order to clarify the influence of central neurological impairment on the mechanical response of the muscle to fatigue. We hypothesised that fatigue would lead to an impairment of active and passive properties, as has already been observed in young adults (Vincent et al. 2024). We also hypothesised that, due to changes associated with spastic paresis, these impairments would be of a lesser magnitude in stroke survivors.

2. Materials and methods

2.1. Participants

Thirty subjects were included in the experimental protocol (**Table 1**). The control group (CG) (n = 15; age = 64.71 ± 12.24 years; BMI = 24.39 ± 3.27 kg/m^2) consisted of healthy individuals with no muscular disorders. The stroke group (SG) (n = 15; age = 67.92 ± 7.66 years; BMI = 25.91 ± 4.25kg/m^2) consisted of stroke survivor in chronic stage with spastic hemiparesis. A minimum torque of 25 Nm was required to take part, to ensure the feasibility of

the fatigue test protocol. All participants were informed of the nature of the study before providing written informed consent. The study was approved by the Person Protection Committee (CPP 2022-038=000117) and was conducted in compliance with the ethical guidelines of the Helsinki Declaration (World Medical Association 2013).

Tab 1. Subject's characteristics

Parameters	Stroke (Mean ± SD)	Control (Mean ± SD)
Age (y)	67.92 ± 7.66	64.71 ± 12.24
Sex (male:female)	8:7	7:8
Height (cm)	167 ± 6.6	167 ± 10.6
Weight (kg)	72.42 ± 10.86	68.85 ± 13.14
BMI (kg.m-²)	25.91 ± 4.25	24.39 ± 3.27
Affected side (L:R)	5:10	NA
Maximal torque (Nm)	55.47 ± 22.14	76.35 ± 28.55
Range of motion (°)	69.52 ± 7.5	78.71 ± 11.15
Spasticity (yes:no)	15:0	NA
Modified Ashworth Scale	1+:3 2:7 3:5	NA

2.2. Materials

2.2.1. Ergometer

An isokinetic dynamometer (Biodex 4 Medical Systems, Inc, Shirley, NY, USA) was used to quantify passive and active forces during all the protocol. The participants were placed in seat position with the knee bent between 10 and 20 degrees of flexion and the ankle joint accurately aligned with the centre of rotation of the ergometer. The position of the ankle joint was adjusted for each participant and defined as the 0° position in the isokinetic system to ensure maximum relaxation of the ankle muscles. The foot was held firmly in place using the non-elastic straps of the ankle module on the ergometer. In addition to the initial setup, a heel cup and non-elastic straps were added to prevent the heel from lifting during plantar and dorsiflexion (**Fig. 1**)



Fig. 1 *Experimental setup. (1) A heel cup is fitted over the foot to prevent heel lift-off. (2) Surface EMG electrodes record the myoelectrical activity of the muscle. (3) A Probefix holder maintains continuous and uniform contact between the ultrasound probe (4) and the gastrocnemius medialis throughout the protocol.*

2.2.1. Shear Wave Elastography

An Aixplorer® ultrasound scanner (Supersonic Imagine, version 6.1, Aix-en-Provence, France) was used to quantify GM shear modulus during passive and active force generation. The shear wave elastography mode was used with the musculoskeletal presets at the penetration level and measurements were scaled to 14.1m/s. An SL15-4 linear probe (4–15 MHz, SL15-4; SuperSonic Imagine, Aix-en-Provence, France) was positioned perpendicular to the skin and held in place using a probe stabilizer (ProbeFix®, Usono, Eindhoven, the Netherlands) to ensure uniform, minimal and continuous contact. Each acquisition was exported in DICOM SWE format and then analysed using a custom script in Matlab software (Matlab R2024a, MathWorks, Natick, USA). The mean Shear Wave Velocity (SWV) values were calculated within a region of interest located in the GM. The shear modulus was calculated, considering the muscle tissue as elastic, incompressible and isotropic, as :

$$\mu = \rho \times SWV^2, where\ \rho\ is\ the\ density\ of\ the\ muscle\ tissue\ (1{,}000\ kg/m^3)$$

2.2.2. Surface electromyography

A surface electromyograph (sEMG, FREEEMG 1000 – BTS Bioengineering, Milan, Italy) was used to record muscle activity. To reduce impedance, the skin was first shaved and cleaned

using an abrasive cleaner (70% alcohol). Four self-adhesive Ag/AgCl electromyography (EMG) sensors (Kendall H124SG, Covidien, Germany) were placed on GM, gastrocnemius lateralis (GL), soleus (SOL) and tibialis anterior (TA) muscles, with a spacing of 20 mm between the electrodes (centre-to-centre). In accordance with SENIAM recommendations (Hermens et al. 2000), their longitudinal positioning relative to the alignment of the muscle bundles and their distance from neighbouring muscles (Lacourpaille et al. 2017; Le Sant et al. 2019a) was verified using the Aixplorer® ultrasound scanner in B-mode (ultrasound mode). The EMG signals were filtered using a second-order Butterworth digital filter. The cut-off frequencies were selected in accordance with the recommendations of (Lim et al. 2021), namely 20 Hz and 450 Hz. All data from the different assessments were collected, rectified and smoothed using a root mean square (RMS) analysis with a 200ms window. Finally, RMS EMG were normalized to RMS EMG during the isometric maximal voluntary contraction (MVC).

2.3. Experimental measurements

2.3.1. Sequential evaluations

The experimental protocol comprised three consecutive phases, during which passive and active outcomes were measured under standardized conditions. The pre-fatigue phase (P1) characterized participant' baseline condition. The post-fatigue phase (P2) involved data collection immediately upon completion of the fatigue protocol. The acute recovery phase (P3) involved a final data collection following a 3-minute recovery period.

Passive outcomes

The maximal ankle range of motion (ROM) was determined by passively moving the joint from plantar flexion (PF) to dorsiflexion. Participants were instructed to remain fully relaxed throughout the maneuver and to indicate verbally when they reached the point of stretch corresponding to their individual comfort limit. SWV was continuously recorded within the GM muscle throughout the entire range of motion. Muscle activity of the GM, GL, SOL and TA was simultaneously recorded during the movement.

Active outcomes

Three MVC were performed on the ergometer. Participants were instructed to develop maximal plantarflexion force as rapidly and forcefully as possible. Each contraction was sustained for five seconds, with sufficient rest allowed between trials. EMG and SWV were recorded continuously throughout the MVC testing procedures. Rate of force development (RFD) was calculated from the linear slop of the torque – time curve (Δ torque/Δ time) during MVC.

Early RFD was obtained from each MVC contraction onset to 50 ms (RFD0-50). Late RFD was acquired from 0 to 200 ms (RFD0-100, RFD0-200, RFD100-200).

2.3.2. Fatigue protocol

Each participant had to perform 60 maximal isometric contractions lasting 4 seconds, interspersed with 1-second rest periods. Throughout this strenuous task, the participants were guided by an audible signal and verbal instructions. To maximise effort, verbal encouragement was provided throughout. Finally, the participants were not told the exact duration of the test to prevent them from adopting effort-management strategies (**Fig. 2**)

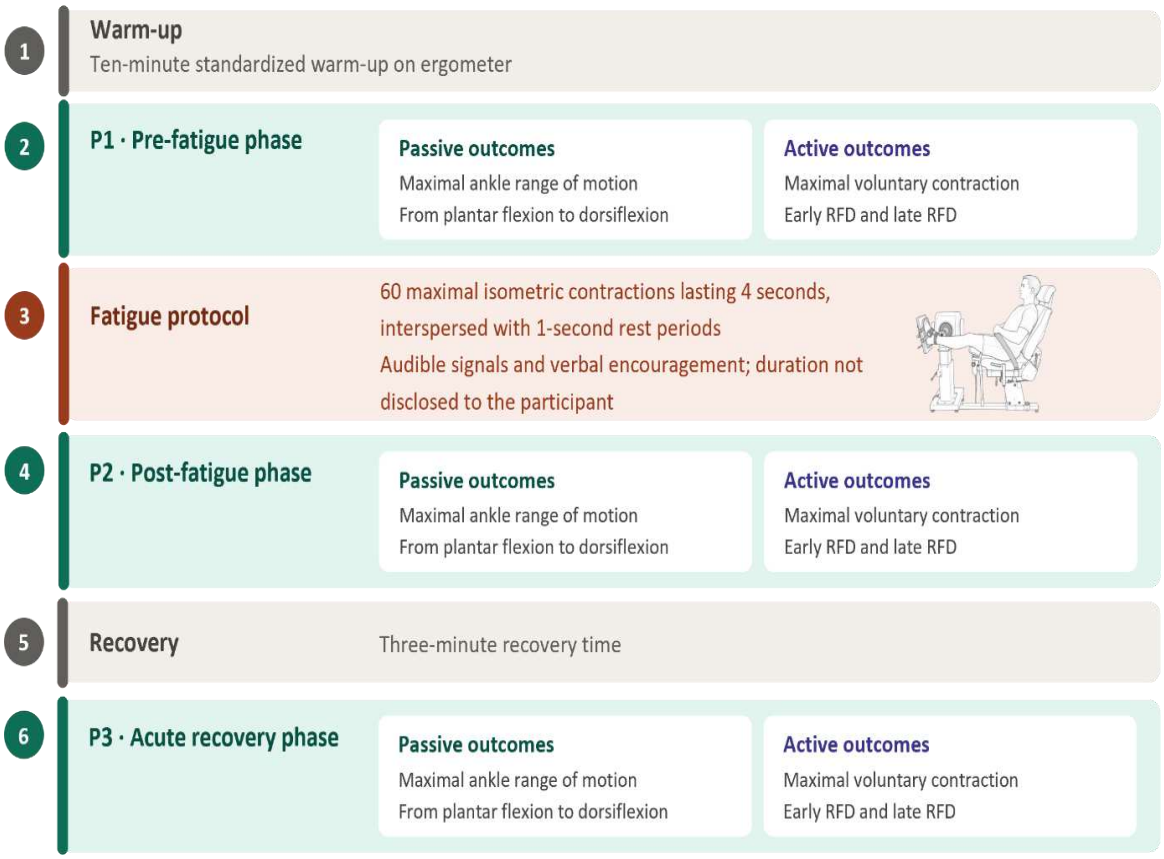


Fig. 2 *Set-up of the fatigue protocol.*

2.4. Statical analysis

Sample size was calculated using G*Power software (version 3.1.9.7) (Faul et al. 2007). The calculation was based on the study of Le Sant et al. (2019b), who comparing lower leg muscle stiffness on stroke and healthy population and reported an effect size Cohen's d = 1.52. Assuming similar effect size, a power of 95% and a significant level of 0.05, our sample size

calculations indicated that a minimum of 12 participants per group would be required (Verma and Verma 2020).

Statistical analyses were performed using Prism 11 Software (GraphPad Software, Inc., San Diego, USA). The normality of variance was assessed using the Kolmogorov-Smirnov test. Difference concerning MVC and sEMG signal were evaluated using the student's t-test. In addition, ANOVA was used for RFD, stiffness and shear modulus.

The values are presented as mean (± standard deviation (SD)). The significance level was set at 5% ($p < 0.05$). The SD/mean ratio (expressed in %) was used to quantify inter-individual variability among participants.

3. Results

3.1. Effect of fatigue on passive parameters

3.1.1. Comparison intra-group

Stiffness increased significantly in the CG (+12% $p<0.01$) between P1 and P2 and between P1 and P3 (+15.8%; $p<0.001$). In the SG, no effect of fatigue was reported (**Fig. 3.a and c**). The angle-dependent passive torque increased as the PF was stretched (-30° to +20°) in both groups at all three measurement time points ($p<0.001$). The passive shear modulus significantly changes during the protocol. Overall, the significance values increased as the muscle was stretched in plantar flexion for both groups with a fall between P1 and P2 (CG = -12% to -13.6% ; $p<0.05$ / SG = -14.6% to -17.3% ; $p<0.05$) at all angles (except 0%–20% for the SG) and between P1 and P3 (CG = -16.8% to -19.6% ; $p<0.01$ / SG = -21.7 % to -28.2% ; $p<0.01$). Regarding the time between P2 and P3, the analyses showed a significant decrease at the 40-60% (-7.2% ; $p<0.01$) and 60-80% (-6.9% ; $p<0.01$) for the CG, and a significant reduction at all angles for the SG (-8.4% to -19.9% ; $p<0.05$) (**Fig. 3.b and d**).

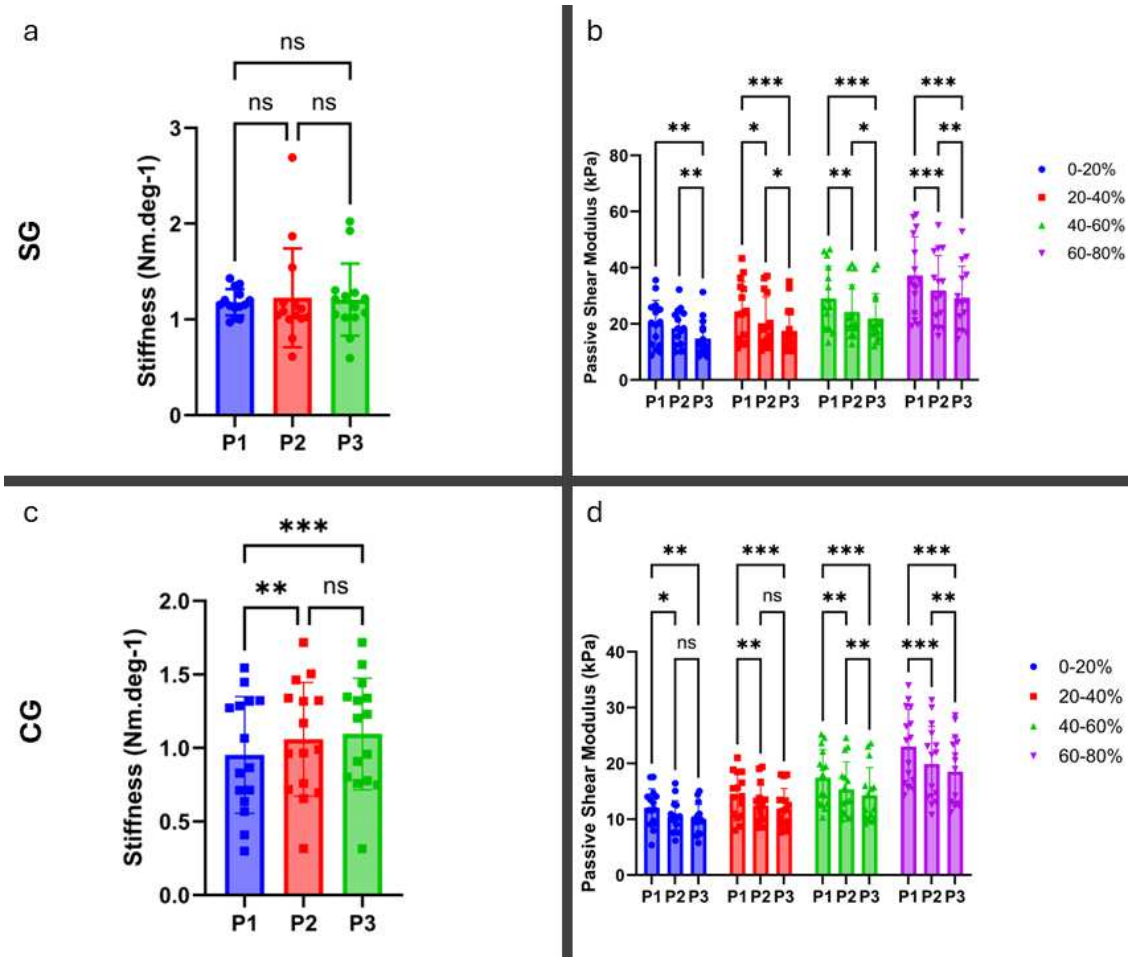


*Fig. 3 Comparative analysis of the passive stiffness of the plantar flexor muscle–tendon unit in the SG (a) and the CG (c), and passive shear modulus of the gastrocnemius medialis during passive dorsiflexion in the SG (b) and the CG (d) across P1, P2 and P3. *: Significant difference, p<0.05; **: Significant difference, p<0.01; ***: Significant difference, p<0.001; ns: no significant difference.*

3.1.2. Comparison inter-group

Prior to the fatigue protocol, stiffness was higher in the SG compared to the CG (+24.6% ; $p<0.05$). However, no significant differences were reported after the protocol nor after recovery between group (**Fig. 4.**).

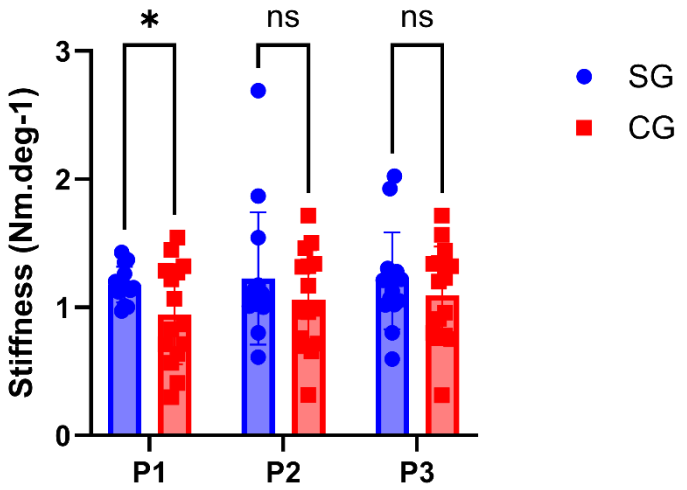


*Fig. 4 Comparative analysis of the stiffness of the plantar flexor muscle-tendon unit between SG and CG across P1, P2 and P3. *: Significant difference, p<0.05; ns: no significant difference.*

The angle-dependent passive torque was significantly higher in the SG at all angles prior to the fatigue protocol (+19.8% to +35.8%; $p<0.05$). No significant differences were subsequently observed in either P2 or P3 (**Fig. 5.a to c**).

The passive shear modulus was significantly higher in the SG compared with the CG, regardless of time or angle (P1 = +38.8% to +41.5% ; $p<0.01$ / P2 = +36.5% to +43.1% ; $p<0.01$ / P3 = +32.1% to +36.6% ; $p<0.05$) (**Fig. 5.d to f**).

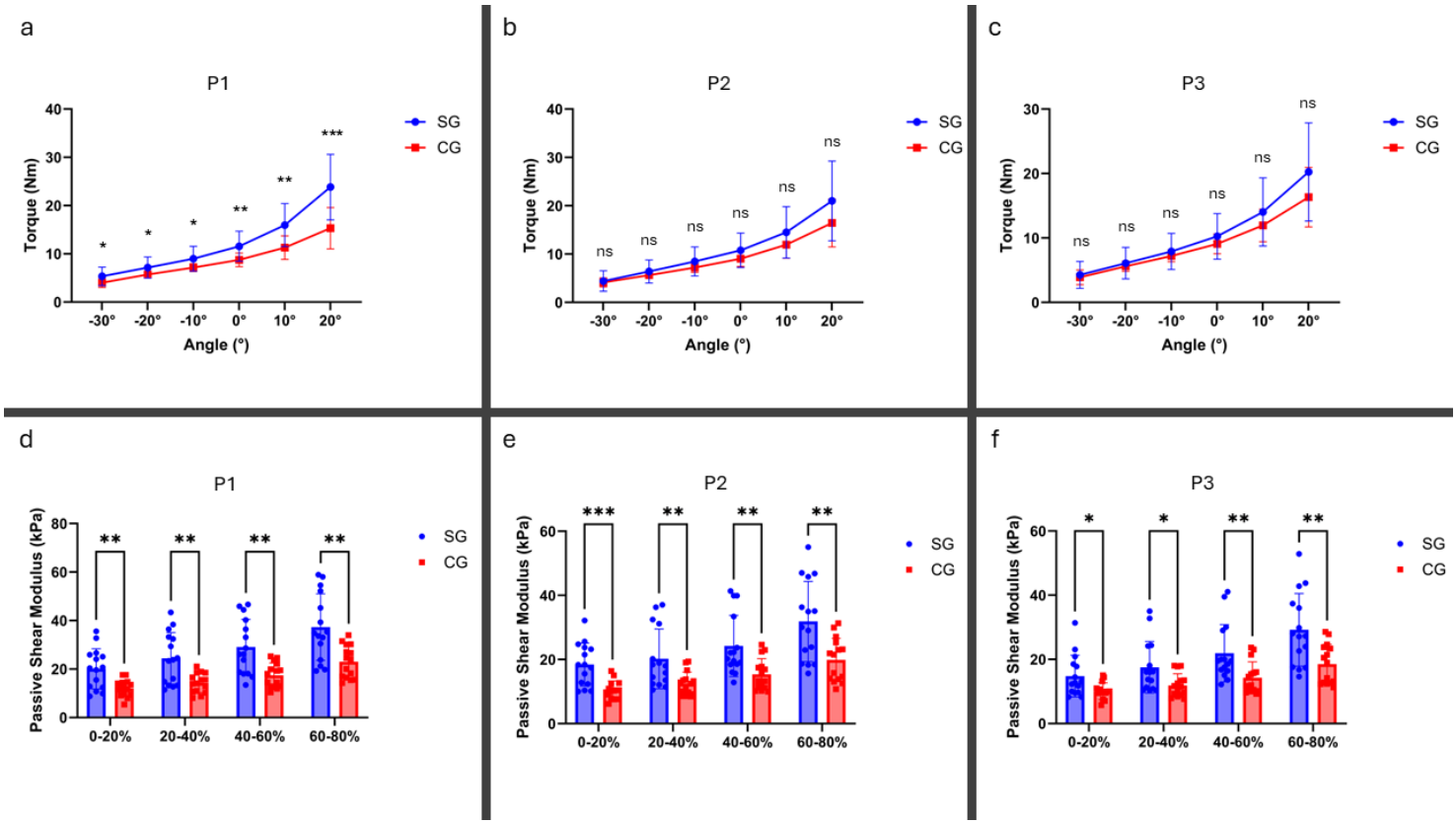


*Fig. 5 Comparative analysis of the passive ankle depended torque of the plantar flexor muscle–tendon unit between SG and CG (a,b,c), and passive shear modulus of the gastrocnemius medialis during passive dorsiflexion between SG and CG (d,e,f) across P1, P2 and P3. *: Significant difference, p<0.05; **: Significant difference, p<0.01; ***: Significant difference, p<0.001; ns: no significant difference.*

3.2. Effect of fatigue on active parameters

3.2.1. Comparison intra-group

In both groups, torque decreased significantly following the protocol, between P1 and P2 (CG = -45.4% ; p<0.0001 / SG = -43.1% ; p<0.001). Between P2 and P3 we observed an increase in torque (CG = +59.2% ; p<0.0001 / SG = +51.9% ; p<0.001). Nevertheless, at P3, the values remained significantly lower than at P1 (CG = -13.1% ; p<0.001 / SG = -14.7% ; p<0.001) (**Fig. 6.a and d**).

The sEMG amplitude followed the same pattern as the torque, with a decrease in amplitude between P1 and P2 (CG = -57.7% ; p<0.001 / SG = -58.1% ; p<0.001), followed by an increase between P2 and P3 (CG = +63.8% ; p<0.0001 / SG = +62.2% ; p<0.01). The values at P3

relative to P1 also remained lower (CG = -30.8% ; p<0.01 / SG = -32.1% ; p<0.001) (**Fig. 6.b and e**).

The active shear modulus decreased significantly in both groups. The shear modulus decreased between P1 and P2 (CG = -44.6% ; p<0.001 / SG = -37.5% . p<0.001) and between P1 and P3 (CG = -14.7% ; p<0.01 / SG = -20.7% ; p=0.001). Between P2 and P3, the shear modulus increased (CG = +53.8% ; p<0.001 / SG = +26.9% ; p<0.05) (**Fig. 6.c and f**).

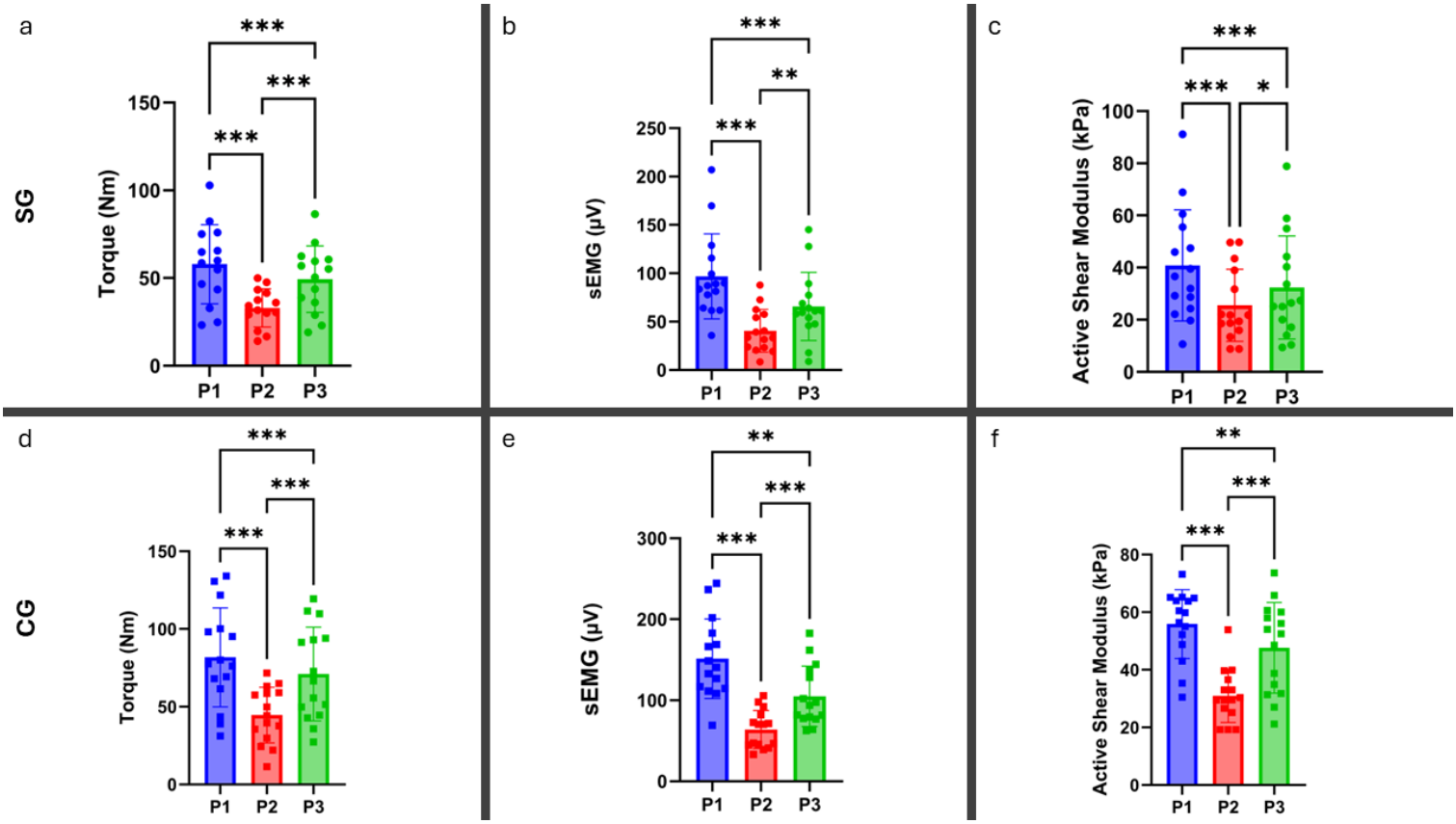
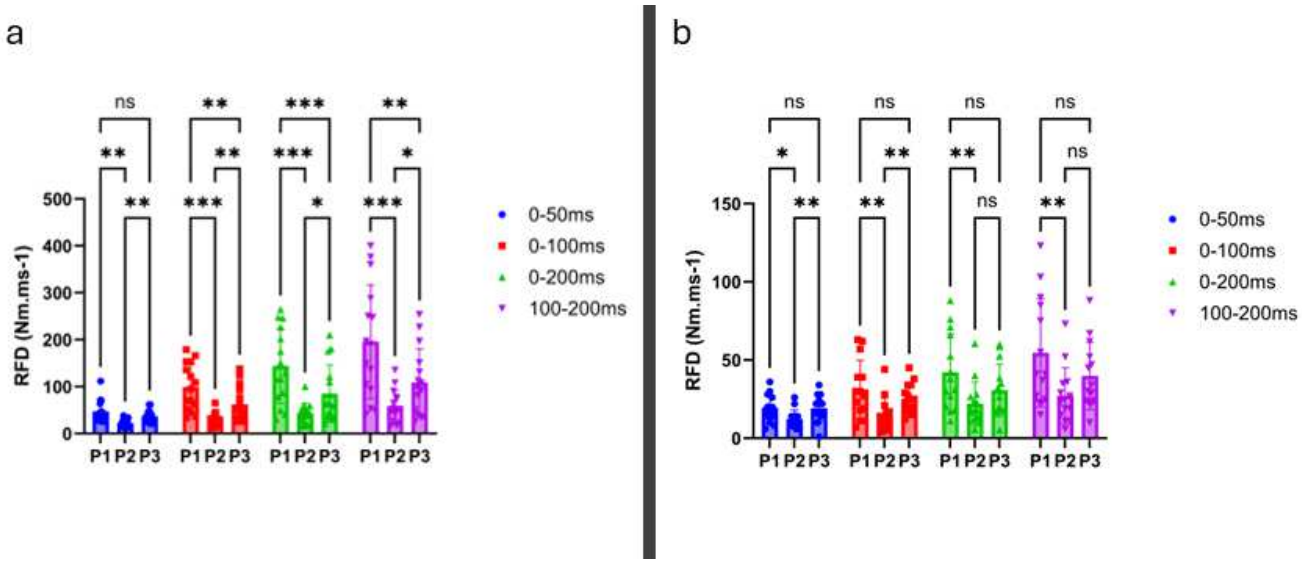


*Fig. 6 Comparative analysis of the maximal torque in the SG (a) and the CG (d), the peak activity of the gastrocnemius medialis in the SG (b) and the CG (e), the active shear modulus of the gastrocnemius medialis during maximal voluntary contraction in the SG (c) and the CG (f) across P1, P2 and P3. *: Significant difference, p<0.05; **: Significant difference, p<0.01; ***: Significant difference, p<0.001.*

The RFD decreased significantly in both groups at all time points, showing a reduction from P1 to P2 (CG = -53.1% to -70.4% ; p<0.01 / SG = -36.4% to -51.1% ; p<0.05). Regarding the interval between P2 and P3, the analysis demonstrated a notable increase across all intervals for the CG (+65.2% to +103.5% ; p<0.05), but only at 0-50 ms (+51.5% ; p<0.01) and 0-100 ms (+66.4% ; p<0.01) for the SG. In P3, the values remained below those of P1 for the CG at 0-100 ms, 0-200 ms and 100-200 ms (-37.0% to -44.3% ; p<0.01) but not at 0-50 ms. Furthermore, there were no significant differences compared with P1 for the SG (**Fig. 7**).



394

395 *Fig. 7 Comparative analysis of the rate force development of the plantar flexor muscle–tendon unit for*
396 *the CG (a) and the SG (b) across P1, P2 and P3. *: Significant difference, p<0.05; **: Significant*
397 *difference, p<0.01; ***: Significant difference, p<0.001; ns: no significant difference.*

398

399 3.2.2. Comparison inter-group

400 The SG torque remained statistically lower regardless of the time point (P1 = -41.5% / P2 = -
401 38% / P3 = -44.6% ; p<0.05) (**Fig. 8.a**).

402 The sEMG amplitude differed significantly between the groups. The values for the SG group
403 were lower for P1 (-56.4% ; p<0.01), P2 (-57.8% ; p<0.05) and P3 (-59.4% ; p<0.01) (**Fig.**
404 **8.b**).

405 Statistical analysis of the active shear modulus revealed that the P1 values were lower in the
406 SG group (-36.9%; p<0.05) and in P3 (-47.1%; p<0.05). However, the values at P2 were not
407 significantly different (**Fig. 8.c**).
408 The RFD was generally significantly lower in the SG group at all time points (P1 = -151.7% to
409 -259.5% ; p<0.001 / P2 = -81.2% to -113.4% ; p<0.01 / P3 = -91.5% to -176.4% ; p<0.01)
410 (**Fig.8.d to f**).

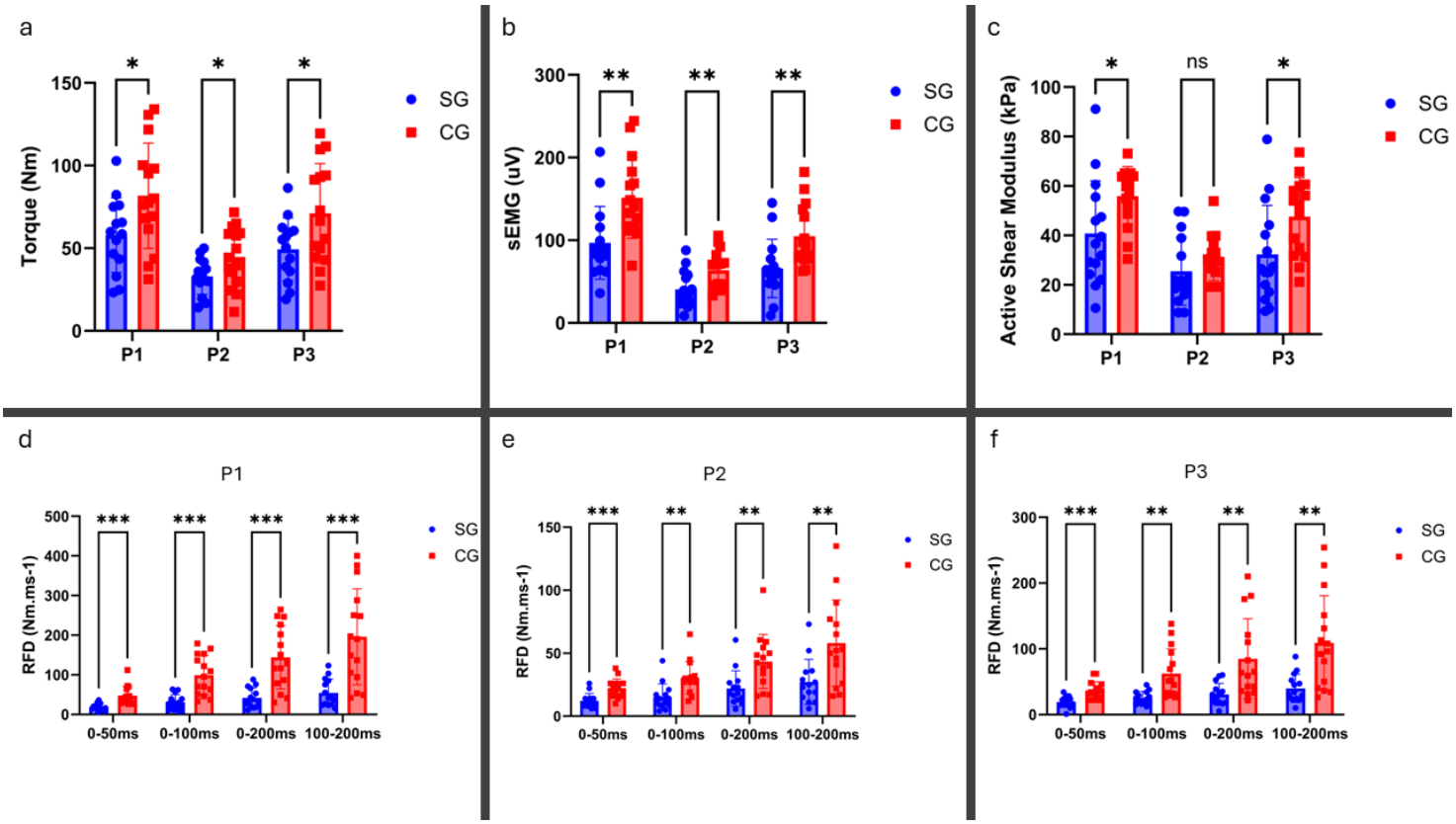


*Fig. 8 Comparative analysis of the maximal torque between SG and CG (a), the peak activity of the gastrocnemius medialis between SG and CG (b), the active shear modulus of the gastrocnemius medialis during maximal voluntary contraction between SG and CG (c), the rate force development of the plantar flexor muscle–tendon unit between SG and CG (d,e,f) across P1, P2 and P3. *: Significant difference, p<0.05; **: Significant difference, p<0.01; ***: Significant difference, p<0.001, ns: no significant difference.*

4. Discussion

The present study aimed to investigate the acute effects of a maximal isometric fatigue protocol on the passive and active mechanical properties of the muscle-tendon complex in individuals with chronic stroke and to compare fatigue-induced responses with those observed in healthy controls. Overall, the fatigue protocol induced substantial alterations in both passive and active muscle-tendon complex properties in both groups, although the magnitude and pattern of these responses differed between individuals with chronic stroke and healthy controls.

Changes in passive and active outcomes in stroke patients

The results showed that the fatigue protocol induced distinct changes in both passive and active GM properties in individuals with chronic stroke. The progressive reduction in passive shear modulus from P1 to P3 may initially appear counterintuitive in the context of

post-stroke spastic myopathy, which is typically associated with increased muscle stiffness (Gracies 2005a; Roots et al. 2022). However, this finding may be better understood within the framework of the time-dependent viscoelastic behaviour of skeletal muscle (McHugh et al. 1992; Van Loocke et al. 2008). Indeed, following repeated loading and sustained positioning, passive muscle structures may undergo stress relaxation and redistribution of internal tension, which may have contributed to the progressive reduction in measured shear modulus (Maganaris et al. 2002; Morel et al. 2019). Furthermore, at the cellular level, recent evidence from isolated muscle fibres suggests that changes in the passive Young's modulus may be related to modifications in the phosphorylation state of the elastic domain of titin, whereas the stiffness of fibre bundles with an intact extracellular matrix may remain relatively unaffected (Privett et al. 2024). These findings highlight the contribution of both intracellular and extracellular structures to passive muscle mechanics and may provide a mechanistic framework for interpreting the present results. Importantly, the absence of concomitant changes in passive stiffness of the plantar-flexor and tendon complex suggests that the reduction detected locally within the GM did not translate into a measurable change in the mechanical behaviour of the muscle–tendon unit as a whole. Such a discrepancy between local measurements obtained using shear-wave elastography and global assessments performed using an ergometer has been reported previously (Le Sant et al. 2017) and likely reflects the integration of mechanical contributions from multiple muscles, tendons, aponeuroses and other elastic structures within the muscle-tendon complex.

In contrast to the progressive passive changes, the fatigue protocol produced a pronounced and immediate deterioration in active muscle function. Immediately after the protocol, torque, sEMG amplitude, RFD, and active shear modulus were markedly reduced, demonstrating a substantial impairment of force-generating capacity. The concomitant reduction in sEMG amplitude and active shear modulus are consistent with a multifactorial impairment of neuromuscular function potentially involving both neural and peripheral mechanisms (Bigland-Ritchie and Woods 1984; Enoka and Duchateau 2008; Nojiri et al. 2025). The reduction in sEMG amplitude may reflect a decrease in the neural activation of the muscle during maximal contractions, whereas the decline in active shear modulus may reflect a reduction in the mechanical response of the muscle fibres to neural activation. At the peripheral level, this response may be related to alterations in excitation–contraction coupling, including reduced Ca^{2+} release from the sarcoplasmic reticulum, decreased myofilament Ca^{2+} sensitivity, and a reduction in the force produced by individual actin–myosin cross-bridges (Debold et al. 2016; Olsson et al. 2020). The simultaneous reduction in neural activation and active muscle

mechanical properties therefore suggests that the observed loss of torque cannot be attributed exclusively to a single site of fatigue but rather reflects the combined contribution of central and peripheral mechanisms.

Following three minutes of recovery, torque, sEMG amplitude, and active shear modulus increased significantly, indicating a substantial but incomplete restoration of neuromuscular function. However, these variables remained below their pre-fatigue values, suggesting that the recovery period was insufficient to fully restore force-generating capacity. This partial recovery is compatible with the relatively rapid restoration of metabolic homeostasis and excitation–contraction coupling following cessation of the fatiguing task, although some residual impairment may persist over the short term (Carroll et al. 2017a). The apparent recovery of RFD should be interpreted with greater caution. Given the very low baseline RFD values observed in the stroke group, the absence of a significant difference between P3 and P1 may partly reflect a floor effect rather than complete physiological recovery. Overall, these findings indicate that acute fatigue transiently compromises both neural activation and the mechanical expression of muscle contraction in chronic stroke, whereas passive mechanical changes appear to follow a distinct, time-dependent trajectory that is not necessarily coupled to the acute loss of active force production.

Changes in passive and active outcomes: stroke vs healthy controls

Individuals with chronic stroke initially exhibited greater passive muscle-tendon stiffness and angular torque than healthy controls, indicating increased resistance to passive ankle rotation (Gao et al. 2009; Huang et al. 2022; García-Bernal et al. 2023). However, these between-group differences were no longer evident immediately after fatigue or following the recovery period. Importantly, this apparent convergence was not attributable to a reduction in passive mechanical properties in the stroke group, but rather to an increase in stiffness in the healthy controls following the fatiguing protocol. This response contrasts with our previous findings in young healthy adults, in which the same protocol induced a significant reduction in muscle-tendon complex stiffness (Vincent et al. 2024). The discrepancy may be related, at least in part, to the substantially older population investigated in the present study. Age-related modifications of the muscle–tendon unit, including increased connective tissue content, altered tendon mechanical properties, changes in muscle architecture and fibre composition, may reduce the capacity of the ageing muscle–tendon complex to undergo creep and stress relaxation during repeated loading (Ryall et al. 2008; Aagaard et al. 2010; Wu et al. 2020). However, this interrogation remains speculative, as age was not investigated in the present work.

Thus, the disappearance of the initial between-group difference after fatigue suggests that the passive mechanical response of the whole muscle–tendon complex is not determined solely by the elevated baseline stiffness associated with chronic stroke, but also reflects the acute mechanical adaptations induced by the fatiguing task. In contrast, the local assessment of the GM revealed a persistent and markedly different mechanical phenotype in the stroke group. The passive shear modulus was consistently higher in the stroke group across all ankle angles and at all measurement time points, with between-group differences ranging from approximately 32% to 43%. This result is in agreement with previous elastography studies showing increased passive stiffness of the paretic gastrocnemius after stroke and with systematic evidence indicating that higher muscle stiffness is a common feature of post-stroke paretic muscles (Roots et al. 2022; Devis et al. 2024; Belghith et al. 2024). Such an increase likely reflects the chronic structural remodelling associated with spastic paresis, including alterations in the extracellular matrix, increased connective tissue accumulation, changes in fascicle architecture, and persistent alterations in muscle resting state (Gracies 2005a; Jalal et al. 2020; Azzollini et al. 2021; Kim et al. 2021). It must be emphasized that, although the GM remained mechanically stiffer in the stroke group throughout the protocol, the magnitude of the between-group difference progressively decreased over time. This pattern suggests that the paretic muscle retains a capacity for time-dependent stress relaxation despite its chronically altered mechanical phenotype. The distinction between the persistent local increase in GM shear modulus and the transient group difference observed at the level of the whole muscle-tendon complex further emphasizes that local muscle GM mechanical behaviour and global joint-level mechanical behaviour represent related but non-equivalent constructs. Indeed, SWE provides a localized estimate of tissue mechanical properties, whereas passive torque and complex stiffness integrate the mechanical contributions of several muscles, tendons, aponeuroses, and other series and parallel elastic structures. The moderate relationship between SWE measures and clinical or joint-level manifestations of post-stroke spasticity reported in the literature further supports this interpretation (Kim et al. 2026).

The contrast between groups was substantially more pronounced for active than for passive outcomes. Across all measurement periods, the stroke group demonstrated lower torque, sEMG amplitude, active shear modulus, and RFD, confirming the persistent impairment of voluntary force production and rapid force generation that characterizes chronic post-stroke motor dysfunction. The lower active shear modulus suggests a reduced mechanical expression of muscle activation in the paretic GM, which may reflect the combined effects of impaired neural drive, altered motor-unit recruitment and discharge, muscle atrophy and architectural

remodelling, and intrinsic contractile dysfunction (Le Sant et al. 2019b; Aze et al. 2023). Similarly, the consistently lower sEMG amplitude indicates that the ability to generate an effective neural drive during MVC remained impaired throughout the protocol. These findings are consistent with the established pathophysiology of chronic stroke, in which impaired descending activation and altered motor-unit behaviour coexist with structural and contractile adaptations within the paretic muscle (Gracies 2005b; Aze et al. 2023). The particularly large deficit in RFD is also noteworthy, as rapid force production depends strongly on the early recruitment and discharge of motor units and on the intrinsic contractile properties of the muscle fibres (Canning et al. 1999; Freire et al. 2015). Consequently, RFD may be more sensitive than maximal torque to the neuromuscular limitations imposed by chronic stroke. This observation is particularly relevant from a functional perspective, because impaired rapid force generation may compromise the ability to respond to sudden perturbations or rapidly stabilize the body during gait and balance-related tasks. Despite these substantial differences in absolute performance, the relative magnitude of fatigue was remarkably similar between groups for torque and sEMG amplitude. Torque decreased by approximately 40–45% in both groups, whereas sEMG amplitude decreased by approximately 55–60%, indicating that the stroke group did not experience a disproportionately greater loss of maximal force or voluntary activation during the fatiguing task. This finding is important because it suggests that the lower performance of individuals with chronic stroke does not necessarily imply an exaggerated susceptibility to acute fatigue when fatigue is expressed relative to their own baseline capacity. Rather, the stroke group appears to begin the task from a markedly reduced functional level but undergoes a broadly comparable relative deterioration during the standardized fatiguing stimulus. The active shear modulus provides further support for this interpretation. Immediately after fatigue, the between-group difference disappeared because the reduction was greater in healthy controls than in the stroke group. One explanation is that the convergence of active shear modulus values at P2 may therefore reflect the greater absolute mechanical reserve available for deterioration in the healthy muscle, whereas the stroke group started from an already compromised mechanical state.

Finally, recovery was less complete in the stroke group for torque and active shear modulus, suggesting that the consequences of fatigue persisted beyond the immediate post-exercise period. This pattern may reflect a reduced neuromuscular reserve and a slower restoration of voluntary motor output following fatigue in individuals with chronic stroke (McManus et al. 2017). Taken together, these findings indicate that chronic stroke is characterized by a dissociation between persistently elevated local passive muscle stiffness,

markedly reduced active mechanical and neuromuscular capacity, and a broadly preserved relative acute fatigue response, but with a particular vulnerability of rapid force production and incomplete recovery.

Potential clinical implications

From a clinical perspective, the present findings suggest that a brief bout of repeated isometric contractions may transiently reduce passive muscle stiffness in individuals with chronic stroke, potentially creating a short therapeutic window for stretching, mobilisation, positioning, or orthotic fitting. However, this potential benefit must be balanced against the concomitant reduction in torque and RFD observed immediately after fatigue. Thus, although fatigue may temporarily increase passive muscle compliance, it may simultaneously reduce the patient's ability to generate rapid corrective forces, which is particularly relevant for postural control and fall risk (Mademli et al. 2008). Clinically, isometric exercise should therefore be carefully dosed and, if used to facilitate subsequent mobilisation, performed in a controlled and supervised context. Given the limited three-minute recovery period investigated here, the duration and functional relevance of this potential therapeutic window remain to be established (Coroian et al. 2018).

Limits & perspectives

This study has several limitations. Firstly, the use of the SWE involves several computational assumptions. Indeed, in this study, the muscle is assumed to be elastic, linear, incompressible, homogeneous and isotropic. Furthermore, the hypotheses of a density of 1,000 kg/m^3 is a simplification arising directly from the assumption of incompressibility. Although these assumptions are commonly accepted in the literature (Gennisson et al. 2013; Siracusa et al. 2019; Belghith et al. 2024), they become an additional source of bias in the case of a post-stroke population, as pathological muscular changes (spasticity, atrophy, accumulation of non-connective tissue, ...) directly alter these properties. Moreover, although the present study measured the impact of fatigue following a recovery period, this was limited to 3 minutes. Given that complete neuromuscular recovery takes place over a much longer period (Carroll et al. 2017b), particularly in post-stroke individuals (Knorr et al. 2011), future research spanning a longer period would provide a better understanding of the recovery process. Finally, this study focused on the GM in terms of SWE analysis. However, we observed a different response from the plantar flexor complex. Thus, SWE analysis of the GL, the SOL and also tendon under identical experimental conditions would provide a better understanding of the complex's

overall response. Despite these limitations, this study sheds new light on the mechanical and neuromuscular responses in post-stroke individuals, paving the way for potential clinical implications with a view to improving the care of this population.

5. Conclusion

Acute maximal isometric fatigue induced distinct changes in the mechanical properties passive and active of the muscle-tendon complex in individuals with chronic stroke. Passive GM shear modulus progressively decreased following the fatiguing protocol, while active mechanical and neuromuscular performance was markedly impaired, with incomplete recovery after three minutes. Compared with healthy controls, individuals with chronic stroke exhibited persistently greater passive muscle stiffness and substantially lower torque, sEMG amplitude, active shear modulus, and RFD, despite a broadly comparable relative fatigue response. These findings highlight a dissociation between the time-dependant response of passive muscle mechanics and the accurate impairment of active force-generating capacity following fatigue. They further suggest that fatigue may transiently modify muscle stiffness while simultaneously reducing functional neuromuscular reserve. From clinical perspective, the transcient decrease in passive GM shear modulus raises the possibility that fatigue could transform the mechanical conditions. However, it remains to be determined whether these changes translate into clinically functional benefits.

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7. Data avaibility

The data underlaying this study can be shared on request to the corresponding author.

8. Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

9. Authors contributions

This work was equally contributed by LV and WM. MZ: conceptualization, resources, validation; LV and WM: writing original draft, methodology; MZ and PP: instructing, review; LV, KB and WM: data collection and analysis; WM and MZ: instructing; MZ: review. MG: software development. All authors contributed to the article and approved the submitted version.

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