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Altered muscle synergy structure in patients with poststroke stiff knee gait

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Stiff knee gait (SKG) occurrence after a stroke is associated with various abnormal muscle activities; however, the interactions among these muscles are unclear. This study aimed to elucidate the muscle synergy characteristics during walking in patients with SKG after a stroke. This cross-sectional study included 20 patients with poststroke SKG (SKG group), 16 patients without poststroke SKG (non-SKG group), and 15 healthy adults (control group). Participants walked a 10-m distance at a comfortable speed, and electromyographic data were recorded from six lower-limb muscles. Non-negative matrix factorization was employed to derive time-varying activity (C), muscle weights (W), and the percentage of total variance accounted for (tVAF) for muscle synergies. The SKG group showed a higher tVAF than the control group. The initial stance module (including knee extensors) showed increased activity during the swing phase. The initial swing module (including hip flexors and ankle dorsiflexors) exhibited a higher activity during the single-support phase but a lower activity during the swing phase. The synergy structure in patients with SKG after stroke was simplified, with specific abnormalities in synergy activities. SKG may arise from several synergy alterations involving multiple muscles, indicating that approaches focused on controlling individual muscle activities are unsuitable.

Keywords Hemiparetic, Kinematics, Electromyography, Muscle coordination, Walking

Stiff knee gait (SKG) is a common occurrence after stroke, characterized by reduced flexion of the knee joints during the swing phase, which decreases foot clearance and subsequently increases the risk of falls[1–3]. In SKG, compensatory movements, such as ipsilateral hip circumduction or contralateral vaulting, occur to clear the foot during the swing phase, resulting in increased vertical displacement of the center of body mass and greater energy expenditure[3,4]. Hence, SKG is a major factor that increases the risk of falls while walking in patients with stroke and represents an important therapeutic target to improve daily life[5]. Owing to upper motor neuron damage after a stroke, the rectus femoris exhibits improper activity with spasticity[1]. Rectus femoris hyperactivity related to muscle activity control disorders, such as stroke and Parkinson's disease, causes excessive knee extension moments, which are primarily observed during the initial preswing phase and possibly during the early and midswing phases[6]. Therefore, SKG has been widely reported to be caused by overactivity of the rectus femoris muscle during forward leg movement[4–8].

SKG treatment focuses on decreasing quadriceps activity during the swing phase. Administering botulinum toxin (BoNT) to the rectus femoris as a treatment for SKG has been extensively reported[1,3,9]. However, after this treatment, the knee flexion angle increases only by < 10° on average during the swing phase[1,3,9]. Therefore, 45%–80% of BoNT treatments to relieve SKG could be inappropriate[10]. In our previous study[5], rectus femoris activity was suppressed by pedaling, but the knee joint angle improved by approximately 5°, which is similar to previous studies[1,3] focusing on BoNT application to the rectus femoris. The effect sizes of treatments in these studies were small–medium, suggesting that treatments targeting the inhibition of rectus femoris activity do not dramatically improve SKG.

During the preswing phase, the rectus femoris muscle is stretched because the knee begins to flex in preparation for toe-off, although the maximum extension of the hip joint is included. The presence of elevated improper quadriceps activity after a stroke is a misinterpretation at the spinal level of sensory (e.g., group I and II afferents) information, originating from hip proprioceptors[11]. At this time, vastus lateralis activity increases because of the multijoint heteronymous excitatory reflexes induced by hip extension, also causing SKG[11]. In our previous study

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examining patients with hemiplegia exhibiting SKG[12], vastus lateralis activity was more strongly associated with decreased knee flexion angle compared to rectus femoris activity. Ankle plantar flexor activity is also involved in SKG development. The gastrocnemius muscle contributes most to the increase in knee flexion velocity during the preswing phase[13]. Insufficient knee flexion velocity at toe-off contributes to kinematic abnormalities that reduce the knee flexion angle during the swing phase[14]. Given that the kinematic chain of the ankle and knee plays a substantial role in the push-off[9,15], ankle plantarflexor activity is also related to SKG occurrence. Furthermore, in patients with stroke, the knee flexion angle increases during the swing phase when functional electrical stimulation is applied to the hamstrings[16], and among the average amplitudes of leg muscles during gait phases, the biceps femoris during the swing phase has the strongest positive correlation with walking speed[17]. Thus, hamstring activity may also be associated with SKG development.

In summary, various muscles are involved in SKG development, and SKG is possibly caused by the interaction of multiple muscles rather than the influence of a single muscle. For example, in patients with stroke, changes in the heteronymous spinal pathway lead to a simultaneous increase in the involuntary activity of the vastus lateralis and the soleus[18]. Additionally, voluntary hip abduction and knee flexion are coupled in patients with stroke in the static position, thereby imitating paretic foot-off[19]. To validate the association between such involuntary intermuscular interactions and occurrence of SKG, a novel experimental approach is required, which investigates the use of the locomotor strategy during walking in individuals with hemiplegia. Recently, an alternative approach to analyzing muscle activity synergies has been used as an innovative method to study motor control in humans[20]. Muscle synergy is the coordinated recruitment of a set of muscles to perform a deliberate movement. Factorization leads to the computation of muscle synergy vectors (W) and temporal activation patterns (C)[20]. In healthy individuals, muscle activities during comfortable walking consist of four independent synergies: hip and knee extensors (Module 1), ankle plantarflexors (Module 2), hip flexors and ankle dorsiflexors (Module 3), and knee flexors (Module 4)[21]. However, modular control is reorganized upon disinhibition and/or hyperexcitation of the brainstem descending pathways and intraspinal motor networks[22]. Normal modules merge after stroke, resulting in fewer modules and suggesting reduced independence of neural control signals[23]. Patients with fewer muscle modules after stroke have greater asymmetry in kinematic parameters such as knee flexion/extension[24]. In addition, the normal four modules in patients who had stroke exhibit a merging of Modules 1 and 2, 1 and 4, or 3 and 4[25,26]. Specifically, Module 3, which acts at the early swing phase, and Module 4, which acts at the terminal swing phase, control the swing of the ipsilateral leg[27]. Thus, the merging of these two synergies may cause an abnormal movement pattern during the swing phase[26].

Therefore, defects in neural signal-descending pathways after stroke alter muscle synergy and affect kinematic patterns. As mentioned in the previous text, the occurrence of SKG is attributed to the abnormal activities of several individual muscles. However, despite various studies on the occurrence of SKG, reports regarding the muscles with the most significant influence have contrasting results. Thus, the most important cause remains unclear. SKG may be attributed to abnormalities in the synergy activities involving multiple muscles, and not to the activity of individual muscles. Hypothetically, in patients with SKG, there may be a simplification of the synergy structure and alterations in the activity patterns and muscle combinations of specific modules that are active during the early swing phase. Hence, this study aimed to elucidate the muscle synergy characteristics during walking in patients presenting with SKG after stroke and to contribute to understanding the causes of SKG occurrence and the development of its treatment methods.

Methods

Study design and participants

This cross-sectional study used baseline data from clinically registered randomized controlled trials (clinical trial no.: UMIN000034270) and additional experimental data to alleviate sample size shortages. Patients aged 40–75 years residing in the same region, who experienced stroke between September 2018 and December 2022, and who are receiving regular physical therapy at the same hospital for chronic stroke sequelae were enrolled in this study. In addition, healthy adults who were age-matched to the abovementioned patients were recruited from the public.

The inclusion criteria were as follows: (i) unilateral cerebral lesions; (ii) at least 6 months since the stroke onset; and (iii) ability to walk independently or under supervision. The exclusion criteria were as follows: (i) decreased knee joint range of motion (ROM) at rest (subjects must be outside the range of 10°–100°); (ii) lower-limb joint surgery history; (iii) ankle foot orthosis requirement to walk independently; and (iv) Mini-Mental State Examination score < 24 points[5]. Out of 53 participants with stroke, 17 were excluded, finally enrolling 36 patients and 15 healthy adults (Table 1). The study was conducted in accordance with the Declaration of Helsinki, and the protocol was approved by the Ethics Committee of Nittazuka Medical Welfare Center (approval no.: 2023–33). We obtained written informed consent from all participants before starting the study.

Experimental setup and protocol

A straight walkway (16 m) was built to measure the walking distance (including 3 m extra at each end). We set up a video camera (HD Pro Webcam; Logicool, Inc., Tokyo, Japan) with a 30-Hz sampling frequency 5 m lateral to the midpoint of the walkway and drew a 1-m line at the midpoint. Participants walked at a comfortable speed, and those who regularly used a cane were allowed to use it during the experiment. Three examiners conducted all the experiments.

Kinematic data during gait were recorded using MyoMotion (Noraxon Inc., Scottsdale, AZ). MyoMotion inertial sensors were placed according to the lower-limb rigid-body model with seven joint segments used in the MR3 software (Noraxon Inc.), positioned on the shoes (top of the upper foot), frontal on the shanks, frontal on the thighs, and bony area of the sacrum[5]. Patients were positioned upright, and calibration was done to determine

		Healthy	Non-SKG	SKG
Age		62.0 (1.0)	57.2 (2.9)	62.3 (1.6)
Height	(cm)	166.3 (2.6)	169.9 (2.0)	168.0 (2.1)
Weight	(kg)	65.6 (3.4)	71.4 (3.7)	66.3 (2.1)
Sex	(Female/male)	4/11	3/13	4/16
Stroke type	(CI/ICH)	N/A	5/11	4/16
Months since the onset		N/A	50.4 (12.4)	55.1 (10.7)
Paretic side	(L/R)	N/A	5/11	5/15
Assistive device	(None/T-cane)	15/0	12/4	11/9
Fugl–Meyer Assessment LE		34.0 (0)	24.4 (0.7)	19.9 (0.9)
Modified Ashworth scale	Hip flexors	0 (0)	1 (1)	2 (2)
	Knee extensors	0 (0)	1 (1)	2 (2)
	Ankle plantar flexors	0 (0)	2 (1.25)	2.5 (2)

Table 1. Characteristics of each group. Values are expressed as mean (standard error) or as median (interquartile range). A Modified Ashworth scale score of 1 + was assigned as 2, and scores of ≥ 2 were revised upward by 1. *SKG* stiff knee gait, *CI* cerebral infarction, *ICH* intracerebral hemorrhage, *L* left, *R* right, *LE* lower extremity.

the 0° angle value in the considered joints. Then, the examiners manually fixed the participants' knee joints to obtain the maximum extension angle. At that time, the hip and ankle joint angles were adjusted to 0° ± 5°. We set the sampling frequency for the inertial sensors at 100 Hz.

TELEmyo DTS (Noraxon Inc.) was used for electromyography (EMG) recording during gait. The sampling frequency was 1,500 Hz, and the bandpass filter was set at 10–500 Hz. Then, the following muscles on the body's paretic side underwent EMG measurement: rectus femoris, vastus lateralis, biceps femoris, tibialis anterior, medial head of the gastrocnemius, and soleus. Next, skin impedance was decreased to ≤ 10 kΩ using alcohol-soaked cotton swabs and an abrasive cream. Furthermore, Ag–AgCl electrodes (EM-272; Noraxon Inc.) were placed in the positions recommended by the Surface ElectroMyoGraphy for the Noninvasive Assessment of Muscle project[28]. To identify the gait phase, we placed foot switches (Noraxon Inc.) on the soles of both feet (each foot = 4 points)[29]. MyoSync and Sync Light devices (Noraxon Inc.) were used to synchronize all instruments and ensure time frame alignment.

Data analysis

The kinematic and EMG waveforms were analyzed using MR3 (version 3.14). First, the initial ground contact on the paralyzed side was identified according to the acceleration information from MyoMotion and the potential signal input from the foot switch. Then, 10 continuous gait cycles (GCs) were extracted as the analysis intervals; if the extraction failed, two data points from five separate GCs were combined. Next, the duration of each GC was normalized after considering one GC to be 100%. We obtained the arithmetic mean of the 10 GCs and calculated 1000-point amplitudes at a 0.1% interval. We identified each gait phase (loading response, single-support, preswing, and swing phases) from foot switch data on both sides. The early swing phase was defined as the interval from the toe-off to the peak knee flexion angle during the swing[30]; if the knee flexion waveform was bimodal, it was defined up to the first peak. We then calculated kinematic parameters, such as the angle and angular velocity, of the knee, hip, and foot related to the swing. Stride length was measured using still images obtained from video data when patients passed through the intermediate points on the walkway[29]. In addition, we calculated the cadence and GC durations using the gait event data and the gait velocity using the measured stride length and cadence.

The raw EMG waveforms of 10 GCs exported from MR3 were imported into MATLAB R2021b (MathWorks, Inc., Natick, MA, USA) to analyze muscle synergy. First, the waveform was passed through a second-order high-pass Butterworth filter (35 Hz) and then full-wave rectified. Next, it was passed through a zero-lag second-order low-pass Butterworth filter (4 Hz), obtaining a linear envelope. Subsequently, each individual EMG channel was normalized in amplitude based on its respective peak activity. Each GC's duration was time-normalized after considering one GC to be 100%.

Processed EMG data were combined using the following formula: m (number of muscles = 6) × t matrix (t = 10 strides × 100 points per GC)[29]. We then used non-negative matrix factorization (NNMF) to derive muscle synergies ($W_{m \times n}$) and the time-varying activation of those synergies ($C_{n \times t}$). In this study, W is the relative weight of each muscle within each synergy, and n is the specified number of modules from 1 to 5 (as this study targets six muscles). The NNMF algorithm was iteratively optimized until it converged on W and C, minimizing the error. To quantify how accurately the derived muscle synergies described the original set of EMG signals, we calculated the total variance accounted for (tVAF) by a given number of synergies[31]. We also calculated the VAF for each muscle to determine the minimum number of modules required to reconstruct the original EMG waveform. The individual's module count was determined based on the condition that the VAF for each muscle was over 90%, and increasing the number of modules further did not result in a VAF increase by more than 5%[23,26].

Statistical analysis

We identified those in the SKG group who had three or more of the following four indicators that were below two standard deviations (SDs) from the mean of control values: peak knee flexion during the early swing phase, knee ROM during the early swing phase, total knee ROM, and duration between toe-off and peak knee flexion during the early swing phase[5,7]. For the control values, we used the data of adults without SKG and stroke history, employing the k-means clustering algorithm for poststroke gait kinematics in a previous study[32] (according to the following mean [SD] values of the four indicators mentioned above: 55.1° [7.6], 25.7° [7.3], 59.4° [7.7], and 13.0%GC [2.5], respectively).

For each group, we pooled the weights (W) and corresponding activations (C) of all participants when they had four modules, which were calculated by NNMF and sorted according to the W values obtained through k-means cluster analysis (1,000 max iterations, $k = 4$)[31]. For those with two modules assigned to the same cluster, we changed the assignment to minimize the sum of the distances from the cluster center. The timing of the peak amplitude of C, the average amplitude of C duration of the single-support phase and the preswing-to-early swing phase, and the Ws of each muscle were compared between the three participant groups using the Kruskal–Wallis test and post hoc tests (Bonferroni correction). VAF, kinematics, and spatiotemporal parameters during walking were also compared between these groups using one-way analysis of variance and post hoc tests (Bonferroni correction). Furthermore, the effect size (r) was calculated and compared between such groups[33]. Given that walking speed or motor paralysis may affect muscle synergies[23,34], we conducted an analysis of covariance using gait velocity (healthy vs. non-SKG vs. SKG) or lower-limb Fugl–Meyer assessment (FMA) score (non-SKG vs. SKG) as covariates. All statistical data were analyzed using SPSS version 25 (IBM Co., Ltd., Armonk, NY, USA) and BellCurve for Excel version 3.20 (Social Survey Research Information Co., Ltd., Tokyo, Japan), with $P < 0.05$ indicating statistical significance. However, many of the variables calculated by NNMF were relative values, and much of the data was not normally distributed. Therefore, we imported the data into the Python package statsmodels and then conducted the Yeo–Johnson transformation[35] before performing covariance analysis.

Results

Muscle synergies

All participants (36 with stroke and 15 healthy adults) had no missing measurement data. Among the patients with a stroke history, 20 had SKG and 16 did not. All healthy adults had four modules determined by VAF. In the non-SKG group, 62.5% had four modules and 37.5% had three modules, whereas in the SKG group, 35%, 40%, and 25% had four, three, and two modules, respectively (Fig. 1). Activity C and weighting W of all participants were classified according to the number of modules they owned (Fig. 2). The tVAF of a single module in the SKG group was higher than that in the healthy group ($P < 0.001$; 95% confidence interval [CI], 13.236–22.184) and non-SKG group ($P = 0.010$; 95% CI 1.101–9.888) (Fig. 1). The SKG group also had higher tVAF from two modules than the healthy ($P = 0.002$; 95% CI 1.036–5.594) and non-SKG groups ($P = 0.003$; 95% CI 0.908–5.383). In three or more modules, no significant difference in tVAF was noted between the groups.

Figure 3 shows the mean waveform of C and the median of W for the synergy involving four modules in each group, encompassing all participants. At the peak timing of C in the four-module synergy, Module 2 was significantly earlier in the SKG group than in the healthy ($P < 0.001$, ES = 0.753) and non-SKG groups ($P = 0.031$,

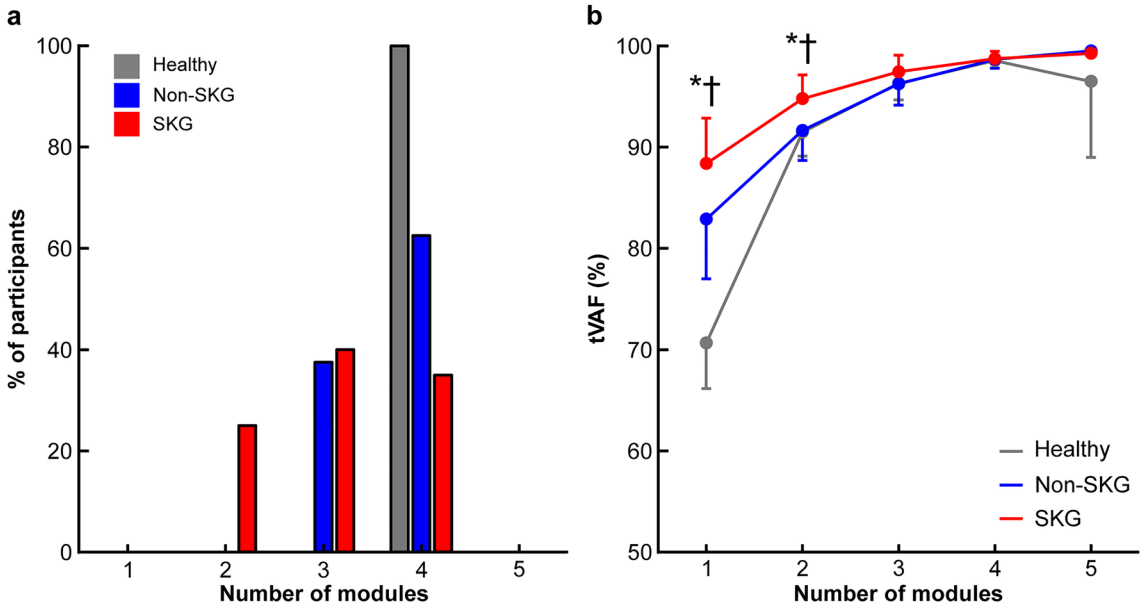

Fig. 1. Number of modules determined by the variance accounted for (VAF) each muscle (**a**) and the total VAF (tVAF) calculated for each module count (**b**). The tVAF is presented with the average and standard deviation for each group. Comparison of values between the groups: versus the healthy group, *$P < 0.05$; versus the non-SKG group, †$P < 0.05$ (Bonferroni test).

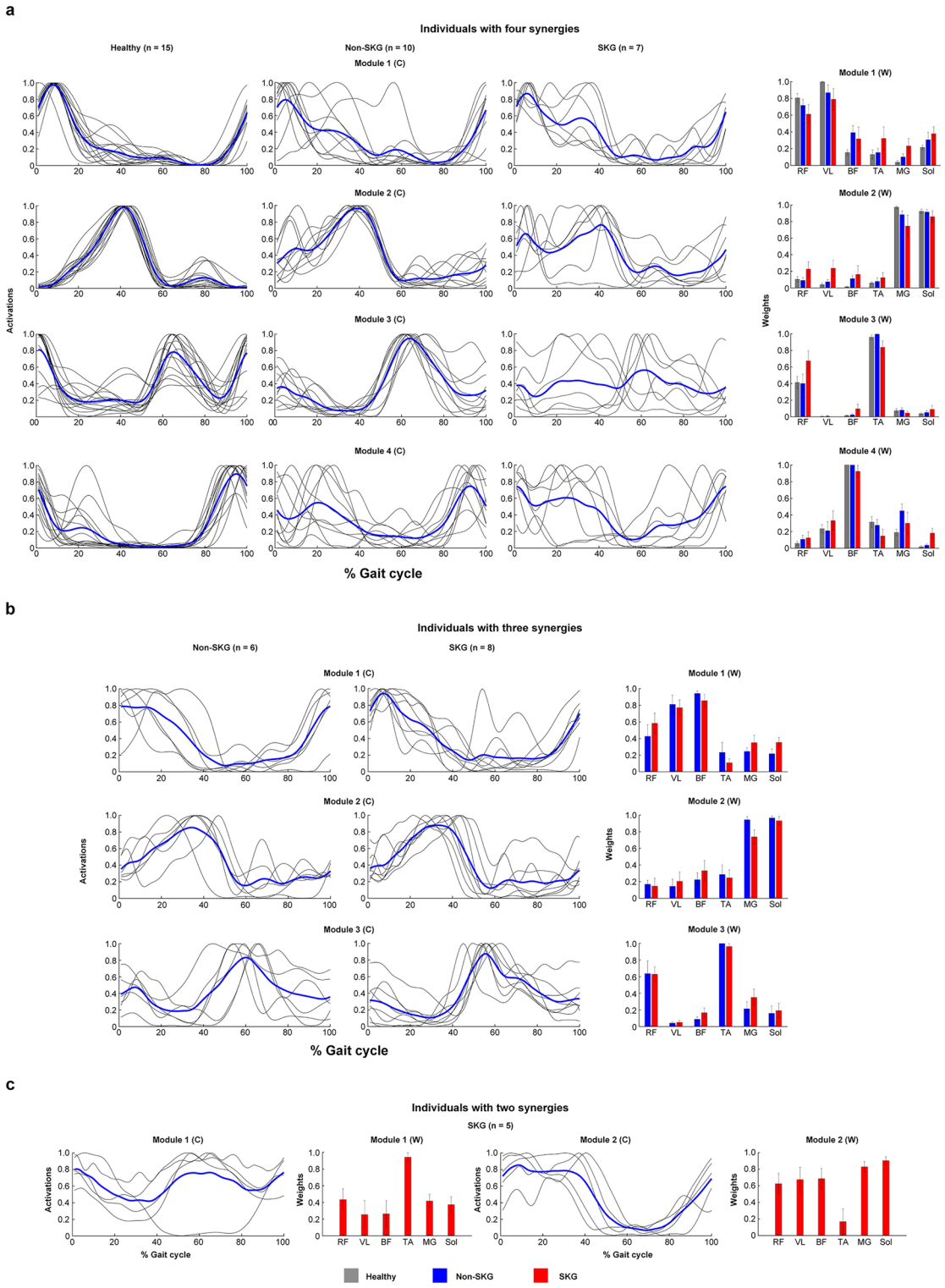

Fig. 2. Profiles of synergy activity (C) and muscle weights (W) for all participants classified by the number of modules determined by variance accounted for (VAF). The black line in the waveform represents individual activity, whereas the blue line indicates the average activity within the group. The weights (W) are presented with the average and standard error for each group. *RF* rectus femoris, *VL* vastus lateralis, *BF* biceps femoris, *TA* tibialis anterior, *MG* medial gastrocnemius, *Sol* soleus.

ES = 0.494) (Table 2). Module 3 in the SKG group was also significantly earlier than that in the non-SKG group (P = 0.022, ES = 0.588). However, the fourth module in the SKG group was significantly later than that in the healthy group (P = 0.024, ES = 0.435).

The mean amplitude of C for the synergy with four modules was significantly higher in Module 1 of the SKG group than in Module 1 of the healthy group during the single-support phase (P = 0.007, ES = 0.513) and the preswing-to-early swing phase (P = 0.025, ES = 0.479) (Table 2). Likewise, it was significantly higher in Module 2 of the SKG group than in Module 2 of the healthy group during the single-support phase (P = 0.026, ES = 0.344) and the preswing-to-early swing phase (P = 0.030, ES = 0.395). As for Module 3, the mean amplitude in the SKG group was significantly higher during the single-support phase (P = 0.039, ES = 0.383) but significantly lower during the preswing-to-early swing phase (P = 0.001, ES = 0.560) than that in the non-SKG group. In the fourth module, the SKG group exhibited significantly higher values during the single-support phase than the healthy

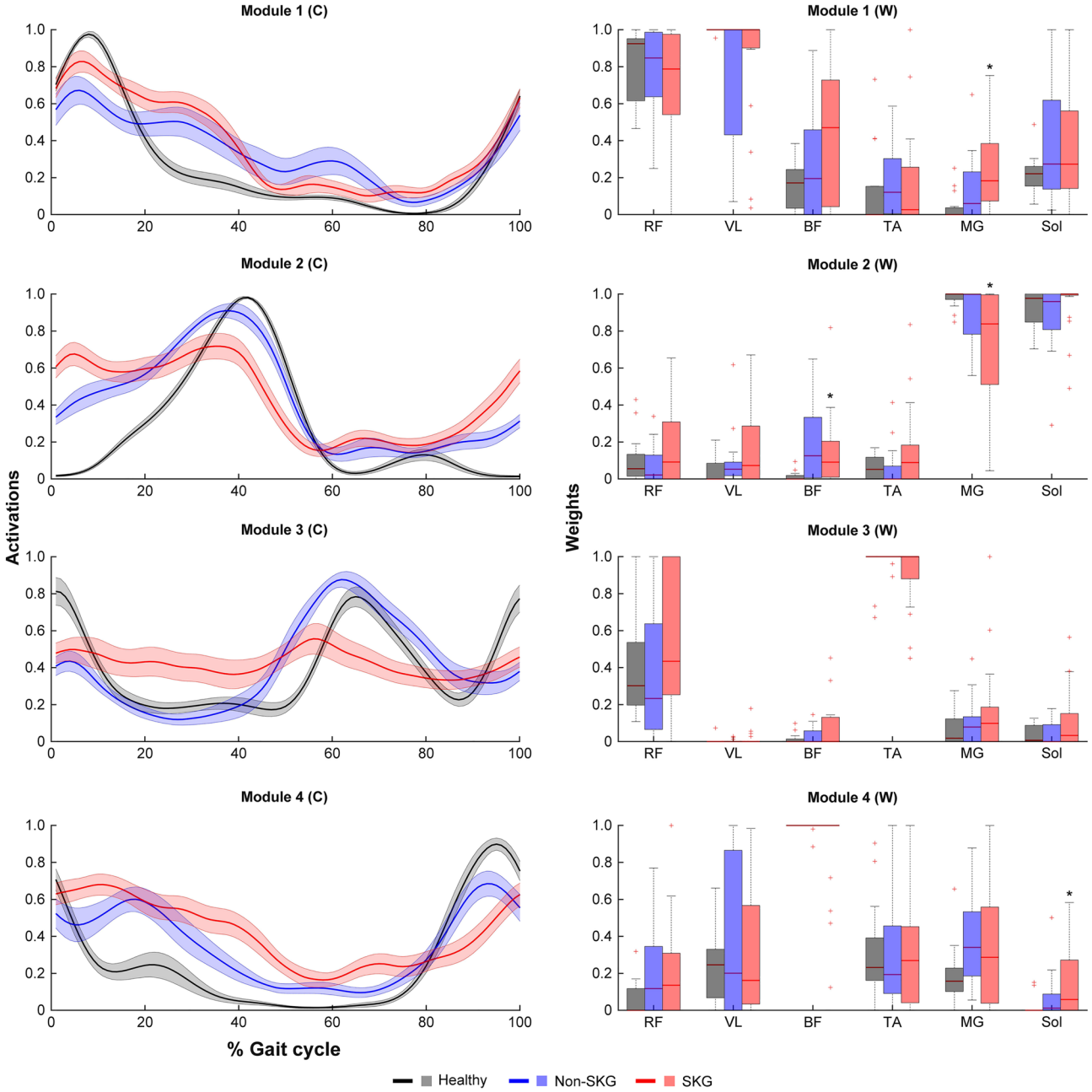

Fig. 3. Synergy activity (C) and muscle weights (W) for each group when all participants are composed of four modules. Synergy activations are plotted as the mean activation over the gait cycle. The shaded bar represents the standard errors. Synergy weights for each muscle are plotted as median and quartile. Comparison of values between the groups: versus the healthy group, *P < 0.05; versus the non-SKG group, †P < 0.05 (Bonferroni test). *RF* rectus femoris, *VL* vastus lateralis, *BF* biceps femoris, *TA* tibialis anterior, *MG* medial gastrocnemius, *Sol* soleus.

	Healthy		non-SKG		SKG		Effect size (*r*)	
							Healthy vs. SKG	Non-SKG vs. SKG
Timing of peak amplitude (%GC)								
Module 1	8.0	(2.0)	16.5	(32.5)	7.5	(13.0)	0.020	0.068
Module 2	41.0	(3.0)	38.5	(6.5)	24.5	(30.5)*†	0.753	0.494
Module 3	2.0	(62.5)	62.0	(5.0)*	47.0	(37.0)†	0.303	0.588
Module 4	94.0	(4.0)	104.5	(23.5)	110.5	(23.3)*	0.435	0.167
Mean amplitudes (%)								
Module 1 SS	23.7	(12.1)	38.8	(41.4)	53.6	(44.1)*	0.513	0.210
Module 1 PSw–ESw	7.1	(5.4)	12.6	(49.0)	18.9	(15.3)*	0.479	0.011
Module 2 SS	59.2	(5.6)	75.7	(8.5)*	75.3	(27.3)*	0.344	0.070
Module 2 PSw–ESw	16.3	(7.6)	30.2	(13.0)*	24.8	(31.3)*	0.395	0.017
Module 3 SS	16.4	(12.9)	13.4	(11.3)	28.9	(58.0)†	0.226	0.383
Module 3 PSw–ESw	61.9	(12.4)	71.3	(15.4)*	53.0	(33.2)†	0.203	0.560
Module 4 SS	6.7	(15.2)	34.8	(41.9)*	50.7	(42.9)*	0.716	0.286
Module 4 PSw–ESw	1.6	(2.0)	11.3	(13.2)*	24.0	(18.3)*†	0.817	0.469

Table 2. Comparisons of synergy activations composed of four modules among the non-SKG, SKG, and healthy (control) groups. Values are expressed as median (interquartile range). *SKG* stiff knee gait, *SS* single support, *PSw* preswing, *ESw* early swing, *GC* gait cycle. Comparison of values between the groups: versus the healthy group, $^{*}p < 0.05$; versus non-SKG, $^{\dagger}p < 0.05$ (Bonferroni test). Effect size, $r = 0.10$ (small); $r = 0.30$ (medium); $r = 0.50$ (large).

group (P < 0.001, ES = 0.716) and during the preswing-to-early swing phase than both the healthy (P < 0.001, ES = 0.817) and non-SKG groups (P = 0.033, ES = 0.469).

Moreover, the W of the gastrocnemius for the synergy with four modules was significantly higher in Module 1 of the SKG group than that of the healthy group (P = 0.001, ES = 0.593) (Fig. 3). In Module 2, it was significantly higher in the biceps femoris (P = 0.006, ES = 0.554) and significantly lower in the gastrocnemius (P = 0.009, ES = 0.498) of the SKG group than those of the healthy group. In the fourth module, the soleus of the SKG group was significantly higher than that of the healthy group (P = 0.006, ES = − 0.503).

In the analysis of covariance with walking speed as a covariate, the regression may be assumed to be significant and linear for the W of the gastrocnemius in Module 3 of the synergy with four modules; however, no significant differences were observed among the three participant groups. When the FMA score is used as a covariate for the W of the soleus in the fourth module of the synergy consisting of four modules, a significant and linear regression may be assumed. However, no significant differences were observed between the SKG and non-SKG groups. Regardless of using walking speed or the FMA score as covariates, assuming the significance and linearity of the regression for variables related to all other synergies is not possible.

Kinematic parameters

The SKG group demonstrated significantly smaller knee joint parameters than the healthy group. These parameters included the flexion angle at toe-off (P < 0.001; 95% CI 19.245–29.171), peak flexion angle at early swing (P < 0.001; 95% CI 37.832–46.025), ROM during early swing (P < 0.001; 95% CI 15.280–20.174), ROM during GC (P < 0.001; 95% CI 36.533–45.873), duration between toe-off and peak flexion (P < 0.001; 95% CI 5.681–7.852), and flexion velocity at toe-off (P < 0.001; 95% CI 264.425–330.878). In the hip joint, the SKG group also exhibited a smaller peak extension angle during stance (P < 0.001; 95% CI 7.144–13.901), extension angle at toe-off (P < 0.001; 95% CI 5.219–11.736), and flexion velocity at toe-off (P < 0.001; 95% CI 122.485–183.180). At the ankle joint, the plantar flexion angle at toe-off (P < 0.001; 95% CI 4.933–15.879) and plantar flexion velocity at toe-off (P < 0.001; 95% CI 82.249–161.263) were smaller in the SKG group than in the healthy group (Table 3).

Compared with the non-SKG group, the SKG group displayed significantly smaller values for various parameters. In the knee joint, these parameters included the flexion angle at toe-off (P < 0.001; 95% CI 7.153–17.279), peak flexion angle at early swing (P < 0.001; 95% CI 19.199–28.146), ROM during early swing (P < 0.001; 95% CI 6.948–15.984), ROM during GC (P < 0.001; 95% CI 21.014–30.979), duration between toe-off and peak flexion (P < 0.001; 95% CI 3.287–7.619), and flexion velocity at toe-off (P < 0.001; 95% CI 120.680–199.229). In the hip joint, values for peak extension angle during stance (P = 0.002; 95% CI 1.972–8.310) and flexion velocity at toe-off (P < 0.001; 95% CI 70.088–129.078) were smaller in the SKG group than in the non-SKG group. Additionally, at the ankle joint, plantar flexion velocity at toe-off (P = 0.006; 95% CI 19.638–135.295) was smaller in the SKG group than in the non-SKG group (Table 3).

Spatiotemporal parameters

Compared with the healthy group, the SKG group exhibited significantly smaller gait velocity (P < 0.001; 95% CI 0.689–0.915), cadence (P < 0.001; 95% CI 24.385–40.545), single-support phase duration (P < 0.001; 95% CI 9.071–13.883), paretic-side step length (P < 0.001; 95% CI 23.719–36.350), nonparetic-side step length (P < 0.001; 95% CI 27.658–41.679), and stride length (P < 0.001; 95% CI 51.691–77.713). Conversely, the durations of the loading response phase (P = 0.005; 95% CI − 5.710 to − 1.148), preswing phase (P < 0.001; 95% CI − 7.797

			Healthy		Non-SKG		SKG		Effect size (*r*)	
									Healthy vs. SKG	non-SKG vs. SKG
Knee	Peak extension angle at stance	(degree)	− 1.7	(0.8)	− 1.3	(1.0)	− 1.0	(1.0)	0.066	0.183
	Flexion angle at toe-off	(degree)	47.6	(1.8)	35.6	(1.9)*	23.4	(1.6)*†	0.866	0.644
	Peak flexion angle at ESw	(degree)	67.9	(1.2)	49.7	(1.6)*	26.0	(1.5)*†	0.964	0.880
	Range of motion during ESw	(degree)	20.4	(2.7)	14.1	(1.2)*	2.7	(0.5)*†	0.932	0.793
	Range of motion during GC	(degree)	66.2	(1.3)	51.0	(1.4)*	25.0	(1.9)*†	0.954	0.877
	Duration between toe-off and peak flexion	(%GC)	10.0	(0.2)	8.7	(0.9)	3.3	(0.5)*†	0.928	0.739
	Flexion velocity at toe-off	(degree/s)	382.8	(13.4)	245.1	(17.7)*	85.1	(10.0)*†	0.954	0.818
Hip	Peak extension angle during stance	(degree)	15.3	(1.4)	10.0	(1.2)*	4.8	(1.0)*†	0.741	0.493
	Extension angle at toe-off	(degree)	5.0	(1.2)	− 2.5	(2.0)*	− 3.4	(1.1)*	0.678	0.076
	Flexion velocity at toe-off	(degree/s)	192.3	(13.4)	139.0	(13.1)*	39.4	(5.5)*†	0.926	0.843
Ankle	Plantar flexion angle at toe-off	(degree)	13.2	(1.8)	4.9	(2.0)*	2.8	(1.8)*	0.560	0.134
	Plantar flexion velocity at toe-off	(degree/s)	186.0	(64.2)	141.7	(15.9)	64.2	(11.7)*†	0.739	0.516

Table 3. Comparisons of the kinematics data between the non-SKG, SKG, and healthy (control) groups. Values are expressed as mean ± standard error. *SKG* stiff knee gait, *PSw* preswing, *ESw* early swing, *GC* gait cycle. Comparison of values between the groups: versus the healthy group, $*p < 0.05$; versus non-SKG, $^{\dagger}p < 0.05$ (Bonferroni test). Effect size, $r = 0.10$ (small); $r = 0.30$ (medium); $r = 0.50$ (large).

to − 2.454), and swing phase (P < 0.001; 95% CI − 5.863 to − 1.685) were significantly longer in the SKG group than in the healthy group (Table 4).

Compared with the non-SKG group, the SKG group demonstrated significantly smaller gait velocity (P < 0.001; 95% CI 0.199–0.428), cadence (P < 0.001; 95% CI 14.253–31.301), single-support phase duration (P < 0.001; 95% CI 2.399–8.270), paretic-side step length (P = 0.006; 95% CI 2.957–15.923), nonparetic-side step length (P = 0.006; 95% CI 3.599–19.882), and stride length (P = 0.003; 95% CI 7.952–34.409) (Table 4).

Discussion

To elucidate the characteristics of muscle synergy during walking in patients who developed SKG after a stroke, this study employed stricter methods to identify such patients. In the SKG group, the average maximum knee flexion angle during the early swing was 26° (standard error = 1.5), which is more than 10° smaller than that in previous studies targeting SKG[1,6–8]. Additionally, all knee joint kinematics, including ROM and flexion velocity, from our participants with stroke were similar to the data range of SKG classified by the k-means clustering algorithm from gait kinematics data[32]. A representative study analyzed muscle synergy in the gait of healthy individuals and identified four modules from eight lower-limb muscles[21,36]. Using a methodology similar to Acuña et al.[31], we computed modules focusing on six lower-limb muscles and identified four modules in healthy individuals; these modules comprised activity patterns and muscle groups similar to those in previous studies targeting eight muscles[21,23]. These four modules were as follows: activities of hip and knee extensors in the early stance (Module 1), activities of ankle plantar flexors in the late stance (Module 2), activities of hip flexors and ankle dorsiflexors in the early swing (Module 3), and activities of knee flexors in the late swing (Module

		Healthy		non-SKG		SKG		Effect size (*r*)	
								Healthy vs. SKG	non-SKG vs. SKG
Gait velocity	**(m/s)**	**1.30**	**(0.04)**	**0.81**	**(0.04)***	**0.49**	**(0.04)*†**	**0.929**	**0.691**
Cadence	(steps/min)	111.9	(2.2)	102.2	(2.8)	79.4	(3.0)*†	0.819	0.682
LR period	(%GC)	12.9	(0.4)	13.6	(1.0)	16.3	(1.0)*	0.525	0.313
SS period	(%GC)	37.3	(0.5)	31.2	(0.9)*	25.8	(1.1)*†	0.889	0.536
PSw period	(%GC)	12.6	(0.5)	15.3	(0.9)	17.8	(1.2)*	0.618	0.263
Sw period	(%GC)	37.3	(0.5)	39.9	(1.0)	41.0	(0.9)*	0.571	0.140
Step length (Ps)	(cm)	69.8	(2.2)	49.2	(2.4)*	39.8	(2.1)*†	0.860	0.453
Step length (NPs)	(cm)	70.3	(2.1)	47.3	(2.9)*	35.6	(2.7)*†	0.870	0.450
Stride length	(cm)	140.1	(4.3)	96.6	(4.6)*	75.4	(4.5)*†	0.870	0.488

Table 4. Comparisons of the spatiotemporal parameters between the non-SKG, SKG, and healthy (control) groups. Values are expressed as the mean ± standard error. *LR* loading response, *SS* single support, *PSw* preswing, *Ps* paretic side, *NPs* nonparetic side, *GC* gait cycle. Comparison of values between the groups: versus the healthy group, $*p < 0.05$; versus non-SKG, $^{\dagger}p < 0.05$ (Bonferroni test). Effect size, $r = 0.10$ (small); $r = 0.30$ (medium); $r = 0.50$ (large).

4). Therefore, although this study conducted a synergy analysis with a relatively small number of muscles, the characteristics of muscle synergy among the participants were well understood.

The number of modules in the SKG group decreased compared with that in the healthy and non-SKG groups. In addition, the SKG group had a higher tVAF than the other groups, suggesting that the synergy structure was simplified in the SKG group. In covariance analysis, walking speed or FMA score did not affect the synergy variables. Therefore, in patients with SKG, the synergy structure may be specifically simplified regardless of the decline in walking ability or motor function. The disinhibition of brainstem descending pathways (reticulospinal tract [RST] and vestibulospinal tract [VST]) is particularly associated with the reorganization of simplified modular control and spastic synergistic activation following poststroke conditions[22]. These RSTs and VSTs are disinhibited because of corticoreticular pathway damage[37], suggesting that the hyperexcitability of the extrapyramidal system is more related to the simplified synergy structure in patients with SKG compared to muscle weakness or paralysis caused by reduced descending drive from the corticospinal pathway.

Module 1 of the four-component synergy plays a role in weight acceptance at the early stance through the activity of the knee extensors[21,23]. In this study, the average amplitudes of activity C of Module 1 in the single-support phase and from the preswing-to-early swing phase were significantly higher in the SKG group than in the healthy group. Therefore, the activity of the knee extensor operating in the loading response phase may be maintained thereafter. When the control of the corticospinal pathways is impaired due to stroke, increased reliance on more diffuse output pathways, such as the RST and VST, may lead to abnormal coupling in joint torque generation[23,38]. Consequently, the extensor module may be excessively generated, leading to the decreasing independence of module propulsion and/or deceleration of the leg from the weight acceptance module[23]. In all groups in this study, the primary muscle constituting Module 1 was the lateral vastus. Among the quadriceps, the vastus lateralis and rectus femoris contribute the most to the knee extension moment[39], and the cross-sectional area of the vastus lateralis is the largest of the quadriceps in healthy individuals and those with stroke[40]. Therefore, the extended activity observed in Module 1 in the SKG group may have led to the generation of higher knee extension moments resulting from the inappropriate timing of discharge in the knee extensor. Another potential scenario is that when Module 1 merges with Module 4, its activity initiates earlier in the swing phase, thereby altering its contribution to the swing of the paralyzed leg to further accelerate it[27]. Focusing on Module 1 in individuals with stroke characterized by a synergy composed of three modules, we observed a high incidence of merging between Modules 1 and 4 of the synergy composed of four modules in healthy individuals (Fig. 2b). Hence, when Module 1, acting as the weight acceptance module, merges with the module involved in the swing phase, knee extension might be exaggerated during the swing and knee flexion involvement may be inhibited.

Module 2 of the four-module synergy is composed of the ankle dorsiflexor muscles, which provide support to the body in the late stance phase and forward propulsive force[21,23]. In Module 2 of the SKG group, the peak timing of the activity C amplitude was early and the W of the gastrocnemius was small. Biarticular plantar flexors are important for swing initiation and energy transfer to the leg during preswing[27]. However, when Module 2 merges with Module 1, the push-off energy is compromised, impairing leg swing. Thus, the gastrocnemius contributes the most to the increase in knee flexion velocity during the early swing phase[13] and the lack of push-off is involved in SKG occurrence[9]. In our study, five individuals with SKG only had two modules (Fig. 2c) as well as a merging of Modules 1 and 2; this merging might result in knee flexion inhibition during swing because of the impact of push-off deficiency. In individuals with SKG exhibiting three modules (Fig. 2b), Module 2, displaying an increase in amplitude during the late stance, might function independently, considering is serves as a module solely for ankle plantar flexors due to the low weighting of the vastus lateralis. Instead, the merging of Modules 1 and 4 may have a greater impact on SKG occurrence.

Module 3 of the four-module synergy contributes to leg acceleration by the rectus femoris and foot clearance by the tibialis anterior during the early swing phase[21,23]. In Module 3 of the SKG group, the average amplitude of activity C during single support was high. Boudarham et al.[6] reported that in patients with SKG, rectus femoris overactivity is mainly observed in the early preswing phase and it may persist until the early or midswing phase. In the SKG group of our study, rectus femoris, a major constituent muscle of synergy 3, showed higher activity in the single-support phase than that in previous studies. The prolonged activity of the knee extensors in patients after a stroke may be a compensatory mechanism used to overcome insufficient plantarflexor strength and provide additional body support[27]. In a study involving patients with cerebral palsy who have spasticity, synergy composed of the rectus femoris and tibialis anterior was more activated during late stance[41]. In spastic muscles, synergy is activated during the stance phase for hip and knee extension; subsequently, these joints cannot be bent for foot clearance[22]. In Module 3, the mean amplitude of active C during the preswing-to-early swing phase was lower in the SKG group than in the non-SKG group, with minimal fluctuations in amplitude within one cycle, thereby indicating an overall absence of on–off activity. In other words, the limitation of knee flexion may not be caused by an increased activity in Module 3, which includes the rectus femoris, but rather by the activity in Module 3 starting and persisting earlier than originally planned. Moreover, the merging of Modules 3 and 4 in patients with stroke may restrict the angle of lower-limb flexion[26]. However, in the SKG group of our study, individuals with the merging of Modules 3 and 4—indicating high activity throughout the swing phase and significant weights on the rectus femoris, tibialis anterior, and biceps femoris—were scarcely found (Fig. 2b and c).

In summary, the SKG group demonstrated several synergy alterations that appear to be associated with deficits in knee flexion during walking. Given the modular neuromuscular framework related to walking biomechanics[24], muscle synergy analysis may be particularly useful in identifying the causes of abnormal gait occurrences. In healthy individuals, the accuracy of foot clearance during the swing phase is highly reproducible[42], as determined by the direct activation of the lower-limb muscles and the indirect coordinated activation between the limbs and trunk[22]. Considering that the combinations of joint kinematics to achieve a certain objective are infinite[43], efficient neural control may be necessary, especially in movements that require repeated high levels of reproducibility (e.g., walking).

This study has several limitations. Synergy analysis is often conducted by measuring the EMG of eight or more muscles. However, on the basis of previous studies[30,44], we conducted synergy analysis using six muscles, which excluded the gluteus medius and medial hamstrings. There is no evidence linking these muscles, which act on hip internal rotation, to SKG. Based on previous research, this study conducted a synergy analysis using 10 gait cycles[21,23,36]. Recent studies have adopted 20 gait cycles[26], and this method may have provided more stable muscle synergy estimates. Moreover, the muscle weighting of modules did not considerably differ between the SKG and control groups. The previous study[31] that conducted the cluster analysis we referred to was based on weighting muscle synergies. If the clusters are based on combined weighting and activation, different conclusions might be obtained. However, in previous studies[23,45], the weighting of muscle modules during walking was strikingly similar between the healthy and poststroke groups. Therefore, alterations in poststroke synergy activity may be more evident in activity patterns than in weighting. Further, this study did not consider follow-up experiments over time. Due to chronic disuse of limbs and central nervous system plastic reorganization over time, muscle overactivity may occur[46]. Thus, the kinesiology and etiology of SKG can vary based on the duration from stroke onset. Moreover, strategies that address incomplete follow-up were not included in this study. Finally, the specific alterations in some synergies in the SKG group that contribute the most to the onset of SKG remain unclear. Further increasing the sample size and conducting a multivariate analysis are necessary to clarify this.

Conclusion

Individuals exhibiting SKG showed simplified muscle synergy structures and had specific synergy alterations that may be associated with SKG occurrence. The early stance module, which includes the knee extensors, exhibited increased activity during the swing phase, and the late stance module, which includes the ankle plantarflexors, showed earlier timing of activity. In addition, the early swing module involving the hip flexors and ankle dorsiflexors demonstrated high activity during the single-support phase but exhibited lower activity during the swing phase, indicating a lack of on–off activity. SKG is probably triggered by several synergy alterations involving multiple muscles. Therefore, treatment approaches focused on controlling individual muscle activity may be unsuitable.

Data availability

All data generated or analyzed during this study are included in this published article.

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Competing interests

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Additional information

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