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A method to differentiate the causes of stiff-knee gait in stroke patients

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ABSTRACT

Stiff-knee gait (SKG) is a common abnormal gait pattern in patients after stroke characterized by insufficient knee flexion (KF) during swing. Overactivity of the rectus femoris (RF) is considered the primary cause of SKG. Inadequate push-off has been indicated as an additional cause in the recent literature, as KF depends on knee flexion velocity in preswing (KFV).

We used the peak of vertical acceleration of the malleolus (PMVA) as a kinematic-based indirect measure of push-off and studied its relationship with KF and KFV in a sample of 20 healthy subjects walking fast ($v = 95 \pm 5\%$height s^{-1}), at self-selected speed ($v = 74 \pm 5\%$height s^{-1}), slow ($v = 54 \pm 6\%$height s^{-1}) and very slow ($v = 38 \pm 5\%$height s^{-1}) and in a sample of 52 stroke patients with SKG (age 60 ± 11, $v = 20 \pm 11\%$height s^{-1}).

In healthy subjects PMVA occurred before knee flexion acceleration ($p < 0.001$) and hip flexion acceleration ($p < 0.001$). KF appeared as a bottom-up mechanism driven by the ankle push-off. From a regression analysis, the PMVA-KFV cause–effect relationship resulted strictly linear, with $R^2 = 0.967$, KFV = $0 + 7.1 \times$ PMVA, $P < 0.0001$.

Data from SKG patients were compared to this normal cause–effect model. For 44/52 patients the reduced KFV was combined with lack of push-off. Data from 8/52 patients only were statistically outside the 95%CI of the model, thus requiring for a braking mechanism to explain KFV reduction.

In stroke adults of our sample the push-off impairment (85% of cases) and not the inappropriate knee extension moment produced by the thigh muscles was the primary cause of SKG. This result could explain the low average efficacy ($<10°$) of focal and surgical treatments at the quadriceps.

The presented model could be used to differentiate the primary cause of SKG between inadequate push-off and braking activity of the thigh muscles, thus increasing the effectiveness of the selected treatment.

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1. Introduction

SKG is a common result of central nervous system lesions that may arise after stroke, traumatic brain injury, spinal cord injury, cerebral palsy (CP), and multiple sclerosis [1]. It is characterized by reduced or delayed KF during the swing phase of the gait cycle. It determines an increased energy cost [2] leading to an inefficient gait pattern [3,4]. SKG is thought to be mainly caused by the inappropriate swing-phase activity of the RF muscle [3,5,6] typically referred to as spasticity [1]. A second mechanism that has been linked to SKG is the abnormal foot kinematics at toe-off, as pointed out by Kerrigan in Ref. [7]. The lack of push-off power at the ankle results in an insufficient KFV at toe-off with a consequent lack of passive KF [6,8,9]. A third mechanism that has been linked to SKG is the insufficient hip pull-off [10] that should produce hip flexion during swing.

As RF spasticity is considered the main cause of SKG, treatments focus on reducing RF activity by either surgery or botulinum toxin injection (BTI) [11,12]. RF can be surgically released or transferred to sartorius or gracilis thus converting its action to a knee flexor [5,13,14]. RF surgery is considered as the treatment of choice in CP children [1], with most studies reporting an average increase in KF between 7 and 10°, as summarized in Ref. [15]. RF treatment by BTI is reported in the literature as an effective intervention for both CP children and stroke adults, as it leads to a statistically significant increase in KF [1,12]. However, the average KF increase is lower than 10° [1,12] and the flexion peak remains on average lower than 30° [1,12].

In this study, we present a method to differentiate among the causes of SKG, i.e. the lack of push-off from the presence of muscular activity preventing flexion.

2. Material and methods

We retrospectively analyzed the gait kinematics of 52 adult stroke patients (38 male and 14 female, age 60 ± 11) with SKG, available in the database of our laboratory, acquired using the Conventional model [16] (Smart-D system, BTS Bioengineering, Milan, Italy). The inclusion criteria were: age ≥ 18 years, hemiparesis

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secondary to stroke, time since stroke >6 months, knee flexion peak in swing lower than 45° [7], independent walking with or without walking aids, available gait analysis data from at least three trials. The exclusion criteria were: orthopedic surgery or casting to lower limbs, BTI or phenol neurolysis within the past year, other neurological problems not caused by the cerebrovascular accident.

Left and right gait kinematics of 20 healthy adults were also analyzed in this study. Five trials of spontaneous, fast, slow and very slow gait were acquired for all subjects. Standardized instructions were given to subjects as follows: "walk as if you are late" for fast gait, "walk at your self-selected speed" for spontaneous gait, "walk slower than before" for slow gait and "walk as you go window-shopping with friends" for very slow gait.

In order to indirectly quantify the ankle push-off from kinematic data only, we computed the peak of malleolus vertical acceleration (PMVA) during preswing. PMVA was obtained by differentiating the vertical trajectory of the marker placed on the lateral malleolus. The differentiation was performed as reported in Ref. [17] over the whole calibrated volume. On average three strides per trial were available, thus leading to approximately 15 values per subject. The median value over strides was used in further analysis.

To investigate the temporal sequence of events that determine knee flexion, we computed and compared the occurrence times of PMVA (tPMVA), of the peak of knee flexion acceleration (tPKFA) and of hip flexion acceleration (tPHFA). Angular accelerations were obtained by differentiation from knee and hip sagittal kinematic, as for PMVA. Statistical comparisons among temporal events and between groups were performed by the Mann–Whitney U-Test.

The time-course of knee flexion throughout the gait cycle was fully described by computing the range of KF during terminal stance (TSt), preswing (PSw) and swing (Sw), the peak of KF in Sw and the average KFV during preswing. Comparisons between patient and control values were performed by the Mann–Whitney U-Test. Statistical significance was set at 5% for all analysis. The correlation between KFV and walking speed was also investigated.

As the push-off mechanism is supposed to determine the fast knee flexion in PSw [6–9], we used a regression analysis to model the effect of PMVA on KFV. Model coefficients and their 95% confidence interval (CI) were computed and the coefficient of determination R^2 was used as goodness-of-fit indicator. Healthy subject kinematics were used to model the relationship between push-off and KF at different velocities and in the absence of spasticity. Patient data were then compared to the model to identify subjects whose limited KFV was explained by a limited push-off only and patients whose KFV was reduced more than the reduced push-off would explain. In those subjects it was presumed that a braking mechanism due to muscle activity was present.

Finally, the effect on knee flexion of a diagnostic femoral nerve (motor branch of rectus femoris) block by lidocaine was analyzed for three patients with data lying at a progressively increasing distance from the normal model.

3. Results

Mean values for all investigated variables are reported in Table 1. In healthy subjects, walking speed, knee flexion during TSt, PSw and Sw, the peak of knee flexion in Sw, PMVA and KFV significantly varied from fast to very slow gait ($P < 0.001$). For
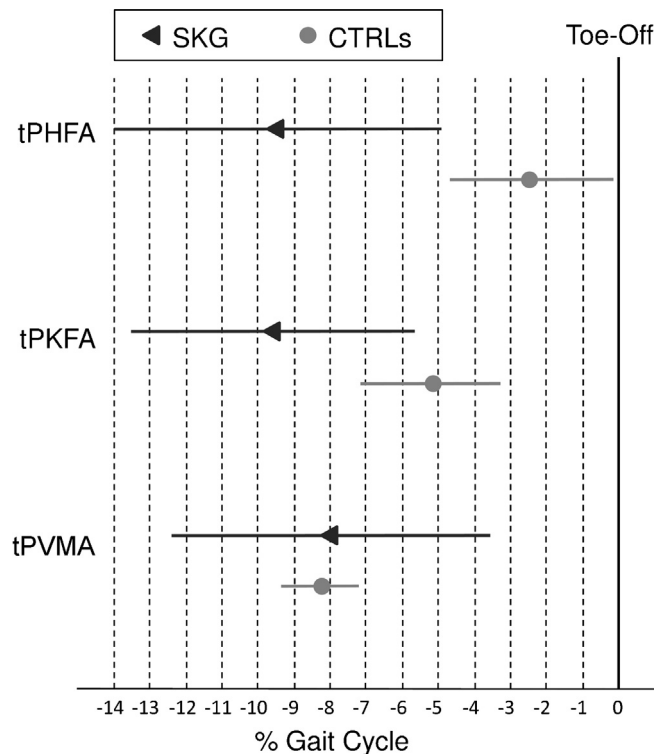

Fig. 1. Timing (mean and 95%CI) of the peak of malleolus vertical acceleration (tPMVA), knee flexion acceleration (tPKFA) and hip flexion acceleration (tPHFA) during preswing for SKG patients and healthy controls walking spontaneously (CTRLs). Both patients and controls time events are aligned to the foot-off. In healthy subjects knee flexion is a bottom-up mechanism, driven by the push-off at the ankle. This temporal evolution is lost in SKG patients, with a large variability among subjects.

these variables, patient values were lower than those of very slow walking controls ($P < 0.001$). In healthy subjects, PMVA was the first event in the knee flexion mechanism at all velocities (Table 1). As shown in Fig. 1, knee flexion was a bottom-up mechanism which started by the push-off at the ankle. In patients this bottom-up mechanism was lost and the movement was more or less synchronous (or top-down for a few patients), with a large variability among patients.

Table 1

Mean values ± standard deviations for all variables related to knee flexion investigated in this study. Values are reported for the affected side of a sample of 52 adult stroke patients with SKG and for both sides of 20 healthy age-matched subjects walking at fast, spontaneous, slow and very slow speed, following standardized instructions (see text for more details). To allow for values comparison, all time occurrences are referred to the toe-off instant. Thus, for instance, tPMVA = 8%GC means that PMVA takes place 8% of the gait cycle duration before toe-off.

	SKG patients	Healthy subjects at different walking speed			
		Very slow	Slow	Spontaneous	Fast
Walking speed (% height/s)	20 ± 11	38 ± 5**	54 ± 6**	74 ± 5*	95 ± 5
Knee flexion peak in Sw (°)	25 ± 9	56 ± 6	56 ± 4	58 ± 4	62 ± 5*
Knee flexion in TSt (°)	2 ± 2	4 ± 3**	6 ± 2*	8 ± 2**	10 ± 2**
Knee flexion in PSw (°)	17 ± 8	29 ± 4**	26 ± 4*	23 ± 4*	22 ± 3
Knee flexion in Sw (°)	6 ± 5	21 ± 4**	24 ± 5*	27 ± 4	29 ± 4
KFV in TSt (°/s)	3 ± 10	17 ± 10**	41 ± 12**	64 ± 23**	99 ± 25**
KFV in PSw (°/s)	53 ± 39	128 ± 28**	191 ± 27**	231 ± 30**	298 ± 30**
KFV in Sw (°/s)	40 ± 38	158 ± 17**	184 ± 27*	220 ± 27**	266 ± 34**
PMVA (cm/s²)	12 ± 7	17 ± 4**	27 ± 6**	31 ± 5*	34 ± 7
tPMVA (%GC before toe-off)	8 ± 4	8 ± 4	8 ± 1	8 ± 1	6 ± 2**
tPKFA (%GC before toe-off)	10 ± 4	5 ± 2**	6 ± 2††	5 ± 2††	6 ± 2
tPHFA (%GC before toe-off)	9 ± 5	4 ± 1**	3 ± 3†	3 ± 2††	3 ± 3††

† Statistically different from the correspondent value in the previous line, $P < 0.05$.
†† Statistically different from the correspondent value in the previous line, $P < 0.001$.
* Statistically different from the correspondent value in the previous column, $P < 0.05$.
** Statistically different from the correspondent value in the previous column, $P < 0.001$.