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NON-REFLEX MEDIATED CHANGES IN PLANTARFLEXOR MUSCLES EARLY AFTER STROKE

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ABSTRACT. The aims of this study were to determine whether changes in the non-reflex component of spastic plantarflexors had developed 2 and 4 months after stroke and to study their relationship with the level of impairment. One group of adults with hemiparesis (HPs) was tested 2 and 4 months after the onset of stroke, and data were compared with a control group (CTLs) tested once. Twenty-two patients (14 males) admitted over a 4-month period in a rehabilitation centre (mean = 62 yrs ±14), and 11 (6 males) non-disabled (CTLs) subjects (mean = 57 yrs ±12.8) agreed to participate in the study. The resistive torque (RT) recorded with a myometer during slow (8–10°/s) passive dorsiflexions imposed manually served as the primary outcome, whereas, the Ashworth score (spasticity), ankle ROM and Fugl-Meyer motor subscore were used as secondary measures to determine the level of impairment. The mean RT values measured at 0° dorsiflexion on the affected and unaffected sides were compared with those in CTLs. As expected, the RT values 2 and 4 months post-stroke on the unaffected side did not differ from corresponding values in CTLs. Significantly higher RT values on the affected side when compared to the unaffected side were found both at 2 months (39%; $p < 0.05$) and at 4 months (43%; $p < 0.01$). No significant difference existed on the affected side between the 2nd and 4th months. A high ($r = 0.80$) and significant ($p < 0.0001$) correlation coefficient was calculated between the changes in RT values recorded at 2 and 4 months. Low and not significant correlations were computed between these RT changes and factors such as the ROM ($r = -0.24$), the Ashworth score ($r = 0.23$) and the Fugl-Meyer lower extremity motor subscore ($r = -0.26$). Present results indicate that: (1) changes in the non-reflex component are already present 2 months after stroke but do not increase significantly between the 2nd and 4th months; (2) these changes are not related to the level of impairment; and (3) myometry testing at 2 months could be used as a preventive measure to detect patients more at risk of developing severe passive muscle stiffness.

Key words: hand-held dynamometry, muscle stiffness, plantarflexors, resistive torque, spastic hypertonia, stroke.

Classically, increased resistance to passive stretching of spastic muscles has been attributed to the velocity-dependent hyperactive tonic stretch reflex (16, 20, 24). Over the past 10 years, however, mechanical or non-reflex mediated changes in spastic muscles have frequently been linked to the increased resistance to passive movement found in spastic hypertonia. More and more experimental evidence suggests spastic hypertonia to be associated not only with an increased sensitivity of the stretch reflex (reflex component), but also with changes in the properties of the muscular and connective tissues (non-reflex components) (1, 10, 17, 18, 30–34). The contribution of the non-reflex components to hypertonia is based on the fact that the mechanical response to muscle stretch increases without a parallel increase in the electromyographic activity of the stretched muscles (1, 10, 17, 18, 31). For instance, the overactivation in the tibialis anterior muscle (TA) during gait (swing phase) that could not be explained by the coactivation of the antagonists led to the conclusion that the TA worked against an increased resistance of non-reflex origin (10). Moreover, based on the discrepancy between the clinical assessment of tone and reflex-evoked EMG activity in subjects with long-lasting spasticity (large increase in tone and low EMG levels), the contribution of non-reflex components to hypertonia is believed to develop with the duration of spasticity (17, 32, 33). The finding of lower reflex-evoked EMG activity in the more chronic spastic subjects further supports the idea that factors other than increased reflex sensitivity are involved in

Table I. *Characteristics of the two groups of subjects*

	Patients ($n = 22$)	Controls ($n = 11$)
Age (yrs)		
Mean	62.0	57.1
Median	65	59
±1 SD	14.0	12.8
Weight (kg)		
Mean	70.9	68.6
Median	74.5	72.7
±1 SD	17.6	17.7
Height (cm)		
Mean	166.5	165.8
Median	168.8	165.0
±1 SD	12.6	11.3
Gender		
Male	14	6
Female	8	5

spastic hypertonia when spasticity is established for one year or more (17, 30, 32, 33).

The present study was thus intended to determine whether changes in the non-reflex components have already taken place at the time the patients enter a rehabilitation centre and at about the time of their discharge by measuring resistive torque to slow passive dorsiflexion, at 2 and 4 months after stroke. The resistive torque was measured with a hand-held dynamometer (or myometer) during manually imposed passive movement, a method that has been shown to be reliable (3, 9, 23, 26). The objectives of the present study were (1) to determine whether changes in the non-reflex components of the plantarflexors had taken place two months after stroke, by comparing resistive torque values recorded during slow passive ankle dorsiflexion in both sides of subjects with hemiparesis with those of control subjects, (2) to examine the evolution of these changes by re-evaluating resistive torque two months later; and (3) to study the relationship between these changes and the level of impairment (flexibility, spasticity and motor function).

MATERIALS AND METHODS

Subjects

Twenty-two patients (14 males, 8 females) with a mean age of 62 years (Table I) consecutively admitted between August 1993 and December 1993 to the François-Charon Rehabilitation Centre in Quebec City for rehabilitation following a stroke participated in this study. The 22 patients had suffered an infarct of tromboembolic ($n = 18$) or haemorrhagic ($n = 4$) origin in the region of the right or left middle cerebral artery ($n = 20$) or in the region of the posterior cerebral artery. Confirmation of the diagnosis was made by computerized tomography. The lesion resulted in a paretic syndrome on the left ($n = 13$) or the right ($n = 9$) side of the body. The patients with hemiparesis (HPs) entered the study if they had no prior history of stroke, had no pain, no aphasia, no contracture (at least 0 degrees of passive dorsiflexion) or any other problems rendering them unable to cope with the assessment procedures. In addition (Table I), 11 non-disabled subjects (6 males, 5 females), matched for age and gender and without any history of neurological or musculo-skeletal dysfunction, served as controls (CTLs). All the subjects (HPs and CTLs) signed an informed consent prior to entering the study.

Design

The HPs were assessed twice (test 1 and test 2); the mean time since stroke at the first evaluation was 59.2 days and the second evaluation was carried out about 2 months later (see Table II). The evaluation at test 2 was carried out by the same evaluator and at the same time of the day. The order of testing was kept similar in tests 1 and 2. The myometry testing was followed by the Ashworth test. The Fugl-Meyer Assessment scale (FMA) was administered on a separate day. Myometry testing was not repeated in the CTLs.

Testing procedures

The hand-held dynamometer used in this study was the Penny and Giles[#] (#LAM Associates, 1001 Sierra Blvd, Missisauga, Ont. L4Y 2E3) myometer. This myometer can record forces up to 300 Newtons (N) with a precision of 1 N. The myometer gives the maximal force recorded. For the needs of the present study, a special device was used to maintain the distal end of the transducer over the head of the metatarsals. The cord connecting the transducer to the recording system was lengthened to ease the evaluation procedures. Similar testing procedures that were standardized in a previous study (26) were applied. During testing, the subject lay on a low table; the knee was maintained at 20° of flexion by placing a small pillow underneath the knee (Fig. 1). The evaluator stabilized the lower leg with one hand, while dorsiflexion of the foot was imposed by the other hand holding the myometer. The head of the myometer was held perpendicular to the sole of the foot

Table II. *Clinical information: hemiparetic subjects ($n = 22$)*

	Time after stroke (days)		FMA (%) Lower extremity		FMA (%) Upper extremity		FMA (%) Standing balance	
	$Test_1$	$Test_2$	$Test_1$	$Test_2$	$Test_1$	$Test_2$	$Test_1$	$Test_2$
Mean	59.3	115.6	64.1	70.0	48.9	60.9	49.4	59.1
±1 SD	20.6	18.8	25.0	24.4	31.2	32.0	22.5	21.3

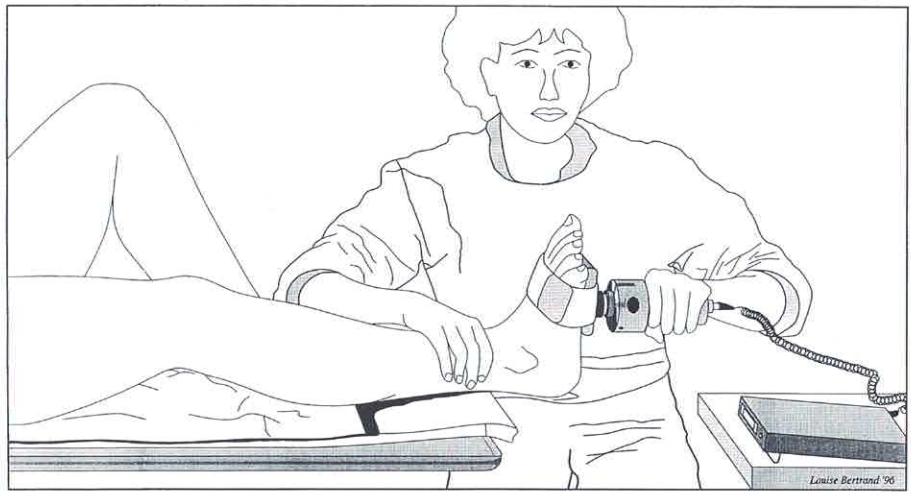

Fig. 1. The standardized testing procedure used for recording resistive torque with the hand-held dynamometer (see text for details).

under the head of the metatarsals throughout the passive dorsiflexions, starting from a resting position of about 30°–35° plantarflexion (PF) to a 0° of dorsiflexion (DF) position. The 0° DF was controlled by monitoring the endpoint of movement with a mirror placed parallel to the subject's leg and facing the evaluator (26). Four passive DFs were executed at low velocity on the affected and then the unaffected sides of the HPs and on one side of the CTLs. The velocity of the limb displacement was controlled by counting mentally so that total movement time would be 4 seconds, corresponding to a velocity of about 8°/s–10°/s. Each passive DF was interspersed by a 10 second rest.

The modified Ashworth scale (2) was used as a clinical measure of spasticity, whereas, a French version of the Fugl-Meyer Assessment (FMA) scale translated and adapted by Dutil et al. (11) from the original article by Fugl-Meyer (14) was used to assess the motor function. Three FMA subscales were used: the motor upper extremity (maximal score = 66), the motor lower extremity (maximal score = 34) and the standing balance (maximal score = 8). For comparison purposes, the score from each subscale was transformed in percent of its respective maximal value. The ankle ROM was measured with a manual goniometer.

Evaluators

Three physiotherapists who had between 10 and 14 years' experience in stroke rehabilitation administered the Ashworth test, the myometry testing and the Fugl-Meyer Assessment (FMA) scale. They all had experience in the use of the myometer and two of them had participated in previous clinical studies where these protocols had been used (4, 26).

Data analysis and statistical tests

The maximal force values recorded with the myometer were converted to torque and the resistive torque (RT) values (N.m) recorded for three of the trials (those with the closest values) were averaged. It was assumed that the maximal force corresponded to the force recorded at 0° dorsiflexion. Indeed, visual monitoring of the read-out showed that the force increased with dorsiflexion and was greatest at the end of the range (0° in this case). The increase of force with dorsiflexion was also confirmed in a previous study using isokinetic devices (3, 33). Student's *t*-test was used for comparison of age, weight and height between the two groups. An analysis of variance (one-way) was used to compare values recorded from the unaffected side of the patients to that in the CTLs (one side) at tests 1 and 2. Determination of the changes on the affected and unaffected sides at tests 1 and 2 was made by a two-factor (side and time) analysis of variance. The ANOVAs were followed by a Tukey post-hoc test when indicated. The Pearson correlation coefficient was used to determine whether changes in resistive torque were associated with the level of spasticity, motor function and flexibility. The probability level was set at 0.05.

RESULTS

The subject characteristics are provided in Tables I and II. As can be seen from Table I, subjects from the two groups had comparable values for age, weight and height. Table II gives the Fugl-Meyer (FMA) motor and standing balance subscores at tests 1 and 2, as well as the elapsed time after stroke at tests 1 and 2. Test 1 was conducted about 2 months after stroke and there was almost a 2-month interval between tests 1 and 2. The mean FMA subscores indicate a residual motor function ranging from 49% to 64% at test 1; at test 2, corresponding scores ranged from 60% to 70% and the increase between tests 1 and 2 was a significant (paired *t*-test: $p < 0.001$) for both motor function (upper and lower extremities) and standing balance.

As expected, the mean (±2SE) RT values recorded at 0° of DF during slow passive dorsiflexion on the unaffected side of the HPs at tests 1 (5.8 N.m ± 0.74) and 2 (6.0 N.m ± 0.68) were not different from corresponding values in CTLs (5.2 N.m ± 0.14). Further comparisons are therefore made between the values

recorded on the affected and unaffected sides of the HPs; the differences between sides were used to calculate the percent changes (increase or decrease) in the affected side. Results from the two-way ANOVA indicated a significant effect between sides but not between tests. Larger resistive torque values were recorded on the affected side (Fig. 2) at test 1 (39.2% ± 62.5) and test 2 (43.5% ± 54.9%). The mean RT values recorded at test 2 were slightly larger compared to test 1 on both the affected (11% ± 20.6) and the unaffected (6.8% ± 17.4) sides, but this increase was not significant (Fig. 2). To give an overview of the changes over time, Fig. 3 illustrates the individual RT values recorded at test 1 and test 2 relative to the mean (2SE) RT values recorded on the unaffected side. Lastly, a significant ($p < 0.001$) correlation ($r = 0.80$) was found between the changes in RT measured at tests 1 and 2.

Fig. 4 illustrates the changes in RT relative to the Ashworth scale (Fig. 4A), to the range of motion (ROM) at the ankle (Fig. 4B) and to the Fugl-Meyer lower extremity motor subscore (Fig. 4C). The correlations between the changes in RT and the Ashworth score ($r = 0.23$), the ankle ROM ($r = -0.24$) and the lower extremity motor score ($r = -0.26$) were low and not significant.

DISCUSSION

Increased mechanical response

The results indicate that significant changes in the mechanical response of the plantarflexors of HPs are present during slow stretch on the affected side as early as 2 months after stroke. The mean RT values measured 2 and 4 months after stroke were 39% and 43% higher than corresponding values in the unaffected side. Stiffness increase of 50% and 94% in paretic plantarflexors have been previously reported in a group of hemiparetic subjects (30, 33) who had suffered a stroke at least 12 months earlier. In light of the present results, however, it appears that significant changes (43%) in the non-reflex components have already taken place within the first 4 months after stroke. The lack of any significant difference in RT between the unaffected side of HPs and corresponding values in CTLs confirms previous observations made in HPs within two years after stroke (mean of 20 months) HPs (33). In contrast, in more chronic HPs (mean of 30 months) a 95% increase in passive stiffness has been measured on the unaffected limb (30). In the present study, although not significant, an increase of 6.8% was observed between the RT values recorded at 2 and 4 months on the unaffected side. The latter observations warrant caution in the choice of reference values with hemiparetic subjects, particularly in the more chronic stage. Using the unaffected side as control may mask the concomitant changes developing in the unaffected side and also give an underestimation of the changes on the paretic side.

Our results also confirm previous findings that stiffness can increase even in subjects free of contractures (33). Although, the present study was not intended to determine the effects of physiotherapy on the development

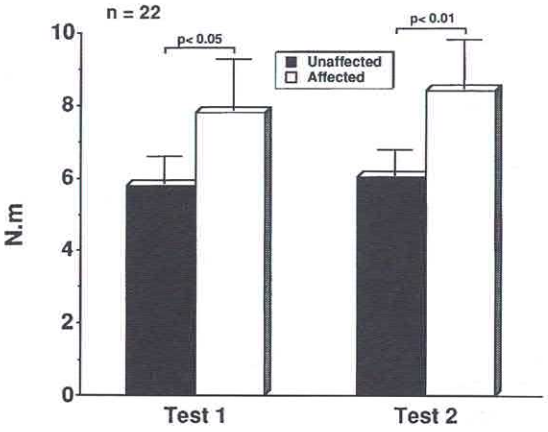

Fig. 2. Bar graph illustrating the mean resistive torque values recorded at 0° of dorsiflexion (DF) during slow passive DF in patients with hemiparesis ($n = 22$). The resistive torque recorded in the affected side was significantly higher (39% and 43%) than that of the unaffected side. Although the mean values on the affected side increased from test 1 and test 2 (at 2 and 4 months post-stroke) this increase was not significant. Vertical lines indicate two standard errors (2SE).

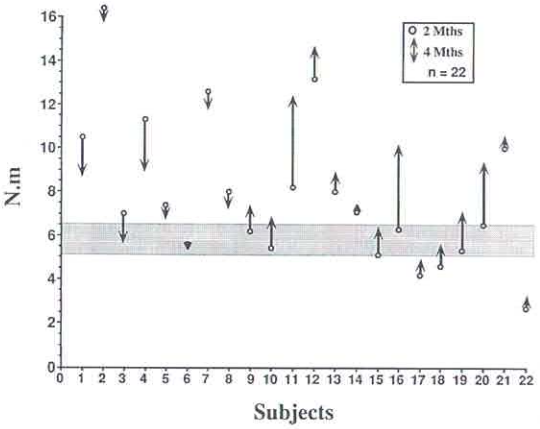

Fig. 3. The RT values recorded at 2 and 4 months after stroke are illustrated for each of the 22 subjects. The line and the arrow connecting individual sets of values indicate the direction of changes between the 2nd and 4th months (tests 1 & 2). The grey area represents the mean (±2SE) RT values (99% confidence limit interval).

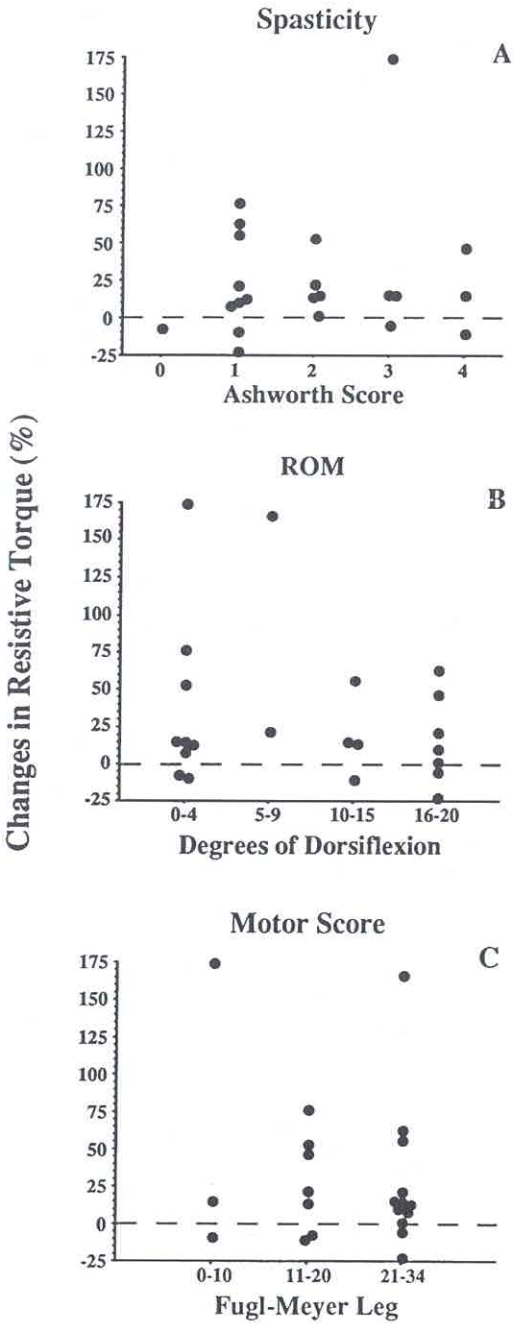

Fig. 4. Changes in the resistive torque values relative to the Ashworth score (Fig. 4A), the ankle passive range of motion (Fig. 4B) and the Fugl-Meyer lower extremity motor score (Fig. 4C) in 22 patients with hemiparesis. Values correspond to the differences (in percent) in the resistive torque calculated between sides for each patient.

of the non-reflex components of SH, it is clear that physiotherapy did not reverse the changes that had already occurred within the first 2 months after stroke. The ongoing physiotherapy may, however, have slowed the pathological process since the increase in RT values (11%) between the 2nd and 4th months was not significant. It remains to be demonstrated whether a more aggressive therapeutic approach after stroke would help prevent or reverse the development of these changes. Answers to such questions could be obtained through randomized controlled trials designed to clarify the role of physiotherapy in controlling the development of muscle stiffness.

Pathological mechanisms

There is no evidence to support a single mechanism to account for the changes of the non-reflex components in SH. After stroke, there is a loss of functional motor units (27, 35), denervation occurs in more distal muscles (6, 15) and a preferential atrophy of type II muscle fibres has been documented (12, 13). Since stiffness is directly related to the muscle volume, the RT would be expected to decrease in spastic atrophied muscles rather than increase, unless muscle fibres were replaced by a less compliant tissue (19, 21, 29). Pathological changes in the mechanical properties of the contractile elements of the muscles as well as alterations of the connective tissue (in the tendon or muscle) have usually been proposed to explain the origin of the abnormal resistance (1, 10, 17, 18, 31–33). The synthesis and transformation of collagen fibres as well as the role of collagen fibres on muscle tensile properties have been studied mainly in animal muscles after immobilization or denervation (19, 21, 29, 34). Thus, any transfer of these observations to human spastic muscles requires considerable caution.

The trophic influence exerted by the supraspinal drive on motoneurones in the development of functional contractures has been proposed (25). The main evidence for the contribution of such a neural influence on the development of functional or myostatic contractures in chronic conditions is the finding that contractures were reduced under anaesthesia or after motor nerve section. It was argued that such a reversal would not be expected if the connective tissue rather than the contractile mechanism were responsible for the contracture. In fact, it was proposed that changes of a physiological (affecting the contractile mechanism) rather than a structural nature (increased fibrous tissue) are responsible for the development of contractures (25). Recent

observations further support the involvement of neural descending input in the development of muscle stiffness. Indeed, the finding that muscle stiffness does not increase in chronic spinal-cord injured patients with a complete motor lesion (22) suggests that muscle stiffness does not develop when the spinal cord is isolated from supraspinal centres. To date, no study has succeeded in demonstrating that the increased passive stiffness of spastic plantarflexors is due to unwanted tonic muscle activity. For instance, no muscle activation was detected during slow passive stretching of the plantarflexors (33); moreover, increased stiffness was also reported to persist despite neural block (28). Perhaps surface EMG may not be sufficient to detect low or localized EMG, or the neural block may have been incomplete or not maintained long enough (28) to be conclusive? Until these questions have been directly addressed, the mechanisms involved in the development of increased passive stiffness in spastic muscles will remain, at best, speculative. Finally, in a recent review of both human and animal studies, it was proposed that adaptations involving the formation of a higher proportion of binding crossbridges could result in abnormal stiffness in the spastic muscles (7). It is possible that more than one mechanism is involved in the development of passive muscle stiffness.

Study limitations

In this study, the torque was measured at the 0° DF. This angle was chosen for clinical, methodological and physiological reasons. First, it is relatively simple and easy to standardize force measurements with the myometer at that angle in a clinical setting (3, 4, 26). Secondly, since the force increases with dorsiflexion, the end point of 0° corresponds to the maximal value (3, 33) recorded with the myometer. Thirdly, force values recorded at this angle are known to be highly reproducible in hemiparetic subjects (26). Lastly, the resistive force recorded at that angle is high enough to detect differences between sides in hemiparetic subjects (33). Results are limited to the present sample ($n = 22$) of adults who suffered a unilateral cerebral lesion 2 months and 4 months earlier and cannot be generalized to other spastic conditions in children (cerebral palsy; brain injury) and adults (spinal-cord lesion; multiple sclerosis). Although no recording of muscle activations was made during slow muscle stretch, based on previous observations where surface EMG was recorded during similar manoeuvres (3, 33), it is unlikely that the reflex threshold was attained to affect the RT and that the increased RT could be reflex-mediated. In order definitively to rule out any low tonic unwanted muscle activation from the plantarflexors, however, more invasive methods such as multiple intramuscular recording or a neural block would be required.

Clinical significance

Present findings also confirm previous observations in chronic HPs (33) to the effect that no correlation could be found between either the flexibility, spasticity or the motor function of chronic HPs and ankle stiffness. Attempts to relate the ankle ROM to changes in RT resulted in very low and not significant correlations, indicating that increased RT can be expected in any patient regardless of the severity of the impairment. The very low correlations also suggest that, individually, these clinical features (spasticity, motor function, flexibility) are poor predictors. A multi-factorial analysis in a larger sample would be required to identify whether combined clinical indicators would be better predictors. Meanwhile, the strong correlation ($r = 0.80$) computed between the changes in RT at tests 1 and 2, is interesting. The difference in RT measured between the two sides early after stroke could be used, for instance, as an indicator to pinpoint the subjects at risk of developing muscle stiffness. The prevention of severe biomechanical alterations at the ankle is important for the retraining of gait-related activities.

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