

# Rate of force development and torque production assessment in spastic stroke survivors

## *Avaliação da taxa de produção de força e torque em indivíduos com espasticidade pós-avc*

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**Abstract** – The aim of this study was to compare the rate of force development (RFD) and maximum torque in spastic stroke survivors and healthy individuals. Fifteen stroke survivors (57.3 ± 11.2 years) with ankle spasticity and fifteen healthy individuals (59.3 ± 6.4 years) participated in this study. An isokinetic dynamometer was used to maximum voluntary isometric contraction (MVC) and RFD assessment of plantar flexors muscles of ankle, which the individuals were instructed to produce maximum torque as fast as possible. The absolute RFD was normalized by MVC (relative RFD). In results were observed significant differences in RFD of affected limb (43.3 ± 8.5 Nm/s) and unaffected limb (98.9 ± 20.4 Nm/s) compared to healthy (186.2 ± 25.2 Nm/s), but with no differences between affected and unaffected limbs (p=0.15). In relation to relative RFD, the affected limb (9.76 ± 1.1 %MVC/s) was significant different than healthy (13.08 ± 1.5 %MVC/s). The MVC produced by affected limb (46.55 ± 7.98 Nm) was significant lower than unaffected limb (84.29 ± 8.47 Nm) and, the two limbs of stroke survivors were weakness than healthy individuals (128.02 ± 9.36 Nm). Lastly, the spasticity level present higher negative correlation in relation to RFD (R= -0.725; p= 0.002) and MVC (R= -0.717; p=0.003). The spasticity promotes alterations in capacity to produce maximum force and fast force in affected and unaffected limbs of stroke survivors compared to healthy.

**Key words:** Dynamometer; Muscle power; Muscle spasticity; Strength.

**Resumo** – O objetivo desse estudo foi comparar a taxa de produção de força (TPF) e o torque máximo em indivíduos com espasticidade e saudáveis. Participaram do estudo 15 sujeitos pós-AVC (57,3 ± 11,2 anos) com espasticidade de tornozelo e 15 sujeitos saudáveis (59,0 ± 6,4 anos). Um dinamômetro isocinético foi utilizado para a avaliação da contração isométrica voluntária máxima (CVM) e da TPF dos flexores plantares do tornozelo, na qual os participantes foram instruídos a produzir força máxima o mais rápido possível. A TPF absoluta também foi normalizada pela CVM (TPF relativa). Como resultados, foram encontradas diferenças significativas na TPF absoluta do lado afetado (43,3 ± 8,5 Nm/s) e não afetado (98,9 ± 20,4 Nm/s) quando comparados com os saudáveis (186,2 ± 25,2 Nm/s), porém sem diferenças entre os membros espásticos (p=0,15). Em relação a TPF relativa, apenas o lado afetado (9,76 ± 1,1 %CVM/s) apresentou diferença em relação aos saudáveis (13,08 ± 1,5 %CVM/s). A CVM produzida pelo lado afetado (46,55 ± 7,98 Nm) foi significativamente menor quando comparado ao lado não afetado (84,29 ± 8,47 Nm) e, os dois lados foram mais fracos em comparação aos indivíduos saudáveis (128,02 ± 9,36 Nm). Por fim, o nível de espasticidade apresentou alta correlação negativa em relação a TPF (R= -0,725; p= 0,002) e a CVM (R= -0,717; p=0,003). A espasticidade gera alterações na capacidade de produzir força máxima e rápida tanto no membro afetado quanto no não afetado em indivíduos que tiveram AVC em relação a indivíduos saudáveis.

**Palavras-chave:** Espasticidade muscular; Dinamometria; Força; Potência muscular.

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## INTRODUCTION

Stroke is the main cause of acquired motor disability in adults<sup>1</sup>, which begins suddenly and commonly promotes weakness and hemiparesis on the contralateral side of the body in relation to the side of the cerebral injury<sup>2</sup>. Among complications, damage to the upper motor neurons that in turn affect the corticospinal tract responsible for the inhibitory projection in neurons of the spinal cord motor is commonly found<sup>3</sup>. Thus, spasticity is developed.

Spasticity is defined as the increase in tendon hyperreflexia at rest, which is the increasing myotatic reflex response coupled with increased muscle tone<sup>4</sup>. This complex motor disorder stems from a dysfunction in the central nervous system and promotes alterations at all levels of the locomotor system including muscles and joints<sup>5</sup>. Adaptations secondary to spasticity have been observed such as increased amounts of type-I muscle fibers and muscle cell stiffness<sup>6</sup>, smaller fascicle length and cross-sectional area<sup>7,8</sup> decreased muscle volume<sup>9</sup> and reduction in voluntary muscle activation, which compromises balance, causing deficient voluntary control<sup>10</sup>.

Previous studies have evaluated the ability of maximal voluntary force production in stroke survivors with spastic hemiparesis. Klein et al.<sup>11</sup> observed decreased plantar flexor strength of approximately 60% in ankle joint on the affected limb compared to the unaffected limb, and McCrea et al.<sup>12</sup> observed an average reduction of 53% in isometric torque on the affected arm compared to the unaffected arm. There are several causes that can be indicated as responsible for strength deficit secondary to spasticity such as muscle atrophy, decreased number of motor units, alterations on muscle recruitment order, decreased firing rate and muscle activation capacity<sup>13,14</sup>.

A parameter related to force production capacity and important for the functional evaluation of fast muscle contraction is the rate of force development (RFD). This index has been the most widely used to represent the explosive force and is obtained by the force-time variation ratio, which is also an important neuromuscular performance parameter<sup>15</sup>. RFD is relevant precisely because, according to Suetta et al.<sup>16</sup>, the ability of individuals to produce fast force is related with daily activities such as walking, climbing and going down stairs. Also, the increase in RFD reflects higher level of muscle strength in the initial muscle contraction phase<sup>17</sup> which enables a fast response in balance disturbance situations, preventing falls. Considering the alterations in motor control promoted by spasticity and on muscles in spastic individuals, it is important to study RFD and the maximal force production in this population. However, few studies have assessed these variables in stroke survivors with spasticity.

Thus, the aim of this study was to compare the rate of force development (RFD) and maximum torque production of plantar flexors between stroke survivors with spasticity and healthy subjects and correlate the spasticity level with the variables investigated.

## METHODS

Fifteen stroke survivors with ankle spasticity participated in this study. The spasticity level was assessed by the Modified Ashworth scale. Inclusion criteria were: (1) presence of hemiparesis spasticity for at least one year; (2) being able to walk without any assistance; (3) no history of orthopaedic surgery in any lower limb; (4) no use of medication to treat spasticity or use of orthoses; (5) cognitive capacity to perform assessments; (6) being able to remain seated for at least one hour. Fifteen healthy and sedentary individuals with no neurological or muscle disorder and with similar age participated in the control group. All subjects signed an informed consent form to participate in the study and the experimental protocol was approved by the Ethics Committee in Human Research of the institution where the study was conducted (Protocol number 18440 UFRGS and Protocol number 10-5179 PUCRS).

### Experimental design

The experiment consisted of two sessions with interval of one week between them for all participants. Each limb was tested in one session using the same protocol. Only the dominant leg of healthy subjects was considered for analysis and used as healthy limb.

### Evaluation procedures

An isokinetic dynamometer (Biodex Medical System, Shirley – NY, USA) was used for the positioning of the ankle joint and assessment of RFD and torque production. Participants were seated on the dynamometer chair and positioned with trunk and hips fixed by adjustable straps, with knee fully extended and ankle at 0° (foot perpendicular to leg). The ankle joint rotation axis (defined by the center of the medial/lateral malleolus) was aligned with the dynamometer axis to minimize rotations out of the intended motion plane.

Subsequently, participants performed three maximal voluntary isometric contractions (MVC) of plantar flexion at 0° for 5 seconds. Participants were oriented to exert maximal force as fast as possible and maintain this effort for at least one second. The maximum active torque recorded among the three contractions was used for further analysis. Before data acquisition protocols, all participants performed three submaximal voluntary isometric contractions of plantar flexion as familiarization protocol, and a 2-min resting time was respected between contractions (familiarization and evaluation) to avoid potential fatigue effects in torque production.

Absolute RFD was defined as the slope of the torque–time curve ( $\Delta\text{moment}/\Delta\text{time}$ ) in incrementing time period of 0–250 ms from the onset of maximal isometric contraction. A routine in MATLAB software (MATLAB version 7.3.0.267, MathWorks, Inc., Natick, MA) was used to obtain torque and RFD variables during CVM. Also, the relative RFD that was obtained with the absolute RFD normalized by MVC (RFD/MVC, present in %MVC/s) was measured at same time period.

## Data analysis

Data homogeneity was tested using Levene's test and distribution normality was tested and confirmed by the Shapiro-Wilk test. For unpaired samples, the student t test was used to compare age and anthropometric variables between groups. One-way ANOVA was used to determine the existence of significant differences in RFD and torque between limbs. Correlation test was performed by the Pearson test. Statistical analysis was performed using SPSS software version 17.0 with significance level of  $\alpha = 0.05$ .

## RESULTS

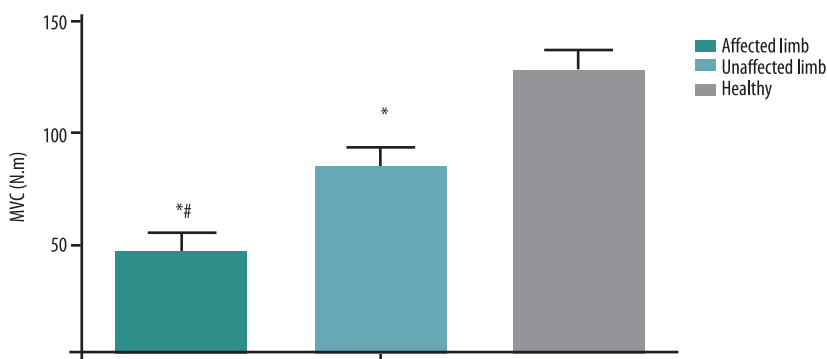
There were no significant differences for age, body mass and height ( $p=0.60$ ,  $p=0.65$  and  $p=0.46$ , respectively) between groups (stroke vs. healthy). Anthropometric and clinical variables are shown in table 1.

**Table 1.** Characteristics of participants (mean  $\pm$  SD).

	Stroke (n=15)	Healthy (n=15)
Age (years)	57.3 $\pm$ 11.2	59.0 $\pm$ 6.4
Time since stroke (years)	7.4 $\pm$ 5.8	-
Male/female (n)	10/5	10/5
Body mass (kg)	76.6 $\pm$ 14.4	74.5 $\pm$ 11.5
Height (cm)	169 $\pm$ 0.05	167 $\pm$ 0.10
ASW	1.5 $\pm$ 0.6	-

ASW = Modified Ashworth scale

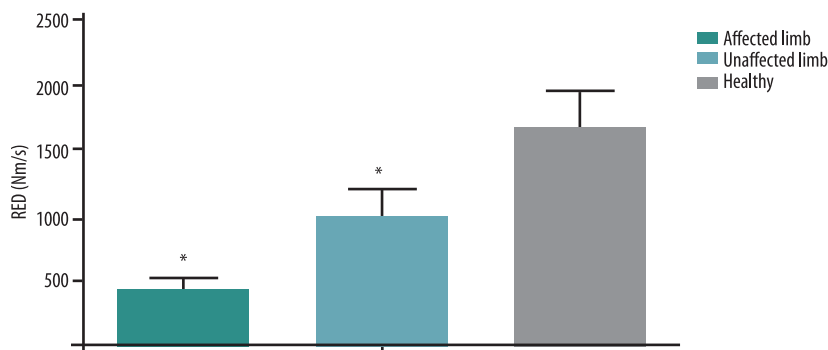
The affected limb showed significantly lower torque production compared with the unaffected limb ( $46.55 \pm 7.98$  Nm and  $84.29 \pm 8.47$  Nm, respectively;  $p \leq 0.001$ ). Healthy individuals ( $128.02 \pm 9.36$  Nm) were stronger in maximum isometric torque production than the affected ( $p \leq 0.001$ ) and unaffected limb ( $p \leq 0.001$ ) of stroke survivors. MVC results are shown in figure 1.



**Figure 1.** Maximum voluntary isometric contraction (MVC) of plantar flexors in affected and unaffected limbs of stroke survivors and dominant limb of healthy subjects. \*Difference to healthy limb; #Difference between affected and unaffected limbs. Data expressed as mean  $\pm$  ED.  $p < 0.05$ .

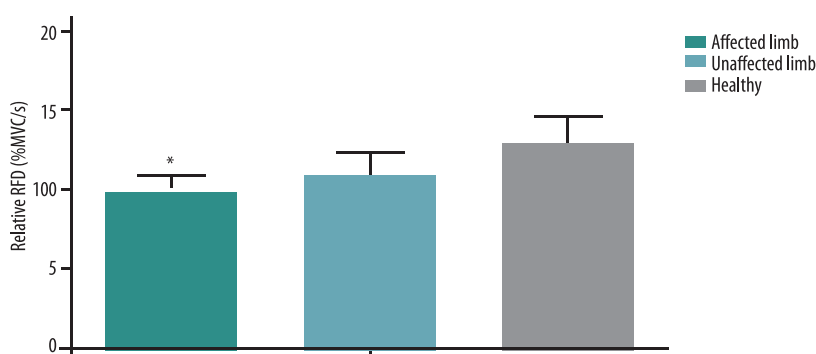
In relation to absolute RFD, affected limb ( $43.3 \pm 8.5$  Nm/s) and unaffected limb ( $98.9 \pm 20.4$  Nm/s) showed significant lower values ( $p \leq 0.01$  and

$p=0.04$ , respectively) compared to healthy limb ( $186.2 \pm 25.2$  Nm/s). On the other hand, there were no significant differences ( $p=0.15$ ) between affected and unaffected limbs of stroke survivors. Results are shown in figure 2.



**Figure 2.** Absolute rate of force development (RFD) of plantar flexors in affected and unaffected limbs of stroke survivors and dominant limb of healthy subjects. \*Difference to healthy limb; Data expressed as mean  $\pm$  ED.  $p < 0.05$ .

When normalized by MVC (relative RFD), affected limb ( $9.76 \pm 1.1$  %MVC/s) was significant lower compared to healthy individuals ( $13.08 \pm 1.5$  %MVC/s). There were no significant differences between unaffected limb ( $10.87 \pm 1.4$  %MVC/s) and healthy limb. Results of relative RFD are shown in figure 3.



**Figure 3.** Relative rate of force development (RFD) of plantar flexors in affected and unaffected limbs of stroke survivors and dominant limb of healthy subjects. \*Difference to healthy limb; Data expressed as mean  $\pm$  ED.  $p < 0.05$ .

High negative correlation ( $R = -0.717$ ;  $p = 0.003$ ) between MVC and spasticity level (ASW scale) and high negative correlation ( $R = -0.725$ ;  $p = 0.002$ ) between RFD and spasticity level were observed.

## DISCUSSION

The aim of this study was to investigate possible changes in muscle force production capacity and ankle joint power in order to identify functional impairment secondary to spasticity compared to healthy subjects. The findings demonstrate reduction in RFD and maximum force production of ankle muscles in spastic individuals but with no significant difference of muscle power between limbs of stroke survivors, which identify functional

changes in both affected and unaffected limbs in relation to healthy subjects.

In this context, results of previous studies have shown decreased RFD of 64%<sup>18</sup> and 70%<sup>19</sup> in spastic children compared to children with typical development, suggesting loss of muscle efficiency caused by spasticity. The decrease in RFD observed in affected and unaffected limbs demonstrated the lower speed with which maximum force can be generated by plantiflexor muscles; however, this difference was not observed between affected and unaffected limbs. Dissimilarly, Fimland et al.<sup>20</sup> found reduction in RFD of affected limbs compared to unaffected limbs in stroke survivors. The authors found reductions ranging from 54% to 67% between limbs and justify the findings due to low neuromuscular activity and atrophy promoted by the commitment time of 6.5 (0.8 to 20.9) years of stroke, on average. However, the present study showed longer stroke commitment time ( $7.4 \pm 5.8$  years), but the stroke commitment time may not be the proper justification. A previous study<sup>20</sup> also showed results of maximum contraction of plantiflexor muscles not normalized by the mass of each individual, which may have been responsible for differences among findings in these studies. In the same way, the spasticity level of participants evaluated by Fimland et al.<sup>20</sup> was not described, thus the difference in RFD can have other reasons.

The fact that the unaffected limb had lower absolute RFD compared to healthy subjects can be justified by functional and morphological differences caused by spasticity. Malaya et al.<sup>21</sup> observed lower medial gastrocnemius muscle length in the unaffected limb of hemiplegic cerebral palsy children ( $0.165 \pm 0.028$  m) compared to children with typical development ( $0.191 \pm 0.035$  m). The authors could not explain the results, but the present study reported reduction in RFD and in maximum force production, demonstrating the inability acquired by the unaffected limb in relation to healthy subjects, which could be a result of morphological or functional adaptation. While the unaffected limb can take higher level of daily activity resulting from increased functional dependence, spasticity leads patients to reduce their overall mobility, which results in decreased functional capacity of apparently healthy muscle<sup>22</sup>. In this context, further studies should be carried out in order to assess the relationship of the unaffected side with spasticity and possible adaptations.

RFD is an aspect influenced by several factors, among which fiber muscle length and thickness, fiber type and composition<sup>17,19</sup>, which are features that can be modified by spasticity. Kwah et al.<sup>8</sup> assessed spastic stroke survivors using ultrasound and observed lower length of gastrocnemius medialis (436 mm) and fascicle muscles (44 mm) compared to healthy subjects (444 and 50 mm, respectively). Other studies have also shown lower length of fascicle muscles in hemiplegic spastic individuals<sup>23,24</sup>. All authors related the lower length of fascicle muscles with increasing stiffness in muscle tissue due to shortened position of the joint and with muscle spasticity<sup>8,24</sup>. Friden and Lieber<sup>25</sup> had previously found lower lengths of spastic fascicle muscles *in vitro*, while Svantensson et al.<sup>26</sup> observed increased muscle stiffness in the affected limb compared to the unaffected limb in stroke survivors. The ratio

between fascicle length and RFD occurs through an increase in number of sarcomeres in series, indicated by fascicle length results in an increase of fiber shortening velocity, and consequently increase of RFD<sup>19</sup>. In addition, the concentration and type of fiber can be altered, histopathological studies found atrophy of type-2 muscle fibers in spastic stroke individuals<sup>27</sup>. This type of fiber is responsible for fast contraction, which can modify RFD.

Maximum torque is representative of the maximum capacity of an individual to generate force, and is associated with intrinsic muscle ability and muscle activation capacity<sup>28</sup>. The results of this study corroborate those obtained by Klein et al.<sup>11</sup>, who found lower torque values, by about one third, in plantarflexor muscles of the affected limb ( $56.7 \pm 57.4$  Nm) compared to unaffected limb ( $147 \pm 35.7$  Nm) in stroke survivors. Additionally, studies justify that neuromuscular weakness is a common finding in the affected limb of spastic hemiplegic individuals<sup>20,29</sup> and unaffected limb in relation to healthy subjects<sup>22,30</sup>, therefore, a decrease in force production resulting from spasticity was expected. Furthermore, maximum force production appears to be an important aspect in muscle power, since when RFD was normalized by MVC (relative RFD), the difference between affected and healthy limb was not statistically significant. Fimland et al.<sup>20</sup> explain that there can be no difference in the neuromuscular recruitment capacity between limbs, but morphological alterations such as muscle atrophy explain the difference observed in absolute and not in relative RFD on unaffected limb compared to healthy subjects.

The high negative correlation between spasticity level with RFD and maximum torque suggests the functional impairment of spastic individuals. The adaptations in reflex sensibility to which spinal motor neurons increase the muscle activation<sup>22</sup> associated with interrupt motor stimulation from the supraspinal centers developed by spasticity results in impairment of force modulation and production<sup>14</sup>. Thus, the ability to produce maximum force and/or muscle power is severely impaired by spasticity.

It is noteworthy that there are some limitations to consider in the findings of this study. Neuromuscular impairment due to spasticity is not restricted to proper muscle contraction but also to the sensory system, which participants reported loss of sensation and proprioception in the affected limb. The position on the dynamometer can have enhanced this limitation with the stretching of the posterior leg muscle. In addition, the level of muscle activation through electromyography (EMG) was not measured. It is suggested the use of EMG to analyze the alterations in motor recruitment and muscle activation.

## CONCLUSION

Spasticity promotes adaptations in muscle functional capacity, reducing its performance through the decreased rate of force development and maximum force production, affecting both affected and unaffected limbs. The high negative correlation between the rate of force development and maxi-



mum force production with spasticity level suggests that spastic muscles without appropriate intervention can become increasingly less functional.

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