Co-activation of upper limb muscles during reaching in post-stroke subjects: An analysis of the contralesional and ipsilesional limbs

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A B S T R A C T

The purpose of this study was to analyze the change in antagonist co-activation ratio of upper-limb muscle pairs, during the reaching movement, of both ipsilesional and contralesional limbs of post-stroke subjects. Nine healthy and nine post-stroke subjects were instructed to reach and grasp a target, placed in the sagittal and scapular planes of movement. Surface EMG was recorded from postural control and movement related muscles. Reaching movement was divided in two sub-phases, according to proximal postural control versus movement control demands, during which antagonist co-activation ratios were calculated for the muscle pairs LD/PM, PD/AD, TRIlat/BB and TRIlat/BR. Post-stroke’s ipsilesional limb presented lower co-activation in muscles with an important role in postural control (LD/PM), comparing to the healthy subjects during the first sub-phase, when the movement was performed in the sagittal plane (p < 0.05). Conversely, the post-stroke’s contralesional limb showed in general an increased co-activation ratio in muscles related to movement control, comparing to the healthy subjects. Our findings demonstrate that, in post-stroke subjects, the reaching movement performed with the ipsilesional upper limb seems to show co-activation impairments in muscle pairs associated to postural control, whereas the contralesional upper limb seems to have signs of impairment of muscle pairs related to movement.

Keywords: Stroke; Reaching; Antagonist co-activation ratio; Ipsilesional limb

1. Introduction

Disorders of the central nervous system often result in conditioned motor function as a consequence of atypical patterns of muscle recruitment. Besides being the leading cause of persistent neurological impairment, stroke causes seriously functional limitations, being considered a major health problem with enormous economic implications in several countries all over the world (WHO, 2011).

Currently, there are several accepted rehabilitation approaches based on different principles (Tate, 2006). However, we believe that it is imperative that physiotherapists perform a movement analysis-based clinical reasoning for designing a subject-directed rehabilitation intervention. Therefore, understanding the mechanisms related to atypical versus typical movement pattern is an emerging need, so that decisions regarding the most appropriate rehabilitation strategies can be taken. Moreover, in order to support the clinical reasoning, motor performance assessment should rely on standard and easy applicable instruments. However, standardized clinical motor performance assessment scales are not sensitive enough for the measurement of certain quantitative features (e.g., intersegment coordination, quality and smoothness of movement), not providing accurate information about motor synergies (Patel et al., 2010).

Despite the difficulty of incorporating reliable assessment instruments amongst clinical setting scenarios, surface electromyography (sEMG) seems to represent a valid and relatively easy-to-use tool for motor performance assessment. In fact, signal acquisition through sEMG allows an extensive and in-depth characterization of motor control patterns, which is extremely important to orientate the clinical reasoning process (Hughes et al., 2009, 2010).
In stroke patients, the contralesional motor deficits have been extensively described (Bobath, 1990; Bourbonnais et al., 1989; Burke, 1988). Nonetheless, based on the current neuroscience knowledge regarding the neurophysiology of movement organization, ipsilesional deficits should also exist, although these have been less described among the clinical and research community (Desrosiers et al., 1996; Sunderland et al., 1999). Indeed, in an unilateral stroke, with the brain lesion located at a sub-cortical level (such as the frequently affected internal capsule), it is highly probable that the lesion interferes with the neuronal connection between the motor cortex and the reticular formation, which clearly justifies an ipsilesional postural control dysfunction (Schepens, 2004; Schepens et al., 2008). Considering the ventromedial system disposal, which, although bilateral, presents a predominant ipsilateral projection, the ipsilesional deficits can no longer be ignored. Thus, research about the motor behavior of the ipsilesional upper limb, should not only address the hand’s dexterity, but proximal components as well (i.e. trunk, shoulder and elbow), which constitutes a still unexplored field.

Given that the major role of the ventromedial system is associated with postural control, it is expected to find signs of dysfunction in stability-related muscles. The inherent specificity of postural control study imposes some demands about the selection of the task and the respective sub-phases, as well as the muscles to be studied. For example, to study the reaching movement it becomes important to assess latissimus dorsi and pectoralis major, as opposed to those mainly related to movement execution, such as deltoid or triceps (Dickstein et al., 2004; Geuze, 2005; Zattara et al., 1988). The importance of the evaluation of postural control muscles during movement tasks is sustained by the evidence that purposeful and orientated distal movement requires the ability to recruit proximal stability (Champion et al., 2009; Shumway-Cook et al., 2007; Yanga et al., 2002). In this sense, the reach movement can be divided in two sub-phases: (a) the first sub-phase, here named as the elbow flexion phase, which includes the period from the movement’ beginning until the maximum elbow flexion; this phase requires a highly demanding proximal postural control; and (b) the second sub-phase, here named as the shoulder flexion phase, starts when the elbow reaches its maximum flexion and ends when the target is reached; this phase is predominantly movement demanding (Fig. 1).

As previously stated, the postural control dysfunction occurring at the ipsilesional side of stroke subjects can only be considered when lesions interfere with the cortico-reticular network. At the neuromuscular level, the deregulation of neurophysiologic mechanisms, such as the reciprocal innervation, is one of the alterations that can exist, being expressed with altered co-activation levels between muscle pairs – (antagonist co-activation ratio). Indeed, the mechanism of reciprocal innervation has been highlighted as one of the altered mechanisms following stroke, mainly in the form of high levels of antagonist co-activation ratio (Hammond et al., 1988; Stoeckmann et al., 2009). Despite the large amount of research about co-activation ratios in stroke subjects (Fellows et al., 1994; Higginson et al., 2006; Lamontagne et al., 2002, 2000; Stoeckmann et al., 2009), the study of the behavior of the muscle pairs related to postural versus movement functions has not been explored so far. The researches in this area are mainly focused on an analytical study of shoulder and/or elbow muscle pairs activity, often demonstrating higher levels of co-activation ratios in the contralesional side. However, these studies did not considered the different motor control dimensions (postural control and movement), and because of that, only movement related muscles have been studied (el-Abd et al., 1993; Gowland et al., 1992; Stoeckmann et al., 2009) (Fellows et al., 1994). To the best of our knowledge, no study has assessed the functional behavior of postural control and movement control related muscles in subjects with postural control and movement control dysfunction, such as in the case of post-stroke subjects with lesions at sub-cortical level at the middle cerebral artery (Matsuyama et al., 2004; Silva et al., 2012a,b; Sousa et al., 2013).

Therefore, the purpose of this study was to analyze the change in antagonist co-activation ratio of upper-limb muscles pairs of either ipsilesional and contralesional limbs of stroke subjects during the reaching movement.

2. Methods

2.1. Participants

Eighteen subjects, included in two distinct groups (healthy, n = 9, mean age: 52.3 ± 4.9, mean body mass index: 26.1 ± 1.8; and post-stroke, n = 9, mean age: 55.0 ± 9.6, mean body mass index: 27.1 ± 4.0), participated in this study. The protocol was approved by the Ethics Committee of Escola Superior de Tecnologia da Saúde do Porto (ESTSP). The investigation conforms to the principles outlined in the Declaration of Helsinki.

For general group’s inclusion, subjects must be above 45 years old. Exclusion criteria comprised musculoskeletal pathology, neck and/or upper limb pain, cerebellar, basal ganglia or brain stem lesions, and a Mini Mental State Examination score below 25.

For the post stroke group inclusion, participants must meet the additional following criteria: unilateral stroke at the subcortical...
middle cerebral artery territory, confirmed by neuroimaging; a
time evolution up to 6 months and a score between 30 and 50 of
the Fugl-Meyer Assessment Scale (moderately impaired) (Scheidt
et al., 2007). As exclusion criteria were considered: hemispatial
neglect; visual (uncorrected), perceptual or cognitive deficits;
and active range of motion of the contralateral shoulder and
eyb joints inferior to 15° (Zackowski et al., 2004).
Post-stroke group’s characterization, namely age, gender,
height, weight, time post stroke and location of lesion is presented
in Table 1.

2.2. Instruments
Surface electromyography (sEMG) was recorded using two
bioPLUX® (Plux, Portugal) devices with a sampling frequency of
1000 Hz, common mode rejection ratio 110 dB, input impedance
greater than 100 MΩ and a 12 bits analog-to-digital signal conver-
sion. The bipolar sensor configuration was selected and auto-
dhesive pediatric electrodes were used (Correia et al., 2004;
Matias et al., 2006). The sEMG signals were analyzed through the
Ariel Dynamics Inc., Canyon, USA), version 12.1.0.10, was used
for data analysis. This software allows to analyze data captured
with SENIAM references, Table 2 (Hermens et al., 2000). Electrode
placement was confirmed by voluntary muscle contraction.
Ground electrodes were placed over both olecraneums (Correia
et al., 2004; Hermens et al., 2000).
In the present study, kinematic analysis was only used to assess
movement onset and offset, as well as to assess maximum elbow
flexion. The selected acquisition setup involved the placement of
the cameras at a 4 m distance from the subject, two in the frontal
plane and two at a 45° between the frontal and the sagittal planes
from each shoulder (Vandenbergh et al., 2010).
The EMG and kinematic data were synchronized by a simulta-
neous trigger input signal (led).

2.4. Data processing
Kinematic data was digitally low-pass filtered using a second
order Butterworth filter with a cutoff frequency of 7 Hz, and ana-
lized in terms of hand peak velocity and maximum elbow flexion.
Movement onset and offset were defined as the times at which third
metacarps’ tangential velocity exceed or fell below 10% of its
maximal value, respectively (Cirstea and Levin, 2000). Maximum
elbow flexion was defined as the lowest angle between the arm
and forearm segments.
The EMG data was offshore processed with AcqKnowledge 3.9.0
software (Biopac Systems, Inc., California, USA). Signals were
band-pass filtered at 20–500 Hz, amplified and root-mean-square
(RMS) processed for consecutive segments of 100 ms. Muscular
onset was determined as the time when the EMG activity exceeded
the baseline for three standard deviations, for a minimum period of
50 ms (Hodges et al., 1996).
The reach movement was divided into two sub-phases, using the
kinematic data. The first sub-phase was defined as the interval
between movement onset (velocity exceeded 10% of its maximum
during the task), until maximum elbow flexion, in this sub-phase
of movement performed at the sagittal plane, the glass was
positioned in front of the ipsilateral shoulder, and when evaluating
the scapular plane it was placed, also ipsilateraly, at 30° from
the frontal plane. Subjects were instructed, after a verbal cue, to
reach and grasp the glass. Three valid repetitions were executed, with
an interval of one minute each. All the verbal commands were given
equitatively and by the same researcher.

Surface EMG was recorded from seven trunk and upper-limb
muscles, namely the latissimus dorsi (LD), sternal head of pectoral-
is major (PM), anterior (AD), and posterior (PD) segments of del-
toid, biceps brachii (BB), brachioradialis (BR), and triceps brachii
lateral head (TRIlat).
Prior to data collection, the skin was shaved and wiped down
with alcohol, after which skin impedance was measured and con-
firmed less than 5 kΩ. Disposable pediatric Ag/AgCl electrodes,
with a skin contact surface of 10 mm², and inter-electrode distance
of 20 mm, were placed parallel to the muscle fibers and according
with SENIAM references, Table 2 (Hermens et al., 2000). Electrode
placement was confirmed by voluntary muscle contraction.

Table 1
Post-stroke group characterization regarding age, gender, weight (kg), height (m), time post stroke (years) and location of lesion.

<table>
<thead>
<tr>
<th>Age/gender</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>E</th>
<th>F</th>
<th>G</th>
<th>H</th>
<th>I</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight</td>
<td>82</td>
<td>105</td>
<td>70</td>
<td>75</td>
<td>60</td>
<td>68</td>
<td>68</td>
<td>69.3</td>
<td>74</td>
</tr>
<tr>
<td>Height</td>
<td>1.70</td>
<td>1.69</td>
<td>1.63</td>
<td>1.75</td>
<td>1.63</td>
<td>1.68</td>
<td>1.65</td>
<td>1.58</td>
<td>1.63</td>
</tr>
<tr>
<td>Time post stroke</td>
<td>1</td>
<td>8</td>
<td>1</td>
<td>1</td>
<td>4</td>
<td>5</td>
<td>2</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>Location of lesion</td>
<td>LMCA</td>
<td>RMCA</td>
<td>RMCA</td>
<td>LMCA</td>
<td>LMCA</td>
<td>RMCA</td>
<td>LMCA</td>
<td>RMCA</td>
<td>RMCA</td>
</tr>
</tbody>
</table>

A-I represent each subject; M – male; F – female; LMCA – left medial cerebral artery; RMCA – right medial cerebral artery.
between maximum elbow flexion until movement offset (velocity fell below 10% of its maximum during the task), where shoulder girdle and elbow muscles act to produce upper limb forward displacement. In each sub-phase, the antagonist co-activation ratios of the muscle pairs (LD/PM, PD/AD, TRIlat/BB, TRIlat/BR) were evaluated. The antagonist co-activation ratio was calculated according to (Kellis et al., 2003):

\[
C(\%) = \frac{\text{antagonist activity}}{\text{agonist + antagonist activity}} \times 100
\]

This approach provides an estimate of the relative activation of the pair of muscles, as well as the magnitude of the co-activation. Considering that differences in upper limb motor patterns have been demonstrated between dominant and non-dominant limbs, and that post-stroke subjects tend to use the ipsilesional limb as dominant limb, the values obtained from the ipsilesional limb were compared to those obtained from the dominant limb while the values obtained from the contralesional limb were compared to those obtained from non-dominant limb (Sainburg et al., 2000; Sainburg et al., 2004; Sainburg, 2002).

### 2.5. Statistics

Statistical Analysis was performed using IBM SPSS Statistics software v20 with a significance level of 0.05. Due to the small number of participants (n = 9 per group), Mann–Whitney U test was used to compare healthy versus post-stroke groups, and Friedman ANOVA test, with Dunn’s test as Post-Hoc, to compare sides within each group. Median and interquartile deviation were used as descriptive statistics (Marôco, 2010).

### 3. Results

The following results characterize the antagonist co-activation ratios of the muscle pairs LD/PM; AD/PD; TRIlat/BB and TRIlat/BR of the ipsilesional limb of post-stroke subjects compared to the dominant limb of the healthy group and the contralesional limb compared to the non-dominant limb. Still, no statistical differences were observed between dominant and non-dominant limbs of healthy subjects (data not shown).

#### 3.1. Antagonist co-activation ratios in the ipsilesional limb of post-stroke subjects in the first sub-phase of the reaching movement

In the first sub-phase of the reaching movement, the antagonist co-activation ratio of the muscle pair LD/PM was significantly lower in the post-stroke’s ipsilesional limb, in relation to the dominant limb of healthy subjects, when the movement was performed on the sagittal plane (p = 0.04). Conversely, on the scapular plane of movement no statistical differences (p > 0.05) were observed. The antagonist co-activation ratios of the other muscle pairs (PD/AD, TRIlat/BB and TRIlat/BR) did not evidenced a statistical different (p > 0.05) behavior between both groups (Table 3).

#### 3.2. Antagonist co-activation ratios in the contralesional limb of post-stroke subjects in the first sub-phase of the reaching movement

In the first sub-phase of the reaching movement, the contralesional limb showed a significant increase of the antagonist co-activation ratio of the muscle pair TRIlat/BB when the movement was performed on the sagittal plane (p = 0.02). Conversely, no significant differences (p > 0.05) of the antagonist co-activation of the muscle pairs LD/PM, PD/AD and TRIlat/BR were detected between the contralesional limb of post-stroke subjects and the non-dominant limb of healthy subjects for both sagittal and scapular planes (Table 3).

#### 3.3. Antagonist co-activation ratios in the ipsilesional limb of post-stroke subjects in the second sub-phase of the reaching movement

In the second sub-phase of the reaching movement, no statistical differences (p > 0.05) were observed between the ipsilesional limb of post-stroke subjects and the dominant limb of the healthy group in all of the studied muscle pairs, namely LD/PM, PD/AD, TRIlat/BB and TRIlat/BR (Table 4).

#### 3.4. Antagonist co-activation ratios in the contralesional limb of post-stroke subjects in the second sub-phase of the reaching movement

In the second sub-phase of the reaching movement, the contralesional limb of post-stroke subjects presented higher antagonist co-activation ratios of the muscle pairs LD/PM (p = 0.019), in the sagittal plane of movement, and of the PD/AD pair in both sagittal (p = 0.008) and scapular (p = 0.04) planes of movement, comparing to non-dominant limb of healthy subjects. The other analyzed muscle pairs, TRIlat/BB and TRIlat/BR showed no statistical differences (p > 0.05) (Table 4).

### 4. Discussion

#### 4.1. Antagonist co-activation ratio in the ipsilesional limb of post-stroke subjects

In this study we demonstrated that the ipsilesional limb of post-stroke subjects shows an altered antagonist co-activation ratio of the LD/PM muscle pair in the first sub-phase of the reaching movement, when performed in the sagittal plane. Moreover, no significant differences were observed between the ipsilesional limb of post-stroke subjects and the dominant limb of healthy subjects in the second sub-phase of the movement, in both sagittal and scapular planes. These findings are in agreement with our postulated hypothesis, i.e., that the ipsilesional limb of post-stroke subjects presents a motor dysfunction of postural muscles. Given that the first sub-phase of the reaching task imposes a higher demand of the proximal postural control, in relation to the second sub-phase that imposes higher demands in terms of proximal mobility, the different behavior between the ipsilesional side of stroke subjects and healthy subjects may be explained by the dysfunctional neuronal systems that present mostly an ipsilateral disposal/projection. Moreover, the fact that stroke subjects evidenced subcortical lesions compatible with alterations of the cortical-reticular circuits reinforces this possibility. Concerning the upper-limb analysis, little or no evidence corroborant with these findings.

### Table 2

Anatomical references used to locate the electrodes. Electrode locations were confirmed by palpation of the muscular belly with the subject in the test position.

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Electrode placement</th>
</tr>
</thead>
<tbody>
<tr>
<td>LD</td>
<td>One centimetre laterally to the lateral edge of the scapula</td>
</tr>
<tr>
<td>PM</td>
<td>Two fingers below the collarbone and at two fingers from the sternum</td>
</tr>
<tr>
<td>AD</td>
<td>One finger width distal and anterior to the acromion, in the direction of the line between the acromion and the thumb</td>
</tr>
<tr>
<td>PD</td>
<td>Two finger width behind the angle of the acromion, in the direction of the line between the acromion and the little finger</td>
</tr>
<tr>
<td>BB</td>
<td>On the line between the medial acromion and the fossa cubiti</td>
</tr>
<tr>
<td>BR</td>
<td>At 1/3 from the fossa cubiti</td>
</tr>
<tr>
<td>TRIlat</td>
<td>At 50% on the line between the posterior crista of the acromion and the olescranon at 2 finger widths lateral to the line</td>
</tr>
<tr>
<td>Ground</td>
<td>Both olecraneums</td>
</tr>
</tbody>
</table>
Antagonist co-activation ratio in the first sub-phase of reach movement. Median values and interquartile range of the co-activation ratio in both sagittal and scapular planes are presented for the dominant limb of healthy subjects versus the ipsilesional limb of post-stroke subjects and the non-dominant limb of healthy subjects versus the contralateral limb of post-stroke subjects, for LD/PM, PD/AD, TRlat/BB and TRlat/BR muscle pairs.

<table>
<thead>
<tr>
<th>Muscle pairs</th>
<th>Healthy dominant limb</th>
<th>Ipsilesional limb</th>
<th>p-value</th>
<th>Healthy non-dominant limb</th>
<th>Contralateral limb</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sagittal plane</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LD/PM</td>
<td>57.7(±11.4)</td>
<td>46.0(±8.6)</td>
<td>0.04</td>
<td>46.1(±9.7)</td>
<td>54.1(±8.2)</td>
<td>0.094</td>
</tr>
<tr>
<td>PD/AD</td>
<td>21.0(±9.7)</td>
<td>23.7(±7.3)</td>
<td>0.546</td>
<td>20.3(±11.7)</td>
<td>39.8(±16.2)</td>
<td>0.190</td>
</tr>
<tr>
<td>TRlat/BB</td>
<td>41.8(±12.0)</td>
<td>42.4(±14.3)</td>
<td>0.489</td>
<td>30.6(±6.48)</td>
<td>46.4(±11.2)</td>
<td>0.02</td>
</tr>
<tr>
<td>TRlat/BR</td>
<td>35.0(±7.6)</td>
<td>26.5(±14.1)</td>
<td>0.796</td>
<td>34.9(±21.9)</td>
<td>54.0(±20.1)</td>
<td>0.340</td>
</tr>
<tr>
<td><strong>Scapular plane</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LD/PM</td>
<td>56.7(±8.8)</td>
<td>56.7(±11.6)</td>
<td>1.000</td>
<td>54.3(±10.3)</td>
<td>59.0(±4.4)</td>
<td>0.136</td>
</tr>
<tr>
<td>PD/AD</td>
<td>41.8(±11.9)</td>
<td>36.3(±18.9)</td>
<td>0.937</td>
<td>31.8(±21.4)</td>
<td>35.1(±18.5)</td>
<td>0.0605</td>
</tr>
<tr>
<td>TRlat/BB</td>
<td>47.0(±12.3)</td>
<td>42.3(±12.3)</td>
<td>0.863</td>
<td>40.8(±8.3)</td>
<td>46.0(±14.4)</td>
<td>0.258</td>
</tr>
<tr>
<td>TRlat/BR</td>
<td>32.9(±10.7)</td>
<td>27.9(±13.0)</td>
<td>0.546</td>
<td>34.2(±18.8)</td>
<td>55.5(±20.4)</td>
<td>0.065</td>
</tr>
</tbody>
</table>

4.2. Antagonist co-activation ratio in the contralateral limb of post-stroke subjects

Concerning the analysis of antagonist co-activation ratios at the contralateral side, our results are in agreement with the widespread scientific evidence that stroke subjects exhibit atypical co-activation levels between muscle pairs, when compared to healthy subjects (Higginson et al., 2006; Lamontagne et al., 2002, 2000). However, it is important to explore this overall tendency to higher co-activation levels, relating the role of the muscle pairs according to the movement sub-phase. So, at the first sub-phase of the movement, statistical differences between groups were only observed in the muscle pair TRlat/BB, which is considered to be the mobility pair for the elbow joint. Indeed, at this sub-phase, the elbow flexion demands the co-activation of these muscles (flexors/extensors), which is, in these participants, altered. This finding may be explained by the lesion at the corticospinal system, which influences the activity of agonist muscles. Since impairment of this system is associated to lower muscle activation capacity during movement tasks, the increased antagonist co-activation ratio can be sustained by this influence.

When analyzing the second sub-phase, and according with the above stated, it is important to understand that, since at this sub-phase, the shoulder is the joint which contributes the most to provide the upper-limb displacement towards the goal, the muscles at this joint need to be considered mainly mobility orientated. So, statistical differences were only observed in the muscle pairs LD/PM (at the sagittal plane) and PD/AD (at both planes). It is important to highlight that although proximal muscles frequently assume a stability role, the reaching gesture incorporates continuous shifts in their roles, according to the upper-limb’s segment presenting higher mobility.
4.3. Limitations

Despite, like other authors (Lamontagne et al., 2000), we opted to use absolute EMG levels, this methodological decision has as drawback the fact that, as we did not measure the thickness of the subcutaneous fat layer, we cannot guarantee that this variable did not influence the co-activation levels obtained. Nevertheless the EMG normalization in post-stroke subjects can be questionable given the abnormal EMG pattern frequently exhibited by these subjects. As a consequence, the EMG normalization in post-stroke subjects can lead to distortion of co-activation values and to higher variability (Yang et al., 1984).

4.4. Clinical implications

These results should be taken into account in the rehabilitation field, i.e., clinicians should be aware of the fact that the ipsilesional side of stroke subjects may also present a motor control dysfunction. For this reason ipsilesional side of stroke subjects should not be overlooked and involved in the rehabilitation strategy. On the other hand, the possible adverse influence of the atypical behavior found in the ipsilesional side (regarding postural control function) over the contralesional side has not yet been fully explored. The pertinence of such (to be addressed in future research) is based on the knowledge that the type II fibers present bilateral connections, justifying thus, the possible interdependence between both sides of the stroke subjects (Jankowska et al., 2005).

5. Conclusion

Our findings demonstrate that, in post-stroke subjects, the ipsilesional upper limb might show signs of postural control dysfunction, whereas the contralesional upper limb might have signs of movement dysfunction. Although further research is still needed, the global motor dysfunction after an unilateral brain lesion has been gaining evidence.

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