Anticipatory Postural Adjustments During Seated Reaching Movements in Post-Stroke Subjects

Abstract:

The study assessed the effect of velocity of arm movement on the generation of APAs in the contralateral and ipsilateral muscles of individuals with stroke in the sitting position. In the sitting position, 10 healthy and 8 post-stroke subjects reached for an object placed at the scapular plane and mid-sternum height at self-selected and fast velocities. Electromyography was recorded from the anterior deltoid (AD), upper (UT) and lower trapezius (LT), and latissimus dorsi (LD). Kinematic analysis was used to assess peak velocity and trunk displacement. Post-stroke subjects presented a delay of APAs on both sides of the body compared to healthy subjects. Differences were found between the timing of APAs on the ipsilateral and contralateral LD and LT in both movement speeds and in the ipsilateral UT during movement of the non-affected arm at a self-selected velocity. A delay in the contralateral LD in the reaching movement with the non-affected arm at fast velocity was also observed. Trunk displacement was greater in post-stroke subjects. In the sitting position, APAs were delayed in both fast and self-selected movements on both sides in post-stroke subjects, which also presented a higher trunk displacement.

Introduction

The central nervous system (CNS) counteracts the expected mechanical effects of the perturbation induced by movement in a feedforward manner through anticipatory postural adjustments (APAs), which are changes in the background muscle activity (Shiratori & Aruin, 2004; Bonnefoy, Louis & Gorce, 2009). Their main goal is to assist motor performance in order to minimize the pertrubation of balance. The behavior of APAs can be described by a temporal parameter that has been vastly explored in studies related to postural control and is involved with the timing of muscular activation (Van der Fits et al., 1998). Regarding the "time window" of APAs, these adjustments occur 100 milliseconds (ms) before to 50 ms after the prime mover's activation (Yoshida et al., 2008).

The generation of APAs can be challenging in persons with neurologic disorders such as stroke (Aruin, 2002). Following a stroke in the middle cerebral artery territory, damages can occur in the internal capsule (Miyai et al., 1999), leading to cortico-pontine network impairment, which involves the output of supplementary motor areas related to the temporal aspect of APAs. Yet studies concerning APAs in post-stroke subjects are scarce. Current evidence suggests that these subjects have a delay in APAs on the paretic side compared to healthy subjects (Slijper et al., 2002). However, the unilateral nature of APA impairment is doubtful and still unclear due to the bilateral vs. ipsilateral disposal of neuronal systems to axial and trunk muscles (Dickstein et al.,

2004). Owing to the bilateral impairment of APAs, post-stroke subjects may not be prepared for the environmental changes and for the constant disturbances of the center of mass during voluntary movement, affecting their functionality.

Beyond neurological diseases, other factors influence APA behavior, such as posture and movement velocity (Aruin & Shiratori, 2003). In the seated position, which is the most-frequent postural set for reaching activities in post-stroke subjects, the center of mass is closer to the base of support. Therefore, keeping the projection of the center of mass within the limits of the basis of support (Aruin & Shiratori, 2003) is less challenging, which could lead to a decrease of APAs (Yoshida et al., 2008). In addition, the APAs seem to be larger when the movement velocity is higher (Yoshida et al., 2008). Furthermore, according to Mochizuki et al. (2004), the increase of velocity of arm movement leads to a higher modulation of the amplitude with lower modulation of the timing of APAs. Nevertheless, the velocity's influence on the timing of APAs remains controversial and understudied, particularly in respect to post-stroke subjects.

It is known that APAs play an important role in controlling flexor movement and trunk orientation prior to arm movement (Cirstea & Levin, 2000; Lee et al., 2009). However, post-stroke subjects exhibit an excessive trunk displacement while performing a reach movement, which could result from APA impairment or elbow extension, shoulder flexion, or adduction impairment (Levin et al., 2002).

The present study aims to assess the effect of velocity of reach movement while sitting on APA generation in the contralateral and ipsilateral muscles of post-stroke individuals; to compare the APAs of the ipsilateral and contralateral sides to the affected hemisphere in post-stroke subjects with healthy subjects; and to compare the trunk displacement of post-stroke individuals with healthy individuals. Based on the exposed, it is hypothesized that the APAs in post-stroke subjects would be delayed on both sides of the body compared to those of healthy individuals and at all speeds. It would also be expected that trunk displacement is higher in post-stroke subjects.

Methods

Sample

This cross-sectional observational study involved two groups. Group 1 included healthy controls (n=10) and group 2 consisted of individuals with stroke (n=8). Group 1 included 3 males and 7 females (age 51.5±5yrs; height 1.64±0.085m; weight 72.5±11.1kg; medina ± interquartile deviations). The inclusion criterion for group 1 was subjects older than 45 years, and exclusion criteria were the presence of uncorrected visual deficits, neuromusculoskeletal pathologies, and/or pain lasting more than 3 months in the neck and/or shoulder. Group 2 included 2 females and 6 males (age 60.5±5yrs). Table 1 provides a description of the sample. Post-stroke subjects were recruited from three health institutions in Northern Portugal. Inclusion criteria for group 2 were as follows: the presence of subcortical lesions after stroke in the MCA territory as confirmed by computed tomography and having occurred within at least six months; the ability to remain sitting without support; an active range of motion of at least 15° on the shoulder and elbow of the affected arm; and the capacity to follow simple

instructions. Exclusion criteria included the presence of Parkinson's disease, previous stroke, uncorrected visual deficits, apraxia, hemineglect, neuromusculoskeletal pathologies, pain lasting more than three months in the neck and/or shoulder, and a score below 25 in the Mini Mental Test.

Instruments

Modified Barthel Index was validated for the Portuguese population by Lima et al. (1998) (Santos et al., 2005).

Surface electromyography (sEMG) was recorded through two bioPLUX® (Plux, Portugal) devices with a sampling frequency of 1000Hz, common mode rejection ratio 110 dB, input impedance greater than 100 MoMs and analog channels with 12 bits. For sEMG, bipolar sensor configuration was selected and child ECG electrodes were used. The electrodes were Ag/AgCl, circular, with 10 mm of diameter and auto-adhesive (Hermens et al., 2000). The sEMG signals were analyzed through the AcqKnowledge Analysis Software version 3.9 (Biopac Systems, Inc., Goleta, USA).

Skin impedance was measured using the Noraxon® Impedance Checker system (Noraxon, Scottsdale, Arizona).

Kinematic data were recorded using the four-camera Qualisys Motion Capture System (sampling frequency: 100Hz) and analyzed using Qualisys Track Manager (QTM) Software (Qualisys, Sweden). Kinematic analysis with QTM was synchronized with the sEMG.

Procedures

The assessment was performed with subjects in a sitting position without trunk support and with the feet flat on the floor. The seat height was normalized, placing it at a height equivalent to each participant's lower limb length, measured from the lateral line of the knee joint to the ground with the participant standing. At the initial position, 75% of the thigh length (measured from the lateral knee-joint line to the greater trochanter) was supported on the seat. In the starting position, the participants had their knees and hips at approximately 90° of flexion, their shoulders in a neutral position, and their hands resting on their hips.

Subsequent to skin preparation and impedance measurement, the electrodes were placed over the anterior deltoid (AD) (prime mover), upper trapezius (UT), lower trapezius (LT), and latissimus dorsi (LD), with an inter-electrode distance of 20 mm (Hermens et al., 2000). Reference electrodes were placed above the olecranon, and electrodes were placed parallel to the muscle fibers and in accordance with SENIAM references to the AD, UT, and LT muscles (figure 1). Regarding the LD, the electrodes were placed obliquely 4 cm below the inferior angle of the scapula, more precisely at the muscular curve at the T2 level and along the line from the posterior axillary fold to the spinous process of S2 (Swinnen at al., 2012). Then, electrodes were connected to two bioPLUX® devices. Reflective markers were placed over the radial styloid apophysis, lateral epicondyle, acromion, and at the mid-sternum point.

The task consisted of reaching for a water bottle containing 0.5 L water, placed in the scapular plane (30º anterior to the frontal plane) and at mid-sternum height (Michaelsen et al., 2001; Faria et al., 2008). The bottle was placed at a distance corresponding to arm length measured from the acromion to the metacarpophalangeal joint of the forefinger. The participant touches the bottle, returning to the initial position without the bottle. The movement was performed by each arm separately. After a verbal instruction, the participant was requested to reach for the object. For the fast movement, the participant was asked to reach for the bottle as fast as possible. Three trials, separated by 2 min of rest, were performed at self-selected and fast velocities.

Data processing

Offline processing was performed with Acknowledge 3.9 software (Biopac Systems, Inc., Goleta, USA). Signals were filtered with a bandpass filter of 20Hz and 450Hz and rectified. The timing of the EMG activity onset was defined as a moment of time in which the EMG activity exceeded the baseline activity for two standard deviations for a minimum period of 30 ms. Baseline activity consisted of the mean EMG activity measured in a range of 500 ms before the event. Thus, the timing of the EMG activity onset consisted in the average value obtained by the difference between the time of the EMG onset of the UT, LT, and LD, and the onset of the AD activity in the three trials (Dickstein et al., 2004).

Prior to the recording process, each of the reflective markers was identified and named in QTM and reviewed to guarantee that the markers were tracked precisely during the data capture. All marker data were low-pass filtered using a Butterworth filter with a cut-off frequency of 6 Hz. After analyzing the graphical plots from the recordings, the kinematic data analysis was focused on: the peak velocity of the radial marker and the distance travelled of the mid-sternum marker that served to calculate the trunk displacement (Levin et al., 2002; Alt Murphy et al., 2006). The distance travelled by the mid-sternum marker was analyzed only from 100 ms before until 50 ms after the AD activation.

Ethics

The ethics committee of the School of Allied Health Sciences of Porto (ESTSP) approved the study, and all the participants signed an informed consent based on the Helsinki Declaration.

Statistics

Predictive Analytics Software (PASW) Statistics 18 (IBM SPSS, Chicago, USA) with a confidence interval of 95% ($\alpha = 0.05$) was used. The Shapiro-Wilks test was applied in the variables of group 1. Then, t-test for paired samples for the intra-group comparison of timings of EMG activity onset of group 1was applied. To compare the variables of group 1 that did not follow the normality and to perform the intra-group comparison of group 2, the Wilcoxon test was used. The Mann-Whitney test was performed for the inter-group comparations. Spearman correlation was used to verify the correlation between trunk displacement and timings of EMG activity onset (SPSS, 2007).

Results

In group 1, the movement at the self-selected velocity presented an average peak velocity of 1.654 ± 0.24 m/s and 1.594 ± 0.363 m/s when performed by the dominant limb (DL) and non-dominant limb (NDL), respectively, while the fast movement was executed at 2.306 ± 0.31 m/s by DL and 2.296 ± 0.350 m/s by NDL. The average speeds of self-selected movements in group 2 were 0.881 ± 0.164 m/s and 0.99 ± 0.278 m/s when executed by the affected limb (AL) and the non-affected limb (NAL), respectively. The fast movement presented an average peak velocity of 1.028 ± 0.162 m/s when performed by AL and 1.448 ± 0.412 m/s by NAL.

Timing of EMG activity onset in healthy individuals

The timings of EMG activity onset of the ipsilateral muscles were not different from those of the contralateral muscles regardless of limb handedness and movement velocity, suggesting that handedness did not have an influence on the timing of APAs. No differences were found in the velocity of movement with limb dominance.

Timing of EMG activity onset in post-stroke individuals

Tables 2 and 3 present the median (Md) and interquartile deviations (IQR) of the timing of EMG activity onset during movements performed at self-selected and fast velocities by affected and non-affected limbs, respectively.

Regarding post-stroke subjects, a delay was verified in EMG activity onset of LD contralateral to the movement compared to the ispsilateral LD during the fast movement of the non-affected limb (table 2). However, no differences were found between ipsilateral and contralateral muscles to the movements of the affected limb and to the movement performed at self-selected velocity by the non-affected limb.

Intergroup comparison of the timings of EMG activity onset during reach movement performed at self-selected velocity

Because no differences were found in the timings of EMG activity onset with the limb handless in group 1, the timings of EMG activity onset during the movement performed at self-selected velocity by the dominant limb were selected to compare the timings of EMG activity onset of post-stroke subjects with those of the healthy group.

During reach movement with the affected limb at a self-selected velocity, post-stroke subjects presented a delay on EMG activity onset of LD contralateral (U=8; p=0.003) and ipsilateral (U=7.5; p=0.002) to the movement compared to group 1. A delay in EMG activity onset was also found in contralateral (U=17; p=0.037) and ipsilateral (U=17; p=0.037) LT, compared to healthy individuals (Figure 2). Figure 2 represents the Md and IQR as well as the p value and Mann-Whitney test used to compare the timings of APAs during the movement of the dominant limb in group 1 and the affected limb in group 2.

A delay on APAs was also confirmed in the movement executed at a self-selected velocity by the non-affected limb, as shown in figure 3. This delay was verified at the onset of EMG activity of the ipsilateral (U=1; p<0,001) and contralateral (U = 11; p = 0.033) LD, ipsilateral (U=18; p=0.046) and contralateral (U=9; p=0.004) LT, and

ipsilateral UT (U=9.5; p=0.009). Figure 3 presents the intergroup comparison of the timings of EMG activity onset during movement, performed at a self-selected velocity by the dominant limb and the non-affected limb.

Influence of velocity on timing of APAs in post-stroke subjects

A delay was verified on LD contralateral to the movement performed by the non-affected arm at a fast velocity (Z=-2.727; p=0.002) when compared with EMG activity onset of the same muscle while executing the movement at a self-selected velocity. No differences were found between the onset of EMG activity of the ipsilateral muscles in the movements at the self-selected velocity and the fast velocity. The same happened with the contralateral muscles, suggesting that movement velocity does not have an influence on the timing of APAs in post-stroke subjects.

Trunk displacement during reach movement performed at a self-selected velocity

For the analysis of trunk displacement in group 1, it was necessary to consider the data from two individuals as missing values, since their anthropometric properties made the analysis of the mid-sternum marker impracticable. This group had a trunk displacement of 0.84± 0.675 mm (md±IQR) in the movement performed by the dominant limb at a self-selected velocity, and a displacement of 0.75 ± 0.275 mm during the same movement performed by the non-dominant limb. However, no differences were found in trunk displacement between the movements of the two limbs in group 1.

In general, post-stroke subjects used greater trunk displacement to reach the bottle. They recruited a trunk displacement of 1.98± 1 mm when reaching with the affected limb, and a displacement of 0.68 ± 0.235mm when the movement was performed by the non-affected limb. Regarding trunk displacement, it was verified that trunk displacement was higher during the movement performed by the affected limb compared to the movements of the non-affected limb (Z=-1.859; p=0.0039) and the dominant limb of healthy individuals (U=16; p=0.049). Although there was higher trunk displacement during the reaching movement with the affected limb, there was no evidence to conclude a correlation between this displacement and the timings of EMG activity onset.

Discussion

The main finding of this study was the delay in EMG activity onset of LT and LD bilaterally in the reach movement of both upper limbs in post-stroke individuals compared to healthy subjects. Apart from the delay in APAs, it was also evident that post-stroke subjects recruited a higher trunk displacement compared to the healthy subjects, but only in the movement performed by the affected limb.

The delay in APAs at LD in post-stroke subjects compared to healthy subjects verified in this study was also noted by Dickstein et al. (2004) in their assessment of APAs in LD, bilaterally, in 50 post-stroke individuals during flexion of both arms in a seated position and by Slijper et al. (2002) who evaluated APAs in 10 post-stroke patients when the participants, in a standing position, dropped a load anteriorly. The

authors concluded that post-stroke subjects had a delay in APAs in the contralateral muscles to the affected hemisphere compared with non-affected muscles and healthy individuals, but they found no differences between the non-affected muscles and healthy subjects. However, this study showed a delay in APAs in LD and LT in both sides of the body of post-stroke subjects compared to healthy subjects, and there were no differences between the non-affected and affected muscles, suggesting an impairment of inputs from ventromedial pathways including reticulospinal that is bilateral (Dickstein et al., 2004). The differences between these findings and the results of Dickstein et al. (2004) and Slijper et al. (2002) could be explained by the muscles assessed, the task selected, and the magnitude of perturbation. Post-stroke subjects also presented a delay in both LT when compared to healthy subjects. These findings are in agreement with Ferreira et al. (2010) who evaluated, in 7 post-stroke subjects, the LT ipsilateral to the fast movement of both upper limbs separately, noting significant differences in the timing of LT activity onset in both sides. Ferreira et al. (2010) also evaluated the reach movement, which explains the similarity of the results. In addition, a delay was observed in the ipsilateral UT during the movement of non-affected limbs compared with healthy individuals. Conversely, in the affected limb movement, the ipsilateral UT was recruited earlier, and there were no differences compared to healthy individuals, which may have resulted from a possible change in the alignment of the scapula (Seitz & Uhl, 2012).

The results demonstrated that changes in the velocity of arm movement do not affect the timing of APAs in post-stroke subjects. This fact was also verified by Ustinova et al. (2004), who concluded that post-stroke subjects are less capable of adapting their APAs to different speeds. Indeed, the perturbation induced by fast movement depends on the acceleration and subject capacity to perform fast movements (Slijper et al., 2002), which is impaired in post-stroke individuals; consequently, the acceleration achieved by these individuals is not enough to induce a change in the timing of APAs. However, the results of this experiment verified a delay in LD of the affected side during fast movements of non-affected limbs. One possible explanation might be that during fast movements, the displacement of the center of mass is larger, increasing the amplitude of EMG activity and decreasing the onset timing of the activity of the ipsilateral LD. This indicates that LD has an essential role in postural control when ipsilateral to the arm movement (Dickstein et al., 2004).

Subsequent to a stroke in the MCA territory, the subjects may present an impairment of the internal capsule, leading to an impairment of APAs (Chang et al., 2010; Miyai et al., 1999). Nevertheless, according to this study, post-stroke subjects generate APAs, which may result from the preservation of SMA and PMC of the affected hemisphere or from the activation of these areas on the intact hemisphere (Riecker et al., 2010; Seitz et al., 1998), with consequent preservation of some cortical input to the rubrospinal, reticulospinal, and vestibulospinal systems (MacKinnon et al., 2007; Shelton & Reding, 2001), which are essential for postural control (Lalonde & Strazielle, 2007).

Another important finding was the use of greater trunk displacement as a compensatory strategy by post-stroke subjects during movement of the affected limb. In

the absence of changes in motor control, when placing the bottle at arm's length, it's assumed that it is not necessary to recruit degrees at the trunk to achieve the task. Indeed, healthy subjects recruit only shoulder and elbow activity. Similar to current results, Robertson et al. (2011), Levin et al. (2002), and Cristea and Levin (2000) also concluded that during the movement of the affected limb for distances corresponding to arm's length or less, post-stroke subjects recruit a greater trunk displacement earlier due to the decreased active range of motion at the elbow and shoulder. Nevertheless, if the excessive trunk displacement was due merely to the impairment of distal activity in the affected limb, this should not occur so early (-100 ms and 50 ms after DA onset) and may be associated with the delay in the APAs of the trunk muscles. However, it was not possible to verify this relationship.

One limitation of this study was the small sample, which did not allow comparisons between the right hemisphere, related to postural control, and the left hemisphere, associated with control of trunk movement (Robertson & Roby-Brami, 2011). Thus, further research is suggested to evaluate the influence of the damaged hemisphere on APAs.

Conclusions

Post-stroke subjects have a delay in the APAs on both sides of the body when performing reaching movements at fast and self-selected velocities in a seated position compared to healthy individuals. The timing of onset activity of the trapezius and latissimus dorsi bilaterally is altered in both sides of the body in post-stroke subjects. Post-stroke subjects may be less capable of adapting their APAs to different speeds and may not be prepared for the constant disturbances of the center of pressure inherent in daily life activities and ambient changes. In addition, it was verified that post-stroke subjects recruit greater trunk displacement compared to healthy subjects. The results of this study are clinically relevant, and it may be important to design APA-based training programs that involve the variable practice of daily life activities and focus on modifying parameters such as velocity, movement plane, and object.

Figure 1. Placement of the electrodes and reflective markers.

Figure 2. Intergroup comparison of EMG activity onset for the movements at a self-selected velocity performed by the affected limb of post-stroke subjects and the dominant limb of healthy subjects. [*]Denotes significant differences at alpha level of 0.05.

Figure 3. Intergroup comparison of EMG activity onset for the movements at a self-selected velocity performed by the non-affected limb of post-stroke subjects and the dominant limb of healthy subjects. [*]Denotes significant differences at alpha level of 0.05.

Table 2. Median (Md) and interquartile deviation (IQR) of timing of EMG onset (ms) for affected limb movements, p values, and Wilcoxon test.