

# Intended rather than actual movement velocity determines the latency of anticipatory postural adjustments

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**Abstract** The literature reports that anticipatory postural adjustments (APAs) are programmed according to movement velocity. However, the linkage between APAs and velocity has been highlighted within single subjects who were asked to voluntarily change movement velocity; therefore, till now, it has been impossible to discern whether the key factor determining APA latency was the *intended* movement velocity or the *actual* one. Aim of this study was to distinguish between these two factors. We analyzed the APA chain that stabilizes the arm during a brisk index finger flexion in two groups of subjects: (1) 29 who composed our database from previous experiments and were asked to “go-as-fast-as-possible” (*go-fast*), but actually performed the movement with different speeds (238–1,180°/s), and (2) ten new subjects who performed the *go-fast* movement at more than 500°/s and were then asked to *go-slow* at about 50 % of their initial velocity, thus moving at 300–800°/s. No correlation between APA latency and actual movement speed was observed when all subjects had to *go-fast* ( $p > 0.50$ ), while delayed APAs were found in the ten new subjects when they had to *go-slow* ( $p < 0.001$ ). Moreover, in the speed range between 300 and 800°/s, the APA latency depended only on movement instruction: subjects *going fast* showed earlier APAs than those *going slow* ( $p < 0.001$ ). These data suggest a stronger role of the *intended* movement velocity versus the *actual* one in modifying the timing of postural muscles recruitment with respect to the prime mover. These results also strengthen the idea of a shared postural and voluntary command within the same motor act.

**Keywords** APA · Speed · Motor control · Posture · Voluntary movement · Human

## Introduction

About 5 years ago, we started investigating the anticipatory postural adjustments (APAs) that develop in the same limb in which the voluntary movement occurs (Caronni and Cavallari 2009). In fact, a brisk finger flexion, driven by the prime mover Flexor Digitorum Superficialis (FDS), is accompanied by an APA chain in the upper limb, consisting of an excitatory burst in triceps brachii (TB) and in an almost contemporary inhibition in biceps brachii (BB) and anterior deltoid (AD). These anticipatory postural activities allow to counteract the elbow and shoulder flexion induced by the upward perturbation that the index finger flexion causes on the metacarpophalangeal joint (MP). Although resulting from the motion of a tiny mass, these intra-limb APAs behave similarly (Caronni and Cavallari 2009; Bolzoni et al. 2012; Bruttini et al. 2014) to the well-known inter-limb APAs of movements involving large masses (see Bouisset and Do 2008 for a review).

The APAs originate from a feed-forward command (Belen’kii et al. 1967; Friedli et al. 1984; Aruin and Latash 1995; Massion et al. 1997), and therefore, APAs are tuned depending on several kinematic aspects of the primary movement. Of particular interest for the understanding of APA programming is the dependence of their latency from movement velocity, illustrated by Horak et al. (1984), Lee et al. (1987), and also appreciable in figure 2a of Shiratori and Aruin (2007). In those papers, information about the linkage between APAs and speed of voluntary movement was obtained within single subjects, by comparing their behavior when instructed to change the movement velocity;

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therefore, it was impossible to discern whether the key factor determining the modification of APA latency was the change of the *intended* or the *actual* movement speed. The aim of this study was to distinguish between these two factors.

To address this issue, we analyzed the well-known intra-limb APA chain that stabilizes the arm during a brisk index finger flexion, in two groups of subjects: 29 composing our database of previous experiments, who received the same “go-as-fast-as-possible” (*go-fast*) instruction but actually performed the movement at different velocities (238–1,371°/s) and 10 new subjects who performed the *go-fast* flexion at more than 500°/s and were then asked to *go-slow* at about 50 % of their initial speed, so that they moved faster than 250°/s. Assuming that all subjects actually obeyed the *go-fast* and *go-slow* instructions by planning a movement at 100 and 50 % of their maximal speed, respectively. The change of movement *instruction* should have been reflected into a parallel change of the *intended* movement speed.

In the *go-fast* population, we tested the correlation between APA latency and *actual* movement speed, while the *go-fast* versus *go-slow* behavior of the 10 new subjects allowed us to assess the effect of the *intended* movement speed. Moreover, a last comparison was drawn between subjects moving at the same speed but obeying the two different instructions, i.e., planning two different speeds. Results from these experiments allowed us to properly distinguish whether APA latency depends on *actual* or *intended* movement velocity, or on both.

## Materials and methods

Two groups of subjects were analyzed. The first group was composed by our database of 29 subjects (12 females), recorded in previous studies. All of them performed experiments in which they were asked to briskly flex the index finger as fast as possible (*go-fast* instruction). The actual velocity of their movements ranged from 238 to 1,180°/s. Their mean ( $\pm$ SD) anthropometric characteristics were: age  $26.2 \pm 8.9$  years, weight  $65.8 \pm 11.6$  kg, height  $172 \pm 16$  cm, index finger length  $8.7 \pm 0.8$  cm and upper limb length  $70.4 \pm 6.4$  cm.

The second group was obtained by collecting ten new subjects (four females) who were able to perform the *go-fast* finger flexion at more than 500°/s. These subjects were then asked to *go-slow*, at about 50 % of their initial speed. Their mean ( $\pm$ SD) anthropometric characteristics were: age  $28.1 \pm 5.7$  years, weight  $68.4 \pm 13.4$  kg, height  $174 \pm 13$  cm, index finger length  $9.2 \pm 0.9$  cm and upper limb length  $72.3 \pm 5.8$  cm.

In both groups, no subject had any history of orthopedic or neurological disease and all of them gave written

consent to the procedure, after being informed about the nature of the experiments. The procedure was approved by the local Ethics Committee in accordance to the 1964 Declaration of Helsinki.

## Experimental procedure

The ten new subjects underwent the same experimental procedure described in Caronni and Cavallari (2009): they sat on a chair with both arms along the body, the right elbow flexed at 90°, the right hand prone and in axis with the forearm. The right index finger was kept extended and in contact with a proximity switch (Pepperl and Fuchs, CJ10-30GK-E2), so that the MP joint angle was about 180°, all other fingers hanging. Subjects were explicitly asked to keep their back supported, the arm and forearm still and both feet on the ground throughout the experiment. The chair was height adjustable and the proximity switch screwed on an articulated arm (Manfrotto 143 MAGIC ARM® + 035 Super-clamp Kit®); both were adapted to the different body dimensions of the subjects. The position of the subject was always visually controlled by the experimenter.

Subjects were asked first to flex their index finger at the MP joint. Each movement was self-paced and performed after an acoustic signal delivered every 7 s. The time interval between the beep and the movement onset varied according to the will of the subject. This procedure was adopted to exclude any reaction time. Subjects performed two sequences of 30 finger flexions in which they were instructed to *go-fast*, followed by two more sequences in which they were instructed to *go-slow*, i.e., to reduce their speed to about 50 % of the fast value. A rest time of about 5 min was allowed between each session. Subjects never complained about fatigue. Movement speed was monitored by the experimenter, who alerted the subjects to speed-up or slow-down when necessary.

## Movement and EMG recordings

The onset of the fingertip movement was monitored by the proximity switch. Flexion of the right MP joint was recorded by a strain-gauge goniometer (mod. F35, Biometrics Ltd®, Newport, UK) taped to the joint. Angular displacement was DC amplified (P122, Grass Technologies®, West Warwick, Rhode Island, USA), and gain was calibrated before each experimental sequence. Pairs of pregelled surface electrodes, 24 mm apart, (H124SG, Kendall ARBO, Tyco Healthcare, Neustadt/Donau, Germany) were used to record the EMG signal from the right prime mover FDS and from some of the ipsilateral postural muscles: BB, TB and AD. A good selectivity of the EMG recordings was achieved both by a careful positioning of the electrodes and by checking that the activity from the recorded muscle,

during its phasic contraction, was not contaminated by signals from other sources. EMG was AC amplified (IP511, Grass Technologies®, West Warwick, Rhode Island, USA; gain 2–10 k) and band-pass filtered (30–1,000 Hz, to minimize both movement artifacts and high-frequency noise). Goniometric and EMG signals were A/D converted at 2 kHz with 12-bit resolution (PCI-6024E, National Instruments®, Austin, Texas, USA), visualized online, and stored for further analysis.

### Data analysis

On each sequence, the 30 EMG traces of the prime mover and those simultaneously recorded from the postural muscles were digitally rectified and integrated (time constant: 25 ms). Traces collected from each muscle were then averaged in a fixed temporal window: from  $-1,000$  to  $+300$  ms with respect to the onset of the FDS EMG, identified by a software threshold set at  $+2$  SD of the reference signal level (from 1,000 to 500 ms prior to movement onset). On each experiment, latency of the postural activity was measured off-line on the averaged traces. The EMG onset in each postural muscle was identified by a software threshold set at  $\pm 2$  SD of the reference signal level, and visually validated. Latency of the APA was referred to the FDS EMG onset, with negative values indicating a time advance.

### Statistics

Pearson's product-moment correlations was used to assess the relationship between APA latency and actual movement speed in BB, TB and AD muscles, in all subjects who were instructed to *go-fast* or to *go-slow*.

Two-way repeated measures ANOVA was employed to test the effect of *instruction* (*go-fast* vs. *go-slow*) and *muscle* (BB vs. TB vs. AD) on APA latency, in the 10 new subjects.

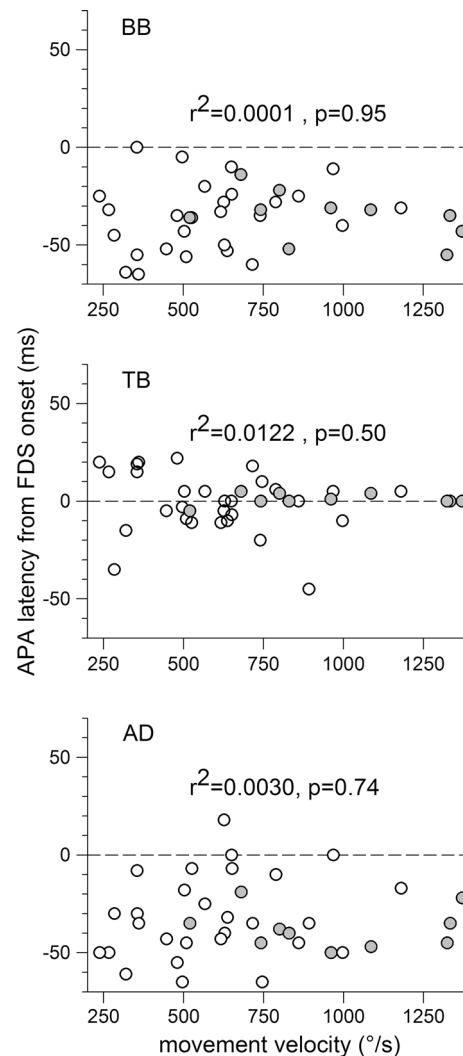
Two-way mixed ANOVA was used to test the effect of *instruction* (between-groups factor) and *muscle* (within-subjects factor) in the 10 new subjects when they had to *go-slow* versus those from our database who had to *go-fast* but actually moved in the same speed range. Movement speed was compared by an unpaired *t* test.

For all tests, significance threshold was set at 0.05.

## Results

### Anticipatory postural adjustments prior to a fast index finger flexion

Despite the large range of their actual movement speeds (from 238 to 1,371°/s), both the 29 subjects from our database and the new 10 subjects, who were instructed to

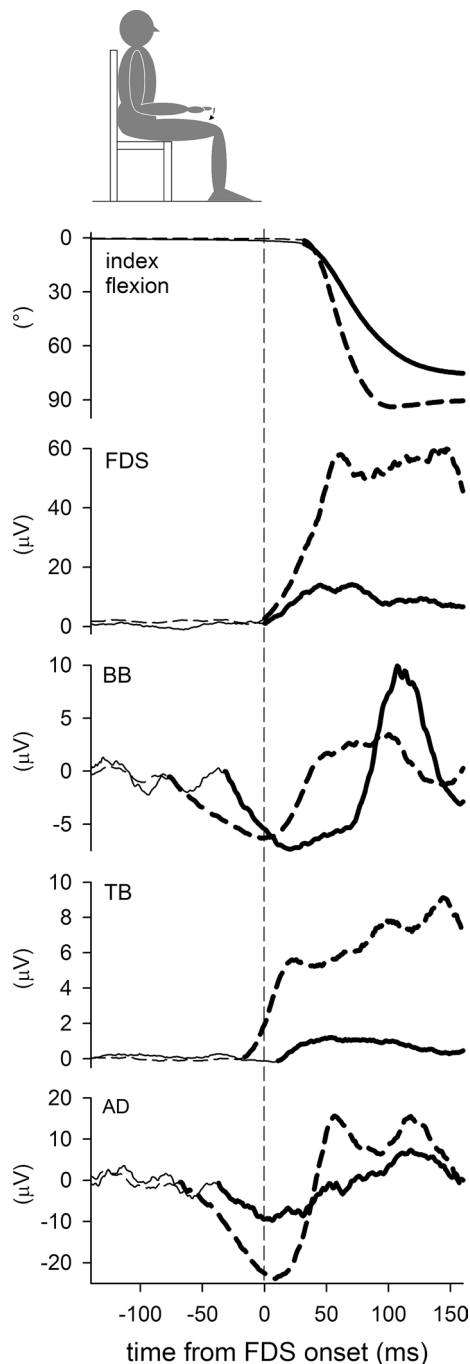


**Fig. 1** Relation between the APA latency in the three postural muscles (biceps brachii, BB; triceps brachii, TB; and anterior deltoid, AD) and the actual movement velocity. Data from subjects from our database (*white circles*) and from the new ten subjects (*gray circles*) are plotted. Time 0 (*dashed line*) refers to prime mover EMG onset. No correlation between APA latency and movement velocity was found in the whole population

*go-fast*, showed no correlation between APA latency and actual movement velocity (BB:  $r^2 = 0.0001$ ,  $p = 0.95$ ; TB:  $r^2 = 0.0122$ ,  $p = 0.50$ ; and AD:  $r^2 = 0.0030$ ,  $p = 0.74$ , see Fig. 1). On average, the TB muscle activation was almost synchronous (mean  $\pm$  SE  $-0.3 \pm 2.2$  ms) to prime mover FDS, while inhibition of BB and AD clearly preceded it ( $-37.5 \pm 2.9$  and  $-34.2 \pm 3.2$  ms, respectively).

### Anticipatory postural adjustments prior to a slow index finger flexion

The different behavior between the *go-fast* and *go-slow* instruction is depicted in Fig. 2 for a representative subject.



**Fig. 2** Go-fast and go-slow movements in a representative subject. The inset depicts the position of the subject in the experimental setup. Goniometric recording of the index finger flexion (top panel) and rectified and integrated (25 ms) EMG from the prime mover FDS and from BB, TB and AD. Note that when going fast (dashed traces) the prime mover onset was preceded by APAs in BB, TB and AD. APAs (emboldened) were instead clearly delayed when going slow (solid traces). Time 0 (vertical dashed line) refers to prime mover EMG onset

The latency lag during *go-slow* movement may be easily appreciated by matching the bolded lines in the three postural muscles. When *going slow*, the movement speed

was reduced from 1,324 to 590°/s, i.e., to about 50 % of its maximal value.

The APA latencies obtained in *go-fast* and *go-slow* movements of the 10 new subjects are shown in Fig. 3. On the left, latency is plotted against actual movement speed. Note that the range of the *go-slow* movements (from 309 to 794°/s) fell within the range of the *less fast* subjects plotted in Fig. 1. In this case too, no correlation was found between APA latency and movement velocity (BB:  $r^2 = 0.0063$ ,  $p = 0.83$ ; TB:  $r^2 = 0.00001$ ,  $p = 0.99$ ; and AD:  $r^2 = 0.013$ ,  $p = 0.75$ ). Mean latencies and individual values are plotted in the right panels, showing that when instructed to *go-slow* subjects clearly delayed their postural activities of about 20–25 ms.

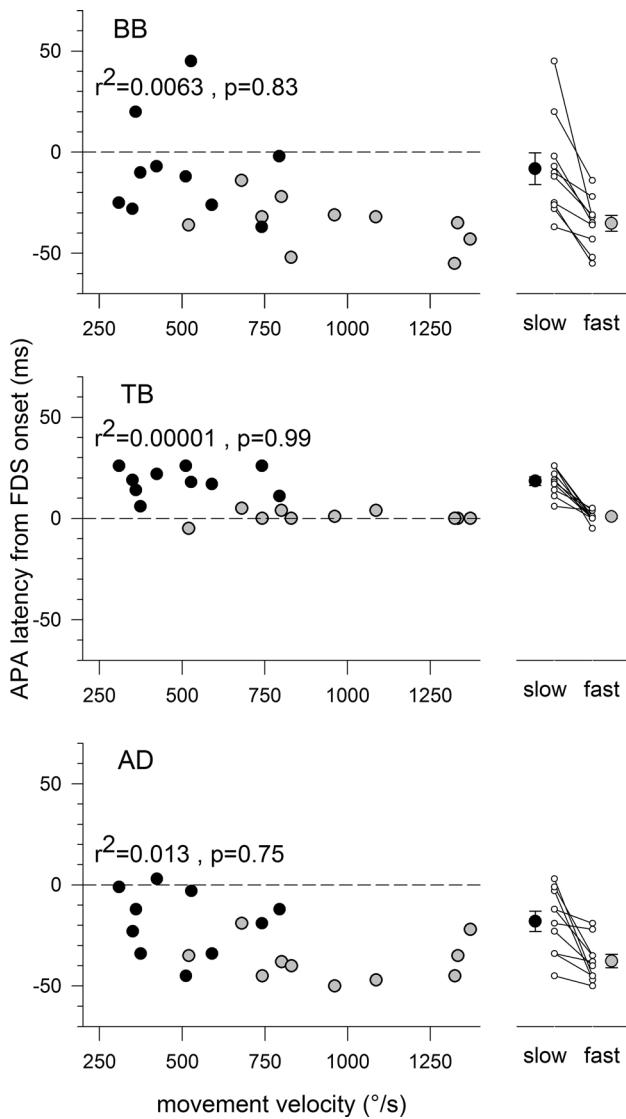
A two-way repeated measures ANOVA showed a significant effect of movement instruction (*go-fast* vs. *go-slow*,  $F_{1,9} = 38.6$ ,  $p = 0.0002$ ) and muscle ( $F_{2,18} = 33.9$ ,  $p < 0.0001$ ), while interaction was not significant ( $F_{2,18} = 1.2$ ,  $p = 0.32$ ), i.e., the change in latency was similar in the three muscles.

Finally, the results from the 10 subjects who had to *go-slow* were matched with those who performed the *go-fast* task at a similar velocity, i.e., 300–800°/s. The mean APA latency was clearly different in the two groups, witnessing that movement instruction, not actual movement speed, was the most significant factor in determining the APA timing.

Table 1 reports the movement speed and the mean APA latencies in the two groups. Despite similar velocities, it is apparent that the jump of latency is due to the different instruction. Unpaired *t* test showed no difference in movement velocity between the two groups ( $t_{29} = 1.05$ ,  $p = 0.30$ ); instead, a two-way mixed ANOVA showed a significant effect of movement instruction (*go-fast* vs. *go-slow*:  $F_{1,29} = 18.1$ ,  $p = 0.0002$ ) and muscle (BB vs. TB vs. AD:  $F_{2,58} = 35.4$ ,  $p < 0.0001$ ), while the interaction was not significant ( $F_{2,58} = 1.8$ ,  $p = 0.17$ ).

## Discussion

This study showed that the key factor determining the modification in APA latency when performing a voluntary movement was the change in the movement instruction (*go-fast* vs. “go slow”), not the actual movement velocity. This conclusion stemmed from three observations: (1) There was no correlation between APA latency and movement speed when all subjects had to follow a *go-fast* instruction, as shown in Fig. 1, (2) APAs were delayed when subjects reduced their movement velocity because they had to follow a *go-slow* instruction (Fig. 3), and (3) in a large range of speeds, the APA latency depended exclusively on movement instruction: subjects *going fast* showed earlier APAs than those *going slow* (Table 1). Under the assumption



**Fig. 3** Go-fast and go-slow APA latency in the ten new subjects. The left panel illustrates the relation between movement velocity and mean latencies of the three postural muscles when subjects were asked to go-fast (gray circles) or to go-slow (black circles). Time 0 (dashed line) refers to prime mover onset. No correlation between APA latency and movement velocity was found when subjects were asked to go-slow. Right panel compares individual and mean ( $\pm$ SE) APA latency in fast and slow movements

**Table 1** Effect of movement instructions on APA latency

	Speed (°/s)	BB (ms)	TB (ms)	AD (ms)
Go-fast	558 $\pm$ 30	-39.4 $\pm$ 4.5	1.1 $\pm$ 2.7	-32.4 $\pm$ 5.3
Go-slow	498 $\pm$ 53	-8.2 $\pm$ 7.8	18.5 $\pm$ 2.1	-18.0 $\pm$ 5.0

Mean APA latency  $\pm$  SE in biceps brachii (BB), triceps brachii (TB) and anterior deltoid (AD) in two groups of subjects who were instructed to go-slow or go-fast, but actually performed index finger flexion at 300–800°/s. First column shows that they actually moved at similar velocities. Statistics (see text) found no difference in movement speed, but a significant effect of instruction on all muscles

that all subjects actually obeyed the go-fast and go-slow instructions by planning a movement at 100 and 50 % of their maximal speed, respectively, the change in movement instruction was paralleled by a change in the intended movement speed; hence, the latter should have been the key factor for determining the APA latency.

The previous literature considers APAs as pre-programmed according to several task parameters, such as velocity, load and direction (for a review, see Bouisset and Do 2008). In particular, for what concerns the relationship between APA latency and movement velocity, several studies (Horak et al. 1984; Lee et al. 1987; Ito et al. 2003, see also in figure 2a of Shiratori and Aruin 2007) found delayed APAs when the subjects voluntarily slowed their movement, in agreement with the second of our above observations. It might appear strange that Ito et al. (2003) found no change in APA latency between fast and slow movements. However, these authors measured latency with respect to movement onset, not to prime mover recruitment, and since, in general, the delay between prime mover activation (EMG) and movement onset increases when slowing the movement, and this could have compensated the reduction in APA latency with respect to prime mover onset. Note also that in all the above studies, the latency–speed relation was observed within single subjects who were explicitly compelled to change the speed of their movement. Such an approach, i.e., studying subjects who planned different movement speeds, did not allow to distinguish whether the APA latency changed in function of the intended movement velocity or of the actual one. In this regard, the novelty of the present paper is to have discerned between these two factors.

It may be argued that the lack of inter-subjects correlation between APA latency and actual movement speed could have been ascribed to a subject-dependency of APAs, like the walking speed in elderly vs. young subjects (cfr. Schimpl et al. 2011). This could be true, even if in our subjects neither the APA latencies nor the maximal movement speed were correlated with age (in all cases,  $r^2 < 0.044$ ;  $p > 0.20$ ). However, this would not affect the main message of our study: the literature on APAs reports that their latency is scaled according to the movement speed, our study (1) showed that such relation held only within-individuals, while no significant correlation was observed between-subjects and (2) concluded that the intended movement speed is a key factor for determining the APA latency because it was the only factor which systematically changed within-individuals and not between-subjects.

Given that the CNS is able to adapt APAs to the postural demand of the forthcoming mechanical perturbation, one may ask whether postural control and voluntary recruitment stem from two separate control centers or they instead result from a shared motor command. In the former

case, it was expected that after few trials, APAs would have adapted their latencies according to the *actual* movement velocity, as shown, when changing the postural context (Cordo and Nashner 1982; Hall et al. 2010; Bruttini et al. 2014). In fact, the postural controller would have overcome the *intended* command because of the proprioceptive feedback. Instead, in the case of a shared motor command, the *intention* would have prevailed, so that APA latency would have always been tailored to it. Present results clearly agree with the latter view.

Such view is not new: it had been already forwarded for justifying the persistence of APAs even after a forearm ischemia, which suppressed (1) the prime mover EMG, (2) the ensuing finger movement and (3) the related mechanical perturbation (Bruttini et al. 2014). In that condition, the CNS did not adapt APAs to the absence of mechanical perturbation, seemingly because the motor command was unchanged. A result indirectly suggested that the recruitment of postural and prime mover muscles was driven by a shared motor command. The concept of a shared postural and voluntary command within the same motor act was also envisaged by Caronni et al. (2013), who showed that APAs properly tailored to the prime mover activation contribute to make the focal movement accurate by securing the position of the proximal joints. Leonard et al. (2011) reached similar conclusions showing that the CNS employs a predictive mode of postural control and consistently adapts the postural muscle activity before correcting the prime movers recruitment. These authors concluded that the postural corrections could be described as being a component of the voluntary movement, rather than being aimed to ensure the maintenance of equilibrium. Present results are also in agreement with Davidson and Wolpert (2005), who illustrated a stronger role of predictive feed-forward internal models versus sensory feedback in several aspects of human motor control, such as oculomotor and skeleto-motor control, perceptual processing, mental imagery and also postural control (see also Wolpert et al. 1995, 2011).

Little is known about the neural sub-systems governing APAs, but several studies suggested a superposition of the neural structures for APAs and those for voluntary recruitment, thus indirectly supporting the above hypothesis of a shared motor command. Severe APA impairments in patients with Parkinson's disease suggested a role of the basal ganglia in the anticipatory postural control (Viallet et al. 1987). In particular, beyond their role in shaping the movement, basal ganglia may be involved in the intentional movement selection, through the pathway involving the anterior mid-circulates cortex (see Hoffstaedter et al. 2013). Anticipatory brain activity before the execution of a bimanual load-lifting task was recently localized in basal ganglia, supplementary motor area (SMA), and thalamus in the hemisphere contralateral to the load-bearing arm (Ng

et al. 2012). It is worth noting that these areas are component nodes of the basal ganglia-thalamo-cortical network implicated in well-learned finger movements (Boecker et al. 1998). The possible involvement of the SMA in the APA network was suggested by several human and primate experiments (Brinkman 1984; Viallet et al. 1992; Yoshida et al. 2008; Jacobs et al. 2009). A change in firing rate, depending on speed instruction, had also been shown in the pre-motor cortex of rhesus monkey (Shenoy et al. 2003), another area involved in motor program selection (see for references Hoffstaedter et al. 2013). This result is of particular interest for us because the movement paradigm closely replicated many aspects of our task, such as the delayed movement onset with respect to the go signal, in order to avoid a reaction time movement, and the two different speed instructions.

Finally, the hypothesis of a *functionally unique motor command* deserves a brief consideration within the framework proposed by Bouisset and Do (2008) in their review on APAs. According to these authors, the voluntary movement is any motor act in which the intention of the subject is to perform a given task. These authors distinguished two aspects of the “task”: First, the task to be performed, which depends on the environmental context and the category of the intended movement, such as pointing, tapping, and throwing. Second, the real task, i.e., the outcome of the motor command, which may satisfy the intended movement by a various degree of efficiency. As these authors stated: “Efficiency is measured by the actual parameter values (speed, precision, etc.) with respect to the intended ones and depends on the neural and muscular–skeletal properties of each subject. Therefore, a voluntary movement is part of a more general process, called the motor act. In other words, a voluntary movement is the mean to complete a motor task”. In this perspective, the present results strengthen the idea that APAs belong to the same *motor act* as that of the *voluntary recruitment*. Indeed, just as voluntary movement, APAs may be considered “the mean to complete a motor task”, as they provide the proper fixation chain.

## Conclusion

This study showed: (1) a lack of correlation between APA latency and actual movement speed in subjects who planned the same movement, i.e., an as-fast-as-possible flexion of their index finger, despite a wide range of actual movement velocities, and (2) that APAs were delayed when 10 subjects were asked to repeat the movement at about 50 % of their maximal speed. These data suggest a stronger role of intended versus actual movement speed in modifying the timing of postural muscles recruitment with respect

to prime mover, thus strengthening the idea of a shared postural and voluntary command within the same motor act.

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