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# Physiological Mechanisms for Stabilizing the Limb when Acting Against Physical Constraints\*

P. Senot, L. Damm, M. Tagliabue, J. McIntyre

**Abstract**—Smooth physical interaction with our environment, such as when working with tools, requires adaptability to unpredictable perturbations that can be achieved through impedance control of multi-joint limbs. Modulation of arm stiffness can be achieved either increasing co-contraction of antagonistic muscles or by increasing the gain of spinal reflex loops. According to the "automatic gain scaling" principle, the spinal reflex gain, as measured via the H-reflex, scales with muscle activation. A previous experiment from our labs suggested, however, that reflex gains might instead be scaled to the force exerted by the limb, perhaps as a means to counteract destabilizing external forces. The goal of our experiment was to test whether force output, rather than the muscular activity *per se*, could be the critical factor determining reflex gain. Five subjects generated different levels of force at the wrist with or without assistance to dissociate applied force from agonist muscular activity. We recorded contact force, EMG and H-reflex response from a wrist flexor. We did not find a strict relationship between reflex gain and contact force but nor did we observe consistent modulation of reflex gain simply as a function of agonist muscle activity. These results are discussed in relation to the stability of the task constraints.

## I. INTRODUCTION

Control of stiffness rather than force or position of a multi-joint limb is a practical way for the brain to adapt to the environmental physical constraints during movement by taking advantage of the visco-elastic properties of muscles and joints [1]. For example, smoothly following a bumpy surface with the hand or adapting to unpredictable perturbations during free motions can benefit from such control. Modulation of arm stiffness can be achieved through different physiological mechanism. First, the stiffness of a muscle tends to increase with its force output [2]; an increase of stiffness without increase of net torque can be achieved through co-contracting antagonistic muscles around a joint [1]. Limb stiffness can be increased by increasing the gain of spinal reflex loops [3]. The question remains as to how these different mechanisms interact and co-vary depending on task constraints. Levels of co-contraction and reflex gain in the upper limb are known to be modulated depending on task constraints (as in force & position control tasks, [4]–[6])

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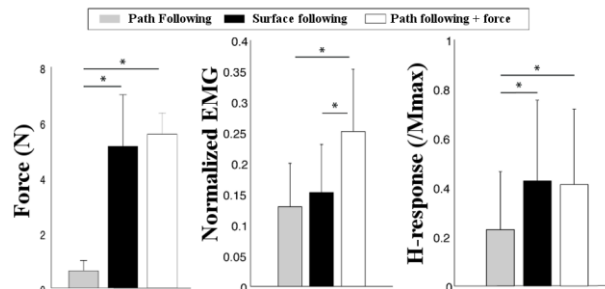


Fig. 1. Averaged (N=8) applied force, agonist EMG activity and H-reflex amplitude as a function of the task constraints, from a previously reported study by Damm and McIntyre [10].

and mechanical properties of the environment (stable vs. unstable environments, [7], [8]). However, the studies cited above either focused on long-latency components of the reflex elicited by mechanical perturbations or showed no modulation of the short-latency response. In contrast, Maluf et al.[9] found a higher H-reflex response for position- than for force-control tasks for equal levels of tonic activity and minimized co-contraction.

In a previous study of our group, we simultaneously looked at the level of muscle co-contraction and at the gain of the stretch reflex while subject performed a dynamic position control task of the arm and hand in a free environment or a constrained motion against a rigid surface [10]. To compare reflex gains as equivalent force outputs, we compared the constrained movements with free movements performed both in the presence or absence of a bias force applied against the hand by a motorized haptic device. Because stiffness is known to increase in unstable environments, we hypothesized that both co-contraction and reflex gain should increase in free relative to constrained movement, the latter being more stable mechanically. As expected we found an increase of co-contraction in the two free movement conditions (with and without bias force) compared to the constrained motion [10]. In those experiments, two very different levels of forces were applied in the path following (without bias force) and the surface following tasks with approximately the same muscle activity in the agonist muscle (FCR). In the same way, similar force levels were achieved in the surface following and in the path following + bias force tasks with very different levels of muscle activity. Consequently, there was dissociation between the forces applied on the environment and the level of muscular activity in wrist flexors. The surprising observation was that the pattern of reflex gains across the three experiment conditions (Fig. 1, right) followed better the pattern of forces applied on the environment (Fig. 1,

left) rather than the pattern of the agonist muscular activation (Fig. 1, middle). This observation fits well with the idea that the critical factor for increasing stiffness for limb stabilization is the force output.

The goal of the experiment to be presented here was to check if this relation between force and reflex gain could be generalized. To this end, one needs to dissociate the amount of muscular activity produced by the participant and the contact forces applied on the environment. In this way one can test if the reflex gain changes with the muscular activity or with the contact force. To achieve this goal, we ask volunteers to flex their wrist and exert a target force under visual feedback while assisting his/her effort or not by attaching a weight to the back of their hand. In this way, the same level of applied force could be attained with different muscular activities, depending on the amount of assistive force.

## II. MATERIAL AND METHODS

### A. Participants

Five subjects (3 males, including author PS and 2 females) volunteered for the experiment and gave their informed written consent to the experimental procedure, which was approved by the local ethics review board (ID RCB: 2011-A00729-32) and carried out in compliance with French law and the Helsinki declaration.

### B. Experimental setup and procedure

Participants were comfortably seated on a chair, their right hand resting pronated on a 5cm diameter wooden hemisphere mounted on the z-axis of a 6-axis force sensor (ATI industrial automation mini40 force/torque sensor). The sensor was placed at shoulder level in the sagittal plane of the participants such that their right arm was lying in the horizontal plane with the elbow flexed at 90°. Participant's wrist and elbow were firmly attached to the chair such that only downward wrist rotation was allowed.

The experiment consisted of three sessions performed on the same day. During the first session, participants were asked to produce maximal force on the z-axis of the force sensor by wrist flexion. Maximal force ( $F_{max}$ ) computed over five trials was used to compute normalized EMG data and define target force levels. After a short pause, a second session consisted of the acquisition of a complete recruitment curve (see below). Then the experimental session began, consisting of three blocks of trials. In this session, participants were asked to push on the sensor by wrist flexion to produce two target force levels (TFL), 5% and 15% of  $F_{max}$ . Instruction was given to the participants to push with the palm of their hand on the sensor without grasping the hemisphere to avoid contraction of the finger muscles.

Participants monitored the force they applied on the sensor in real time via visual feedback on the computer screen. A force target window corresponding to target forces  $\pm 1N$  was also displayed on the screen and at each

trial the participants were instructed to maintain their force within this window for 5 seconds.

### C. Experiment Conditions

For each target force, 3 assistive forces (AF) could be applied by placing a weighted soft pad on the back of their hand. The assistive forces were 0% (null assistive force,  $AF_N$ ), 66% of the lowest target force level (low assistive force,  $AF_L$ ) and 66% of the highest force level (high assistive force,  $AF_H$ ). With such assistive forces, one would expect to observe a specific theoretical pattern of EMG activities reported in Table 1. In particular, one should see similar EMG activity between the lower force target level with a 0% assistive force and the highest target force level with an assistive force equal to 66% of highest force level. One could also expect a null or decreased activity in flexor muscles when the assistive force was superior to the lowest target force level.

TABLE 1. EXPECTED EMG ACTIVITIES (ARBITRARY UNITS) DEPENDING ON TARGET AND ASSISTIVE FORCES

Assistive force	Target Force Level (a.u.)	
	1 (LTFL)	3 (HTFL)
0%	1.00	3.00
66% of LTFL <sup>a</sup> (0.66)	0.33	2.33
66% of HTFL <sup>a</sup> (2.00)	-1.00	1.00

a. LTFL : lowest target force level 15%  $F_{max}$  ; HTFL : highest target force level, 15%  $F_{max}$

For each block corresponding to different medial nerve stimulation intensities (see below), participants performed blocks of 10 trials for each target force level and each assistive force.

### D. Force, EMG and H-reflex recordings

Total applied force and EMG signal were sampled and recorded at 1000 Hz using a data acquisition interface (1401plus Cambridge Electronic Design, UK). Raw voltage from the force sensors were converted to forces and torques using the calibration matrix provided by the constructor. The norm of the 3 force components was then computed as a measure of the total applied force. Wrist flexor muscles activity was assessed through EMG signals (Delsys Bagnoli 8) acquired and amplified using bipolar surface electrodes glued on the skin over the *flexor carpi radialis* (FCR) muscle. For each trial, force and EMG amplitudes were computed by averaging the corresponding signal over a 1s window.

During the experimental session, reflex gain was assessed in the FCR muscle through constant current stimulation of the medial nerve at the elbow of the right arm. (square pulse, 1-ms duration; DS7A, Digitimer, Hertfordshire, UK) Such a stimulation elicits two EMG responses: an early direct motor response (M-wave) which is considered as a reliable indicator of the effective stimulus strength and a later H-reflex response reflecting the excitability of the motoneuron pool by muscular sensory nervous fibers and thus a good indicator of reflex gain [4]. We chose the stimulation location before the experiment by monitoring with an oscilloscope (Tektronix

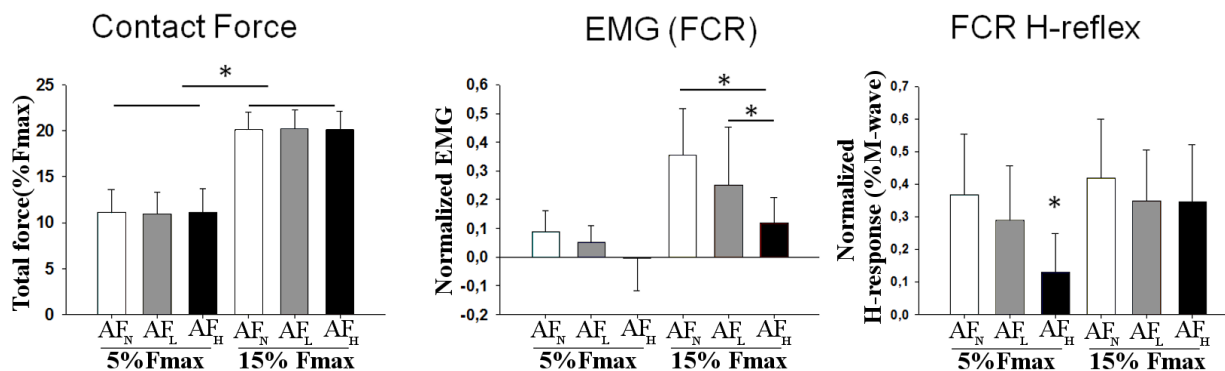


Figure 2. Averaged applied force (left), EMG activity (middle) and H-reflex amplitude (right) as a function of target force (5%  $f_{Max}$  and 15%  $F_{max}$ ) and assistive force (AF<sub>N</sub>: 0% ; AF<sub>L</sub>: 66% of lowest target force; AF<sub>H</sub>: 66% of highest target force). Stars depict significant differences ( $p < 0.05$ )

TDS 210) twitch responses in the target muscle as the stimulating electrode was moved over the skin.

To elicit the H-reflex response during the experiment, constant-current electrical stimuli were applied via computer control at three different current levels. Initially, a complete recruitment curve was acquired for each muscle by applying a range of currents from sub-threshold to that which evoked a maximal M-wave ( $M_{max}$ ). Throughout the acquisition of the recruitment curve subjects were asked to apply a force similar to the force they would apply during the subsequent experimental task. Three different intensities of stimulation on the ascending branch of the recruitment curve were chosen for the experimental session. The peak-to-peak amplitude of M-wave and H-reflex responses for each condition and stimulation intensity was computed by subtracting maximal and minimal EMG values in specific temporal windows (5-15 ms and 16-35 ms after stimulation respectively). M-H pairs were then rank-ordered according to M-wave size and a linear regression was fitted for each data set. Amplitudes of H-reflex for similar M-waves were extracted by inserting values of the same M-wave amplitude (2% of  $M_{max}$ ) into the regression equation for each condition.

Effects of target and assistive force on the measured total applied force, EMG and H-response amplitude were assessed through a 2x3 repeated-measures ANOVA and HSD Tuckey post-hoc test.

### III. RESULTS

As shown on Fig. 2 (left), participants applied the required force during the experiment whatever the assistive force as demonstrated by a significant main effect of target force level ( $F(1,5)=571,61$ ,  $p<0.05$ ). The averaged EMG signal, shown in Fig. 2 (center), significantly depended both on target force ( $F(1,5)=10,01$ ,  $p<0.05$ ) and assisting force ( $F(2,10)=7,66$ ,  $p<0.05$ ). EMG globally increased with target force and decreased with increasing assistive force. Despite the lack of a significant interaction between the two factors, the decrease in EMG activity induced by the assistive force was only present for the highest target force level ( $p<0.05$ ) where the highest assistive force induced significantly lower EMG activity than for the other two. Finally, we found a main effect of target force but not assistive force on H-

responses ( $F(1,3)=14,94$ ,  $p<0.05$ ) (Fig. 2 right). H-reflex amplitude was significantly lower ( $p<0.05$ ) for the lower target force level when the highest assistive force was applied, that is when the total amount of force required was provided by the assistive weight.

### IV. DISCUSSION

In our experiment, we asked subjects to push by wrist flexion on a static force sensor to reach target forces while their effort was assisted or not by a weighted soft pad applied on the back of their hand. The task of the subject was to control the contact force applied on the sensor which was decoupled from the flexor EMG activity and from the corresponding force developed at the wrist because of the assistance. Results showed that when the assistance was present, the muscular activity was lower than what could be expected from the contact force. Because the hand pushed on a fixed object, and because a low level of co-contraction is expected in a stable environment, the force developed at the wrist was coupled to the flexor muscular activity. So in this experiment we dissociated contact force from FCR activity and force developed at the wrist for similar stability constraints.

We asked whether the FCR reflex gain was strictly related to the force produced by FCR activity ("automatic gain scaling" [12]) or to the contact force. The response to this question was mixed. There was a consistent main effect of target force on both EMG activity and H-response, consistent with both hypotheses. To answer the question, one must look at conditions in which contact force and EMG activity were decoupled. As expected from our experiment design (see paragraph II.C), the EMG activity for AF<sub>N</sub> @ TFL = 5%  $F_{max}$  was not significantly different from the EMG for AF<sub>H</sub> @ TFL = 15%  $F_{max}$ . If we specifically look at these two conditions with approximately equal EMG levels but two different contact forces, it appears that the H-reflex amplitude did not increase with contact force *per se*, as there was no significant difference in H-reflex response between these two conditions. This is in apparent contradiction with previous results showing an increase of H-response with increasing applied force [15] but is explained by the decoupling of force and EMG activity in our experiment. In accordance, we observed a good tendency for H-reflex to increase with muscle activation

for low target forces, according to conventional wisdom. However, this was not so for higher target forces, where the H-reflex gain was similar for at least two, if not three, clearly different levels of EMG but the same contact force. We therefore did not find a fixed relationship between reflex gain and contact force but nor did we observe consistent modulation of reflex gain as a function of FCR activity. These results argue for a more complex modulation of reflex gains that is not strictly related to either net force output or to muscle activation.

How to explain the lack of h-reflex amplitude modulation at higher force level? One hypothesis is that reflex responses saturate. This is unlikely because the higher level of force was only 15% of  $F_{max}$ , which is an acceptable value with regard to the literature (see [13]). Second, H-reflex amplitude was estimated for a relatively low constant value of M-wave (2% of  $M_{max}$ ) to prevent saturation of the reflex response. A more likely explanation lies in the inter-relationship between exerted forces, effective limb stiffness and stability. Normally, increased reflex gains should be associated with greater instability, given delays in the reflex loops. But here and in the task used by Damm and McIntyre (2008), where the hand exerted forces against a stabilizing constraint, the H-wave amplitude was equally large for both target forces without assistive force. Because of the constraint, the spinal loop gain could be maintained at a high level without challenging the stability of the effector-environment combination. Thus, exerting a force against a stable environment could favour high reflex gains for any exerted force above a certain threshold. Conversely, McIntyre et al. [14] demonstrated that pushing against the environment provokes instability that grows with the amplitude of the net exerted force, as a function of limb configuration and force direction. Thus, increasing reflex gains with exerted force might serve to counteract this potential source of instability at higher force levels.

What could be the neurophysiological basis of this force stability modulation of the reflex gain? Presynaptic inhibition likely has a crucial effect on H-reflex gain since it particularly affects Ia terminals [16]. Recent studies suggest that modulation of presynaptic Ia inhibition is the likely source of H-response modulation between position and force control tasks at the fingers [6], [15] and is therefore a prime candidate in our tasks as well. Presynaptic inhibition also allows tuning of stretch reflexes at the wrist by peripheral factors. Cutaneous afferent signals tonically inhibit Ia presynaptic inhibitory pathways [17]. This inhibition of inhibition would increase the reflex gain when exerting forces through contact with the environment. But for a given target force the contribution of these cutaneous signals should be constant whatever the assistive force. The change of reflex gain noticed for the low target force according to assistive force therefore argues against this explanation.

#### CONCLUSIONS

Theoretical considerations suggest that limb impedance and reflex gains should vary as a function of

net force applied against the environment. The results described here indicate that reflex gains vary as a complex function of net forces and muscle activations that remains to be elucidated.

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