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Transition between reciprocal activation and co-contraction during wrist posture control

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Abstract

In daily life we perform numerous tasks which require position control against disturbances. We can counter these disturbances by opposing forces using reciprocal activation of muscles, or attenuate their effect by increasing the joint impedance using muscle co-contraction. Increasing impedance is the only possible strategy to minimize the effect of high frequency disturbances, while for low frequency disturbances either reciprocal activation or co-contraction (and hence increased impedance) may be used. We analyze here how the selection between reciprocal activation and co-contraction depends on the frequency and amplitude of a disturbance and how and the control strategy is modified. Our results indicate that with increase of disturbance frequency the reciprocal activation decreases and is gradually replaced by co-contraction. Co-contraction increases with the magnitude and frequency of reciprocal activation even when impedance increase does not directly help the task, suggesting that co-contraction is a prerequisite for reciprocal activation control.

Introduction

Our central nervous system (CNS) achieves mechanical interaction with the environment by controlling the force (Shadmehr and Mussa-Ivaldi 1994) and impedance (Akazawa et al. 1983, Burdet et al. 2001) at the contact points of the limb. Roughly speaking, at a joint, torque is exerted by differential or reciprocal activation of the muscles spanning this joint, while impedance can be regulated by co-contracting antagonist muscle pairs. Therefore, the control of muscle reciprocal activation and co-contraction is central to motor control theories such as

the equilibrium-point hypothesis (Feldman 1966, Bizzi et al. 1984, Levin et al. 1992, Ostry and Feldman 2003) and the internal model theory of simultaneous force and impedance control (Franklin et al. 2008).

Torque provides power to perform a task while mechanical impedance, the resistance to positional perturbations, helps stabilizing the system against disturbances. Therefore in usual movement tasks there are clear distinctions in the role of the reciprocal activation and co-contraction. However, the distinction becomes ambiguous in postural control tasks where both may be utilized to maintain posture against disturbances. While intuitively we may expect that co-contraction is used to attenuate unpredictable or 'high-frequency' disturbances and reciprocal activation for 'low frequency' disturbances, it is unclear when and how transition occurs between the two strategies.

To investigate this question we examined the electromyography (EMG) activity of wrist muscles in a posture control task where subjects experienced sinusoidal perturbations of different frequencies applied by a computer controlled manipulandum while they tried to maintain their wrist position. We compared the position, force and EMG subjects produce in this task with the activity they produce while performing the same movement by choice. The results show that even though subjects are able to control active reciprocal activations over a frequency of 3 Hz, they prefer to slowly shift from reciprocal activation to co-contraction while resisting external disturbance and as the disturbance frequency increases.

Methods and Materials

Subjects and Task

Five males right-handed subject aged between 23-29 years, without known pathology, participated in the study. The experiments were conducted according to the principles in the Declaration of Helsinki. The experiments were approved by the ethics committee at Imperial College and the subjects gave informed consent prior to performing them.

The subjects stood in an upright posture and had their forearm fixed firmly with straps to a wrist manipulandum constraining the movement along the flexion/extension axis (Fig.1A). The subjects performed six 250-seconds long sessions: three *resistive* and three *active* sessions.

In the *resistive sessions* the subjects were given visual feedback of the wrist flexion/extension angle (blue cursor in Fig. 1B) and a target angle (± 3 degrees, area enclosed by the two green polygons in Fig. 1B). They were instructed to “maintain the wrist angular position within and near the boundaries of the target” while the manipulandum imposed sinusoidal torque perturbations in the flexion-extension direction of three torque magnitudes $\{T_1, T_2, T_3\}$ corresponding to 0.2, 0.35 and 0.5 of maximum voluntary co-activation (MVCA), respectively. The frequency of perturbation in each session varied from 0.4Hz to 3.5Hz. The order of frequencies were presented to the subject in a pseudo-random series $\{0.6, 1.9, 1.0, 2.8, 3.5, 0.4, 1.5, 1.2, 2.3, 0.8, 1.0, 3.5, 2.3, 0.6, 1.2, 0.4, 0.8, 1.9, 1.5, 2.8\}$ Hz with each frequency presented twice in each session. Frequencies lower than 1Hz were presented for 10 cycles, while frequencies greater than 1Hz were presented for 10 seconds each.

The three *active sessions* served as control and were designed to compare the reciprocal activation and co-activation values when the actuations of similar load and frequency were made voluntarily by the subjects. In these sessions, the subjects were provided visual feedback not of their hand position, but of their wrist torque as a cursor (Fig. 1B). The subjects actively performed isometric wrist flexions-extensions such that the torque cursor amplitude touched the boundaries of the same target as the resistive sessions. The cursor position was determined by multiplying the instantaneous subject torque by $3/T_1$, $3/T_2$ and $3/T_3$ degree/Nm respectively so as to equalize the load to the resistive sessions. The frequency of the movement was regulated using a metronome with frequencies (and order) similar to the resistive sessions. A rest period of few minutes was provided between sessions in order to avoid fatigue related effects. All subjects performed consecutive resistive and active sessions, though two performed the active sessions first and the rest performed the resistive session first.

Electromyography (EMG)

Surface EMG was measured from four wrist muscles: flexor carpi radialis (FCR), flexor carpi ulnaris (FCU), extensor carpi radialis bravis (ECRB) and extensor carpi ulnaris (ECU) which are the major contributors to wrist flexion and extension in a midway position (Hoffman and Strick 1999; Kakei et al. 1999; Haruno and Wolpert 2005). After electrode position was determined for each muscle using functional movements, the area was cleansed with alcohol and abrasive gel (Nuprep, DO Weaver), and disposable pre-gelled adhesive electrodes were fixed to the subject's skin (inter-electrode distance $\sim 1\text{cm}$) (Fig. 1C). A ground electrode was fixed to the

ankle of the subject. The EMG signals were pre-amplified using active clip connectors (g.GAMMAclip+g.GAMMABox, g.Tec) and amplified using a medically certified g.BSamp amplifier (g.Tec) before being fed into the manipulandum computer through a National Instruments data acquisition card (NI 6221) at 1 KHz and high-pass filtered at 20Hz. The filtered EMG was rectified and low pass filtered using a 2nd order Butterworth filter with a 5Hz cut-off frequency to obtain EMG_{fcr} , EMG_{cu} , EMG_{ecr} and EMG_{ecu} .

Subject specific load calculation and normalization

Each subject started the experiment by four repetitive calibration sessions separated by rest periods of 120 seconds. Each calibration session consisting of four parts:

i) Relaxation – subjects were asked to not move and relax their forearms as much as possible.

The average muscle activity during this part was added up for the flexor and extensor muscles to obtain the rest period activations for the flexor ($FLEX^{rest}$) and extensor (EXT^{rest}) muscles.

ii-iii) Flexion-Extension – subjects were instructed to either flex or extend their wrist and keep a constant level of wrist torque while the device was blocked at 0°. The torque level required from the subjects varied from 1-4Nm (i.e. 1Nm in session 1, 2Nm in session 2, and so on). A cursor was programmed to respond to individual torque measurements (scaling: 1Nm of applied torque moved the cursor of 4°), which provided subjects with visual feedback of the applied torque level. Subjects were asked to apply the force necessary to keep the cursor inside a target.

iv) Co-activation – subjects were asked to co-activate maximally.

Each part of the calibration lasted for 4s, with 30s of rest time between parts. The last 2s of each recording were used to calculate the parameters for the normalization of flexors and extensor muscle groups. *Relaxation*, *flexion* and *extension* tasks were used to compute a linear relationship between torque and EMG activation levels. Next, the mean of the *co-activation* parts through the four calibration sessions was used to determine the maxima of voluntary co-activation (MVCA) of each muscle (EMG_{fcr}^{MVCA} , EMG_{fcu}^{MVCA} , EMG_{ecr}^{MVCA} , EMG_{ecu}^{MVCA}) and hence the MVCA of the flexor ($EMG_{fcr}^{MVCA} + EMG_{fcu}^{MVCA} = FLEX^{MVCA}$) and extensor ($EMG_{ecr}^{MVCA} + EMG_{ecu}^{MVCA} = EXT^{MVCA}$) muscle groups. Levels at 0.2, 0.35 and 0.5 of the maximum activation of the flexor muscle group ($FLEX^{MVCA} - FLEX^{rest}$) were used to compute the torque loads T1, T2 and T3 respectively.

The rest and MVCA values from the calibration sessions were used to determine the normalized flexor (EMG_{flex}) and extensor (EMG_{ext}) muscle activations so as to enable comparison across antagonist muscles and subjects as:

$$EMG_{flex} = \frac{EMG_{fcr} + EMG_{fcu} - FLEX^{rest}}{FLEX^{MVCA} - FLEX^{rest}}$$

$$EMG_{ext} = \frac{EMG_{ecr} + EMG_{ecu} - EXT^{rest}}{EXT^{MVCA} - EXT^{rest}} \quad (2)$$

Fig.1D,E show the normalized flexor and extensor EMG from one of the subject with the corresponding force sensor readings during a section of the experiment. Note the distinctively different EMG patterns in the Resistive and Active sessions. In comparison to the active session, during the Resistive session, co-contraction increases more with the frequency and reciprocal

activation decreases with increase of frequency. We quantify these differences in the following sections of the article.

Data analysis

Task performance

The subject performance, measured as the cursor position (representing manipulandum position and subject torque in resistive and active sessions respectively), was low pass filtered using a zero-lag second order Butterworth filter with 5 Hz cut-off frequency and rectified. As each disturbance period lasted for at least 10 cycles, at least 20 peaks are expected in the rectified cursor position profiles. Of these, the 10 peak values were collected from latter part of each disturbance period and the mean was calculated for each subject, session and for every frequency. The mean and standard error of the subject means was plotted as in Figs. 2 and 3. Note that the latter part of the disturbance period was considered in order to avoid any transition effects from the previous disturbance.

Reciprocal activation and co-contraction

We start the EMG analysis by the definition of these two terms. Note that in the last section we have normalized each muscle EMG by the external torque the muscle produces. Reciprocal activation may be thus defined as the differential antagonist muscle activation which leads to an active external wrist torque. Co-contraction is defined as the common antagonist muscle activation that does not lead to an external torque. Note that the definitions includes the

voluntary activity produced by the subject and the involuntary reflexes in the muscles produced during perturbations.

Reciprocal activation (RA) and co-contraction (CC) are thus calculated as:

$$EMG_{RA,fr} = filter(EMG_{flex} - EMG_{ext}, fr/2)$$

$$EMG_{CC,fr} = \min(EMG_{ext}, EMG_{flex})$$

where $EMG_{RA,fr}$ and $EMG_{CC,fr}$ represent the reciprocal muscle activation and co-contraction at frequency fr . EMG_{flex} and EMG_{ext} represent the normalized flexor (Eq.1) and extensor EMG (Eq. 2). $filter(x,a)$, represents a Butterworth third order high pass filter of x with a cut-off frequency of a and $\min()$ takes the minimum of the two elements at any time instance. The high pass filter helped remove the co-contraction component from the EMG, while changing the cut-off frequency corresponding to the disturbance frequency helps to reduce any frequency dependent bias from the filter.

The RA and CC data from above were processed similar to the cursor position data. The mean of the peak 10 values from the latter part of each disturbance period were collected for each disturbance period and the average was calculated for each subject and for every frequency.

The mean and standard error of the subject averages were then plotted as in Fig. 3.

To compare the torque sensor readings with the reciprocal activation, we looked at the correlation between the two signals. To remove the delay between the muscle activity and the torque sensor signal, at each frequency, the correlation was repeatedly calculated by time shifting the torque signal between 0-100 ms. The maximum correlation during this process was noted for every frequency and subject. The subject average the standard error of the maximum

correlation was plotted for the resistive and active sessions in Fig. 4A. The corresponding subject average of the time –shifts (delay between RA and torque signal) are plotted in Fig. 4B.

Results

Subjects' performance

Figure 2 shows the accuracy of subject performance in the resistive and active sessions. All subjects succeeded in performing the task in both the resistive (A) and active (B) sessions. Irrespective of the task load (different colors) the subject cursor, representing wrist movement in the resistive and wrist torque in the active sessions, remained within the target area (brown dashed trace). In both the resistive and active sessions, while the cursor movement was not different across frequencies (resistive: $p=0.95$, active: $p=0.86$; 2 way ANOVA across all subject sessions and frequency), its amplitude was consistently larger at higher loads (resistive: $p<0.017$, act: $p<0.01$; 2 way ANOVA across all subject sessions and loads). However, across the resistive the active sessions, there was no significant difference in cursor movement at each load (0.2 MVCA: $p=0.93$, 0.35 MVCA: $p=0.77$, 0.5 MVCA: $p=0.16$; 2 way ANOVA across subject sessions of similar load and frequency).

Reciprocal activation and co-contraction

In the resistive sessions (Fig. 3A) the reciprocal activation (upper panel) increased with increase in load (different color traces) and decreased with increasing frequency of disturbance ($p<0.002$ and $p<0.001$ respectively, 2-way ANOVAs across subject sessions and loads and frequencies).

On the other hand, while co-contraction similarly increases with load ($p < 0.001$, 2-way ANOVAs across subject sessions and loads), it also increases with increase of frequency ($p < 0.001$, 2-way ANOVAs across subject sessions and frequencies).

In the active sessions, the reciprocal activation (Fig. 3B, upper panel) increased with load ($p < 0.001$, 2-way ANOVAs across subject sessions and loads) and frequency ($p < 0.006$, 2-way ANOVAs across subject sessions and frequencies), while co-contraction (Fig. 3B, lower panel) increased with frequency ($p < 0.001$, 2-way ANOVAs across subject sessions and frequencies); no significant difference was seen with increase of load ($p = 0.95$, 2-way ANOVAs across subject sessions and loads).

Co-contraction was found to be consistently higher in the resistive sessions in comparison to the active sessions (0.2MVCA: $p < 0.001$; 0.35 MVCA: $p < 0.001$; 0.5 MVCA: $p < 0.001$; 2 way ANOVA across subject sessions of similar load and frequency). We note that the trends of the total muscle activation, given by the summation of the flexor and extensor EMGs and corresponding roughly to mechanical impedance, were similar to that observed with co-contraction.

While reciprocal activation indicated the active torque applied by the subjects, we also had a torque sensor which measured the applied torque on the manipulandum handle. The correlation between recorded torque and reciprocal activation (Fig. 4A) indicated that, while in the case of the active sessions the correlation remained high through the frequencies, the correlation steadily decreased with frequency in the case of resistive sessions, indicating again the fading role of reciprocal activation in the perturbation compensation. The delay between the reciprocal activation and torque sensor increased with frequency in both the active and

resistive sessions (Fig. 4B), which is consistent with previous studies on muscle physiology (Patridge 1963).

Discussion

Our result showed that during posture control against random disturbances, the strategy changes with increasing frequency from that of reciprocal activation at low frequencies to co-contraction at higher frequencies. This gradual transition is not due to an impossibility to produce reciprocal activation at higher frequency. The active sessions clearly show that the subjects can perform reciprocal activation at higher frequencies. Interestingly, even in this case, the co-contraction increases with the frequency even when not directly required by the task. This differential choice of the co-contraction and reciprocal activation can explain the trend of correlation between the torque sensor readings and reciprocal activation (Fig.4A). The reading on the torque sensor provides us the external wrist torque measured. In the resistive sessions, this torque is produced by both the reciprocal activation and the stiffness of the wrist, which is related to the co-contraction produced by the subject. With increase of frequency, as the reciprocal activation decreases, the correlation with torque decreases. In contrast, co-contraction does not contribute to the task in the active sessions, correspondingly the torque signal is well correlated to reciprocal activation at all frequencies.

While the strategy employed by subjects differed with sessions, the subjects' performance (Fig. 2) were equally good in the active and resistive sessions. However, the cursor amplitude was consistently larger at larger loads. This observation may be explained by the fact that motor

output variability increases with EMG magnitude, as motor noise generally increases with muscle activity (Slifkin and Newell 1999).

It is interesting to observe co-contraction in the active isometric experiment when, as only reciprocal activation was required to perform this task. Co-contraction was observed (Fig. 3B) to increase with the frequency. Control of reciprocal activation at higher magnitudes and frequencies requires co-contraction due to the physiology of muscle force development. The CNS controls muscle force by modulating the frequency of the action potentials in the motor units. Both build-up and fall off of force (at the onset/conclusion of neural) activity is slow (Burke et al., 1973) and therefore, muscle force cannot follow rapid variations. To compensate for this, when rapid changes are required (like in the active isometric experiment), the CNS commands a higher burst of activity on the agonist muscles; this initial burst of activity would eventually lead to an overshoot in the force production unless it is compensated by a burst of activity in the antagonistic muscle (Kandel et al., 2000), giving rise to co-contraction.

In the resistive sessions, several additional factors could have also contributed to the increase of co-contraction. First, when performing a posture control task, the CNS responds to perturbations with reflexes whose gains can vary according to the task and change in the environment. Delay on the reflex response varies depending on its origin, e.g. 30ms for the monosynaptic reflexes, 50-80ms for the long-loop reflexes (Evarts, 1973; Hammond, 1956) or 110-150ms for visual reflexes (Franklin and Wolpert, 2008). As the nature of the task was to maintain the wrist at a given posture, the CNS may have modified reflex gains and utilized mechanisms of inhibition to the Ia inhibitory interneurons in order to build (or help building) co-contraction automatically (Nielsen, 1993). In addition, the burst of activity produced by these

reflexes on one muscle could start overlapping with the activity on the antagonist muscle when the perturbation changes direction, thus building up co-contraction.

Second, experiments in primates have shown that firing-rate modulation of both slow and fast twitching units depends on the frequency of the perturbation (Humphrey and Reed, 1983). For slow perturbations, slow-twitch units begin to fire, but as the frequency increases, fast-twitch units start to interact and tonic co-activation drive on both flexors and extensors is increased.

Further, it is well known that motor noise increases with muscle activation (Hamilton et al. 2004). However, an increase of co-contraction has been shown to reduce motion variability (Burdet et al. 2001) probably due to the faster increase of the muscle impedance than motor noise (Selen et al. 2005, Tee et al. 2010). In addition, co-contraction is also known to increase the reflex response to perturbation in the stretched (Akazawa 1983, Carter et al. 1993).

Increase of co-contraction may thus also be a strategy utilized by the CNS to aid the control of the reciprocal activation to move the wrist.

Finally, the decrease in reciprocal activation at higher frequencies in the resistive sessions can be explained as follows. With the change of disturbance frequency the increase of co-contraction, due to all the above reasons, also leads to a fast increase of muscle impedance (Selen et al. 2005, Tee et al. 2010). The increased impedance, in turn, 'takes over' the posture control task and enables the CNS to reduce reciprocal activation, which lack in efficiency at higher frequencies due to the muscle dynamics which have a cut off of 1.8Hz (Patridge 1966).

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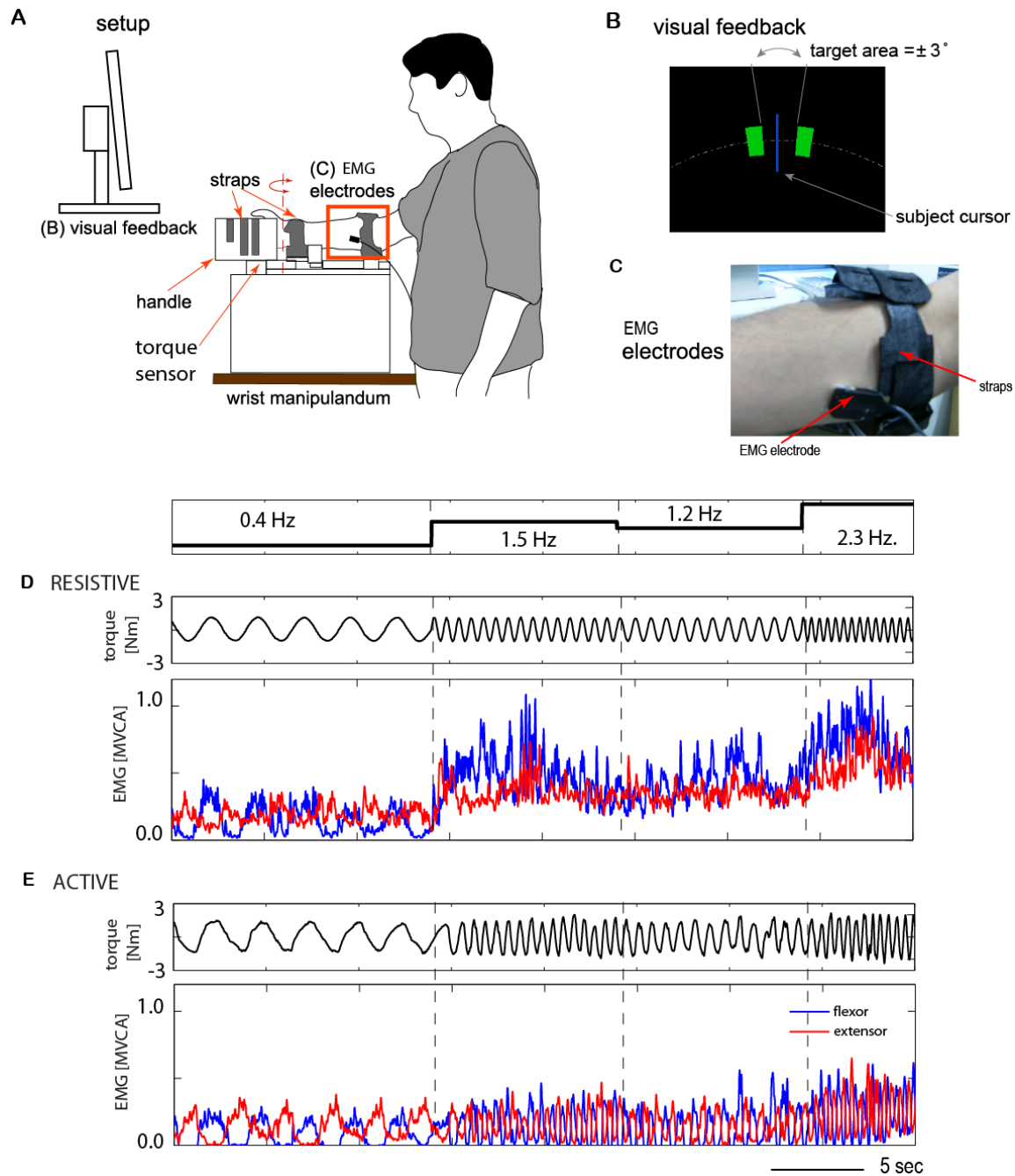


Fig. 1: Experimental setup. Subjects stood in an upright position with their forearm fixed to the wrist interface firmly (A) by straps. (B): They were provided with visual feedback of the target amplitude (enclosed by green squares) and a cursor (blue line) corresponding to their wrist position (in resistive sessions) or wrist torque (in active sessions). The enclosing green squares

turned red if the cursor moved over them indicating to the subjects that they were overshooting the allowed amplitude. EMG (C) was recorded from four wrist muscles. (D) and (E) show the torque and EMG over 40 seconds of the experiment in the resistive and active sessions respectively for one subject.

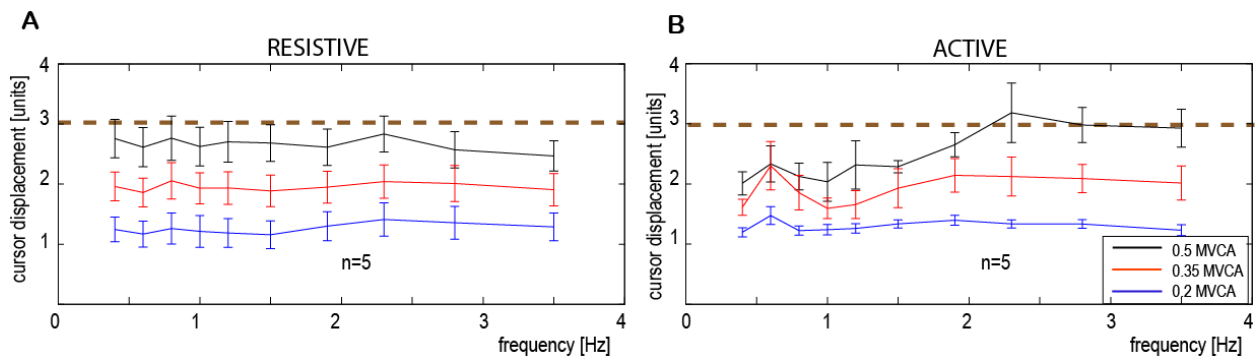


Fig 2: Task performance. The target cursor amplitude across subjects, which corresponds to the subject wrist amplitude in the resistive sessions (A) and to the isometric torque amplitude in active sessions (B), indicates that the subject cursor was maintained on the target (brown dashed trace) at all frequencies and loads. Error bars represent standard error.

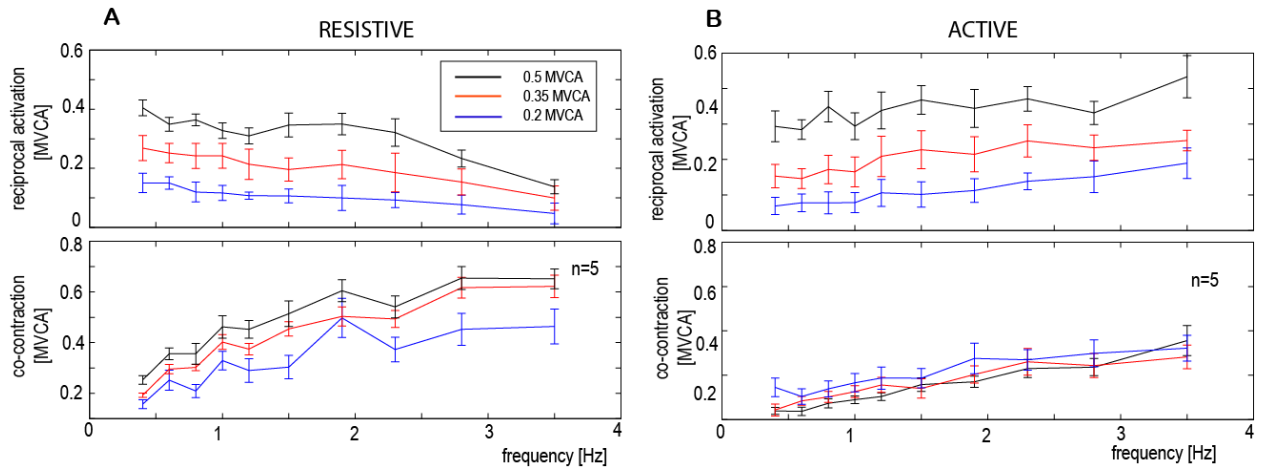


Fig. 3: Active and resistive behaviors. The mean (lines) and standard error (error bars) of reciprocal activation (upper panels) and co-activation (lower panels) exhibited across subjects is plotted for the resistive (A) and active (B) sessions. The values are plotted in units of MVCA.

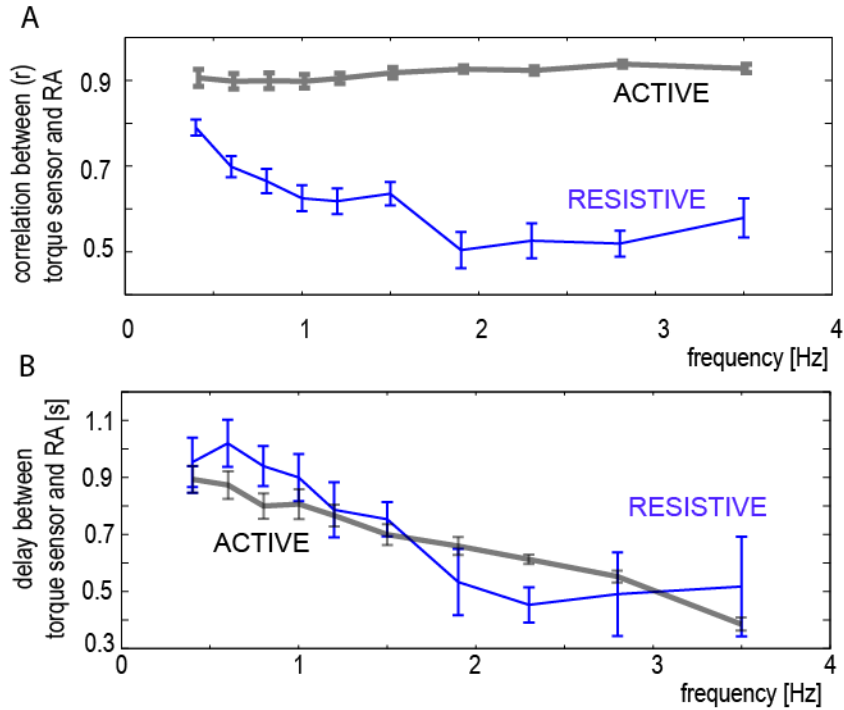


Fig4: A) Correlation between torque sensor signal and reciprocal activation EMG (EMG_{RA}) was plotted for every frequency. The data shows the average across all subjects and sessions. The correlation remains at a constant high value for the active sessions, while it steadily decreases with frequency in the case of the resistive sessions. The correlation plotted in (A) represents the maximum value calculated by time shifting the torque signal with respect to the RA at every frequency in order to remove the temporal delays. (B) shows the across subject and sessions average time shifts in the active and resistive sessions.