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RESEARCH ARTICLE

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Task dependent gain regulation of spinal circuits projecting to the human flexor carpi radialis

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Abstract In humans, the flexor carpi radialis (FCR) and extensor carpi radialis (ECR) muscles act as antagonists during wrist flexion-extension and as functional synergists during radial deviation. In contrast to the situation in most antagonist muscle pairs, Renshaw cells innervated by the motor neurons of each muscle inhibit the motoneurons, but not Ia inhibitory interneurons, of the opposite motor pool. Here we compared gain regulation of spinal circuits projecting to FCR motoneurons during two tasks: flexion and radial deviation of the wrist. We also investigated the functional consequences of this organisation for maximal voluntary contractions (MVCs). Electromyographic (EMG) recordings were taken from FCR, ECR longus and ECR brevis using fine-wire electrodes and electrical stimulation was delivered to the median and radial nerves. Ten volunteers participated in three experiments.

1. To study the regulation of the Renshaw cell-mediated, inhibitory pathway from ECR to FCR motoneurons, forty stimuli were delivered to the radial nerve at 50% of the maximal M-wave amplitude for ECR brevis. Stimuli were delivered during both isometric wrist flexions and radial deviation actions with an equivalent EMG amplitude in FCR (~5% wrist flexion MVC).
2. To explore the homonymous Ia afferent pathway to FCR motoneurons, 50 stimuli were delivered to the median nerve at intensities ranging from below motor threshold to at least two times that which evoked a maximal M-wave during wrist flexion and radial deviation (matched FCR EMG at ~5% wrist flexion MVC).
3. EMG amplitude was measured during MVCs in wrist flexion, extension and radial deviation.

There was no significant difference in the inhibition of FCR EMG induced via ECR-coupled Renshaw cells between radial deviation and wrist flexion. However, the mean FCR H-reflex amplitude was significantly ($P<0.05$) greater during wrist flexion than radial deviation. Furthermore, EMG amplitude in FCR and ECR brevis was significantly ($P<0.05$) greater during MVCs in wrist flexion and extension (respectively) than radial deviation. ECR longus EMG was significantly greater during MVCs in radial deviation than extension. These results indicate that the gain of the Renshaw-mediated inhibitory pathway between ECR and FCR motoneurons is similar for weak flexion and radial deviation actions. However, the gain of the H-reflex pathway to FCR is greater during wrist flexion than radial deviation. Transmission through both of these pathways probably contributes to the inability of individuals to maximally activate FCR during radial deviation MVCs.

Keywords H-reflex · FCR · ECR · Renshaw cell · Wrist muscle · Co-contraction · Maximum voluntary contraction

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Introduction

The organisation of spinal circuits between the extensor and flexor motor pools of the human and feline wrist differ from the standard reciprocal arrangement that exists between most pairs of antagonist muscles (Aimonetti et al. 2000a, 2000b; Aymard et al. 1995, 1997, 2001; Baret et al. 2003; Hultborn et al. 1971a, 1971b, 1971c; Illert and Wietelmann 1989; Rossi et al, 1995). Unlike connections between antagonist motor pools of the lower limb, Renshaw cells innervated by the motor neurons of extensor carpi radialis (ECR), inhibit the motor neurons of the flexor carpi radialis (FCR) (Aymard et al. 1997), but not the Ia inhibitory interneurons that project to FCR

motor neurons (Aymard et al. 1995) as shown in Fig. 1A. An equivalent organisation of circuits was also demonstrated in the reverse direction; that is, between FCR-coupled Renshaw cells and ECR motoneurons. Aymard et al. (1995, 1997) suggested that this difference may be due to the unique functional requirements of the wrist muscles, in that the FCR and ECR act as antagonists during wrist flexion-extension, and as functional synergists during radial deviation.

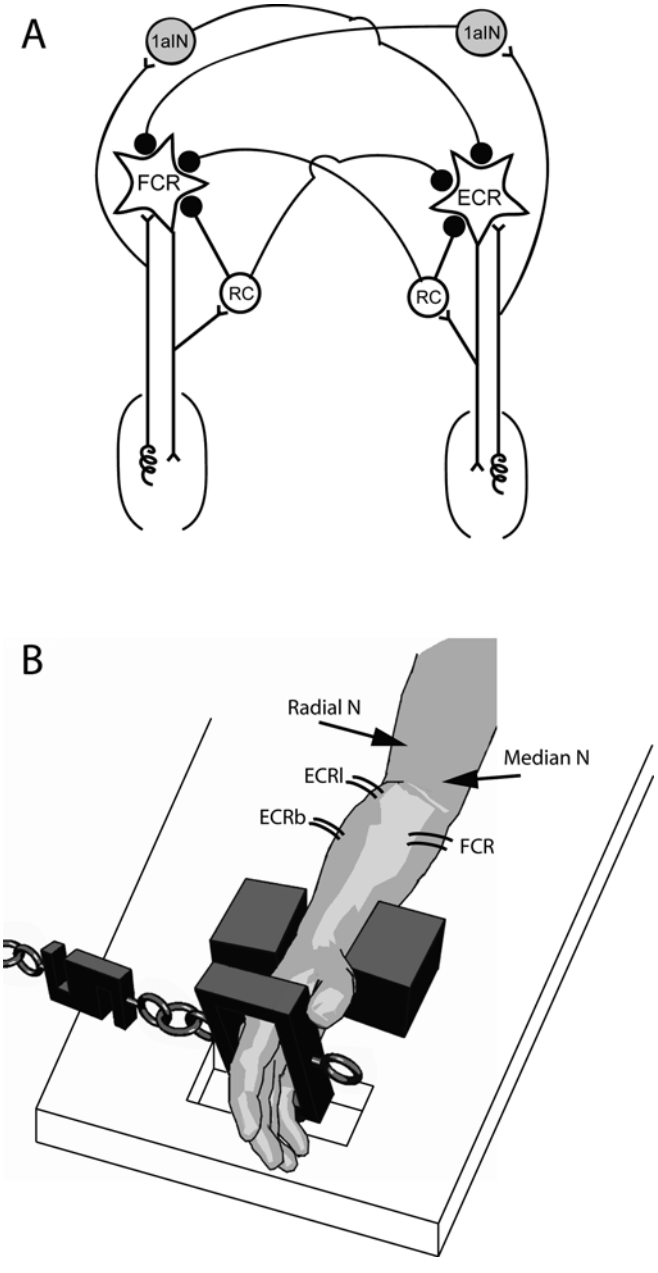

Fig. 1 **A** Schematic representation of the proposed organisation of Renshaw-cell circuitry within the wrist motor pools. 1a inhibitory interneurons (*1aIN*), ECR and FCR motoneurons (*ECR and FCR in star-shaped cells*), and Renshaw cells (*RC*) are displayed. Adapted from Aymard et al. (1997). **B** Illustration of the experimental set-up. The approximate sites of insertion of fine-wire electrode pairs (1 cm inter-electrode distance) are shown for ECR longus (*ECRl*), ECR brevis (*ECRb*) and FCR. The approximate location of stimulating electrodes for the radial and median nerves are illustrated by *arrows*

The purpose of this study was threefold. First, we sought to compare the regulation of the inhibitory Renshaw-cell pathway from ECR to FCR motoneurons between wrist flexion and radial deviation. It is known that recurrent inhibition operates with similar gain during agonist only contraction and co-contraction at low levels of muscle contraction (~10% MVC) in the human leg (Nielsen and Pierrot-Deseilligny 1996). At higher levels of contraction (~50% MVC), however, recurrent inhibition is suppressed during agonist activity but not during co-contraction. In contrast to disynaptic 1a inhibitory (Aimonetti et al. 2000a; Nielsen and Kagamihara 1992) and presynaptic inhibitory (Aimonetti et al. 2000b; Aymard et al. 2001; Nielsen and Kagamihara 1993) pathways, there has been no previous investigation of how transmission in circuits from ECR-coupled Renshaw cells to FCR motoneurons is modulated depending on task. We studied this issue to shed light on the functional role that these circuits might play during normal movement. Second, we compared the amplitude of FCR H-reflex amplitude between wrist flexion and radial deviation, to determine the task-dependent regulation of the (predominantly) 1a afferent reflex pathway within the context of the unique reciprocal arrangement of circuits between the wrist motor pools. Third, we sought to investigate the functional consequences of the unique arrangement of these circuits for maximal muscle activation. Thus, we compared the activity of FCR during maximal wrist flexion versus radial deviation contractions, and of ECR during maximal wrist extension and radial deviation actions. We used fine-wire electromyography (EMG) to selectively record from both ECR longus (ECRl) and ECR brevis (ECRb) because Riek et al. (2000) showed that the two heads of ECR were differentially activated during extension versus radial deviation actions. Some of these data have been reported previously (Carroll and Baldwin 2004).

Methods

Subjects

Ten individuals (seven males, three females) with no documented neurological disease volunteered for this experiment. The participants ranged in age from 21 to 39 years. The procedures conformed to the Declaration of Helsinki and were approved by the Human Research Ethics Board at the University of Alberta.

Procedure

The study consisted of three parts. In the first part we compared the extent of Renshaw cell-mediated inhibition from ECR to FCR, by comparing the degree to which ongoing FCR EMG was reduced by a radial nerve (RN)

stimulus at the elbow between wrist flexion and radial deviation. In the second part, FCR H-reflex amplitudes were compared between trials in which participants maintained a small EMG contraction (~5% MVC) while producing isometric wrist flexion or radial deviation torque. Third, we compared EMG activity in three wrist muscles (FCR, ECRl and ECRb) during trials in which participants performed wrist flexion, wrist extension and radial deviation MVCs.

General set-up

Participants were seated comfortably with their right arm resting on a table as shown in Fig. 1B. The right wrist was held in a neutral orientation, such that flexion-extension movements occurred in the horizontal plane. Padded blocks were positioned just proximal to the wrist joint to restrict motion of the forearm. Torques exerted during isometric flexion, extension, and radial deviation actions of the wrist were measured by an S-type load cell (SSM100, Interface, Scottsdale, AZ, USA) that was connected in series with a metal chain. The chain was attached to the appropriate side of a padded, metal rectangle that fit snugly around the hand just proximal to the metacarpophalangeal joint to record flexion, extension and radial deviation forces at the wrist. This arrangement permitted contractions about the wrist without the application of a grip force. Participants were provided real-time visual feedback of the torque and FCR EMG activity via a computer monitor positioned in front of them.

Nerve stimulation

The median nerve (MN) and RN were stimulated with single 1-ms pulses applied through bipolar surface electrodes using a Grass S88 stimulator, connected in series with a Grass SIU5 isolator and a Grass CCU1 constant current unit (Grass Instruments, AstroMed, West Warwick, RI, USA). The MN was stimulated just proximal to the medial epicondyle of the humerus and the RN was stimulated ~3–5 cm proximal to the lateral epicondyle of the humerus in the spiral groove. The current provided by the stimulator was measured (mA-2000 Noncontact Milliammeter, Bell Technologies, Orlando, FL, USA).

EMG

Two electrodes, each consisting of a single strand of insulated fine wire, were inserted into each of the three forearm muscles (FCR, ECRl, ECRb) via a hypodermic needle (27 gauge) to a depth between 0.5 and 1 cm. Approximately 2–3 mm of the insulation was removed from the tip of each wire. The inter-electrode distance was ~1 cm. Electrode sites for ECRl and ECRb were determined according to the methods described by Riek et al. (2000). EMG signals were preamplified (gain of 200–1000; P511 Grass Instruments, AstroMed), and band-pass filtered (30–1000 Hz). Subjects maintained a 5% MVC contraction using real time visual feedback of the full-wave rectified and low pass filtered (3 Hz) EMG signals.

Reciprocal-Renshaw cell inhibition experiments

Forty stimuli were delivered to the RN in separate trials in which participants exerted either wrist flexion or radial deviation torque. Stimulus intensity was adjusted to evoke an M-wave in ECRb at ~0.5 times the maximal M-wave (M-max) according to the procedures described by Aymard et al. (1997). The same current was applied to the stimulus electrodes for flexion and radial deviation trials. In both trials, participants matched EMG in FCR to ~5% of the peak EMG activity recorded during their wrist flexion MVC.

H-reflex experiments

In separate trials, participants exerted either wrist flexion or radial deviation torque while matching FCR EMG activity to ~5% of the peak EMG activity recorded during their wrist flexion MVC. Fifty stimuli were applied to the MN (interstimulus interval 3–5 s) to elicit M-waves and H-reflexes in the FCR muscle. Stimulus intensity was adjusted pseudo-randomly from below the motor threshold to at least two times that required to elicit M-max, in such a way that most of the stimuli elicited responses on the ascending limb of the H-reflex recruitment curve.

MVC experiments

Each participant performed two MVCs in each of the three directions of wrist action: flexion, extension and radial deviation. To familiarise themselves with the experimental set-up participants performed up to five submaximal efforts in each direction before the MVCs were performed. The order in which the different directions were tested varied randomly between subjects and at least 1 min elapsed between subsequent MVCs. Participants were provided real-time feedback of their torque and were verbally encouraged to produce a maximal effort. Each attempt lasted for ~5 s.

Data acquisition and analysis

Data were sampled at 5000 Hz with a 12-bit National Instruments (Austin, TX, USA) A/D board interfaced with a computer running custom-written Labview (National Instruments) software. For the experiments involving nerve stimulation, data were collected from 150 ms prior to stimulation to 250 ms after stimulation. Data analyses

were performed off line using custom-written Matlab (The MathWorks, Natick, MA, USA) software. For the Renshaw cell inhibition experiments, the FCR EMG was full-wave rectified and averaged over the 40 trials. The mean EMG amplitude prior to stimulation, the minimum EMG amplitude in the period of inhibition (i.e. peak inhibition), and average EMG amplitude during the period of inhibition (i.e. average inhibition) were measured. The period of inhibition was specified by manual placement of cursors at the departure and return of the post-stimulus EMG to the pre-stimulus baseline. The degree of inhibition was expressed as a percentage of the pre-stimulus EMG amplitude. For the H-reflex experiments the amplitude of each response and the current delivered were calculated. The three largest H-reflex amplitudes were averaged to calculate the maximal H-reflex amplitude (H-max). The H-reflex amplitudes of all trials in which the M-wave amplitude was close to (within ±2.5% M-max) half of the M-wave amplitude at H-max were also averaged to provide a measure of H-reflex amplitude on the ascending limb of the recruitment curve (H-ascend). For the MVC experiments, data were only processed for the trial in which the greatest torque was exerted. Cursors were manually set 0.5 s either side of the time of peak torque, and the peak torque, and mean rectified EMG amplitude in the 1 s window were measured.

Statistics

Paired *t*-tests were used to compare pre-stimulus EMG amplitudes, the degree of reciprocal-Renshaw cell-inhibition and H-reflex amplitudes between flexion and radial deviation trials. A two-way ANOVA with planned comparisons was conducted to compare EMG amplitudes in FCR between the wrist flexion and radial deviation MVCs, and in ECRl and ECRb between wrist extension and radial deviation (data were log transformed prior to analysis due to skewness). Statistical significance was set at $P<0.05$ for all tests. All descriptive statistics are reported as the mean±SEM.

Results

The direction of torque applied at the wrist significantly affected the EMG activity of the three muscles studied during MVCs and the amplitude of the FCR H-reflex. In contrast, Renshaw inhibition, assessed by RN stimulation, was unaffected.

Renshaw cell experiments

Inhibition of FCR motoneurons by ECR-coupled Renshaw cells was assessed by measuring the inhibition in ongoing FCR EMG activity evoked by stimulation of the RN. Figure 2A shows that the magnitude of inhibition (indicated by the shaded region) was similar when a single subject performed wrist flexion and radial deviation. For the group there was no significant difference in the extent to which the FCR EMG was inhibited by ECR-coupled Renshaw cells between radial deviation and wrist flexion (Fig. 2B). This was true both for the peak inhibition (flexion; 72.1±3.5%, radial deviation; 70.5 ±2.9%; $P=0.61$) and the mean inhibition (flexion; 46.4 ±3.3%, radial deviation; 42.7±2.2%; $P=0.31$). The amplitude of pre-stimulus EMG activity was also not different between tasks (6.5±1.3% of flexion MVC EMG during flexion, 6.2±1.2% of flexion MVC EMG during radial deviation; $P=0.12$).

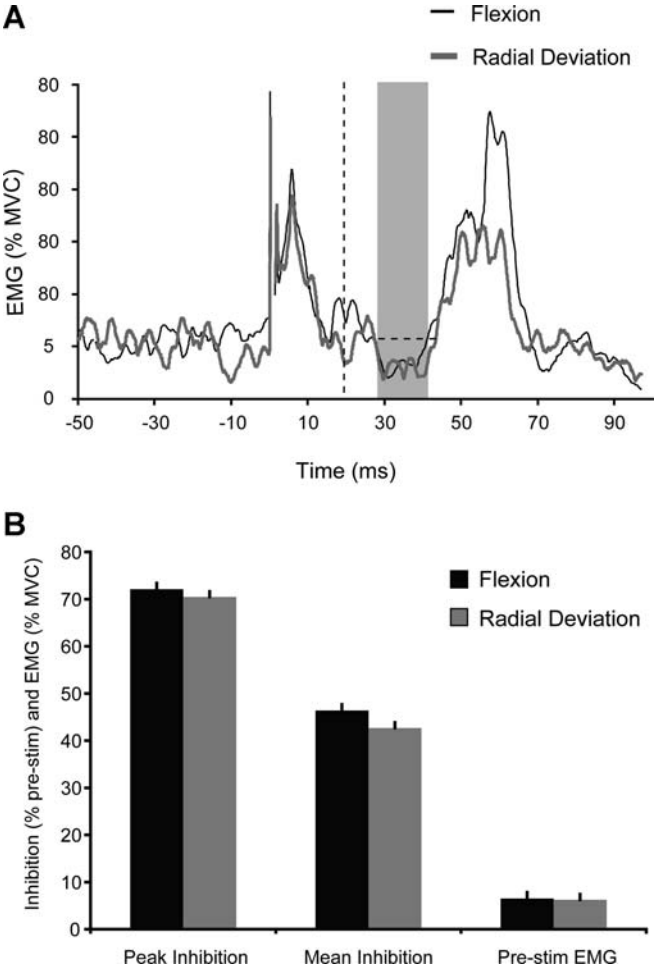

Fig. 2 **A** An example of the ECR-coupled, Renshaw cell-mediated inhibition of FCR motoneurons during flexion and radial deviation muscle actions. Each trace is the mean of 40 sweeps for a single participant. The *vertical and horizontal dashed lines*, respectively, depict the H-reflex latency for this participant and the mean pre-stimulus EMG amplitude. The *shaded area* represents the period over which the mean inhibition was calculated. **B** Mean (±SEM) values for the peak Renshaw cell-mediated inhibition, the average Renshaw cell-mediated inhibition, and the pre-stimulus EMG amplitudes during flexion and radial deviation muscle actions for the group ($n=10$)

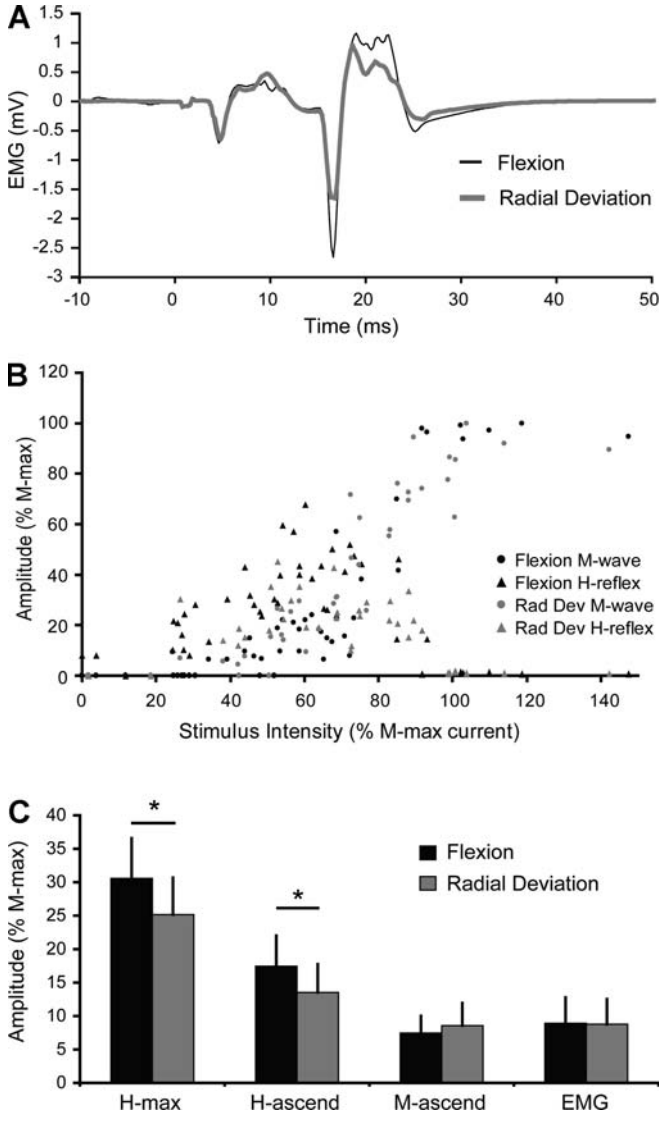

Fig. 3 **A** Raw H-reflex traces recorded during flexion and radial deviation at matched M-wave amplitudes for a single participant. **B** Example of H-reflex recruitment curves during wrist flexion and radial deviation for a single participant. The amplitudes of the evoked potentials are scaled to the M-max amplitude recorded during each task, and the stimulus intensity is scaled according to the current level at motor threshold and the current required to elicit M-max in each task. (*Rad. Dev.*=radial deviation). **C** Mean (±SEM) H-max, H-ascend and pre-stimulus EMG amplitudes during flexion and radial deviation muscle actions for the group (n=10). The H-reflex and M-wave values are expressed relative to M-max, and the EMG values are expressed relative to the EMG recorded during MVC

H-reflex experiments

Results of the H-reflex experiments are summarised in Fig. 3. Panel A shows the attenuation of H-reflexes sampled from the ascending limb of the recruitment curve during radial deviation compared to wrist flexion for one subject. Data were compared at similar stimulus intensities as inferred from the matched M-wave amplitudes. Panel B shows M/H recruitment curves recorded from a representative subject during wrist flexion and radial deviation. H-reflex amplitudes are generally smaller at similar M-wave amplitudes during radial deviation than wrist flexion. Panel C summarises the data for the group of ten subjects. Mean amplitudes of maximal FCR H-reflexes were significantly (P<0.05) greater during wrist flexion than radial deviation, while pre-stimulus EMG amplitudes were not significantly different between tasks for these trials. H-reflexes sampled from the ascending limb of the recruitment curve (H-ascend) were also significantly (P<0.05) greater during wrist flexion than radial deviation. Pre-stimulus EMG and M-wave amplitudes for the trials averaged to yield H-ascend data were not significantly different between tasks.

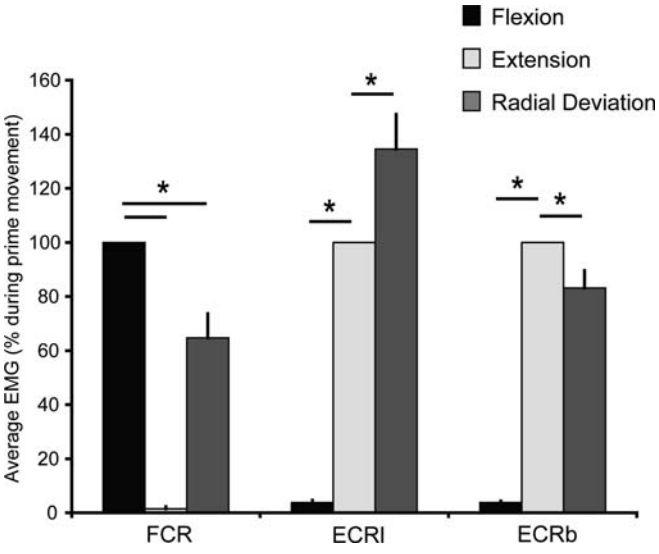

Fig. 4 Mean (±SEM) rectified EMG amplitude during flexion, extension and radial deviation MVCs for the group (n=10)

MVC experiments

Figure 4 shows the mean rectified EMG activity recorded during MVCs in wrist flexion, extension and radial deviation for the entire group (n=10). Data are expressed relative to MVCs recorded during flexion for FCR and extension for the two heads of ECR. EMG activity in FCR was largest during wrist flexion, was significantly smaller during radial deviation (P<0.05), and was negligible during wrist extension. ECRb EMG activity was greater during wrist extension MVCs than radial deviation (P<0.05) and was minimal during wrist flexion. EMG from ECRl was significantly greater during radial deviation MVCs than extension MVCs (P<0.05), and was smallest during wrist flexion.

Discussion

The results of this study show that the inhibitory pathway between ECR-coupled Renshaw cells and FCR motoneurons operates with a similar gain during weak flexion and radial deviation tasks of the human wrist. In contrast,

transmission from homonymous Ia afferents to FCR motoneurons is depressed during radial deviation compared with wrist flexion. Furthermore, for ECRb and FCR, greater EMG activity occurs during extension and flexion MVCs (respectively) than during radial deviation MVCs. This suggests that a degree of inhibitory coupling between the two muscles persists when they are activated together as functional synergists. During radial deviation MVCs it is likely that decreased transmission through the homonymous Ia pathway to FCR motoneurons, combined with the inhibition from ECR-coupled Renshaw cells, play a role in the inability to maximally activate the FCR when compared to flexion MVCs.

Renshaw cell experiments

The current experiments employed the conditioning technique of Aymard et al. (1997) to assess the gain of the pathway from ECR-coupled Renshaw cells to FCR motoneurons. Aymard et al. (1997) presented evidence from a number of sources to establish that Renshaw cell inhibition is the major mechanism responsible for the long-lasting inhibition of multi-unit FCR EMG induced by supra-threshold RN stimulation. It remains possible, however, that some contribution from afferent stimulation is involved in the EMG inhibition, particularly at high stimulus intensities. Indeed, there was relatively short lasting inhibition of single motor unit firing probability induced by 0.95 motor threshold stimulation in 11 of the 27 FCR motoneurons studied by Aymard and colleagues. Aimonetti et al. (2001) also showed relatively small reduction in firing probability of single ECR motor units after MN stimulation.

There was no task-dependent difference in the expression of reciprocal-Renshaw cell inhibition in this study, which is consistent with data obtained by Nielsen and Pierrot-Deseilligny (1996) on recurrent Renshaw cell inhibition during weak contractions in the lower leg. Nielsen and Pierrot-Deseilligny (1996) also found that recurrent inhibition was suppressed during strong agonist contraction, but not strong co-contraction. Although we have no data on Renshaw cell inhibition during strong contractions, and the available evidence came from situations in which the target of Renshaw cell inhibition was different (i.e. homonomous versus antagonist motor pools), it appears that for weak contractions there is a similar central control of Renshaw cell excitability during co-contractions at the ankle and wrist. We only studied here the effects of RN conditioning on FCR EMG, but not the effects of MN conditioning on ECR EMG. It would be particularly interesting to compare the effects of median nerve conditioning on ECRl and ECRb, in light of the different recruitment of the two muscles during MVC in wrist flexion versus radial deviation (see below).

H-reflex experiments

Our finding of a reduction in H-reflex amplitude during co-contraction of antagonist muscles compared to during a purely agonist contraction mirrors the findings of Nielsen and co-workers (1993, 1994) from the lower leg. The mechanisms underlying this reduction in H-reflex during radial deviation actions are unclear, however, our findings are unlikely to be due to post-synaptic effects on the FCR motoneurons. This is because the level of EMG in FCR was matched between the radial deviation and flexion tasks. If similar neural control mechanisms operate in the lower and upper limbs, it would follow that the task-dependent changes in H-reflex amplitude were mediated, at least in part, by increased pre-synaptic inhibition at the Ia afferent terminals, because Nielsen and Kagamihara (1993) demonstrated increases in presynaptic inhibition during co-contraction of the flexors and extensors of the ankle. However, increased presynaptic inhibition of wrist muscles was not observed during tasks requiring co-contraction of antagonists, or prior to pure antagonist contraction (Aimonetti et al. 2000b; Aymard et al. 2001), and alternative possibilities could explain the reductions in H-reflex amplitude in our current study. Electrical stimulation of a peripheral nerve does not selectively excite group Ia afferents, but rather group Ib, and II afferents may contribute to the H-reflex (for a review see Misiaszek 2003). Modulation of transmission in spinal circuits involving these afferents might therefore have contributed to our results. Furthermore, the group 1a effects on FCR motoneurons are not entirely monosynaptic (Burke et al. 1984; Marchand-Pauvert et al. 2002). It is possible that non-monosynaptic pathways that contribute to the FCR H-reflex were modulated differently for the radial deviation and wrist flexion tasks, and that this was the reason for the reduced H-reflex amplitude during radial deviation.

MVC experiments

There was greater EMG activity during radial deviation than wrist extension in ECRl in the current study, but less EMG during radial deviation than wrist extension for ECRb. These results are consistent with those of Riek et al. (2000), who studied sub-maximal contractions of ECR. Here, we extend the observations of Riek et al. (2000) to the FCR muscle and make quantitative comparisons of muscle activity during maximal contractions. For FCR and ECRb, our results show that participants were unable to fully activate these muscles together during a maximal radial deviation effort. Whether this inability to fully activate wrist muscles leads to a reduction in torque could be assessed by using the interpolated twitch technique. A reduction in EMG activity relative to pure agonist contractions has also been shown during maximal co-contractions of the biceps and triceps brachii at the elbow (Tyler and Hutton 1986). This is a somewhat different situation, however, because the elbow flexors and extensors are strict anatomical antagonists, and their

mutual activation leads only to an increase in joint stiffness. It is possible that EMG activity was sub-maximal in this case because of the requirement to balance the flexor and extensor torques at the elbow.

Our current results indicate that, for muscles that can act as antagonists and functional synergists, the inhibitory coupling that exists for antagonist contractions persists to some extent during synergistic activation. A number of circuits are likely to be responsible for this inhibitory coupling. Our finding that the gain of the reciprocal-Renshaw-cell pathway between ECR and FCR is not reduced during weak radial deviation suggest that this pathway might inhibit FCR activity during radial deviation, but not during pure wrist flexion, when ECR, and thus presumably the ECR-coupled Renshaw cells, are silent. The recurrent effects of FCR-coupled Renshaw cells on their own motoneurons is also likely to be involved, because recurrent inhibition is suppressed during strong agonist contraction, but not strong co-contraction (Neilsen and Pierrot-Deseilligny 1996). The reduced amplitude of the FCR H-reflex during radial deviation versus wrist flexion suggests that reduced feedback from homonymous Ia inputs to FCR motoneurons might also contribute to the lower FCR EMG during radial deviation MVCs. Reduction in descending drive to FCR and ECRb during radial deviation could also be a contributing factor.

In contrast to ECRb and FCR, the ECRl muscle was more active during radial deviation than wrist flexion or extension. Riek et al. (2000) suggested that the differences in recruitment between ECRl and ECRb might be related to their anatomical characteristics. ECRl has a greater moment arm for radial deviation than wrist extension because it inserts at the base of the second metacarpal, whereas ECRb has a greater moment arm for wrist extension due to its insertion at the base of the third metacarpal. However, irrespective of the anatomical features of these muscles, it is clear that the organisation of the neural circuits responsible for controlling them is different. The finding that ECRl was activated to a greater extent when the wrist extensors and flexors were co-activated than when participants exerted pure wrist extension torques suggests a fundamental departure from the typical reciprocal arrangement of anatomical antagonists. It might be that the reciprocal, inhibitory connections between neural elements involved in the recruitment of wrist flexors and ECRl are weaker than those for ECRb. It is also possible that facilitation from spinal and/or supra-spinal sites that drive other heteronomous muscles involved in radial deviation is required to fully activate the ECRl motor pool. Irrespective of these speculations, the current results strongly argue that ECRl and ECRb should be independently considered in future investigations on the neural control of wrist movement.

Future directions

This study has enhanced our understanding of the neural control of human wrist muscles, however, a number of issues remain unresolved. Future studies should determine whether there is a similar gain of the reciprocal pathways from ECR-coupled Renshaw cells to FCR motoneurons for alternate tasks that involve co-contraction of wrist flexor and extensor muscles (such as hand clenching). Comparisons of the degree of inhibition of ECRl and ECRb motoneurons by FCR-coupled Renshaw cells should also be included in studies designed to elucidate the neural organisation responsible for the functional differences in recruitment of these muscles. Finally, the gain of other spinal circuits such as presynaptic and 1a reciprocal inhibition during radial deviation contractions should be determined.

Conclusions

This study shows that the gain of the reciprocal pathways from ECR-coupled Renshaw cells to FCR motoneurons is similar during weak radial deviation and wrist flexion contractions but the gain of the H-reflex pathway to FCR is attenuated during radial deviation. Transmission through both pathways probably contributes to the inability of participants to fully activate FCR during radial deviation MVCs. These results suggest that the control system for the human wrist is not optimised for the production of maximal force in radial deviation, when co-contraction of flexors and extensors is required. It is possible that neural organisation of the wrist circuitry might be influenced largely by its primary functional requirements; such as to exert grip forces.

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