Voluntary motor commands reveal awareness and control of involuntary movement

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Abstract

The capacity to inhibit actions is central to voluntary motor control. However, the control mechanisms and subjective experience involved in voluntarily stopping an involuntary movement remain poorly understood. Here we examined, in humans, the voluntary inhibition of the Kohnstamm phenomenon, in which sustained voluntary contraction of shoulder abductors is followed by involuntary arm raising. Participants were instructed to stop the involuntary movement, hold the arm in a constant position, and 'release' the inhibition after ~2 s. Participants achieved this by modulating agonist muscle activity, rather than by antagonist contraction. Specifically, agonist muscle activity plateaued during this voluntary inhibition, and resumed its previous increase thereafter. There was no discernible antagonist activation. Thus, some central signal appeared to temporarily counter the involuntary motor drive, without directly affecting the Kohnstamm generator itself. We hypothesise a form of "negative motor command" to account for this novel finding. We next tested the specificity of the negative motor command, by inducing bilateral Kohnstamm movements, and instructing voluntary inhibition for one arm only. The results suggested negative motor commands responsible for inhibition are initially broad, affecting both arms, and then become focused. Finally, a psychophysical investigation found that the perceived force of the aftercontraction was significantly overestimated, relative to voluntary contractions with similar EMG levels. This finding is consistent with the hypothesis that the Kohnstamm generator does not provide an efference copy signal. Our results shed new light on this interesting class of involuntary movement, and provide new information about voluntary inhibition of action.

Keywords: Motor control; Involuntary movement; Inhibition; Action awareness; Bilateral movement; Negative motor command

1. Introduction

The capacity both to initiate actions, and to inhibit them, is central to cognitive motor control. Previous studies of action inhibition focussed on stopping a latent but prepotent voluntary response (Aron & Verbruggen, 2008), or on stopping an ongoing voluntary movement (Pope, Holton, Hassan, Kourtis, & Praamstra, 2007). Action inhibition can involve either global inhibition of all motor output, or selective inhibition of a specific movement (Aron & Verbruggen, 2008). The control mechanisms and subjective experience involved remain poorly understood. Nevertheless, evidence from several neurological conditions, such as Tourette's syndrome, suggests that involuntary movements can, in fact, be voluntarily inhibited (Prado et al., 2008).

Involuntary movements in neurotypical individuals are normally very transient. Reflexes in response to an external perturbation provide one obvious example, and are usually quite brief (<120 ms; Pruszynski et al., 2011). It is not possible to bring these movements under voluntary control *once the stimulus has been delivered*. Therefore, studies of voluntary inhibition need to focus on longer-lasting responses. The Kohnstamm phenomenon offers one example. Here, a strong, sustained isometric contraction of a muscle produces, upon relaxation, a slow, involuntary aftercontraction that is associated with a subjective feeling of lightness and a lack of agency (Adamson & McDonagh, 2004; Craske & Craske, 1985; Forbes, Baird, & Hopkins, 1926; Kohnstamm, 1915; Salmon, 1916).

There is evidence for central (Duclos, Roll, Kavounoudias, & Roll, 2007; Ghosh & Haggard, 2014; Solopova, Selionov, Zhvansky, Gurfinkel, & Ivanenko, 2016) and peripheral (Hagbarth & Nordin, 1998) contributions to the Kohnstamm phenomenon. Afferent input from the periphery can temporarily 'gate' motor output to the muscle (De Havas et al., 2015), while large changes in visual input have been shown to switch motor output from the muscle active during the induction to its antagonist (Ghafouri, Thullier, Gurfinkel, & Lestienne, 1998; Gilhodes, Gurfinkel, & Roll, 1992). Control processes for the Kohnstamm phenomenon may involve multiple regions of the central nervous system. It is therefore convenient to speak of a 'Kohnstamm generator' when considering how a particular aftercontraction responds to input (De Havas et al., 2015; Ghosh, Rothwell, & Haggard, 2014; Moraitis &

Ghosh, 2014). In this context the Kohnstamm generator is a functionally defined unit whose precise location within the central nervous system is not known.

The neural mechanism of the “Kohnstamm generator” remains unclear. The motor drive passes through the primary motor cortex (Duclos et al., 2007; Ghosh et al., 2014; Parkinson, McDonagh, & Vidyasagar, 2009), and reflects adaptation of a postural control system (Duclos, Roll, Kavounoudias, & Roll, 2004; Gurfinkel, Levik, & Lebedev, 1989). Most interestingly, the Kohnstamm aftercontraction can be voluntarily inhibited without the use of the antagonist muscle (Ghosh et al., 2014), apparently by voluntary inhibition of the drive to the agonist. When voluntary inhibition ceases, the arm involuntarily rises again, and a reduced electromyography (EMG) signal is observed (Fessard & Tournay, 1949; Ghosh et al., 2014). This could either reflect simple temporal decay in the Kohnstamm generator due to elapsed time, or a change in the internal state of the generator caused by the inhibition. These experiments involved bringing the arm down. It is not clear what the effects of inhibiting the arm and keeping it stationary might be. One early report could not detect agonist EMG during this form of inhibition (Pereira, 1925), but another found clear agonist EMG activity (Forbes et al., 1926).

How might voluntary inhibition of the Kohnstamm work mechanistically? We outline three possible scenarios (Fig. 1.). First, participants might simply voluntarily contract the antagonist, thus preventing the involuntary drive to the Deltoid from actually moving the arm. Secondly, cognitive control circuits, presumably in the prefrontal cortex, might turn the Kohnstamm generator off, or withdraw some degree of tonic facilitation that is normally present. This form of inhibitory cognitive control remains controversial (Mostofsky & Simmonds, 2008), but the processes of voluntary suppression of emotions (Kühn, Haggard, & Brass, 2014) and of thoughts (Wyland, Kelley, Macrae, Gordon, & Heatherton, 2003) may provide an analogy. Third, voluntary inhibition might merely suppress the expression of motor output from the Kohnstamm generator, by adding an additional inhibitory drive to a motor output node, but without affecting the generator itself. This possibility, which will be termed “negative motor command” (NMC), will be discussed in more detail later. For now we will define it as a putative neural signal which decreases agonist activity without recruiting the antagonist, and which supresses motor output without ‘cancelling’ the Kohnstamm generator itself.

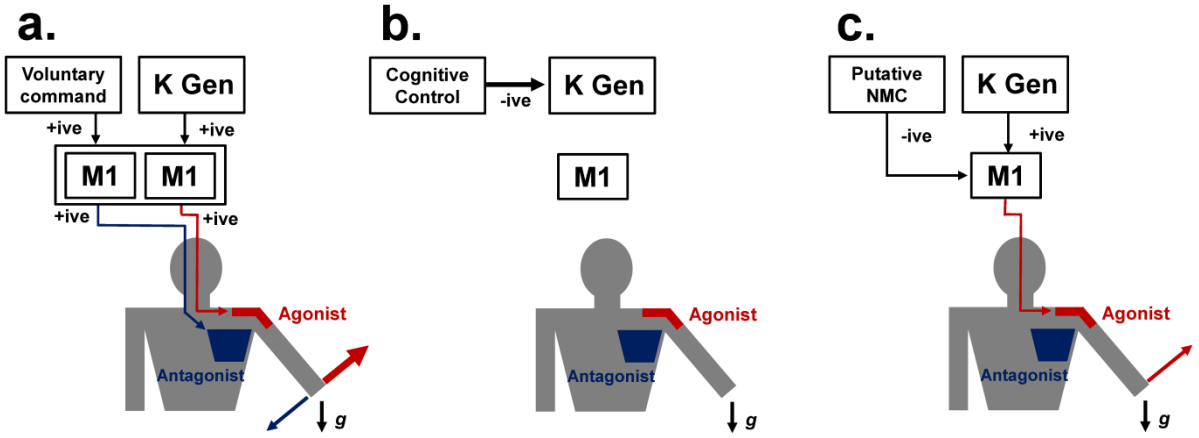

Figure 1. Possible mechanisms for aftercontraction inhibition. *Theoretically the arm could be stopped from moving by activation of the antagonist muscle (a). Motor drive to the muscle could be cut by cognitive control circuits 'switching off' the Kohnstamm generator (b). If this was total the arm would begin to fall due to gravity. Alternatively, inhibitory "negative motor commands" could summate with the excitatory output of the Kohnstamm generator in an output region, such as M1 (c; see discussion for consideration of an alternative locus of integration). With this form of control, the drive to the agonist would be reduced, so as to hold the arm stationary. Interestingly, the Kohnstamm generator itself would remain unaffected.*

Inhibition of Kohnstamm was also associated with a subjective feeling of paradoxical resistance when the arm was voluntarily moved downwards (Ghosh et al., 2014). This curious sensation could be due to a lack of the efference copies that normally accompany voluntary movement. These efference copies are thought to cancel the sensory inflow from the arm (Blakemore & Frith, 2003; Blakemore, Goodbody, & Wolpert, 1998; Blakemore, Wolpert, & Frith, 1998; Frith, Blakemore, & Wolpert, 2000; Shergill, Bays, Frith, & Wolpert, 2003). The aftercontraction has been labelled involuntary because it subjectively feels so (Allen, 1937; Allen & O'Donoghue, 1927; Parkinson & McDonagh, 2006; Rothmann, 1915; Salmon, 1925; Salomonson, 1921; Schwartz & Meyer, 1921). However, it resembles a voluntary movement physiologically (Fessard & Tournay, 1949; Henriques & Lindhard, 1921; Mathis, Gurfinkel, & Struppler, 1996; Pinkhof, 1922).

Previous experiments showed that the involuntarily rising arm could be brought down without contracting antagonist muscle, and that this downward movement was associated with a feeling of resistance. However, the movement of the arm *after* the end of instructed inhibition was not investigated in detail in that study. For example, it was unclear whether, after the instruction to inhibit is ended, the arm continues to rise because of persistent output of an involuntary motor command, and whether this involuntary motor command specifies the same final

position as in no-inhibition trials. Previous studies thus could not decide between four alternative possibilities regarding the effects of voluntary inhibition on the Kohnstamm generator: permanent interruption of the generator, temporary pause in generation, continued generation with a transient disconnection from the motor output pathway, or summation with an additional inhibitory signal so as to cancel the motor outputs driven by the generator. Finally, the specificity of the inhibitory process, and the subjective experience it produces, remain largely unexplored.

2. Methods

2.1. Equipment

Electromyography (EMG) was recorded from bipolar, surface electrodes placed over the middle of the lateral deltoid, parallel to the orientation of the muscle fibres. Data was also collected from the antagonist muscle (pectoralis) in a subgroup of participants. Although not comprehensive, this sample size (n = 4) is fairly typical of the field (Fessard & Tournay, 1949; Kozhina, Person, Popov, Smetanin, & Shlikov, 1996; Marsden, Merton, & Morton, 1976), and could suffice to check whether any major recruitment of the antagonist is involved in voluntary inhibition. An earlier study found that the involuntarily rising arm could be brought down via inhibition without the use of the antagonist muscle (Ghosh et al., 2014). The authors found no evidence of the antagonist muscle countering the agonist to bring about downward movement in any of the nine participants tested. The electrodes were connected to a 1902 amplifier (Cambridge Electronic Design, Cambridge, UK), which was controlled via custom Labview scripts (sample rate = 2000 Hz, gain = 1000, 50 Hz online notch filter). An adjustable doorframe was built using two vertical metal poles, positioned such that each participant could comfortably stand between them and push outwards with both arms 10 degrees abducted. Arm kinematics were recorded via a video camera (30 fps) and LEDs attached to the participant's arm at the shoulder (fixed point) and upper arm (moving point). Participants wore goggles to limit visual input and wrist and elbow splints to ensure their arms stayed straight while the shoulder rotated. Task instructions were signalled using an auditory buzzer (6 V, Maplin, London) controlled by the experimenter. A strain gauge (Mecmesin Advanced Force Gauge, West Sussex, UK) fitted with a flat circular metal disc (diameter = 2 cm) was used to calculate total applied force in the weight estimation task, in which participants matched the force generated by adding 50 g weights to the participant's palm.

2.2. Participants

In total 21 participants (9 female, age: Mean = 23.1, SD = 3.42 yrs, 4 left handed) were recruited for the experiment. However, 7 participants were not included in the final analysis because they either: 1) voluntarily withdraw from the experiment (n=1), 2) did not display an aftercontraction (n = 5), or 3) displayed a small aftercontraction that disappeared after the first trial (n = 1). This left 14 participants (7 female, Mean = 22.21, SD = 2.58 yrs, 2 left handed) whose data was analysed. Experiments were undertaken with the understanding and written consent of each subject in accordance with the Code of Ethics of the World Medical Association (Declaration of Helsinki).

2.3. Procedure

First, a voluntary weight estimation task was administered. Participants were instructed to abduct one of their arms to ~20° of angular displacement. The experimenter then applied a downward force to the forearm using a strain gauge and participants were instructed to counter the force, in order to keep the arm stationary (Fig. 5A). Based on piloting work it was estimated that the average upwards force of a Kohnstamm aftercontraction was ~7 N. Five forces were selected centred on this value (~1, 4, 7, 10, 13 N). The experimenter pushed with one of these force levels. The strain gauge was braced against a rigid surface. A buzzer signalled that participants should remember the amount of upward force they were applying. They were then instructed to hold out the other arm in front of them with the elbow bent and the palm flat, facing upwards. A box was then placed on their hand and weight was slowly added (50 g/s). They were instructed to indicate when the weight became sufficient to have countered the upward force they had been generating when the buzzer sounded. This procedure thus estimated the perceived weight-bearing capacity associated with different degrees of voluntary contraction. For each trial the level of EMG, exact force and perceptual estimates of that force were recorded (see Fig. 5A&C). Trials alternated between arms and the order of forces was randomized.

At the start of each Kohnstamm trial, participants were instructed to stand upright with their palms facing medially and their arms relaxed and by their sides. The first buzzer signalled participants to begin a continuous, unimanual, isometric contraction of the lateral deltoid at ~70% maximal isometric voluntary contraction (MVC). After 30 s the buzzer signalled participants to stop pushing, step forward and

relax. The aftercontraction of the lateral deltoid then caused the arm to abduct. During control trials the arm was allowed to rise unimpeded. In the 'Inhibition' trials an auditory signal was presented when the arm reached ~20° of angular displacement. Participants were instructed to stop the arm from rising any further, but not to bring it down. They were also told to remember the feeling of the arm being stationary. After ~2 s the buzzer was turned off and participants were instructed to allow the arm to rise once more. They were explicitly told not to voluntarily raise their arm, only to 'stop preventing it from rising'. Once the aftercontraction had finished, the experimenter administered a weight estimation task (Fig. 5B). This was identical to the voluntary weight estimation task, with the exception that participants were now asked "when your arm became stationary after the buzzer, how much weight could it have supported?". After every Kohnstamm trial there was a 3 minute rest. Unilateral Kohnstamm trials alternated between the left and right arm (4 unilateral trials; 2 control trials, 2 inhibition trials).

Voluntary unilateral trials followed Kohnstamm unilateral trials. Participants were told to replicate the speed and final arm position of the preceding unilateral Kohnstamm control trials, regardless of the specific Kohnstamm trial that immediately preceded the voluntary movement (Kohnstamm and voluntary trials separately randomised). As before they were told that if the buzzer came on they should stop the arm. However, unlike the Kohnstamm trials they were told that on such trials when the buzzer turned off they should resume the voluntary abduction of the arm. A total of four voluntary trials was performed, two with the buzzer instructing inhibition, and two without, in randomized order.

After the unilateral trials, participants performed bilateral trials, in which both arms simultaneously performed the Kohnstamm induction, and both experienced the involuntary lift. On these trials, a 'target arm' was specified at the start of each trial. If the buzzer sounded during the bilateral aftercontraction, participants were instructed to stop only the target arm, and to do nothing to the other arm. Once again when the buzzer turned off (after ~2 s) they were told to 'stop stopping the target arm'. Participants completed 2 bilateral inhibition trials and 1 bilateral control trial, without inhibition. Voluntary replication trials immediately followed each bilateral trial, as in the unilateral trials. Each participant therefore experienced 5 left arm and 5 right arm aftercontractions during the entire experiment. The number of trials per participant is therefore much lower than most voluntary movement experiments. However, this is

typical of Kohnstamm experiments, because of the need to avoid effects of fatigue (Danielopolu, Radovici, & Carniol, 1921; Parkinson & McDonagh, 2006; Zigler, Martin, Smith, & Stadeker, 1948).

The voluntary weight estimation task administered at the start of the experiment was repeated at the end of the experiment, to control for effects of fatigue. Finally, participants completed a questionnaire about the subjective experience of the task (Table 1). They rated each statement from -3 (strongly disagree) to 3 (strongly agree) on a 7-point Likert scale.

2.4. Analysis

Kinematics analysis was performed by determining the angle between the two body-mounted LEDs over time using IMAGEJ (Schneider, Rasband, & Eliceiri, 2012) and an object tracker (SPOTTRACKER, Switzerland; IMAGEJ plug-in). The *latency* of the movement was defined based on the time from the end of the induction period (or instruction to move on voluntary trials) to the point when the velocity first reached 10% of the maximum velocity for that trial (Irlbacher, Voss, Meyer, & Rothwell, 2006). *Onset* of inhibition was defined as the time from the buzzer coming on to the point when velocity fell below 10% of the max velocity. Likewise *offset* of inhibition was the time from the buzzer turning off to the point when the arm again reached 10% of the max velocity. On bilateral trials 'transient bilateral cessations of movement' were deemed to occur if the non-target arm velocity fell below 10% of the max velocity while the buzzer was on. This 10% criterion has been used in previous research (Irlbacher et al., 2006) and allowed us to make unbiased statistical comparisons across movement types.

EMG was band pass filtered (10-500 Hz) and rectified. On unilateral inhibition trials analysis was time-locked to the onset of the buzzer. Four 250 ms bins were created either side of this inhibition instruction. The mean EMG in each bin across all inhibition trials was then calculated for every participant. Next, using the kinematics data, the angular displacement at inhibition onset was calculated, and its mean was used to identify the corresponding point in control trials, and four similar EMG bins were created before, and four after this point. To determine the progression of EMG, we used linear trends (Howell, 2010) across these four bins with coefficients -3, -1, 1 3 in each condition. A 2x2 within subjects ANOVA with the variables 'time relative to *onset* of inhibition' (before vs. after) and 'presence of inhibition' (inhibition vs. control)

was then performed on the linear trends, in order to investigate how the instruction to inhibit affected EMG. The same analysis was used to determine how EMG changed in the two conditions as a function of the end of the inhibition period. Analysis windows were time-locked to the offset of inhibition. Here, the 2x2 within subjects ANOVA had the variables 'time relative to *offset* of inhibition' (before vs. after) and 'presence of inhibition' (inhibition vs. control).

Bilateral data was analysed in the same manner as unilateral data. However, in this case there were three conditions: control trials, 'inhibition arm' and 'no inhibition arm' (the latter two coming from inhibition trials). Voluntary movements were analysed in the same way as Kohnstamm trials. All bilateral trials were included in the EMG analysis, including trials with transient bilateral cessation of movement.

Antagonist data was filtered and rectified in the same manner as agonist data. ECG artefacts were manually identified and removed by replacing affected EMG time points with data from immediately before each heartbeat. Mean antagonist EMG was calculated before (-1000-0 ms) and after (0-1000 ms) the point of inhibition onset. A 2x2 within subjects ANOVA with the variables 'time relative to inhibition onset' (before vs. after) and 'presence of inhibition' (inhibition vs. control) was then performed. EMG was low-pass (4 Hz) filtered for display purposes.

The experience of aftercontraction was quantified as follows. First, mean deltoid EMG (filtered and rectified) levels and force levels (strain gauge signal) were calculated from the voluntary weight estimation task (Fig. 5A). An analysis window of 500 ms, starting from when the buzzer sounded, was used to quantify the EMG and *force applied* for each of the 10 trials. For each participant *force applied* was plotted against *perceived force* (the amount of weight they estimated would counter their upward voluntary force; Fig. 5C; left scatter plot). Two subjects were excluded because they did not show a significant linear relationship between these variables, indicating that they were not able to perform the task. Next, to quantify if participants were aware of the involuntary aftercontraction during the inhibition period, the amount of weight they thought their arm could support during this period was plotted on the same graphs (Fig. 5B&C). An estimate of the perceptually-equivalent *force applied* was then calculated based on the *perceived force* of these two trials and the individual's perceptual function relating actual to perceived force in the voluntary task (Fig. 5C; left scatter plot). This perceptually equivalent *force applied* during each Kohnstamm trial was then used to calculate the level of EMG that *would* have been

required to achieve those forces, had they been veridical (Fig. 5C; right scatter plot). This was termed the *perceived aftercontraction* (Fig. 5D). The *actual aftercontraction* was calculated from the mean EMG during the Kohnstamm inhibition period (0.5 – 2 s post instruction to inhibit; Fig 5B&D). *Perceived aftercontraction* was compared to *actual aftercontraction* across participants via a paired sample t-test (Fig. 5D).

Each item in the questionnaire was analysed separately, using one sample t-test to determine if there was significant agreement (>0) or disagreement (<0) with each statement across participants.

3. Results

3.1. Voluntary inhibition gates output from Kohnstamm generator to the muscle

When the buzzer instructed the participants to inhibit the aftercontraction, the arm stopped rising (mean response time = 674 ms, SD = 227 ms). Data from the 4 participants in whom the antagonist muscle was measured showed that this was always achieved without antagonist activity (Fig. 2). Mean antagonist EMG was very low, and uniform across conditions and time (control condition, before inhibition onset = 0.0046 mV, SD = 0.001 mV; control condition, after inhibition onset = 0.0048 mV, SD = 0.00056 mV; inhibition condition, before inhibition onset = 0.0041 mV, SD = 0.001 mV; inhibition condition, after inhibition onset = 0.0042 mV, SD = 0.00071 mV). There was no significant main effect of 'presence of inhibition' ($F(1,3) = 0.675$, $p = 0.471$) or 'time relative to inhibition onset' ($F(1,3) = 0.333$, $p = 0.604$) and no significant interaction ($F(1,3) = 0.035$, $p = 0.864$). Due to the small sample size we cannot exclude the possibility that some participants recruited the antagonist muscle. However, previous studies using larger samples found no evidence for antagonist recruitment (Ghosh et al., 2014). Thus, antagonist contraction seems unlikely to account for voluntary inhibition of involuntary movement.

Importantly, the inhibition condition showed a reduced agonist EMG trend relative to the control condition (Fig. 3). This manifested as a significant main effect of 'time relative to inhibition onset' (before vs. after; $F(1,13) = 10.01$, $p = 0.007$) and a significant 'time relative to inhibition onset' x 'presence of inhibition' interaction ($F(1,13) = 15.12$, $p = 0.002$) on the linear EMG trends. There was no main effect of 'presence of inhibition' ($F(1,13) = 2.36$, $p = 0.15$). Simple effects paired t-tests showed no significant difference between the conditions before inhibition ($t(13) = 0.17$, $p = 0.87$), but after inhibition the linear trend was lower in the inhibition than in

the control condition ($t(13) = 2.6$, $p = 0.022$). We also compared EMG trends before and after the inhibition onset within each condition: there was a significant change in the inhibition condition when comparing before to after ($t(13) = 4.7$, $p = 0.0004$, but not in the control condition: ($t(13) = 0.49$, $p = 0.63$).

When the inhibition instruction was removed, the arm began to rise again (mean response time = 496 ms, SD = 240 ms) with a resumption of the previous pattern of EMG increase. This is shown by a significant interaction between 'time relative to inhibition offset' (before inhibition offset vs. after inhibition offset) and 'presence of inhibition' ($F(1,13) = 4.76$, $p = 0.048$) in the linear EMG trends. There was no main effect of 'time relative to inhibition offset' ($F(1,13) = 0.015$, $p = 0.9$) or 'presence of inhibition' ($F(1,13) = 1.51$, $p = 0.24$). Simple effects t-tests showed no significant difference between the conditions before inhibition offset ($t(13) = 1.83$, $p = 0.09$) and no significant difference between the conditions after inhibition offset ($t(13) = 1.2$, $p = 0.25$). Further, the control condition did not change from before to after the inhibition offset ($t(13) = 1.2$, $p = 0.25$). These null results may reflect variability in Kohnstamm speed across participants: in some the arm was still rising at the time of inhibition instruction, while in others it had already reached its maximum angular displacement. Importantly, however, there was a significant difference between these two time points in the inhibition condition ($t(13) = 4.02$, $p = 0.001$), showing that the removal of inhibition caused the linear trend of the EMG to increase.

In kinematic recordings, there was a trend towards *offset* response time being faster than *onset* response time (Mean = 496, SD = 240 vs. Mean = 674, SD = 227 ms; $t(13) = 2.16$, $p = 0.05$; Bonferroni corrected $\alpha = 0.017$). Interestingly, *offset* response time was faster than the latency for movement onset at the start of the Kohnstamm response time (Mean = 496, SD = 240 vs. Mean = 3082, SD = 1211 ms; $t(13) = 8.04$, $p < 0.001$; Bonferroni corrected $\alpha = 0.017$). This shows that there was not a 'second latent period'. Instead it seems the Kohnstamm generator remained active during inhibition and was not 'reset' back to its starting level. Final arm angle did not differ significantly between the control and inhibition condition, both for unilateral (Mean = 50.12°, SD = 23.43° vs. Mean = 44.03°, SD = 19.90°; $t(13) = 1.83$, $p = 0.09$) and bilateral (Mean = 44.37°, SD = 22.93° vs. Mean = 41.61°, SD = 19.82°; $t(13) = 1.62$, $p = 0.13$) Kohnstamm movements. Final arm angle is known to depend on the activity level of the Kohnstamm generator, notably because it varies with the duration and force of the induction period (Allen, 1937; Allen & O'Donoghue, 1927;

Brice & McDonagh, 2001; Fessard & Tournay, 1949; Matthaei, 1924). Therefore, the consistency of final arm position despite inhibition suggests that voluntary inhibitory commands did not alter the activity level of the Kohnstamm generator itself.

To assess whether sensory function was altered in the Kohnstamm condition, we asked participants to voluntarily replicate their final arm position after the end of each Kohnstamm control trial. These tests were perfomed in the absence of visual information, in order to test whether position sense is affected during aftercontractions. The results showed no significant difference in position sense between Kohnstramm and voluntary trials. Since these analyses are distinct from the main focus of this paper on inhibition, full details are shown in supplementary materials.

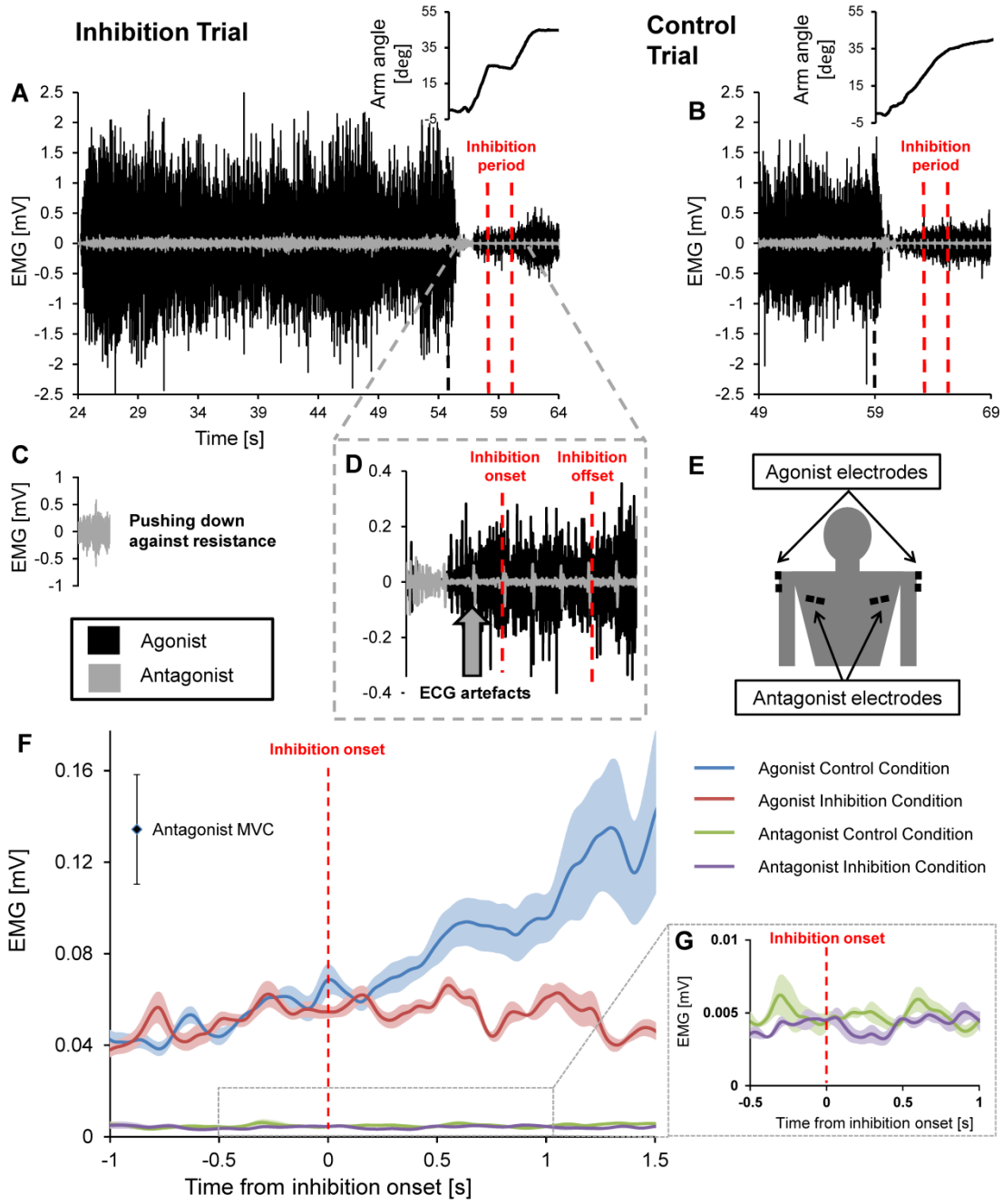

Figure 2. ***The effect of inhibiting a unilateral Kohnstamm aftercontraction****. Agonist and antagonist EMG and kinematics from a single representative participant during a right arm unilateral inhibition (A) and control (B) trial. Note that antagonist activity was always much lower across both trials than during a comparison condition where the participant was instructed to adduct (C). (D). Instructions to briefly voluntarily inhibit the aftercontraction produced a plateau in the normal rising agonist EMG profile, followed by resumed increase after participants were instructed to cease inhibiting. Note that antagonist EMG remained low and constant throughout inhibition. (E) Schematic showing electrode placement. Lower panel shows mean rectified and smoothed agonist and antagonist EMG during inhibition of unilateral Kohnstamm aftercontraction (F). Data from four participants are shown. For the deltoid muscle (agonist) there was an increase in EMG as the arm rose. At the point of inhibition the EMG began to diverge in the two conditions. However, after removal of ECG artefacts, pectoralis (antagonist) EMG was flat and low relative to MVC. Note that antagonist activity was slightly lower in the inhibition condition than the control condition (G). If the antagonist muscle had been used to stop the movement, the reverse should have been the case. Error bars show SEM.*

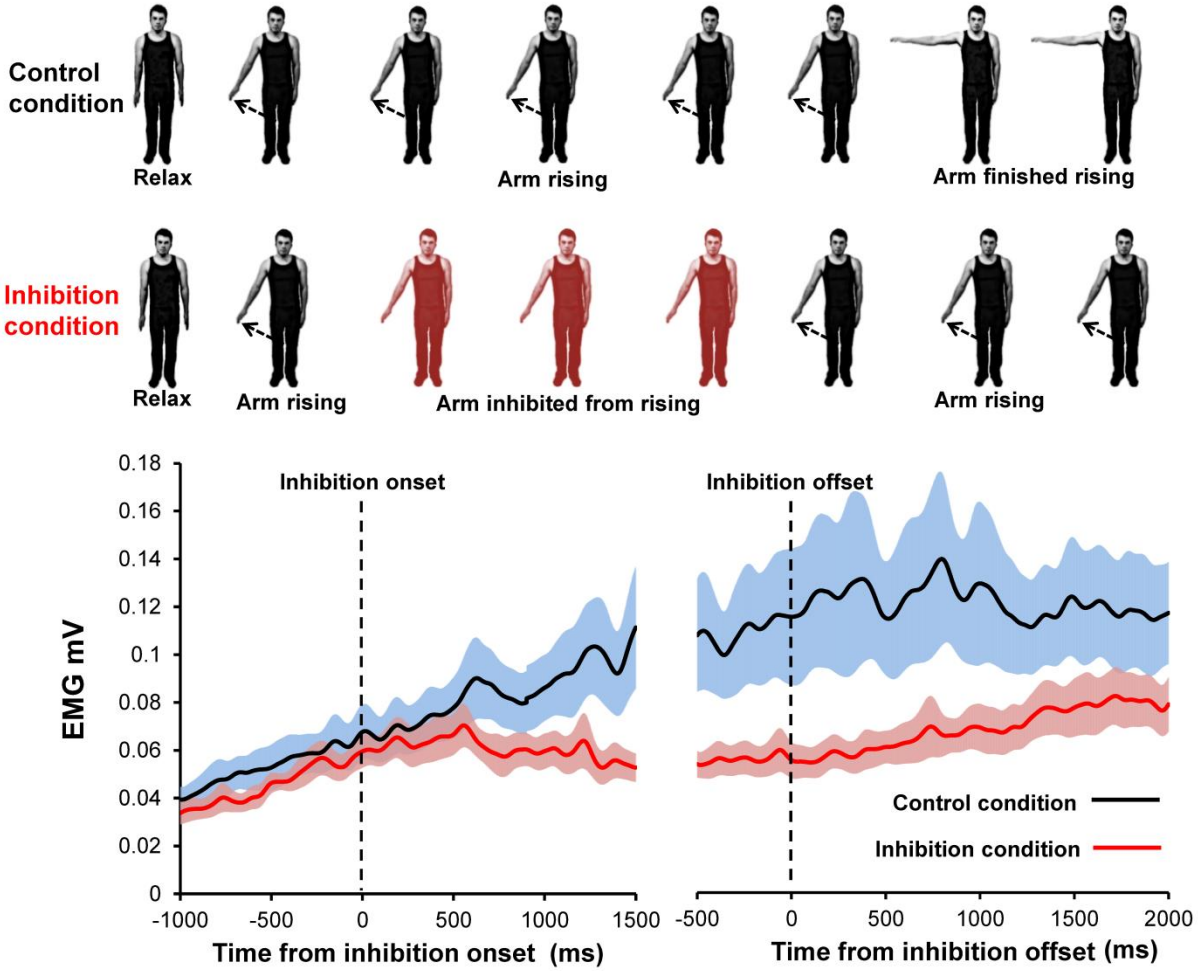

Figure 3. ***The effect of inhibiting and releasing inhibition of a unilateral Kohnstamm aftercontraction on rectified, smoothed deltoid EMG across participants****. Dashed lines show the time of the onset of the inhibition instruction and offset of inhibition instruction. Error bars show SEM.*

3.2. Separate Kohnstamm generators in each hemisphere not affected by voluntary inhibitory command

During bilateral Kohnstamm movements, voluntarily stopping one arm did not affect the EMG signal in the other arm (Fig. 4). A significant interaction ($F(1,13) = 7.83$, $p = 0.015$) was found between Arm (inhibition arm vs. no inhibition arm) and 'time relative to inhibition onset' (before vs. after). There was also a main effect of 'time relative to inhibition onset' ($F(1,13) = 7.72$, $p = 0.016$), but no main effect of Arm ($F(1,13) = 1.18$, $p = 0.3$). Simple effects paired t-tests showed no significant difference between the arms before inhibition onset ($t(13) = 1.99$, $p = 0.07$) and the EMG trend for the 'no inhibition arm' did not change from before to after inhibition onset ($t(13) = 0.38$, $p = 0.71$). The difference between the arms after inhibition onset

was significant (t(13) = 2.44, p = 0.03). Importantly, a significant difference in the inhibition arm when comparing before to after was found (t(13) = 3.41, p = 0.005). As a further test of whether the 'no inhibition arm' EMG was affected by the voluntary inhibition command, this data was compared to a bilateral control condition. No main effect of 'presence of inhibition' (F(1,13) = 0.63, p = 0.44) or 'time relative to inhibition onset' (F(1,13) = 0.46, p = 0.51) was found and the interaction was also not significant (F(1,13) = 0.05, p = 0.83).

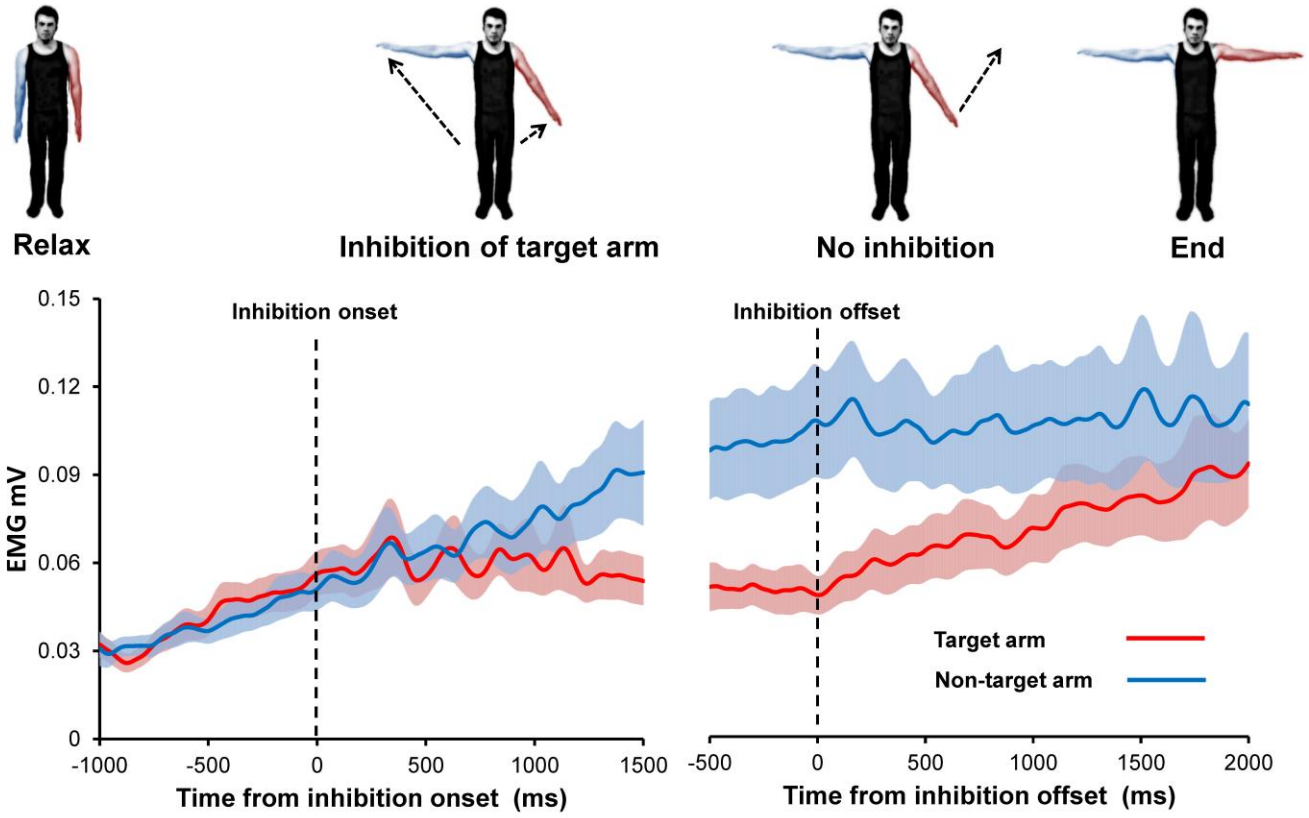

Figure 4. ***The effect of inhibiting, and releasing inhibition, of a single 'target' arm during bilateral Kohnstamm aftercontraction on rectified, smoothed deltoid EMG.*** *Dashed lines show time of inhibition onset and offset. Note the continued increase in EMG for the non-target arm, together with plateauing EMG in the target arm, beginning approximately 500 ms after the instruction to inhibit. Error bars show SEM.*

At the offset of voluntary inhibition, EMG began to rise again, as in unilateral conditions. There was no main effect of 'time relative to inhibition offset' ($F(1,13) = 0.68$, $p = 0.43$) or Arm ($F(1,13) = 0.09$, $p = 0.77$), but a significant 'time relative to inhibition offset' x Arm interaction ($F(1,13) = 23.49$, $p = 0.0003$). Simple effects t-tests showed the inhibition arm had a significant *increase* in the linear trend of the EMG from before offset to after offset of inhibition ($t(13) = 3.12$, $p = 0.008$). There was a significant *decrease* in the EMG linear trend of the 'no inhibition arm' between before and after inhibition offset ($t(13) = -4.62$, $p = 0.0005$). The linear trend of EMG was lower in the 'no inhibition arm' than the 'inhibition arm' after inhibition offset ($t(13) = -2.18$, $p = 0.048$), due to EMG naturally levelling off as the arm reached its maximum position in the 'no inhibition arm'. Before inhibition offset the two arms showed a trend towards being significantly different ($t(13) = 2.12$, $p = 0.054$).

3.3. Stopping both arms: Voluntary inhibitory commands have broader focus than modulations of existing motor commands

The combination of bilateral Kohnstamm and unilateral voluntary inhibition allowed us to probe the nature of the voluntary inhibitory command. Mean response times for the onset of inhibition were similar between unilateral and bilateral Kohnstamm movements (Mean = 674, SD = 227 vs. Mean = 721, SD = 320 ms; $t(13) = 0.59$, $p = 0.59$; Bonferroni corrected $\alpha = 0.025$). There was no significant difference between unilateral and bilateral Kohnstamm response times to the offset of inhibition either (Mean = 496, SD = 240 vs. Mean = 541, SD = 627 ms: $t(13) = 0.25$, $p = 0.81$; Bonferroni corrected $\alpha = 0.017$). There was also no significant difference in onset of inhibition response times between bilateral Kohnstamm and matched voluntary movements (Mean = 721, SD = 320 vs. M = 672, SD = 239 ms; $t(13) = 0.63$, $p = 0.54$; Bonferroni corrected $\alpha = 0.025$). The maximum angular displacement of the arm did not differ between Kohnstamm and Voluntary control trials (Mean = 44.37°, SD = 22.93° vs. Mean = 48.37°, SD = 20.38°: $t(13) = 1.33$, $p = 0.21$). Additionally, on inhibition trials the angle of the arm at inhibition did not differ between Kohnstamm and Voluntary movements (Mean = 18.94°, SD = 7.69° vs. Mean = 18.92°, SD = 8.36°: $t(13) = 0.1$, $p = 0.99$). However, the proportion of trials that featured a '*transient bilateral cessation of movement'* (i.e. trials in which the non-target arm also stopped moving at the inhibition instruction) was significantly higher in bilateral Kohnstamm than bilateral voluntary movements (0.5 vs. 0.18; $\chi^2(1, N = 56) = 6.45$, p

= 0.011). The proportion of participants that showed at least one 'transient bilateral cessation of movement' was also significantly higher in bilateral Kohnstamm than bilateral voluntary movements (0.79 vs. 0.29; $\chi^2(1, N = 28) = 7.04$, $p = 0.008$). These analyses suggest that the voluntary inhibition of the aftercontraction was initially directed to the non-target arm as well as the target arm. For the 11 participants who had 'transient bilateral cessations of movement' during Kohnstamm trials, the mean response times to inhibition onset for the non-target arm did not differ significantly from the response times of stopping the target arm (Mean = 689, SD = 429 vs. Mean = 761, SD = 353 ms; t(10) = 0.42, p = 0.68). Finally, 'transient bilateral cessations of movement' were brief, with mean duration of 511 ms (SD = 221 ms), before the kinematics showed resumed movement of the non-target arm (Fig. 6), perhaps explaining why they did not cause any change in the EMG trend for the non-inhibited arm overall.

3.4. Involuntary aftercontraction is overestimated

Participants could perceive the aftercontraction caused by the Kohnstamm generator. The involuntary aftercontraction was perceived as being able to support an external load of 3.02 N (SD 0.66) during the inhibition period (Fig. 5B & C). For the participants who successfully performed the weight estimation task (n = 12; Fig. 5A & C), we found that for a voluntary contraction to be perceived to support a similar external load, the voluntary contraction would in fact need to generate a force of 8.61 N (SD 6.55). This suggests that the perceived force generated by Kohnstamm aftercontractions was equivalent to a perceived force generated by a much higher EMG (mean actual aftercontraction = 0.0528 mV, SD = 0.0232 mV; mean voluntary EMG level perceptually equivalent to this aftercontraction = 0.0972 mV, SD = 0.0465 mV; t(11) = 4.20, p = 0.0015). That is, participants appeared to experience the aftercontraction as almost twice as strong as a voluntary contraction with an equivalent EMG level (Fig. 5D).

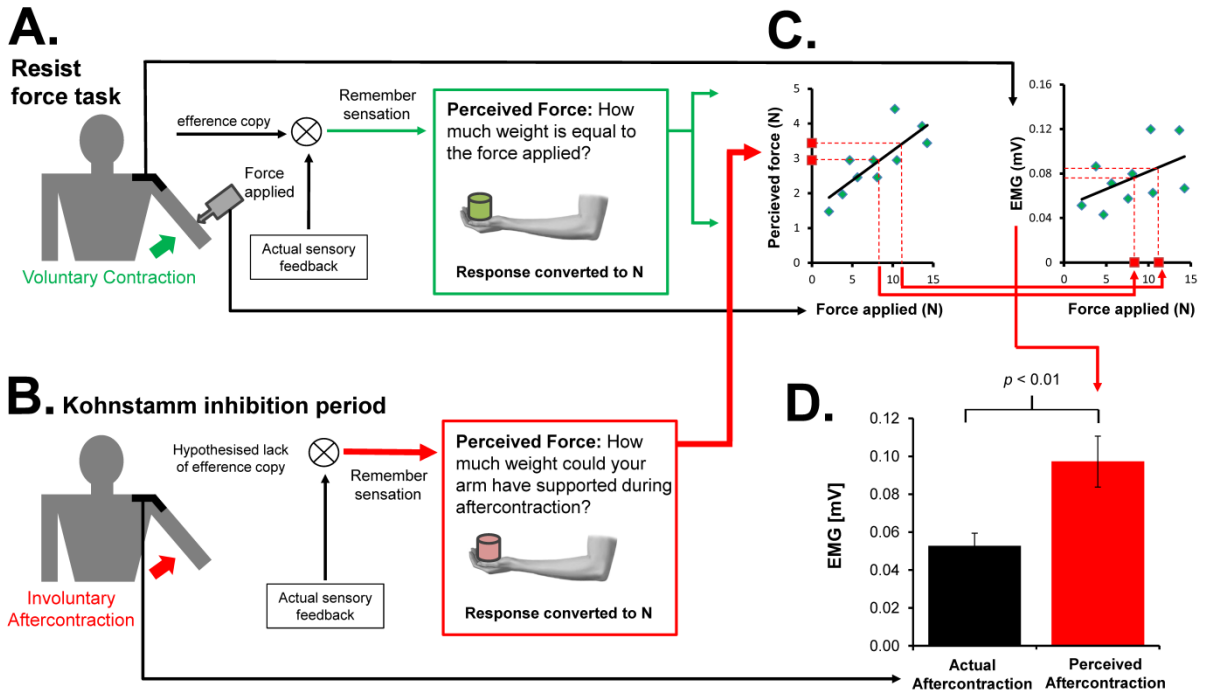

Figure 5. ***Subjective awareness of the involuntary aftercontraction during voluntary inhibition***. *The methods for estimating perceived force are shown for Voluntary trials (A), and Kohnstamm trials (B), along with the hypothesised difference in sensorimotor attenuation. Results from one illustrative participant (C), judging the weight that their arm could support during inhibition phases from two Kohnstamm trials (red squares). The data is plotted together with the relation between perceived and actual force from voluntary trials (green diamonds). Interpolating this relation allowed us to estimate the equivalent Kohnstamm forces that would be required to generate percepts similar to those on voluntary trials. The level of voluntary EMG required to generate the equivalent Kohnstamm force was calculated, using the relation between EMG and actual force for voluntary trials. The subject reported that during inhibition their arm could resist 2.94 and 3.43 N of downward force. For a voluntary contraction to be perceived as resisting the same downward force, it would need to generate 8.35 and 11.22 N of upward force respectively. (C left panel). Such forces would require 0.08 and 0.09 mV of EMG activity if they had been voluntary (C right panel). The actual aftercontraction EMG during these trials was 0.04 and 0.06 mV. Thus this subject was representative of the group. Repeating this procedure across participants allowed us to calculate a perceptually equivalent involuntary aftercontraction during inhibition, based on judgements of weight-supporting capacity. This was significantly greater than the actual involuntary aftercontraction (mean EMG) during the inhibition period (D).*

3.5. Questionnaire data supports subjective and physiological findings

The questionnaire data are shown in Table 1. Participants' experience of the Kohnstamm phenomenon agreed with previous reports. Briefly, the aftercontraction was experienced as involuntary (Q04, 08, 24), automatic (Q01), lacking agency (Q09, 12, 13, 17) and associated with feelings of lightness in the arm (Q02, 05, 14, 22). Interestingly, inhibition of the aftercontraction was accompanied by a feeling that involuntary aftercontraction had to be continuously opposed (Q33, 38) and was accompanied by an urge to allow the arm to move again (Q37).

Table 1. The subjective experience of the Kohnstamm phenomenon *(section 1), inhibition of unilateral Kohnstamm aftercontractions (section 2), and bilateral Kohnstamm aftercontractions (section 3). Participants rated each statement from -3 (strongly disagree) to 3 (strongly agree) on a 7-point Likert scale.*

Question	Mean rating	SD of rating	t-value	p-value
01) The movement seemed to begin automatically	2.64	0.63	15.61	< 0.001
02) My arm seemed lighter than normal	2.36	0.74	11.84	< 0.001
03) I found the experience of my arm moving interesting	2.36	0.74	11.84	< 0.001
04) I had to will my arm to begin the movement	-2.29	0.73	-11.78	< 0.001
05) It seemed like gravity was not acting on my arm	1.79	0.89	7.49	< 0.001
06) I found the experience of my arm moving boring.	-2.21	1.12	-7.39	< 0.001
07) The rest of my body felt normal during the movement	1.93	1.07	6.73	< 0.001
08) It seemed the movement was involuntary	2.14	1.23	6.51	< 0.001
09) It seemed like my arm was being buoyed up by water	1.71	0.99	6.45	< 0.001
10) I found the experience of my arm moving pleasant	1.71	0.99	6.45	< 0.001
11) I had the sensation of pins and needles in my arm	-2.00	1.41	-5.29	< 0.001
12) It seemed like a cushion of air was lifting my arm	1.43	1.02	5.26	< 0.001
13) It seemed like my arm was being pulled upwards by a rope	1.36	1.08	4.69	< 0.001
14) My arm seemed heavier than normal	-1.86	1.51	-4.60	< 0.001
15) I felt a greater sense of freedom during this movement than normal movements	1.50	1.22	4.58	< 0.001
16) I found the experience of my arm moving frightening	-2.00	1.66	-4.50	< 0.001
17) It seemed like my arm was being lifted by a helium balloon	1.36	1.15	4.41	< 0.001
18) The experience seemed dreamlike	1.36	1.15	4.41	< 0.001
19) I experienced a sense of relief when my arm started to move	1.29	1.20	3.99	0.002
20) The movement seemed smoother than normal movements	1.43	1.55	3.44	0.004
22) It seemed like my arm was full of helium	1.07	1.49	2.69	0.019
23) I felt like I could control the speed of my arm	-1.00	1.47	-2.55	0.024
24) It seemed like I was in control of the moving arm	-1.00	1.52	-2.46	0.029
25) I knew where my arm was during the movement	1.07	1.64	2.45	0.029
26) As my arm began to move I had the sensation that it would not stop	0.79	1.42	2.06	0.059
27) It seemed like my arm was out of my control	0.79	1.53	1.92	0.077
28) I found the experience of my arm moving strange	0.93	1.82	1.91	0.078
29) It seemed like the moving arm did not belong to me	-0.43	1.74	-0.92	0.374
30) It seemed the experience of my arm was less vivid than normal	0.21	1.42	0.56	0.583
31) I had the sensation that my arm was numb	0.07	1.27	0.21	0.836
32) It seemed like I couldn't really tell where my arm was in space	0.00	1.71	0.00	1.000
33) I had to keep telling my arm to stay still	1.71	0.83	7.77	< 0.001
34) It seemed like my arm was pulled upwards and I was pulling against that force	1.57	1.34	4.38	< 0.001
35) When I stopped my arm I felt like upward drive was put on hold	1.43	1.22	4.37	< 0.001
36) It was a relief when my arm stopped moving	-1.29	1.14	-4.22	< 0.001
37) When my arm was stationary I had an urge to allow it to move again	1.64	1.55	3.97	0.002
38) I only had to tell my arm to stop once and then it did not move	-1.14	1.23	-3.47	0.004
39) When stationary, it seemed like my arm was resting on a cushion of air	-0.71	1.33	-2.02	0.065
40) It was difficult to maintain my arm in a stationary position	0.57	1.74	1.23	0.241
41) When I stopped my arm I felt like upward drive ended	0.36	1.55	0.86	0.404
42) When stationary, it seemed like my arm was resting on water	-0.21	1.25	-0.64	0.533
43) I found it easy to make my arm stop moving	0.07	1.38	0.19	0.850
44) When stationary, it seemed like my arm was resting on a solid object	0.00	1.41	0.00	1.000
45) It was easy to stop one hand without affecting the other	-0.93	1.64	-2.12	0.054
46) When I stopped one hand the other hand also briefly stopped	0.50	1.65	1.13	0.278
47) This task was easier than doing the same task with voluntary movement	0.43	1.65	0.97	0.349

4. Discussion

A prolonged voluntary contraction of the shoulder abductors produced the sustained involuntary aftercontraction known as the Kohnstamm phenomenon. Interestingly, although the aftercontraction was involuntary, participants could voluntarily counter it, leading the arm to hang stationary in mid-air, with a plateau in deltoid EMG. We showed for the first time that when participants stopped inhibiting, EMG resumed its previous pattern of increase. Further, the time taken for the arm to resume moving was significantly faster than the time it took the involuntary movement to first begin after relaxation, and final position of the inhibited arm did not differ from the control condition. Participants were aware of the involuntary aftercontraction, but overestimated its strength. During bilateral aftercontractions, inhibiting one arm did not have an effect on the slope of the EMG recorded from the other arm. However, these commands were associated with brief cessations of movement in *both* arms on some trials. There were significantly more transient bilateral cessations of movement during Kohnstamm than during matched voluntary movements.

The notion that the Kohnstamm phenomenon can be voluntarily inhibited is hinted at in older literature (Fessard & Tournay, 1949; Forbes et al., 1926; Pereira, 1925), but was not systematically measured. Theoretically, inhibition could be achieved by voluntarily contracting the antagonist, or by a cognitive control signal supressing the Kohnstamm generator, or by some form of negative motor command (Fig. 1.). We found no evidence of antagonist involvement in inhibition, in line with previous reports (Forbes et al., 1926; Ghosh et al., 2014). We also found that at the offset of inhibition the arm began again to rise involuntarily. This suggests that voluntary inhibition does not involve a cognitive control signal simply shutting down the Kohnstamm generator. A similar finding has been previously reported in experiments where inhibition caused adduction followed by additional aftercontractions (Fessard & Tournay, 1949; Ghosh et al., 2014).

Therefore, we may postulate a novel neural signal, the “negative motor command” to explain the data (Fig. 1C). Several cortical areas have been reported to cause slowing and cessation of movement when directly stimulated (Brown & Sherrington, 1912; Filevich, Kühn, & Haggard, 2012b). The negative motor command could be implemented by a putative area for voluntary control that makes synaptic contacts on to the same motor output neurons that the Kohnstamm

generator excites. An M1 location for this integration of excitatory and inhibitory signals is consistent with the finding that the Kohnstamm generator outputs via the primary motor cortex (Ghosh et al., 2014).

Alternatively, integration may occur at the spinal cord. Inhibitory control could be mediated by spinal interneurons, which receive signals from both sensory afferents and descending motor commands (Rossignol, Dubuc, & Gossard, 2006; Sherrington, 1913). If the aftercontraction is strongly driven by afferent signals, as has been suggested (Hagbarth & Nordin, 1998; Parkinson & McDonagh, 2006), then voluntary inhibition could, in principle, be achieved by interneuron-mediated gating of this afferent drive (Nielsen, 2004; Rudomin, 1999). However, a purely spinal account of the Kohnstamm phenomenon is difficult to reconcile with numerous lines of evidence pointing to a cortical origin (Duclos, Roll, Kavounoudias, & Roll, 2007; Ghosh, Rothwell, & Haggard, 2014; Mathis, Gurfinkel, & Struppler, 1996; Parkinson, McDonagh, & Vidyasagar, 2009; Sapirstein, Herman, & Wallace, 1936; Sapirstein, Herman, & Wechsler, 1938). Integration could also occur in the hindbrain. Work with cats has found distinct excitatory and inhibitory regions in the brainstem which modulate tonic postural drive (Takakusaki, 2008). In particular, a muscle tone inhibitory region in the pedunculopontine nucleus has been identified (Takakusaki, Habaguchi, et al., 2003; Takakusaki et al., 2004), which receives projections from basal ganglia and motor cortex (Matsumura et al., 2000), and sends projections which suppress postural muscle tone, via either direct postsynaptic inhibitory effects on motoneurons or via activation of inhibitory interneurons (Chase & Morales, 1990; Takakusaki, Kohyama, et al., 2003; Takakusaki et al., 2001, 1994). However, it remains to be seen how this system works in humans and whether it can exert the kind of precise inhibition observed in our experiments.

The inhibitory signal itself may originate from basal ganglia (Majid, Cai, Corey-Bloom, & Aron, 2013), since it can exert strong inhibitory effects on the cortex and postural regions of the brainstem (Takakusaki, 2008). Interestingly, aftercontractions have been found to be of abnormally long duration in patients with Parkinson's disease (Laignel-Lavastine, Chevalier, & Vie, 1927; Salmon, 1915, 1916, 1925, 1929; Sapirstein, Herman, & Wechsler, 1938), perhaps reflecting an impaired ability to end the involuntary movement via inhibition.

The concept of "negative motor command" is a relatively novel one. It does not figure in the ontology of classical motor control, even though cortical neurons

with inhibitory effects on muscle activity are well-known (Kraskov, Dancause, Quallo, Shepherd, & Lemon, 2009). Our study justifies this concept, and reveals several important new features of negative motor commands. Since the arm could be maintained without vision in a stable position against the involuntary aftercontraction, negative commands can apparently be proportional, so as to just balance the involuntary Kohnstamm agonist drive, and can produce a desired target position. This suggests they integrate closely with proprioception in a manner similar to positive motor commands. Secondly, we showed that negative motor commands do not directly affect the Kohnstamm generator. The maximum arm angle resulting from an aftercontraction depends on the activity level of the Kohnstamm generator (Brice & McDonagh, 2001; Fessard & Tournay, 1949; Matthaei, 1924; Sapirstein, Herman, & Wallace, 1937). We found that the maximum arm angle did not differ between inhibition and control conditions. If putative negative motor commands acted on the generator itself, one would expect to see a lower final arm angle in the inhibition conditions, yet this was not found. Furthermore, after the offset of inhibition the amount of time taken for the arm to begin to rise was much lower than for the onset of the initial involuntary movement (latent period). If putative negative motor commands acted on the generator itself, one would expect to see a "second latent period" associated with the generator's restarting, yet this was not found. These findings extend those of Ghosh et al. (2014). That study had shown that the arm could be brought down without the antagonist muscle, and could thereafter rise again involuntarily. However, those results were agnostic regarding the mechanism of inhibition. In particular, previous results could not clarify whether the inhibition acted on the generator itself, or merely on an output relay driven by the generator.

Functional imaging, TMS and early drug and patient studies indicate a cortical location for the Kohnstamm generator (Duclos et al., 2007; Ghosh et al., 2014; Sapirstein, Herman, & Wallace, 1936; Sapirstein et al., 1938). However, there is also evidence for a peripheral component (Hagbarth & Nordin, 1998). We found that during bilateral Kohnstamm, inhibition of one arm did not affect the EMG signal in the other arm. This suggests that there are separate Kohnstamm generators for each arm, potentially located in each contralateral hemisphere, and is consistent with earlier reports (De Havas et al., 2015; but see Brun et al., 2015; Brun & Guerraz, 2015 for evidence of interlimb coupling).

Our use of bilateral Kohnstamm and matched voluntary movements allowed us to compare inhibition across these two conditions for the first time. We found that performance of the two tasks was comparable in all regards except one: there were significantly more transient bilateral cessations of movement in the Kohnstamm condition. For voluntary movement, stopping a prepotent response produces both a rapid global inhibitory effect, followed by a slower, selective inhibition of specific actions. The two processes can be behaviourally dissociated (Aron and Verbruggen 2008). However, even in tasks where selective inhibition is required, there can be global slowing of responses (Coxon, Stinear, & Byblow, 2007; but see Xu, Westrick, & Ivry, 2015 for negation with minimal training), which may be caused by a transient suppression of corticomotor excitability (MacDonald, Coxon, Stinear, & Byblow, 2014; Majid, Cai, George, Verbruggen, & Aron, 2012).. Separate hyperdirect and indirect pathways from the inferior frontal gyrus to the motor output circuits may control rapid, global inhibition and slower, selective inhibition respectively (Aron & Poldrack, 2006). Our tasks would favour engagement of the slower, selective system, because participants knew in advance that they should only stop one arm, and accuracy rather than speed was emphasised. Indeed, we observed few 'transient bilateral cessations of movement' in the voluntary movement task. However, we observed numerous 'transient bilateral cessations of movement' in the Kohnstamm condition, suggesting a different control mechanism.

Transient bilateral cessation of movement when inhibiting the bilateral aftercontraction indicates that the targeting of putative negative motor commands was initially relatively imprecise, but was then refined (Fig. 6.). This again suggests sensory feedback to negative motor commands: the second, selective stage of inhibition might be implemented by monitoring the effects of the earlier, broader inhibition. Our results demonstrate that the Kohnstamm phenomenon can be used to understand action inhibition mechanisms. In studies involving inhibition of voluntary movement, it is difficult to distinguish between inhibiting an action, and not making the action in the first place (Filevich, Kühn, & Haggard, 2012a). The Kohnstamm phenomenon does not suffer from this limitation.

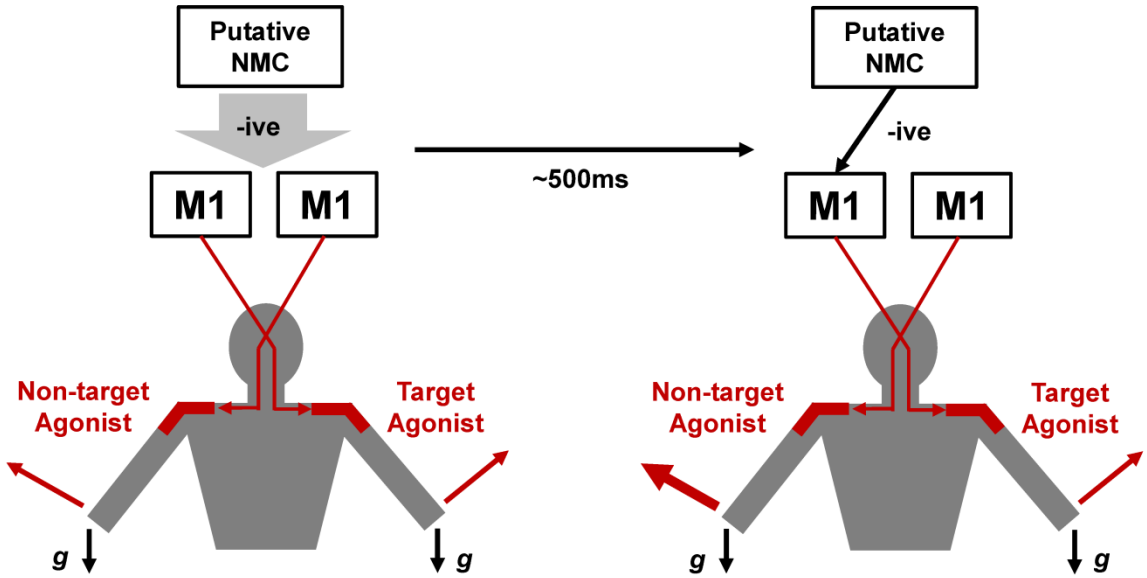

Figure 6. A schematic showing dynamics of putative negative motor commands during bilateral aftercontractions*. Our results suggest that putative negative motor commands have an initially broad focus (left), but are quickly refined to focus on one target effector (right). This progressive focussing explains why both arms sometimes stopped moving, but within ~500ms only the target arm remained stationary (transient bilateral cessation of movement).*

Participants were aware of the aftercontraction, even when the arm was stationary during voluntary inhibition. This suggests that the experience of the aftercontraction was not simply reconstructed from the fact of the arm's movement. Rather, during voluntary inhibition participants reported a sensation like an urge to allow the arm to move. These reports are reminiscent of the urge felt during voluntary tic suppression in people with Tourette's syndrome. The need to tic is described as a build-up of tension, pressure, or energy (Bliss, 1980; Prado et al., 2008). A widespread frontal network seems to be involved in controlling the occurrence of tics (Roessner et al., 2012). Moreover, voluntary tic suppression appears to be independent of the tic generation process, since it does not lead to a subsequent increase in the generation of tics (Specht et al., 2013). The Kohnstamm generator and tic generator clearly differ in several ways. However, we suggest the mechanisms for exerting voluntary control over these involuntary generators could overlap.

We also found implicit evidence regarding the experience of involuntary movements. Participants reported that the 'floating', stationary arm could support surprisingly high weights. This agrees with reports of a sensation of resistance as participants adducted voluntarily against the aftercontraction (Ghosh et al., 2014) and reports that aftercontraction forces are overestimated (De Havas et al., 2015;

Matthaei, 1924). We used a quantitative method to assess experience of the aftercontraction based on weight-perception. Like previous qualitative studies, we also found that the aftercontraction was perceptually overestimated relative to equivalent voluntary contraction. This is consistent with the Kohnstamm generator not producing efference copies of the involuntary movement. Motor control models suggest that, in the absence of an efference copy, nothing can be cancelled against the sensory inflow. The augmented inflow leads to higher ratings of force relative to voluntary movements (Blakemore & Frith, 2003; Blakemore, Goodbody, et al., 1998; Shergill et al., 2003). The primary motor cortex has been identified as a key site in the Kohnstamm circuit (Ghosh et al., 2014). Motor efference copies relevant to perception are thought to be produced higher in the motor hierarchy than M1 (Chronicle & Glover, 2003; Voss, Bays, Rothwell, & Wolpert, 2007). Interestingly, the supplementary motor areas are not active during Kohnstamm aftercontraction (Duclos et al., 2007), yet may play a role in efference copy awareness (Fried et al., 1991; Haggard, 2011). A lack of efference copies might therefore underlie the strange sensation of non-agency during aftercontraction, and feelings of limb lightness (Craske & Craske, 1985; Cratty & Duffy, 1969; Gurfinkel et al., 1989; Hagbarth & Nordin, 1998; Kohnstamm, 1915).

We focussed on interactions between the involuntary aftercontraction and voluntary functions. One view treats the Kohnstamm as an adaptation of a system for maintaining body posture (Duclos et al., 2004; Gurfinkel et al., 1989). The aftercontraction can thus be viewed as amplification into the perceptible range of a normally sub-aware postural control system. Postural control normally proceeds automatically, but can seamlessly be brought under voluntary control, which can then be relinquished once a new posture is adopted. The first state may be experienced as a relatively effortless, agency-neutral default, while the second is a more effortful, precise, high-agency state. The concept of alternation between default and more attentive states is familiar throughout cognition (Baird, Smallwood, Lutz, & Schooler, 2014; De Havas, Parimal, Soon, & Chee, 2012; Feurra et al., 2013; Fox et al., 2005; Kahneman, 2012), and underlies recent models of neuromotor circuits for voluntary action (Jun, Longtin, & Maler, 2014; Murakami, Vicente, Costa, & Mainen, 2014). Such models posit switching between these alternative states. We have shown that an involuntary movement can be voluntarily inhibited via putative negative motor commands. In this case, a more voluntary motor system does not

alternate and time-share with a less voluntary system, and does not suspend the operation of the less voluntary system. Rather, the voluntary system adds a transient overriding input, which prevents the normal expression of its output. Future research should investigate whether this model could also apply to other forms of inhibition.

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References

Adamson, G., & McDonagh, M. (2004). Human involuntary postural aftercontractions are strongly modulated by limb position. *European Journal of Applied Physiology*, *92*(3), 343–351. http://doi.org/10.1007/s00421-004-1091-8

Allen, F. (1937). The post-contraction of the muscles of the arm. *Quarterly Journal of Experimental Physiology*, *26*(4), 305–317.

Allen, F., & O'Donoghue, C. (1927). The post-contraction proprioceptive reflex, its augmentation and inhibition. *Quarterly Journal of Experimental Physiology*, *18*(3), 199–242.

Aron, A. R., & Poldrack, R. (2006). Cortical and Subcortical Contributions to Stop Signal Response Inhibition: Role of the Subthalamic Nucleus. *Journal of Neuroscience*, *26*(9), 2424–2433. http://doi.org/10.1523/JNEUROSCI.4682-05.2006

785 Aron, A. R., & Verbruggen, F. (2008). Stop the presses: dissociating a selective from
786 a global mechanism for stopping. *Psychological Science*, *19*(11), 1146–1153.
787 http://doi.org/10.1111/j.1467-9280.2008.02216.x
788 Baird, B., Smallwood, J., Lutz, A., & Schooler, J. W. (2014). The decoupled mind:
789 mind-wandering disrupts cortical phase-locking to perceptual events. *Journal*
790 *of Cognitive Neuroscience*, *26*(11), 2596–2607.
791 http://doi.org/10.1162/jocn_a_00656
792 Blakemore, S. J., & Frith, C. (2003). Self-awareness and action. *Current Opinion in*
793 *Neurobiology*, *13*(2), 219–224.
794 Blakemore, S. J., Goodbody, S., & Wolpert, D. (1998). Predicting the consequences
795 of our own actions: the role of sensorimotor context estimation. *Journal of*
796 *Neuroscience*, *18*(18), 7511–7518.
797 Blakemore, S. J., Wolpert, D. M., & Frith, C. D. (1998). Central cancellation of self-
798 produced tickle sensation. *Nature Neuroscience*, *1*(7), 635–640.
799 http://doi.org/10.1038/2870
800 Bliss, J. (1980). Sensory experiences of Gilles de la Tourette syndrome. *Archives of*
801 *General Psychiatry*, *37*(12), 1343–1347.
802 Brice, T., & McDonagh, M. (2001). Abduction of the humerus by postural
803 aftercontractions in man: effects of force and duration of previous voluntary
804 contractions. *The Journal of Physiology*, *536P, S214*.
805 Brown, T. G., & Sherrington, C. S. (1912). On the Instability of a Cortical Point.
806 *Proceedings of the Royal Society B: Biological Sciences*, *85*(579), 250–277.
807 http://doi.org/10.1098/rspb.1912.0050
808 Brun, C., & Guerraz, M. (2015). Anchoring the “floating arm”: Use of proprioceptive
809 and mirror visual feedback from one arm to control involuntary displacement

of the other arm. *Neuroscience*, *310*, 268–278. http://doi.org/10.1016/j.neuroscience.2015.09.052

Brun, C., Metral, M., Chancel, M., Kavounoudias, A., Luyat, M., & Guerraz, M. (2015). Passive or simulated displacement of one arm (but not its mirror reflection) modulates the involuntary motor behavior of the other arm. *Neuroscience*, *285*, 343–355. http://doi.org/10.1016/j.neuroscience.2014.11.036

Chase, M. H., & Morales, F. R. (1990). The atonia and myoclonia of active (REM) sleep. *Annual Review of Psychology*, *41*, 557–584. http://doi.org/10.1146/annurev.ps.41.020190.003013

Chronicle, E. P., & Glover, J. (2003). A ticklish question: does magnetic stimulation of the primary motor cortex give rise to an “efference copy”? *Cortex*, *39*(1), 105–110.

Coxon, J. P., Stinear, C. M., & Byblow, W. D. (2007). Selective inhibition of movement. *Journal of Neurophysiology*, *97*(3), 2480–2489. http://doi.org/10.1152/jn.01284.2006

Craske, B., & Craske, J. D. (1985). Muscular after-contraction reveals oscillator mechanisms in the human motor apparatus. *Human Movement Science*, *4*(4), 249–269. http://doi.org/10.1016/0167-9457(85)90013-2

Cratty, B. J., & Duffy, K. E. (1969). Studies of movement aftereffects. *Perceptual and Motor Skills*, *29*(3), 843–860. http://doi.org/10.2466/pms.1969.29.3.843

Danielopolu, D., Radovici, D. A., & Carniol, A. (1921). Sur un phénomène d’automatisme des muscles volontaires chez l’homme. *Bulletins et Mémoires de la Societe médicale des Hôpitaux de Bucarest*, *3*(7), 101–107.

De Havas, J. A., Ghosh, A., Gomi, H., & Haggard, P. (2015). Sensorimotor organization of a sustained involuntary movement. *Frontiers in Behavioral Neuroscience*, *9*(185). http://doi.org/10.3389/fnbeh.2015.00185

De Havas, J. A., Parimal, S., Soon, C. S., & Chee, M. W. L. (2012). Sleep deprivation reduces default mode network connectivity and anti-correlation during rest and task performance. *NeuroImage*, *59*(2), 1745–1751. http://doi.org/10.1016/j.neuroimage.2011.08.026

Duclos, C., Roll, R., Kavounoudias, A., & Roll, J. P. (2004). Long-lasting body leanings following neck muscle isometric contractions. *Experimental Brain Research*, *158*(1), 58–66. http://doi.org/10.1007/s00221-004-1871-8

Duclos, C., Roll, R., Kavounoudias, A., & Roll, J. P. (2007). Cerebral correlates of the “Kohnstamm phenomenon”: an fMRI study. *NeuroImage*, *34*(2), 774–783. http://doi.org/10.1016/j.neuroimage.2006.06.050

Fessard, A., & Tournay, A. (1949). XI. - Quelques données et réflexions sur le phénomène de la post-contraction involontaire. *L’année psychologique*, *50*(1), 217–235. http://doi.org/10.3406/psy.1949.8445

Feurra, M., Pasqualetti, P., Bianco, G., Santarnecchi, E., Rossi, A., & Rossi, S. (2013). State-dependent effects of transcranial oscillatory currents on the motor system: what you think matters. *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, *33*(44), 17483–17489. http://doi.org/10.1523/JNEUROSCI.1414-13.2013

Filevich, E., Kühn, S., & Haggard, P. (2012a). Intentional inhibition in human action: the power of “no.” *Neuroscience and Biobehavioral Reviews*, *36*(4), 1107–1118. http://doi.org/10.1016/j.neubiorev.2012.01.006

858 Filevich, E., Kühn, S., & Haggard, P. (2012b). Negative motor phenomena in cortical
859 stimulation: implications for inhibitory control of human action. *Cortex*, *48*(10),
860 1251–1261. http://doi.org/10.1016/j.cortex.2012.04.014
861 Forbes, A., Baird, P., & Hopkins, A. M. (1926). The involuntary contraction following
862 isometric contraction of skeletal muscle in man. *American Journal of*
863 *Physiology*, *78*(1), 81–103.
864 Fox, M. D., Snyder, A. Z., Vincent, J. L., Corbetta, M., Van Essen, D. C., & Raichle,
865 M. E. (2005). The human brain is intrinsically organized into dynamic,
866 anticorrelated functional networks. *Proceedings of the National Academy of*
867 *Sciences of the United States of America*, *102*(27), 9673–9678.
868 http://doi.org/10.1073/pnas.0504136102
869 Fried, I., Katz, A., McCarthy, G., Sass, K. J., Williamson, P., Spencer, S. S., &
870 Spencer, D. D. (1991). Functional organization of human supplementary
871 motor cortex studied by electrical stimulation. *The Journal of Neuroscience:*
872 *The Official Journal of the Society for Neuroscience*, *11*(11), 3656–3666.
873 Frith, C. D., Blakemore, S. J., & Wolpert, D. M. (2000). Abnormalities in the
874 awareness and control of action. *Philosophical Transactions of the Royal*
875 *Society of London. Series B, Biological Sciences*, *355*(1404), 1771–1788.
876 http://doi.org/10.1098/rstb.2000.0734
877 Ghafouri, M., Thullier, F., Gurfinkel, V. S., & Lestienne, F. G. (1998). Muscular after-
878 contraction and ongoing postural reactions in standing and sitting humans.
879 *Neuroscience Letters*, *250*(1), 61–65.
880 Ghosh, A., & Haggard, P. (2014). The spinal reflex cannot be perceptually separated
881 from voluntary movements. *The Journal of Physiology*, *592*(Pt 1), 141–152.
882 http://doi.org/10.1113/jphysiol.2013.260588

Ghosh, A., Rothwell, J., & Haggard, P. (2014). Using voluntary motor commands to inhibit involuntary arm movements. *Proceedings of the Royal Society B: Biological Sciences*, *281*(1794), 20141139–20141139. http://doi.org/10.1098/rspb.2014.1139

Gilhodes, J. C., Gurfinkel, V. S., & Roll, J. P. (1992). Role of Ia muscle spindle afferents in post-contraction and post-vibration motor effect genesis. *Neuroscience Letters*, *135*(2), 247–251.

Gurfinkel, V. S., Levik, I. S., & Lebedev, M. A. (1989). Immediate and remote postactivation effects in the human motor system. *Neiřofiziologiia = Neurophysiology*, *21*(3), 343–351.

Hagbarth, K. E., & Nordin, M. (1998). Postural after-contractions in man attributed to muscle spindle thixotropy. *The Journal of Physiology*, *506 (Pt 3)*, 875–883.

Haggard, P. (2011). Decision time for free will. *Neuron*, *69*(3), 404–406. http://doi.org/10.1016/j.neuron.2011.01.028

Henriques, V., & Lindhard, J. (1921). Das Salmon-Kohnstammeche Phanomen. *Neurologisches Centralblatt*, *40*(1), 30–35.

Howell, D. C. (2010). *Statistical methods for psychology*. Belmont, CA: Thomson Wadsworth.

Irlbacher, K., Voss, M., Meyer, B.-U., & Rothwell, J. C. (2006). Influence of ipsilateral transcranial magnetic stimulation on the triphasic EMG pattern accompanying fast ballistic movements in humans. *The Journal of Physiology*, *574*(Pt 3), 917–928. http://doi.org/10.1113/jphysiol.2006.108563

Jun, J. J., Longtin, A., & Maler, L. (2014). Enhanced attention precedes self-initiated locomotion in an electric fish. *BMC Neuroscience*, *15*(Suppl 1), P104. http://doi.org/10.1186/1471-2202-15-S1-P104

Kahneman, D. (2012). *Thinking, fast and slow*. London: Penguin Books.

Kohnstamm, O. (1915). Demonstration einer katatoneartigen erscheinung beim gesunden (Katatonusuersuch). *Neurologisches Centralblatt*, *34*(9), 290–291.

Kozhina, G. V., Person, R. S., Popov, K. E., Smetanin, B. N., & Shlikov, V. Y. (1996). Motor unit discharge during muscular after-contraction. *Journal of Electromyography and Kinesiology: Official Journal of the International Society of Electrophysiological Kinesiology*, *6*(3), 169–175.

Kraskov, A., Dancause, N., Quallo, M. M., Shepherd, S., & Lemon, R. N. (2009). Corticospinal neurons in macaque ventral premotor cortex with mirror properties: a potential mechanism for action suppression? *Neuron*, *64*(6), 922–930. http://doi.org/10.1016/j.neuron.2009.12.010

Kühn, S., Haggard, P., & Brass, M. (2014). Differences between endogenous and exogenous emotion inhibition in the human brain. *Brain Structure and Function*, *219*(3), 1129–1138. http://doi.org/10.1007/s00429-013-0556-0

Laignel-Lavastine, M. M., Chevalier, P., & Vie, J. (1927). Le réflexe tonodynamique. *Bulletins et Memoires de la Societé médicale des Hôpitaux de Paris*, *43*(22), 988 – 991.

MacDonald, H. J., Coxon, J. P., Stinear, C. M., & Byblow, W. D. (2014). The fall and rise of corticomotor excitability with cancellation and reinitiation of prepared action. *Journal of Neurophysiology*, *112*(11), 2707–2717. http://doi.org/10.1152/jn.00366.2014

Majid, D. S. A., Cai, W., Corey-Bloom, J., & Aron, A. R. (2013). Proactive Selective Response Suppression Is Implemented via the Basal Ganglia. *Journal of Neuroscience*, *33*(33), 13259–13269. http://doi.org/10.1523/JNEUROSCI.5651-12.2013

933 Majid, D. S. A., Cai, W., George, J. S., Verbruggen, F., & Aron, A. R. (2012).
934 Transcranial Magnetic Stimulation Reveals Dissociable Mechanisms for
935 Global Versus Selective Corticomotor Suppression Underlying the Stopping of
936 Action. *Cerebral Cortex*, *22*(2), 363–371. http://doi.org/10.1093/cercor/bhr112
937 Marsden, C. D., Merton, P. A., & Morton, H. B. (1976). Servo action in the human
938 thumb. *The Journal of Physiology*, *257*(1), 1–44.
939 Mathis, J., Gurfinkel, V. S., & Struppler, A. (1996). Facilitation of motor evoked
940 potentials by postcontraction response (Kohnstamm phenomenon).
941 *Electroencephalography and Clinical Neurophysiology*, *101*(4), 289–297.
942 Matsumura, M., Nambu, A., Yamaji, Y., Watanabe, K., Imai, H., Inase, M., …
943 Takada, M. (2000). Organization of somatic motor inputs from the frontal lobe
944 to the pedunculopontine tegmental nucleus in the macaque monkey.
945 *Neuroscience*, *98*(1), 97–110.
946 Matthaei, R. (1924). Nachbewegungen beim menschen Untersuchungen uber das
947 sog. Kohnstammsche phanomen. I. Vorkommen und Wesen der Erscheinung
948 (Postcontraction in humans(Research concerning the so called Kohnstamm
949 phenomenon)). *Pflugers Archive*, *202*(1), 88–111.
950 Moraitis, T., & Ghosh, A. (2014). Withdrawal of voluntary inhibition unravels the off
951 state of the spontaneous blink generator. *Neuropsychologia*, *65*, 279–286.
952 http://doi.org/10.1016/j.neuropsychologia.2014.09.010
953 Mostofsky, S. H., & Simmonds, D. J. (2008). Response Inhibition and Response
954 Selection: Two Sides of the Same Coin. *Journal of Cognitive Neuroscience*,
955 *20*(5), 751–761. http://doi.org/10.1162/jocn.2008.20500

Murakami, M., Vicente, M. I., Costa, G. M., & Mainen, Z. F. (2014). Neural antecedents of self-initiated actions in secondary motor cortex. *Nature Neuroscience*, *17*(11), 1574–1582. http://doi.org/10.1038/nn.3826

Nielsen, J. B. (2004). Sensorimotor integration at spinal level as a basis for muscle coordination during voluntary movement in humans. *Journal of Applied Physiology*, *96*(5), 1961–1967. http://doi.org/10.1152/japplphysiol.01073.2003

Parkinson, A., & McDonagh, M. (2006). Evidence for positive force feedback during involuntary aftercontractions. *Experimental Brain Research*, *171*(4), 516–523. http://doi.org/10.1007/s00221-005-0299-0

Parkinson, A., McDonagh, M., & Vidyasagar, R. (2009). Brain activation in an involuntary human action. *Brain Research*, *1304*, 57–65. http://doi.org/10.1016/j.brainres.2009.09.092

Pereira, J. (1925). Contraction automatique des muscles stries chez l'homme. *Journal de physiologie et de pathologie générale*, *23*(1), 30–38.

Pinkhof, J. (1922). Contraction residuelle de muscles volontaires apres un raccourcissement tetanique energique (Residual contraction of voluntary muscles after a strong, tetanic shortening). *Archives Neerlandaises de Physiologie de l'Homme et Des Animaux*, *6*, 516–527.

Pope, P. A., Holton, A., Hassan, S., Kourtis, D., & Praamstra, P. (2007). Cortical control of muscle relaxation: a lateralized readiness potential (LRP) investigation. *Clinical Neurophysiology: Official Journal of the International Federation of Clinical Neurophysiology*, *118*(5), 1044–1052. http://doi.org/10.1016/j.clinph.2007.02.002

Prado, H. S., Rosário, M. C., Lee, J., Hounie, A. G., Shavitt, R. G., & Miguel, E. C. (2008). Sensory phenomena in obsessive-compulsive disorder and tic disorders: a review of the literature. *CNS Spectrums*, *13*(5), 425–432.

Pruszynski, J. A., Kurtzer, I., & Scott, S. H. (2011). The long-latency reflex is composed of at least two functionally independent processes. *Journal of Neurophysiology*, *106*(1), 449–459. http://doi.org/10.1152/jn.01052.2010

Roessner, V., Wittfoth, M., Schmidt-Samoa, C., Rothenberger, A., Dechent, P., & Baudewig, J. (2012). Altered motor network recruitment during finger tapping in boys with Tourette syndrome. *Human Brain Mapping*, *33*(3), 666–675. http://doi.org/10.1002/hbm.21240

Rossignol, S., Dubuc, R., & Gossard, J.-P. (2006). Dynamic sensorimotor interactions in locomotion. *Physiological Reviews*, *86*(1), 89–154. http://doi.org/10.1152/physrev.00028.2005

Rothmann, M. (1915). Zum Katatonusversuch (Kohnstamm). *Neurologisches Centralblatt*, *34*(12), 421–425.

Rudomin, P. (1999). Presynaptic selection of afferent inflow in the spinal cord. *Journal of Physiology, Paris*, *93*(4), 329–347.

Salmon, A. (1915). Nuove osservazioni sui movimenti automatici che si compiono dopo gli sforzi muscolari e del loro valore in neuropatologia. *Atti Della Accademia Medico-Fisica Fiorentina*, 78–91.

Salmon, A. (1916). D'un interessant phenomene d'automatisme qu'on remarque apres les efforts musculaires chez les sujets sains (On an interesting phenomenon of automaticity observed after muscular effort in healthy subjects). *Revue Neurologique*, *29*(1), 27–34.

Salmon, A. (1925). Les mouvements automatiques qui suivent les efforts musculaires volontaires chez les sujets sains. *Journal de Physiologie et de Pathologie Générale*, *23*(1), 790–794.

Salmon, A. (1929). Nouvelle observations cliniques et expérimentales sur les mouvements automatiques qui suivent les efforts musculaires volontaires. *Revue Neurologique*, *52*(2), 428–438.

Salomonson, J. K. A. W. (1921). Tonus and the reflexes. *Brain*, *43*(4), 369–389. http://doi.org/10.1093/brain/43.4.369

Sapirstein, M. R., Herman, R. C., & Wallace, G. B. (1936). Effect of certain drugs on after-contraction. *Proceedings of the Society for Experimental Biology and Medicine*, *35*(1), 163–165.

Sapirstein, M. R., Herman, R. C., & Wallace, G. B. (1937). A study of after-contraction. *American Journal of Physiology -- Legacy Content*, *119*(3), 549–556.

Sapirstein, M. R., Herman, R. C., & Wechsler, I. S. (1938). Mechanisms of after-contraction - further studies. *Archives of Neurology & Psychiatry*, *40*(2), 300–312. http://doi.org/10.1001/archneurpsyc.1938.02270080084004

Schneider, C. A., Rasband, W. S., & Eliceiri, K. W. (2012). NIH Image to ImageJ: 25 years of image analysis. *Nature Methods*, *9*(7), 671–675.

Schwartz, A., & Meyer, P. (1921). Un curieux phénomène d'automatisme chez l'Homme. *Comptes Rendus de la Société de Biologie*, *85*(27), 490–492.

Shergill, S. S., Bays, P. M., Frith, C. D., & Wolpert, D. M. (2003). Two eyes for an eye: the neuroscience of force escalation. *Science (New York, N.Y.)*, *301*(5630), 187. http://doi.org/10.1126/science.1085327

Sherrington, C. S. (1913). REFLEX INHIBITION AS A FACTOR IN THE CO-ORDINATION OF MOVEMENTS AND POSTURES. *Quarterly Journal of Experimental Physiology*, *6*(3), 251–310. http://doi.org/10.1113/expphysiol.1913.sp000142

Solopova, I. A., Selionov, V. A., Zhvansky, D. S., Gurfinkel, V. S., & Ivanenko, Y. (2016). Human cervical spinal cord circuitry activated by tonic input can generate rhythmic arm movements. *Journal of Neurophysiology*, *115*(2), 1018–1030. http://doi.org/10.1152/jn.00897.2015

Specht, M. W., Woods, D. W., Nicotra, C. M., Kelly, L. M., Ricketts, E. J., Conelea, C. A., … Walkup, J. T. (2013). Effects of tic suppression: Ability to suppress, rebound, negative reinforcement, and habituation to the premonitory urge. *Behaviour Research and Therapy*, *51*(1), 24–30. http://doi.org/10.1016/j.brat.2012.09.009

Takakusaki, K. (2008). Forebrain control of locomotor behaviors. *Brain Research Reviews*, *57*(1), 192–198. http://doi.org/10.1016/j.brainresrev.2007.06.024

Takakusaki, K., Habaguchi, T., Ohtinata-Sugimoto, J., Saitoh, K., & Sakamoto, T. (2003). Basal ganglia efferents to the brainstem centers controlling postural muscle tone and locomotion: a new concept for understanding motor disorders in basal ganglia dysfunction. *Neuroscience*, *119*(1), 293–308. http://doi.org/10.1016/S0306-4522(03)00095-2

Takakusaki, K., Kohyama, J., & Matsuyama, K. (2003). Medullary reticulospinal tract mediating a generalized motor inhibition in cats: III. Functional organization of spinal interneurons in the lower lumbar segments. *Neuroscience*, *121*(3), 731–746.

1051 Takakusaki, K., Kohyama, J., Matsuyama, K., & Mori, S. (2001). Medullary
1052 reticulospinal tract mediating the generalized motor inhibition in cats: parallel
1053 inhibitory mechanisms acting on motoneurons and on interneuronal
1054 transmission in reflex pathways. *Neuroscience*, *103*(2), 511–527.
1055 Takakusaki, K., Oohinata-Sugimoto, J., Saitoh, K., & Habaguchi, T. (2004). Role of
1056 basal ganglia–brainstem systems in the control of postural muscle tone and
1057 locomotion. In *Progress in Brain Research* (Vol. 143, pp. 231–237). Elsevier.
1058 Retrieved from http://linkinghub.elsevier.com/retrieve/pii/S0079612303430239
1059 Takakusaki, K., Shimoda, N., Matsuyama, K., & Mori, S. (1994). Discharge
1060 properties of medullary reticulospinal neurons during postural changes
1061 induced by intrapontine injections of carbachol, atropine and serotonin, and
1062 their functional linkages to hindlimb motoneurons in cats. *Experimental Brain*
1063 *Research*, *99*(3), 361–374.
1064 Voss, M., Bays, P. M., Rothwell, J. C., & Wolpert, D. M. (2007). An improvement in
1065 perception of self-generated tactile stimuli following theta-burst stimulation of
1066 primary motor cortex. *Neuropsychologia*, *45*(12), 2712–2717.
1067 http://doi.org/10.1016/j.neuropsychologia.2007.04.008
1068 Wyland, C. L., Kelley, W. M., Macrae, C. N., Gordon, H. L., & Heatherton, T. F.
1069 (2003). Neural correlates of thought suppression. *Neuropsychologia*, *41*(14),
1070 1863–1867.
1071 Xu, J., Westrick, Z., & Ivry, R. B. (2015). Selective inhibition of a multicomponent
1072 response can be achieved without cost. *Journal of Neurophysiology*, *113*(2),
1073 455–465. http://doi.org/10.1152/jn.00101.2014
1074 Zigler, M. J., Martin, B., Smith, A., & Stadeker, J. (1948). Post-contractional fatigue:
1075 change in magnitude of involuntary behavior as a function of successive

1076 tensive stimulations. *Journal of Comparative and Physiological Psychology*,
1077 *41*(2), 124–136. http://doi.org/10.1037/h0059394
1078