JNeurosci
THE JOURNAL OF NEUROSCIENCE

Research Report: Regular Manuscript

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https://doi.org/10.1523/JNEUROSCI.0002-20.2020

Cite as: J. Neurosci 2020; 10.1523/JNEUROSCI.0002-20.2020

Received: 1 January 2020
Revised: 16 May 2020
Accepted: 19 June 2020

Title: On stopping voluntary muscle relaxations and contractions: evidence for shared control mechanisms and muscle state specific active breaking

Abbreviated title: Stopping muscle relaxations and contractions

Authors: Jack De Havas[1], Sho Ito[1], and Hiroaki Gomi[1]

[1]NTT Communication Science Laboratories, Nippon Telegraph and Telephone Corporation, Wakamiya 3-1, Morinosato, Atsugi, Kanagawa-pref. 243-0198, Japan

Author contributions: JDH, SI, and HG designed the research. JDH and SI performed the research. JDH Analyzed the data. JDH and HG wrote the paper.

Corresponding author
Jack De Havas
NTT Communication Science Laboratories, Nippon Telegraph and Telephone Corporation, Wakamiya 3-1, Morinosato, Atsugi,
Kanagawa-pref. 243-0198, Japan
jdehavas@gmail.com

Number of Figures: 6

Number of Tables: 6

Number of Multimedia: 0

Number of words in Abstract: 246

Number of words in Significance statement: 102

Number of words in Introduction: 630

Number of words in Discussion: 1495

Acknowledgments: The authors would like thank Maiko Goshima for help with providing illustrations for task design figures and two anonymous reviewers for their useful comments on the manuscript.

Conflict of interest: The authors declare no competing financial interests.

Funding: This work was funded by a JSPS short-term fellowship. HG were further supported by Grants-in-Aid for Scientific Research (JP16H06566) from Japan Society for the Promotion of Science.

Abstract

Control of the body requires inhibiting complex actions, involving contracting and relaxing muscles. However, little is known of how voluntary commands to relax a muscle are cancelled. Action inhibition causes both suppression of muscle activity and the transient excitation of antagonist muscles, the latter being termed active breaking. We hypothesised that active breaking is present when stopping muscle relaxations. Stop signal experiments were used to compare the mechanisms of active breaking for muscle relaxations and contractions in male and female human participants. In experiments 1 and 2, go signals were presented which required participants to contract or relax their biceps or triceps muscle. Infrequent stop signals occurred after fixed delays (0-500ms), requiring that participants cancelled go commands. In experiment 3, participants increased (contract) or decreased (relax) an existing isometric finger abduction depending on the go signal, and cancelled these force changes whenever stop signals occurred (dynamically adjusted delay). We found that muscle relaxations were stopped rapidly, met predictions of existing race models, and had stop signal reaction times that correlated with those observed during the stopping of muscle contractions, suggesting shared control mechanisms. However, stopped relaxations were preceded by transient increases in EMG, while stopped contractions were preceded by decreases in EMG, suggesting a later divergence of control. Muscle state specific active breaking occurred simultaneously across muscles, consistent with a central origin. Our results indicate that the later stages of action inhibition involve

separate excitatory and inhibitory pathways, which act automatically to cancel complex body movements.

Significance Statement

The mechanisms of how muscle relaxations are cancelled are poorly understood. We showed in three experiments involving multiple effectors that stopping muscle relaxations involves transient bursts of EMG activity, which resemble co-contraction and have onsets that correlate with stop signal reaction time (SSRT). Comparison with the stopping of matched muscle contractions showed that active breaking was muscle state specific, being positive for relaxations and negative for contractions. The two processes were also observed to co-occur in agonist-antagonist pairs, suggesting separate pathways. The rapid, automatic activation of both pathways may explain how complex actions can be stopped at any stage of their execution.

Introduction

Control of the body necessitates the rapid cancelling of actions in response to changes in the environment. Such actions often involve multiple muscles in different states of contraction and relaxation. However, most action inhibition experiments have focused on simple button press responses and saccades (Band and van Boxtel, 1999; Verbruggen and Logan, 2008). Less is known about complex actions, particularly those caused by muscle relaxations.

Voluntary isometric and isotonic muscle relaxations are an active process (Toma et al., 2000; Alegre et al., 2003), vital for updating posture and initiating movement (Buccolieri et al., 2003). Relaxation commands activate the

supplementary and primary motor cortex (M1) to a similar degree to matched muscle contractions (Toma et al., 1999; Spraker et al., 2009). Prior to a relaxation command, readiness potentials are observed at fronto-central electrodes (Dimitrov, 1985; Terada et al., 1995; Rothwell et al., 1998), while the execution of the movement itself is preceded by a lateralized readiness potential (LRP) of equal amplitude and latency to those preceding muscle contractions (Pope et al., 2007). Understanding the control of voluntary muscle relaxations is important because muscle relaxation deficits are prevalent in stroke (Chae et al., 2002, 2006; Seo et al., 2009), dystonia (Yazawa et al., 1999; Oga et al., 2002) and Parkinson's disease (Kunesch et al., 1995; Grasso et al., 1996; Labyt et al., 2005).

Given the control similarities to muscle contraction, it should be possible to cancel a 'go command' to relax a muscle if a 'stop command' is issued early enough. The stop signal task involves participants responding to a go signal on every trial and attempting to suppress their responses whenever an infrequent stop signal occurs (Lappin and Eriksen, 1966; Logan et al., 1984). Stop signal reaction time (SSRT) can then be calculated, which is a good predictor of general inhibitory function (Logan et al., 1997; Williams et al., 1999). Stop and go processes race to completion via largely separate pathways in the brain (Logan and Cowan, 1984; Aron et al., 2007). If stopping muscle relaxations conforms to this account, it may be possible to derive the general mechanisms determining how complex actions are cancelled.

Action inhibition involves multiple central and peripheral pathways. In the motor cortex, stopping is associated with increased short-interval intracortical

inhibition (SICI; Coxon et al., 2006; van den Wildenberg et al., 2010), indicating greater inhibitory GABAergic activity. Increased SICI is also observed during muscle relaxation (Buccolieri et al., 2004; Motawar et al., 2012). As such, combining muscle relaxation go commands and action inhibition might produce slowing of SSRT due to interference. However, excitatory pathways likely also contribute to stopping, negating any such slowing. Excitation has been observed at the periphery in the form of bursts of antagonist activity when reaching movements are inhibited (Atsma et al., 2018). This 'active breaking' may also explain how ballistic movements (McGarry and Franks, 1997) and increases of isometric force (de Jong et al., 1990) can be arrested mid-way through their execution. Nevertheless, it is unknown whether active breaking is confined to the antagonist muscle, nor whether it occurs uniformly in relaxing and contracting muscles, or manifests differently in both as a form of muscle state specific response. Relatedly, it is uncertain whether active breaking arises from changes at the agonist which in turn influence the antagonist (peripheral command), or from a single central command, which may be similar to co-contraction.

We therefore designed a series of stop signal experiments to determine the general peripheral mechanisms underpinning the inhibition of relaxing and contracting muscles. We test the hypotheses that: 1) voluntary relaxations are cancelled as rapidly as muscle contractions, in accordance with the principles of the race model, 2) Active breaking is present across movement types, but manifests differently depending on the muscle state, 3) Active breaking involves simultaneous action across muscles.

Methods

Equipment

Participants were seated at an adjustable table, fitted with foam padding. Modified stop signal tasks were run via MATLAB (2017a) and Psychtoolbox (Brainard, 1997). Visual stimuli were displayed via a flat screen monitor (27-inch LCD, 1902 x 1080 pixels, 60 Hz refresh rate). Auditory stimuli consisted of tones delivered via a buzzer (3000 Hz, 50 dB, 160ms).

Electromyography (EMG) was recorded from bipolar, surface electrodes (Ag-AgCl disposable electrode, GE Healthcare Japan, Tokyo, Japan) placed over the middle of the right biceps brachii and right triceps brachii lateral head muscles in experiment 1 and 2, and on the first dorsal interosseous (FDI) muscle in experiment 3. EMG signals were sampled at 2000 Hz and amplified using variable gain (MME-3132, Nihon Kohden, Tokyo, Japan). For the purposes of training participants to relax their muscles, EMG data was displayed via an oscilloscope (TDS2004C, Tektronix, Inc., Oregon, USA) positioned at eye level in front of them. This was removed during the actual task.

In experiments 1 and 2, elbow angle was determined via the use of three infrared reflective markers positioned at the forearm, elbow and shoulder of the right arm. Marker position was detected via two motion tracking cameras (Oqus300, Qualisys AB, Gothenburg, Sweden) with a sampling rate of 250 Hz. In experiment 3, isometric finger abduction force was measured using a force sensor (6-axis force sensor; ThinNANO 5/4-A, BL-Autotech, LTD., Kobe, Japan). Maximum voluntary forces were recorded at the start of the experiment via a different force sensor (Force gauge; FGC-5, Nidec-Shimpo, Kyoto, Japan).

Participants

For experiment 1 we recruited 13 participants (8 were female, mean age = 34.5 yrs, SD = 9.2 yrs). Experiment 2 included 12 participants (8 were female, mean age = 33.83 yrs, SD = 9.42 yrs), 5 of whom had previously participated in experiment 1. We recruited 16 participants for experiment 3. However, 2 participants were excluded before the analysis phase due to equipment failure. This left 14 participants who were included in the analysis (11 were female, mean age = 36.71 yrs, SD = 8.63 yrs). Of these, 5 had participated in exps. 1 and 3 had participated in exps. 2. Experiments were undertaken with the understanding and written consent of each participant in accordance with the Code of Ethics of the World Medical Association (Declaration of Helsinki), and with the NTT Communication Science Laboratories Research Ethics Committee approval.

General procedure

Participants first completed a practice button press version of the stop signal task, similar to those widely used in the stop signal literature (Verbruggen et al., 2019). Maximum voluntary contractions (MVC) of relevant muscles were then recorded via 5 s isometric contractions against a fixed surface. All experiments used a modified version of the stop signal task. Participants maintained a constant level of muscle contraction in the target effector whilst looking at a fixation cross on a screen 70 cm in front of them. On every trial, a visual go signal was presented in the centre of the screen in the form of a coloured shape (subtending 4.7 ° visual angle). In the relaxation condition participants relaxed

the target muscle and in the contraction condition they contracted the target muscle as quickly as possible in response to the go signal. On a randomly selected subset of trials (30%), a stop signal (tone) was presented after a specifically controlled stop signal delay (SSD). Experiments 1 and 2 compared the stopping of relaxation of the biceps to the stopping of contractions of the triceps and biceps muscle respectively. Thus in experiment 1, the movement direction was matched (elbow extension) and in experiment 2 the primary muscle was matched (biceps brachii) across movement conditions. Experiment 3 was conducted using isometric relaxations and contractions of the FDI muscle to determine whether the findings of the previous experiments generalised to other effectors.

Procedure for experiment 1

The target muscle was the right biceps brachii in the relax condition and the triceps brachii in the contract condition. Electromyography (EMG) was recorded from both muscles in all conditions. Uniquely in experiment 1, we tested the relax and contract condition in separate blocks (counterbalanced). Participants were informed of the block type on screen at the start of each block. They rested their elbow on a padded table, supinated and bent at an angle of ~30° from the horizontal (Fig. 1. A.), which required activity in the biceps muscle (~4% MVC). Before each trial, arm angle was adjusted via visual feedback on screen. Successfully matching current to required elbow angle triggered the appearance of a fixation cross.

After a random inter-stimulus interval (ISI = 2-4s) the fixation cross was replaced by green shape subtending 4.7 ° of visual angle (Go/No Go stimulus).

Participants moved their right arm downwards as rapidly as possible whenever a green circle appeared on the screen (Go trials; 80% of trials), and remained stationary whenever a green square appeared (No-go trials; 20% of trials). No go trials ensured participants processed the go stimulus before acting (Verbruggen and Logan, 2009), but were not analysed further. During 'relaxation blocks' participants moved their right arm downwards (elbow extension) by relaxing the biceps muscle, but during 'contraction blocks' extension was achieved by contracting the triceps muscle. On a random subset (30%) of trials a stop signal (tone) was presented. Stop signal onset time in relation to the go signal varied between 0 and 500ms (stop signal delay). There were six tone intervals (0, 100, 200, 300, 400, 500ms; randomized).

Each block consisted of 80 trials (40 go trials, 24 stop trials, 8 no go trials with tone, 8 no go trials without tone). There were 10 blocks in total (5 relax, 5 contract). The experiment lasted ~3 hours.

Procedure for experiment 2

Procedural details were the same as experiment 1, with three important differences. Firstly, the target muscle in the contract condition was the biceps and the go response in this condition involved flexing the elbow (Fig. 3. A.). The relax condition was the same as experiment 1. Secondly, relax and contract conditions were tested within the same blocks. Thirdly, we did not include No go trials. Participants were instructed to relax their right biceps muscle in response to a blue circle appearing on the screen (50% of trials; randomized) and contract their right biceps muscle in response to a blue square (50% of trials; randomized). Stimulus-action pairing was counterbalanced across participants.

Stop signals were the same as experiment 1. There were 10 experimental blocks of 80 trials (28 relax condition go trials, 12 relax condition stop trials, 28 contract condition go trials, 12 contract condition stop trials).

Procedure for experiment 3

Experiment 3 was different to experiment 2 because the target muscle was the right first dorsal interosseous (FDI), the go response involved isometric increases and decreases of force, and the stop signal used dynamic rather than fixed SSDs. Participants rested their arm on a table in a pronated position (Fig. 5. A.) with the outside of their right index finger touching a force sensor. They pushed outwards (abduction) with ~10% MVC to move a small black circle between two boundary lines on screen. These lines, and an additional target line representing a force of 20% MVC, were shown during training, but removed during the actual task once a stable force was achieved at the start of each trial. Go signals were presented after variable ISI (1-4s). Participants had to rapidly decrease FDI muscle activity to 0 if a *circle* appeared (relax condition) and rapidly increase it by ~10% if a *square* appeared (contract condition).

SSD on stop trials (30% of trials) was adjusted from an initial 250ms by increasing it by 50ms every time participants successfully stopped and decreasing by 50ms every time they failed to stop. Staircasing was performed separately for each condition (relax vs contract). This *one-up one-down* method is commonly used in stop signal experiments and requires online trial classification (Logan et al., 1997; Osman et al., 1986; Verbruggen and Logan, 2009). We classified stop trials by setting a force change threshold based on

the mean maximum force 'velocity' (first derivative of force increase/decrease) during the go trials of the previous run. Exceeding 30% of this value during the response window meant a stop trial was classified as a failed stop trial. Trials under threshold were classified as successful stop trials. There were 10 runs of 60 trials (21 relax go trials, 9 relax stop trials, 21 contract go trials, 9 contract stop trials per run). The experiment lasted ~ 3 hours.

Analysis

Elbow angle velocity was determined in exp. 1 and 2 by calculating the angle change (relative to baseline: -2000ms to 0ms) between three reflective marker located on the upper arm, elbow and forearm. The angular change data was smoothed (zero-phase digital filtering; low pass 10 Hz) and the velocity was calculated by taking the difference between successive time points. Acceleration was calculated by taking the difference between successive time points of the velocity data. Force velocity was calculated in exp. 3, by taking the difference between successive time points of the smoothed force data (zero-phase digital filtering; low pass 5 Hz).

Response time (RT) was the point after the Go signal where the angular velocity first rose above 10% of the maximum velocity on that trial (Irlbacher et al., 2006; De Havas et al., 2016). Outliers (> 4 x SD) were re-assigned to the value of the slowest non-outlier trial to ensure SSRT calculations were unbiased (Verbruggen and Logan, 2009). In exp. 3 we used a threshold of 30% because the force signal contained more noise.

EMG data in all experiments was band-pass filtered (zero-phase digital filtering; 10–500 Hz) and rectified, before being smoothed (zero-phase digital

283 filtering; low pass 10 Hz). EMG velocity was calculated by taking the difference
284 between successive time points in the smoothed data. We determined EMG-RT
285 on Go trials via the threshold technique (first time point > 30% of the max, which
286 remained above threshold for at least 25ms).

287 Stop trial success was classified online in exp. 3 (*see procedure*), but in
288 exp. 1 and 2 it was classified offline by first calculating the mean peak go trial
289 velocity separately for each movement type for each participant. Stop trials
290 were coded as successful if the velocity never rose above 10% of the mean
291 max go trial velocity, but as failed if the threshold was exceeded. In all
292 experiments, failed stop trial RT was the point relative to the go signal where
293 the velocity first rose above the stop classifier threshold. Outliers were dealt
294 with in the same manner as for go trials. All calculations were performed
295 independently for the relax and contract conditions. Response times were
296 compared across conditions via within subjects t-tests.

297 In exp. 3 we calculated the mean probability of movement (pMov) in each
298 movement condition (total failed stop trials divided by total stop trials). In exp. 1
299 and 2, pMov was calculated at each SSD, in each movement condition in the
300 same manner. One-way within subjects ANOVAs were used to verify that pMov
301 increased across the six levels of SSD in each movement condition. Inhibition
302 functions were fit to the pMov data using the Weibull method (Hanes and Schall,
303 1995). We used Matlab (2017a) and the Palamedes curve fitting toolkit (Prins
304 and Kingdom, 2018), with a 4 parameter model (α = scale, β = shape, γ = lower
305 bias, λ = upper bias). Lower and upper bias in this context are synonymous with
306 error rate, and in the SST literature consist of trials where stop (trigger failure)

and go (forced inhibition) processes are not initiated (Band et al., 2003). Both bias parameters were restricted to 0.05 (Livesey and Livesey, 2016). Beta values were compared via t-test to determine whether inhibition functions differed in shape across relax and contract conditions.

SSRT was calculated in exp. 3 by subtracting mean SSD from go trial response time in each movement condition (Verbruggen et al., 2019). In exp. 1 and 2, SSRT was calculated using the integration method, which is commonly used for fixed SSDs (Logan and Cowan, 1984) and was necessary because we did not tightly constrain performance, leading to heterogeneity in SSD curves (Verbruggen and Logan, 2009). Go RTs were rank ordered from smallest to largest. For each SSD the total number of Go RT trials was multiplied by the pMov at that SSD. This value was rounded to the nearest integer and the corresponding Go RT in the rank ordered list was selected. The SSD itself was then subtracted from this Go RT to give the SSRT at that SSD. Overall SSRT was then calculated by taking the mean of all the SSRTs obtained, separately for the relax and contract conditions.

SSRTs were compared across movement conditions in all experiments via within-subjects t-tests and Pearson's r. To discount the possibility that experiment 1 was underpowered and to avoid type II errors, we applied a Bayesian analysis (Dienes, 2008, 2014) to experiments 2 and 3 using the mean difference between conditions obtained in experiment 1 (half-normal distribution, 1-tailed).

In all experiments, active breaking was defined as a consistent stop-signal related change in the elbow angle acceleration (exp. 1 and 2), force

velocity (exp. 3) or EMG velocity (exp. 1, 2 and 3) that could not be ascribed to the go signal. In exp. 1 and 2, kinematic active breaking was determined from angle acceleration to control for the large deviations in position and velocity on failed stop trials. In all cases, we first removed any stop trials where early movements occurred in the opposite direction to that which would be expected if it was a Go trial (mean no. trials removed per subject in exp. 1 = 0.6%, exp. 2. = 1.6%, exp. 3. = 2.18%). Next, through visual inspection of group average kinematic, force and EMG data we determined the direction (positive or negative) of active breaking, separately for successful and failed stop trials in each movement condition. Active breaking onset times were calculated separately for elbow angle acceleration, force velocity and EMG velocity. For a given signal, onset time was defined at the individual participant level by taking the first time point 0 – 500ms after the stop signal that reached 30% of its max or minimum value (depending on active breaking direction) within the same window. To determine if active breaking was significant at the group level, we averaged the signal in a 50ms window time-locked to active breaking onset for each participant and compared these values to 0 via one-sample t-tests (see Fig. 2., 4. & 6.). After confirming the presence of active breaking at the group level, the analysis was repeated across trials at the individual participant level and tested via one-sample t-test (1-tailed).

In experiment 1, in the relax condition the primary muscle was the biceps and the secondary muscle was the triceps. Some participants (n = 5) showed evidence of increased triceps activity before the stop signal on some trials and were excluded from the secondary muscle analyses to minimize false positive

detection of active breaking. In the contract condition the primary muscle was the triceps and the secondary muscle was the biceps. Again, we had to remove some participants (n = 5) who showed early pre-stop signal activation of the secondary muscle. In experiment 2, as before the biceps was the primary muscle and the triceps was the secondary in the relax condition (n = 5 excluded from secondary muscle analysis, due to pre-stop signal activity on some trials). Likewise, in the contract condition the biceps was the primary muscle and the triceps was the secondary muscle. One participant was excluded from the secondary muscle analysis because triceps EMG active breaking occurred very late relative to the stop signal (>300ms), and after the kinematic active breaking onset, meaning it was a reaction to the arm stopping rather than a cause. Finally, in experiment 3, the FDI was the primary muscle in both movement conditions.

We performed a further analysis on successful stop trials to determine: 1) if active breaking occurred when kinematics/force traces were flat, and 2) how active breaking magnitude changed with decreasing detected go response. In all experiments, successful stop trials were split into quartiles at the individual participant level after rank ordering them according to the amplitude of go response detected in the smoothed kinematic or force traces prior to the stop signal onset (-1000 – 0ms). Primary muscle active breaking amplitude was then calculated for trials in each of the 4 bins. This was done at the group level in the same manner as described above, using the already derived active breaking onset times (based on all successful stop trials) for time-locking.

Mean individual active breaking onset times were calculated by taking the average between successful and failed stop trials, separately for kinematics force and primary muscle EMG in each movement condition. Mean active breaking times were then correlated to SSRT in all experiments via Pearson's r, under a 1-tailed assumption that all correlations were expected to be positive.

Primary and secondary muscle active breaking onset times were compared via paired sample t-tests in exp. 1 (relax n = 8, contract n = 8) and exp. 2 (relax n = 7, contract n = 11). A time-shifted cross correlation analysis was done to determine if active breaking in one muscle predicted active breaking in the other after some reliable time-delay. Absolute triceps muscle EMG velocity traces were shifted (-50 – 50ms) in 0.5ms increments relative to the Biceps muscle active breaking onset time. After each shift, cross-correlation was performed (Pearson's r) during a window that included the whole active breaking response (EMG velocity traces in both muscles, -10 – 250ms relative to biceps muscle active breaking onset). The reverse calculation was also performed (i.e. biceps shifted relative to triceps) and the results averaged together, after the time-axis had been reversed to ensure results were in the same direction. This was done to control for any biases in how onset times were detected across muscles. Thus in both cases, if correlations peaked reliably before 0ms it indicated that the biceps activity predicted the triceps activity, and if correlations peaked reliably after 0ms it indicated triceps activity predicted the biceps activity. Positive peaks close to 0ms indicated simultaneous action. The analysis was performed at the individual participant level. Successful and failed

stop trial results were then averaged, and group averages were calculated for relax and contract conditions in each experiment (see Fig. 2. M. & Fig. 4. M.).

Figure 1

Table 1

Results

Go trial response times

Go trial response times were faster in the contract condition than the relax condition in experiment 1 (521.28ms vs 554.74ms; t (12) = 2.511, p = 0.027, Cohen's d = 0.57; Table 1) and experiment 2 (549.82ms vs 588.36; t (11) = 3.879, p = 0.003, Cohen's d = 0.77; Table 3), but did not differ in experiment 3 (595.11ms vs 598.01ms; t (13) = 0.26, p = 0.799, Cohen's d = 0.05; Table 5). In experiment 1, for the relax condition the biceps EMG-RT was faster than the triceps (311.01ms vs 390.61ms; t (12) = -3.572, p = 0.004, Cohen's d = -1.17). In the contract condition the biceps muscle began to relax earlier than the triceps muscle started to contract (331.05ms vs 417.18ms; t (12) = -6.036, p < 0.001, Cohen's d = -1.56). In experiment 2, the biceps muscle EMG-RT was again faster than the triceps muscle in the relax condition (331.76ms vs 416.21ms; t (11) = -4.083, p = 0.002, Cohen's d = -1.39). Biceps and triceps muscle EMG-RT did not differ in the contract condition, when both were contracting (437.79ms vs 440.08ms; t (11) = -0.852, p = 0.412, Cohen's d = -

0.07). However, comparing directly between the relax and contract conditions in the biceps showed relaxation was indeed faster ($t(11) = -11.003$, $p < 0.001$, Cohen's $d = -2.99$). Thus relaxation was faster than contraction in the forearm, particularly in the biceps muscle. However, in the FDI muscle (exp. 3) there was only a trend for relaxation being faster than contraction (424.29ms vs 445.21ms; $t(13) = -1.982$, $p = 0.069$, Cohen's $d = -0.41$).

Stopping muscle relaxations not impaired relative to stopping contractions

In experiment 1 contract SSRT (Mn = 238.51ms, SD = 49.42ms) and relax SSRT (Mn = 220.46, SD = 49.42) did not significantly differ ($t(12) = 1.628$, $p = 0.129$, Cohen's $d = 0.401$, observed power = 0.266). There was also no significant difference in SSRT between contract and relax conditions in experiment 2 (Mn = 256.5ms, SD = 31.69 vs. Mn = 268.1ms, SD= 48.31; $t(11) = -0.992$, $p = 0.34$, Cohen's $d = -0.284$, observed power = 0.147) and experiment 3 (Mn = 156.86ms, SD = 69.84 vs. Mn = 170.98ms, SD= 58.94; $t(13) = -1.31$, $p = 0.213$, Cohen's $d = -0.219$, observed power = 0.118).

Bayesian analysis of experiments 2 and 3, using the mean difference between conditions in experiment 1 (half-normal distribution, 1-tailed), was consistent with weak support for the null hypothesis in experiment 2 (mean diff. = -11.599, SE = 13.314; likelihood of theory = 0.0076, likelihood of null = 0.021, Bayes factor = 0.369) and support for the null in experiment 3 (mean diff. = -

14.124, SE = 11.886; likelihood of theory = 0.0048, likelihood of null = 0.0166, Bayes factor = 0.291). This suggests that SSRT does not differ across muscle contractions and relaxations.

Figure 2

Table 2

The race model can be applied to stopping muscle relaxations

Experiment 1 and 2 had a fixed SSD design. In line with the race model, the probability of failing to inhibit a response increased with increasing SSD across movement conditions (Fig. 1. E. and Fig. 2. C.). The effect was strong for the relax ($F(5,60) = 100.937$, $p < 0.001$, $\eta p^2 = 0.89$) and contraction conditions ($F(5,60) = 79.418$, $p < 0.001$, $\eta p^2 = 0.87$) in experiment 1, and for the relax ($F(5,55) = 112.86$, $p < 0.001$, $\eta p^2 = 0.91$) and contract conditions ($F(5,55) = 97.403$, $p < 0.001$, $\eta p^2 = 0.9$) in experiment 2.

Weibull functions showed a high degree of fit to the probability of stopping data (Fig. 1. E. and Fig. 2. C.). In the first experiment, mean adjusted R^2 was 0.90 (SD = 0.13) in the relax condition and 0.81 (SD = 0.18) in the contract condition. In experiment 2, R^2 was 0.89 (SD = 0.15) in the relax condition and 0.84 (SD = 0.24) in the contract condition. The shape of these SD curves was similar across movement conditions. Mean Weibull β parameter

value were 4.23 (SD = 1.95) for the relax condition and 3.34 (SD = 1.48) for the contract conditions in experiment 1 (t (12) = 1.73, p = 0.11, Cohen's d = 0.51), and in experiment 2 were 4.35 (SD = 1.43) for the relax condition and 3.97 (SD = 1.6) for the contract condition (t (11) = 0.88, p = 0.4, Cohen's d = 0.25).

Experiment 3 used a variable SSD design. Despite independent staircasing, the probability of moving (i.e. failed stop trials/total stop trials) did not differ between relax (M = 0.48, SD = 0.04) and contract conditions (M = 0.47, SD = 0.05; t (13) = 1.325, p = 0.21, Cohens d = 0.17). Mean SSD was also similar for muscle relaxations (M = 427.02ms, SD = 55.48ms) and contractions (M = 438.25ms, SD = 59.97ms; t (13) = -0.77, p = 0.45, Cohens d = -0.19).

As predicted by the race model, mean response times were significantly faster on failed stop trials than on go trials across all three experiments. This held for the relax (t (12) = 5.83, p < 0.001, Cohens d = 1.22) and contract conditions (t (12) = 6.69, p < 0.001, Cohens d = 1.15) in experiment 1, for the relax (t (11) = 6.62, p < 0.001, Cohens d = 0.9) and contract conditions (t (11) = 5.22, p < 0.001, Cohens d = 1.21). in experiment 2, and for the relax (t (13) = 10.58, p < 0.001, Cohens d = 1.58) and contract conditions (t (13) = 8.4, p < 0.001, Cohens d = 1.79) in experiment 3.

Figure 3

Table 3

Relax SSRT and contract SSRT positively correlated

We observed a positive correlation between relax SSRT and contract SSRT in all three experiments. In experiment 1 ($r = 0.62$, $p = 0.024$; Fig. 1. F.) and experiment 3 ($r = 0.82$, $p < 0.001$; Fig. 5. C.) the results were significant, while in experiment 2 ($r = 0.55$, $p = 0.061$; Fig. 3. D.) they only showed a trend towards significance.

Stopping involves active breaking

In all experiments significant active breaking was observed at the group level for all conditions. Experiment 1 compared the stopping of elbow extensions caused either by relaxation of the biceps (relax condition) or contraction of the triceps (contract condition; Fig. 1. G-I.). Elbow acceleration showed positive active breaking (flexion direction) when relaxations were successfully ($t(12) = 6.903$, $p < 0.001$, Cohen's $d = 1.91$; Fig. 2. A.) and unsuccessfully ($t(12) = 6.012$, $p < 0.001$, Cohen's $d = 1.67$; Fig. 2. B.) stopped. Biceps EMG velocity showed positive active breaking earlier in these same successful ($t(12) = 6.862$, $p < 0.001$, Cohen's $d = 1.9$; Fig. 2. C.) and failed stop trials ($t(12) = 5.125$, $p < 0.001$, Cohen's $d = 1.42$; Fig. 2. D.). We also tested active breaking at the individual subject level (1-tailed). All participants (13/13) showed significant active breaking for kinematics and EMG on successful and failed stop trials in the relax condition.

In the contract condition, active breaking (flexion direction) was observed when examining elbow acceleration on successful ($t(12) = 6.168$, $p < 0.001$, Cohen's $d = 1.71$; Fig. 2. A.) and failed ($t(12) = 7.188$, $p < 0.001$, Cohen's $d =$

1.99; Fig. 2. B.) stop trials. On failed stop trials, when the triceps was already contracting, negative active breaking was observed ($t(12) = -5.812$, $p < 0.001$, Cohen's $d = -1.61$; Fig. 2. F.). Conversely, on successful stop trials, when the biceps muscle was relaxing, but the triceps had typically not begun to contract (see table 1.), positive active breaking was observed ($t(12) = 3.167$, $p = 0.008$, Cohen's $d = 0.88$; Fig. 2. E.). Positive triceps EMG velocity increases on successful stop trials could therefore have been contaminated with 'go response', since both involved signal increase. This was judged unlikely given the onset timing and shape of the triceps response, which was more closely related to the stop-signal across participants. After positive active breaking, a negative dip was observed (Fig. 2. E., red line), indicative of a bi-phasic response, but also of later voluntary response components. As such, we analysed only the first stop-related components of the signal in each condition. Active breaking as defined above could be detected at the individual participant level in 10/13 participants from the kinematics and 8/13 from the EMG on successful stop trials. For failed stop trials it reached threshold ($p < 0.1$) in 12/13 participants for kinematics and 13/13 for triceps EMG.

In experiment 2 we compared the stopping of biceps relaxations (elbow extension) and contractions (elbow flexion; Fig. 3. E-G.). In the relax condition the results replicated those seen in experiment 1. Positive active breaking was observed from the kinematics for successful ($t(11) = 6.04$, $p < 0.001$, Cohen's $d = 1.74$; Fig. 4. A.) and failed stops ($t(11) = 5.447$, $p < 0.001$, Cohen's $d = 1.57$; Fig. 4. B.) and from the biceps EMG velocity for successful ($t(11) = 6.56$, $p < 0.001$, Cohen's $d = 1.89$; Fig. 4. C.) and failed stops ($t(11) = 4.994$, $p < 0.001$,

Cohen's d = 1.44; Fig. 4. D.). In the contraction condition, negative active breaking was observed from the kinematics for successful ($t(11) = -5.614$, $p < 0.001$, Cohen's d = -1.62; Fig. 4. A.) and failed stops ($t(11) = -8.261$, $p < 0.001$, Cohen's d = -2.38; Fig. 4. B.) and from the biceps EMG velocity for successful ($t(11) = -6.037$, $p < 0.001$, Cohen's d = -1.74; Fig. 4. C.) and failed stops ($t(11) = -6.51$, $p < 0.001$, Cohen's d = -1.88; Fig. 4. D.). At the individual participant level active breaking could again be detected in the majority of cases for the relax condition (kinematics: succ. = 10/12, fail = 12/12, EMG: succ. = 11/12, fail = 12/12) and for the contract condition (kinematics: succ. = 9/12, fail = 12/12, EMG: succ. = 10/12, fail = 12/12).

Experiment 3 had participants stopping isometric relaxations and contractions of the FDI muscle (Fig. 5. D-E). Positive active breaking was found in the relax condition when examining force velocity on successful ($t(13) = 6.972$, $p < 0.001$, Cohen's d = 1.86; Fig. 6. A.) and failed stop trials ($t(13) = 6.742$, $p < 0.001$, Cohen's d = 1.8; Fig. 6. B.), and when examining FDI EMG velocity for successful ($t(13) = 8.139$, $p < 0.001$, Cohen's d = 2.18; Fig. 6. C.) and failed stop trials ($t(13) = 4.936$, $p < 0.001$, Cohen's d = 1.32; Fig. 6. D.). In the contract condition, we found negative active breaking for force velocity on successful ($t(13) =-6.923$, $p < 0.001$, Cohen's d = -1.85; Fig. 6. A.) and failed stop trials ($t(13) = -6.524$, $p < 0.001$, Cohen's d = -1.74; Fig. 6. B.), and for FDI EMG velocity on successful ($t(13) = -7.656$, $p < 0.001$, Cohen's d = -2.05; Fig. 6. C.) and failed stop trials ($t(13) = -4.306$, $p < 0.001$, Cohen's d = -1.15; Fig. 6. D.). Active breaking was also detected in the majority of cases at the individual participant level, both for the relax (Force: succ. = 13/14, fail = 13/14, EMG:

succ. = 13/14, fail = 14/14) and contract conditions (Force: succ. = 13/14, fail = 14/14, EMG: succ. = 14/14, fail = 14/14).

Figure 4

Table 4

Active breaking is muscle state specific

In experiment 1 and 2 we observed active breaking of secondary muscles. In both cases for the relax condition the triceps was considered the secondary muscle, showing significant positive active breaking for successful stop trials ($t(7) = 4.587$, $p = 0.003$, Cohen's $d = 1.62$; Fig. 2. E.) and failed stop trials ($t(7) = 4.96$, $p = 0.002$, Cohen's $d = 1.75$; Fig. 2. F.) in experiment 1 and significant positive active breaking for successful stop trials ($t(6) = 3.901$, $p = 0.008$, Cohen's $d = 1.47$; Fig. 4. E.) and failed stop trials ($t(6) = 2.547$, $p = 0.045$, Cohen's $d = 0.96$; Fig. 4. F.) in experiment 2. In experiment 1, the biceps muscle was the secondary muscle in the contract condition and significant positive active breaking was found on successful ($t(7) = 4.01$, $p = 0.005$, Cohen's $d = 1.42$; Fig. 2. C.) and failed stop trials ($t(7) = 5.125$, $p < 0.001$, Cohen's $d = 1.91$; Fig. 2. D.). Here the active breaking was positive because the biceps muscle was relaxing in response to the go signal. Conversely, in the contract condition in experiment 2, the triceps (secondary muscle) tended to contract in response to the go signal, presumably to stabilise the elbow during

607 rapid flexion. As such, active breaking was significantly negative at the group
608 level on successful ($t(10) = -4.482$, $p = 0.001$, Cohen's $d = -1.35$; Fig. 4. E.) and
609 failed stop trials ($t(10) = -2.826$, $p = 0.018$, Cohen's $d = -0.85$; Fig. 4. F.).

610 In experiment 1, 8/8 participants showed significant (1-tailed) triceps
611 active breaking in the relax condition on successful stop trials and 7/8 showed
612 significant active breaking on failed stop trials, at the individual participant level
613 (1-tailed). For the contract condition, 7/8 showed significant biceps active
614 breaking on successful stop trials and 8/8 showed significant active breaking on
615 failed stop trials. In experiment 2, 5/7 participants showed significant triceps
616 active breaking in the relax condition on successful stop trials and 6/7 showed
617 significant active breaking on failed stop trials. For the contract condition 8/11
618 showed significant triceps active breaking on successful stop trials and 11/11
619 showed significant active breaking on failed stop trials.

620 Thus, with one exception (exp.1 contract triceps condition, successful
621 stop trials; *see discussion*), in all experiment and conditions we found that
622 muscles that relaxed in response to the go signal showed positive active
623 breaking after the stop signal, while muscles that contracted in response to the
624 go signal showed negative active breaking.

625

626 **Active breaking onset time correlated with SSRT**

627 If active breaking is causally involved in stopping its onset time should be
628 positively correlated with SSRT across participants. In experiment 1 (Fig. 2. K.
629 & L.), for the relax condition there was a moderate positive relationship between
630 kinematic active breaking onset time and SSRT ($r = 0.54$, $p = 0.054$) and

between biceps EMG active breaking onset time and SSRT (r = 0.5, p = 0.085), both of which reached significance (1-tailed). We did not observe significant correlations in the contract condition, either for kinematic active breaking and SSRT (r = 0.11, p = 0.714) or triceps EMG active breaking and SSRT (r = 0.13, p =0.6712).

In experiment 2 (Fig. 4. K. & L.), while relax condition kinematic active breaking did not correlate with SSRT (r = 0.2, p = 0.523), biceps EMG active breaking onset time did correlate significantly with SSRT (r = 0.66, p = 0.02). Moreover, in the contract condition there was a significant correlation between kinematic active breaking onset time and SSRT (r = 0.73, p = 0.007), and a trend towards a significant correlation between biceps EMG active breaking and SSRT (r = 0.46, p = 0.131).

Finally, in experiment 3 (Fig. 6. I. & J.) all measures of active breaking onset time significantly correlated with SSRT. This held for force (r = 0.67, p = 0.0088) and EMG (r = 0.67, p = 0.0082) in the relax condition and for force (r = 83, p < 0.001) and EMG (r = 0.7, p = 0.005) in the contract condition. Thus overall we found support for a relationship between active breaking onset time and SSRT for muscle relaxations, but weaker support for this relationship when stopping muscle contractions.

Figure 5

Table 5

Active breaking in the absence of detectable Go response

Splitting the successful stop trials into quartiles based on the amount of putative 'go response' present revealed that even when the arm kinematics or finger force level were flat, active breaking was still detected after the stop signal. This was particularly true in the relax condition, where EMG velocity in the fourth quartile (least go response) was significantly positive in experiment 1 ($t(12) = 3.347$, $p = 0.0058$, Cohen's $d = 0.93$; Fig. 2. G. & I.), experiment 2 ($t(11) = 2.370$, $p = 0.037$, Cohen's $d = 0.68$; Fig. 4. G. & I.) and experiment 3 ($t(13) = 3.564$, $p = 0.0035$, Cohen's $d = 0.95$; Fig. 6. E. & G.). For muscle contractions, EMG velocity in the fourth quartile was significantly negative in experiment 3 ($t(13) = -3.23$, $p = 0.0066$, Cohen's $d = 0.86$; Fig. 6. F. & H.), was not significant in experiment 2 ($t(11) = -0.5$, $p = 0.626$, Cohen's $d = 0.14$; Fig. 4. H. & J.) and showed a trend towards being significant in experiment 1 ($t(12) = 1.958$, $p = 0.074$, Cohen's $d = 0.54$; Fig. 2. H. & J).

Active breaking amplitude decreased as Go response decreased

The quartile analysis showed that amount of active breaking decreased as the amount of detected 'go response' decreased. This was true for the relax ($F(3,48) = 10.361$, $p < 0.001$, $\eta p^2 = 0.61$; Fig. 2. I.) and contract ($F(3,48) = 8.142$, $p < 0.001$, $\eta p^2 = 0.4$; Fig. 2. J.) conditions in experiment 1, the relax ($F(3,44) = 4.734$, $p = 0.007$, $\eta p^2 = 0.3$; Fig. 4. I.) and contract ($F(3,44) = 9.9$, $p < 0.001$, $\eta p^2 = 0.47$; Fig. 4. J.) conditions in experiment 2, and the contract ($F(3,52) = 10.393$, $p < 0.001$, $\eta p^2 = 0.44$; Fig. 6. H.) condition in experiment 3. The

relax condition in experiment 3 had active breaking which did not systematically differ across quartiles ($F(3,52) = 1.328$, $p = 0.279$, $\eta p^2 = 0.09$; Fig. 6. G.). Overall, the results indicate that active breaking amplitude was proportional to the degree of go response at the muscle.

Comparing active breaking in relax and contract conditions

EMG active breaking onset times were around 80-100ms across experiments (Table 2., 4. and 6.). A notable exception were successful stop trials in the contract condition experiment 2, where negative active breaking onset times were around 140ms for both muscles (Table 4.). Direct comparison shows that active breaking onset times for the biceps muscle were significantly slower on successful than on failed stop trials (144.29ms vs 91.42ms; $t(11) = 3.62$, $p = 0.004$, Cohen's $d = 1.51$). However, the comparison is not strictly appropriate, since we did not observe active breaking on all successful stop trials when the data was split into quartiles (Fig. 4. J.). As such, we did not compare active breaking across movement conditions in experiment 1 and 2. The comparison was justified in experiment 3, where active breaking was significant in all cases, but no significant difference was observed when mean onset times were compared between relax and contract conditions (94.41ms vs 97.88ms; $t(13) = -0.732$, $p = 0.477$, Cohen's $d = -0.18$).

Active breaking involves simultaneous action across muscles

In experiment 1 active breaking was detected on average 18ms earlier in the biceps than the triceps muscle ($t(7)) = -3.88$, $p = 0.006$) in the relax condition. However, when the entire active breaking waveform was compared in

both muscles via time-shifted cross-correlation, strong positive correlation was observed peaking at -6ms (mean peak r = 0.83, SD = 0.09; Fig. 2. M.). This suggests that the biceps may have contracted just before the triceps, but that both muscles were likely driven by a single input, rather than the biceps activity causing the triceps activity. This was confirmed in the other conditions. In the contract condition in experiment 1 the mean onset of the triceps and biceps active breaking did not significantly differ ($t(7) = 0.764$, $p = 0.47$). Cross-correlation was weaker than in the relax condition (mean peak r = 0.56, SD = 0.17), but the peak correlation was again close to 0 (2.5ms; Fig. 2. M.), indicating simultaneous action. Moreover, in experiment 2 biceps and triceps active breaking onset times did not differ from one another in the relax ($t(6) = -1.124$, $p = 0.304$) or contract conditions ($t(10) = -0.149$, $p = 0.884$) and cross-correlation suggested simultaneous action in both cases (relax peak = 1ms, r = 0.69, SD = 0.22; contract peak = -3ms , r = 0.61, SD = 0.26; Fig. 4. M.). Thus, regardless of whether active breaking was positive or negative, our results suggest it occurred simultaneously across the observed muscles in each condition.

Figure 6

Table 6

Discussion

Muscle relaxations were inhibited as rapidly as muscle contractions across all experiments. Our behavioural results were consistent with the race model, which argues that stop and go processes separately race to completion (Logan and Cowan, 1984). On this account successful stopping will become less frequent as the delay between go and stop signal increases (Logan, 1994) because increasing the delay gives the go process more time to finish. This was confirmed in experiment 1 and 2. The resulting SSD functions were similar to those obtained in previous studies (Logan et al., 1984; Schall et al., 2017). In all conditions tested, mean response times for unsuccessful stop trials were significantly faster than those for go trials. If there had been prolonged interaction between stop and go processes, then we would have observed the opposite result as the presence of the stop signal would slow the go response. But our results were consistent with stop/go independence. Failed stop trials were faster because for a given probability of stopping (e.g. 0.5), the unsuccessful stop trials must be drawn predominantly from the left side (faster 50% of responses) of the RT distribution, while the go trial RT reflects the mean of the entire distribution.

We found SSRTs on the order of 150-300ms for muscle relaxation and muscle contraction conditions, consistent with previous literature (Atsma et al., 2018; Verbruggen and Logan, 2008). Existing work had suggested shared GABAergic control in M1 might underpin the initiation of muscle relaxation and the stopping of actions (Buccolieri et al., 2004; Coxon et al., 2006; Kato et al., 2019; Motawar et al., 2012). However, we found that relax SSRT did not differ from contract SSRT. This lack of interference-related slowing in the relax

760 condition argues against shared GABAergic control. Other pathways are known
761 to be involved in action inhibition. Of particular importance is the fronto-striatal
762 hyperdirect pathway, which is involved in the reactive breaking of action (Aron
763 and Poldrack, 2006; Aron et al., 2003). A basal ganglia account of stopping
764 maps well onto the race model and can explain the inhibition of diverse
765 movement such as saccades, vocalizations, reaching, grip force and classic
766 finger movements (de Jong et al., 1990; Logan and Irwin, 2000; Hanes and
767 Carpenter, 1999; Mirabella et al., 2006; Xue et al., 2008; Bissett and Logan,
768 2012). Our finding, that relax SSRT and contract SSRT significantly correlated
769 across participants, argues for a shared control mechanism in the stopping of
770 relaxations and contractions, possibly involving this basal ganglia circuitry.

771 Action inhibition is known to involve muscle excitation as well as
772 suppression. During the cancellation of reaching, increases in motor unit firing
773 have been recorded from the pectoralis muscle (Atsma et al., 2018). Such
774 responses are termed active breaking because they involve additional input to
775 the muscle which opposes the go response (Kudo and Ohtsuki, 1998;
776 Goonetilleke et al., 2010, 2012). As predicted, we found evidence that this
777 mechanism operates during the stopping of muscle relaxations. Positive active
778 breaking was consistently found across all three experiments both at the group
779 and individual participant level for failed and successful stop trials. These
780 transient increases in surface EMG could be detected even on the subset of
781 successful stop trials where there was no detectable go signal (i.e.
782 kinematics/force was flat). Moreover, in all experiments we found a significant
783 positive correlation between the onset time of EMG active breaking and SSRT

784 in the relax condition. Active breaking may play a causative role in the stopping
785 of muscle relaxations, akin to the theorised peripheral stopping system, which
786 counters unwanted activity that escapes the motor cortex (De Jong et al., 1995;
787 de Jong et al., 1990).

788 We also found transient *decreases* in EMG when muscle contractions
789 were cancelled. EMG decreases are expected when the excitatory drive to the
790 muscle is interrupted, and the resulting partial responses have been reported
791 during the stop signal task (de Jong et al., 1990; McGarry and Franks, 1997;
792 McGarry et al., 2000). However, our analysis showed that EMG did not simply
793 cease to increase on these stop trials, rather, the velocity of EMG changed
794 direction and quickly became significantly negative, suggesting a suppressive
795 input to the muscle (Coxon et al., 2006). Again the effect was detectable at the
796 group and individual subject level across tasks. We therefore propose that this
797 form of response be termed *negative* active breaking. The relationship between
798 negative active breaking onset time and SSRT was less consistent than that
799 seen for positive active breaking, with correlations being significant in
800 experiment 3, but only showing a trend in experiment 2. Negative active
801 breaking onset times did not differ from positive active breaking times in
802 experiment 3, where a direct comparison was justified due to active breaking
803 being present under all conditions, consistent with the similar SSRTs observed
804 across conditions. However, negative active breaking was noticeably slower in
805 experiment 2 for successful stop trials, perhaps because proximal muscles,
806 unlike distal muscles, only show negative active breaking when the muscle is
807 actively shortening, due to control differences (Serrien and Baeyens, 2017).

However, we cannot rule out other explanations such as increased onset variability at the trial-level.

Active breaking was muscle state specific. If the go command caused the muscle to relax then active breaking was positive, while if the go command caused contraction then active breaking was negative. However, in the contract condition in experiment 1 we found negative active breaking on failed stop trials and positive active breaking on successful stop trials. This was likely because the biceps muscle began to relax significantly earlier in relation to the go signal than the triceps started to contract; an EMG-RT difference that has previously been observed (Buccolieri et al., 2003). Consequently, stop commands on successful trials tended to be executed before the triceps began to contract, but as the relax command was starting to be initiated. The critical factor determining active breaking direction therefore may be whether a relaxation command is present centrally rather than at the muscle itself. Under such conditions the observed active breaking response may be a mix of positive and negative forms, perhaps explaining the lack of a significant correlation with SSRT in experiment 1.

Active breaking was previously observed in antagonist muscles (Atsma et al., 2018). It was therefore unclear if the excitatory signal originates centrally or from the agonist muscle. We found active breaking in the triceps and biceps muscle in all conditions, with independently derived onset times that were similar regardless of the type of go command. When we took the entire active breaking EMG waveform for each muscle and performed time-shifted cross correlation, we found that in all conditions there was a positive correlation and

the peak was close to 0ms. So activity in one muscle did not predict the activity of the other at some time-delay. Rather, both muscles were likely driven by central signals and acted simultaneously.

Simultaneous positive active breaking functionally resembles co-contraction, in in that it prevents unwanted movement by increasing joint stiffness (Osu and Gomi, 1999). However, when active breaking was concurrently negative in one muscle and positive in the other, the analogy with co-contraction is less clear. Positive and negative active breaking may involve a common cortico-striatal pathway, but diverge afterwards. Negative active breaking likely results from activity in the striatal hyperdirect pathway suddenly cutting excitatory drive to M1 (Aron et al., 2007), coupled with downstream inhibitory activity in M1 (Badry et al., 2009; Coxon et al., 2006). Reactive inhibition of this sort has a strong and generalised suppressive effect on action (Aron, 2011), which accounts for why we observed reductions of baseline FDI EMG in the absence of a go command and why negative active breaking amplitude scaled with the current level of excitatory drive to the muscle. Importantly, we show that this cutting of excitatory drive does not extend to the positive active breaking signal, since both mechanisms can be observed on the same trials. Positive active breaking may therefore involve a separate descending pathway, perhaps driven by connections from the striatum to sub-cortical postural control regions (Mena-Segovia and Bolam, 2017; Takakusaki, 2017) or gain increases in the gamma motoneuron system (Johansson et al., 1986).

Conclusion

The cancelling of voluntary muscle relaxations was rapid, conformed to the predictions of the race model, and correlated with the time taken to cancel muscle contractions, suggesting shared high-level control. But in the periphery we found evidence for a later divergence of control. Stopping relaxing muscles involved transient increases in muscle activity, while stopping contractions involved transient decreases in muscle activity. This active breaking correlated with SSRT and occurred simultaneously in the biceps and triceps, indicating a central origin. Negative active breaking, though known to have a general suppressive effect on activity, did not prevent positive active breaking in other, already relaxing muscles, suggesting the two forms of active breaking rely on separate pathways. The automatic coordination of positive and negative active breaking across muscle groups may explain how animals can rapidly inhibit complex actions at different stages of their execution.

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Tables and Figures

Table 1: Response times for experiment 1. Table shows group mean (SD) behavioural response times for go trials and response times for onset of muscle activity changes on go trials. Both muscles were relaxing in the relax condition, but in the contract condition the triceps muscle was contracting and the biceps muscle was relaxing. Behavioural response times on stop trials where movement occurred are also shown along with stop signal reaction time (SSRT).

Condition	Go Trial RT	Go Trial Biceps EMG-RT	Go Trial Triceps EMG-RT	Failed stop trial RT	SSRT
Relax	554.74ms (54.21ms)	311.01ms (32.3ms)	390.61ms (90.3ms)	497.02ms (39.44ms)	220.46ms (49.42ms)
Contract	521.28ms (62.02ms)	331.05ms (51.09ms)	417.18ms (59.17ms)	461.27ms (39.74ms)	238.51ms (40.09ms)

Table 2: Active breaking onset times exp. 1. Mean (SD) onset time of active breaking are shown for arm acceleration and primary and secondary muscles. Active breaking onset was calculated separately for each measure, in each movement condition, for successful and failed stop trials. This was done at the individual participant level. Mean stop trial onset refers to the average of successful and failed stop trial onset times. Secondary muscle data (triceps in relax condition, biceps in contract condition) could only be analyzed in a subset of participants. Note that triceps active breaking in the contract condition is labelled mixed because it was positive on successful and negative on failed stop trials (see text for details).

Movement condition	Type of active breaking	Succ. stop trial onset	Failed stop trial onset	Mean stop trial onset
Relax	Positive arm acceleration	193.23ms (32.92ms)	200ms (26.98ms)	196.62ms (27.27ms)
	Positive biceps EMG velocity	83.12ms (22.04ms)	82.42ms (19.74ms)	82.77ms (18.64ms)
	Positive triceps EMG velocity (n = 8)	86.31ms (14.84ms)	94.69ms (28.31ms)	90.5ms (17.14ms)
Contract	Positive arm acceleration	199.38ms (27.37ms)	166.15ms (37.08ms)	182.77ms (22.44ms)
	Mixed triceps EMG velocity	92.62ms (30.97ms)	85.85ms (25.98ms)	89.23ms (22.5ms)
	Positive biceps EMG velocity (n = 8)	92.94ms (32.23ms)	95.81ms (24.31ms)	94.38ms (26.08ms)

Table 3: Response times for experiment 2. Table shows group mean (SD) behavioural response times for go trials and response times for onset of muscle activity changes on go trials. Both muscles were relaxing in the relax condition and contracting in the contract condition. Behavioural response times on stop trials where movement occurred are also shown along with stop signal reaction time (SSRT).

Condition	Go Trial RT	Go Trial Biceps EMG-RT	Go Trial Triceps EMG-RT	Failed stop trial RT	SSRT

Relax	588.36ms (56.51ms)	331.76ms (36.9ms)	416.21ms (77.43ms)	543.98ms (41.17.ms)	268.1ms (48.31ms)
Contract	549.82ms (42.18ms)	437.79ms (33.97ms)	440.08ms (32.84ms)	504.46ms (32.47ms)	256.5ms (31.69.ms)

Table 4: Active breaking onset times exp. 2. Mean (SD) onset time of active breaking are shown for arm acceleration and primary and secondary muscles. Active breaking onset was calculated separately for each measure, in each movement condition, for successful and failed stop trials. This was done at the individual participant level. Mean stop trial onset refers to the average of successful and failed stop trial onset times. Secondary muscle data (triceps) could only be analyzed in a subset of participants in the relax condition and one participant was removed from the triceps analysis in the contract condition (see text for details).

Movement condition	Type of active breaking	Succ. stop trial onset	Failed stop trial onset	Mean stop trial onset
Relax	Positive arm acceleration	204.33ms (29.42ms)	211.33ms (29.39ms)	207.83ms (24.16ms)
	Positive biceps EMG velocity	67.25ms (12.59ms)	86.96ms (26.46ms)	77.1ms (16.13ms)
	Positive triceps EMG velocity (n = 7)	91ms (34.87ms)	87.79ms (27.47ms)	89.39ms (25.7ms)
Contract	Negative arm acceleration	260.67ms (37.56ms)	193.33ms (28.51ms)	227ms (29.15ms)
	Negative biceps EMG velocity	144.29ms (38.84ms)	91.42ms (30.71ms)	117.85ms (24.18ms)
	Negative triceps EMG velocity (n =11)	140.73ms (42.77ms)	96.64ms (26.63ms)	118.68ms (18.14ms)

Table 5: Response times for exp. 3. Group mean (SD) behavioural response time for go trials, go trial FDI EMG response time, failed stop trials behavioural response time, and stop signal reaction time (SSRT) in the relax and contract condition.

Condition	Go Trial RT	Go Trial FDI EMG-RT	Failed stop trial RT	SSRT
Relax	598.01ms (54.8ms)	424.29ms (47.98ms)	511.11ms (55.52ms)	170.98ms (58.94ms)

Contract	595.11ms (63.59ms)	445.21ms (52.95ms)	489.73ms (53.79ms)	156.86ms (69.84ms)

Table 6: Active breaking onset times exp. 3. Mean (SD) onset time of active breaking are shown for force velocity and FDI EMG velocity. Active breaking onset was calculated separately for each measure, in each movement condition, for successful and failed stop trials. This was done at the individual participant level. Mean stop trial onset refers to the average of successful and failed stop trial onset times.

Movement condition	Type of active breaking	Succ. stop trial onset	Failed stop trial onset	Mean stop trial onset
Relax	Positive force velocity	177.04ms (75.94ms)	212.79ms (62.58ms)	194.91ms (66.01ms)
	Positive FDI EMG velocity	88.21ms (23.85ms)	100.61ms (23.34ms)	94.41ms (20.05ms)
Contract	Negative force velocity	184.07ms (78.34ms)	199.54ms (29.61ms)	191.8ms (51.49ms)
	Negative FDI EMG velocity	99.04ms (14.92ms)	96.71ms (28.54ms)	97.88ms (19.42ms)

Figure 1. Design and results for experiment 1. Task set-up is shown **(A).** In Relax and Contract blocks, participants made elbow extensions in response to go signals and attempted to prevent these movements whenever a stop signal occurred. Elbow extensions were driven by biceps relaxation or triceps activation respectively, as shown in the representative go trials from the Relax **(B)** and Contract **(C)** conditions. Group mean relax and contract condition EMG and kinematic responses to Go trials **(D).** SSD functions for the relax and contract condition **(E)**, showing how the probability of failing to stop on a stop trial increased as SSD increased. SSRT

in relax and contract blocks significantly correlated (r = 0.62, p = 0.024) across participants **(F)**. Group mean relax and contract condition kinematics **(G)**, biceps EMG **(H)**, and triceps EMG **(I)**, for successful and failed stop trials, time-locked to the stop signal. Upper panels show smoothed data, while lower panels show angle acceleration and EMG velocity traces. All error bars are SEM.

Figure 2. Active breaking results for experiment 1. Group mean kinematic active breaking results for successful **(A)** and failed stop trials **(B)** in the relax and contract conditions. Successful stop trials also plotted on the failed stop trial graph for comparison. Traces always aligned to active breaking onset times derived at the individual participant level. Inserts show mean values obtained during the 50ms shaded window immediately after active breaking onset. Group mean biceps EMG active breaking results for successful **(C)** and failed **(D)** stop trials for the relax and contract conditions. Active breaking was significantly positive for all conditions. Group mean triceps EMG active breaking results for successful **(E)** and failed **(F)** stop trials. Active breaking was significantly positive in the relax condition and significantly negative for failed stop trials in the contract condition. But for successful stop trials it was significantly positive (see text for details). Successful stop trial kinematics in the relax **(G)** and contract **(H)** conditions after trials were sorted into quartiles according to the amount of Go response. Positive biceps EMG active breaking was present in the relax condition even when the kinematics were flat **(I)**. A trend was observed for the triceps in the contract condition **(J).** Active breaking amplitude decreased significantly across bins in both cases, as detected go response decreased. Positive correlations between kinematic **(K)** and EMG **(L)** active breaking onset times and SSRT reached significance in the relax, but not the contract condition. Time-shifted cross correlation **(M)** showed that peak correlation between biceps and triceps active breaking was close to 0ms in both conditions, indicating simultaneous action. All error bars show SEM. Results of statistical tests shown as asterisks (*** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$).

Figure 3. Design and results for experiment 2. On relax go trials participants extended the elbow by relaxing the biceps and on contract go trials they flexed the elbow by further

contracting the biceps. They attempted to stop these movements if a stop signal was present **(A)**. Group mean relax and contract condition EMG and kinematic responses to Go trials **(B).** SSD functions for the relax and contract condition **(C)**, showing how the probability of failing to stop on a stop trial increased as SSD increased. SSRT in relax and contract conditions showed a positive trend ($r = 0.55$, $p = 0.061$) across participants **(D)**. Group mean relax and contract condition kinematics **(E)**, biceps EMG **(F)**, and triceps EMG **(G)**, for successful and failed stop trials, time-locked to the stop signal. Upper panels show smoothed data, while lower panels show angle acceleration and EMG velocity traces. All error bars are SEM.

Figure 4. Active breaking results for experiment 2. Group mean kinematic active breaking results for successful **(A)** and failed stop trials **(B)** in the relax and contract conditions. Successful stop trials also plotted on the failed stop trial graph for comparison. Inserts show mean values obtained during the 50ms window immediately after active breaking onset. Group mean biceps EMG active breaking results for successful **(C)** and failed **(D)** stop trials, and mean triceps EMG active breaking results for successful **(E)** and failed **(F)** stop trials. Active breaking was always significantly positive in the relax condition and significantly negative in the contract condition. Successful stop trial kinematics in the relax **(G)** and contract **(H)** conditions after sorting by amount of detected Go response. Positive biceps EMG active breaking was present in the relax condition even when the kinematics were flat **(I)**. In the contract condition **(J)** negative active breaking was not significant in B4. Active breaking amplitude decreased significantly across bins in the relax and contract conditions. Positive correlations between kinematic **(K)** and EMG **(L)** active breaking onset times and SSRT reached significance in the relax condition EMG, but only showed a trend in other cases. Time-shifted cross correlation **(M)** showed that peak correlation between biceps and triceps active breaking was close to 0ms in both conditions, indicating simultaneous action. All error bars show SEM. Results of statistical tests shown as asterisks (*** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$).

Figure 5. Design and results for experiment 3. On relax go trials participants reduced force by relaxing the FDI and on contract go trials they increased force by further contracting the FDI.

1167 They attempted to stop these force changes if a stop signal was present **(A)**. Group mean relax
1168 and contract condition EMG and force responses to Go trials **(B).** SSRT in relax and contract
1169 blocks showed a significant positive correlation ($r = 0.82$, $p < 0.001$) across participants **(D)**.
1170 Group mean relax and contract condition force **(E)** and FDI EMG **(F)** for successful and failed
1171 stop trials, time-locked to the stop signal. Upper panels show smoothed data, while lower panels
1172 show velocity traces. Error bars show SEM.
1173
1174 **Figure 6. Active breaking results for experiment 3.** Group mean force active breaking results
1175 for successful **(A)** and failed stop trials **(B)** in the relax and contract conditions. Inserts show
1176 mean values obtained during the 50ms window immediately after active breaking onset. Group
1177 mean FDI EMG active breaking results for successful **(C)** and failed **(D)** stop trials. Active
1178 breaking was always significantly positive in the relax condition and significantly negative in the
1179 contract condition. Successful stop trial force in the relax **(E)** and contract **(F)** conditions after
1180 sorting by amount of detected Go response. Positive FDI EMG active breaking was present in
1181 the relax condition even when the force traces were flat **(G)** and negative active breaking was
1182 present in the contract condition when force traces were flat **(H)**. Active breaking amplitude
1183 decreased significantly across bins in the contract, but not relax condition. Significant positive
1184 correlations were found between force **(I)** and EMG **(J)** active breaking onset times and SSRT in
1185 the relax and contract conditions. All error bars show SEM. Results of statistical tests shown as
1186 asterisks (*** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$).