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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.

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, Kurtzer, & Scott, 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

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the muscle (De Havas, Ghosh, Gomi, & Haggard, 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.

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

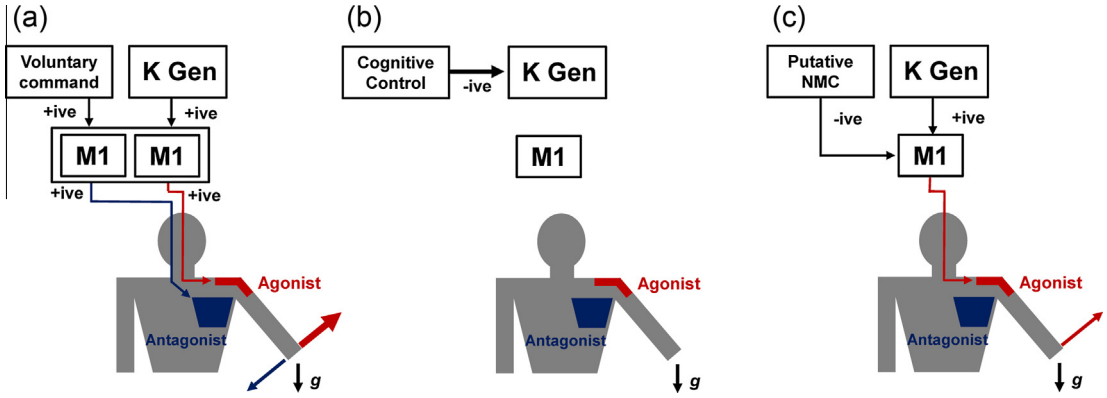

Fig. 1. Possible mechanisms for aftercontraction inhibition. Theoretically the arm could be stopped from moving by activation of the antagonist muscle (a). Motor drive to the muscle could be cut by cognitive control circuits 'switching off' the Kohnstamm generator (b). If this was total the arm would 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 Section 4 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.