# Principles of involuntary vs. voluntary control of human action: investigations using the Kohnstamm phenomenon

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I, Jack De Havas, confirm that the work presented in this thesis is my own[1]. Any information derived from other sources is fully cited and referenced in the thesis.

Signed,

Jack De Havas

1. Experiments reported in chapter 3 of this thesis have been already been published as: De Havas, J. A., Ghosh, A., Gomi, H., & Haggard, P. (2015). Sensorimotor organization of a sustained involuntary movement. *Frontiers in Behavioral Neuroscience*, *9*(185).

**Abstract**

Psychological accounts of human action control strongly distinguish between voluntary and involuntary movements. In the Kohnstamm phenomenon, a sustained voluntary contraction of a muscle is followed by sustained, involuntary aftercontraction of the same muscle. This offers a useful experimental model of the voluntary/involuntary distinction, because aftercontractions physically resemble voluntary movements, while feeling subjectively very different. Despite 100 years of study, many basic questions remain unanswered about the Kohnstamm phenomenon. This thesis presents several experiments addressing these questions, and using the phenomenon to shed light on the voluntary/involuntary distinction. First, the recruitment of the Kohnstamm generator was explored by systematically varying the muscle contractions and task goal during the initial voluntary activity that induces the Kohnstamm phenomenon. This revealed that the Kohnstamm generator is a low frequency integrator. Next, experiments on physical obstruction of the involuntarily rising arm showed that afferent input can temporarily gate output from the Kohnstamm generator. Subjective estimates of contact force against the obstacle were higher than for matched voluntary movements, suggesting that the generator does not produce efference copies. In a further experiment, resistive and assistive perturbations during a horizontal Kohnstamm aftercontraction produced EMG responses, consistent with principles of negative position feedback control operating during voluntary movements, but with lower gains. Experiments in which participants were instructed to inhibit the aftercontraction showed that, though involuntary, Kohnstamm movements could nevertheless be voluntarily controlled, suggesting the novel concept of a “negative motor command”. Such voluntary inhibition caused a strange subjective experience of upward force, again suggesting a lack of efference copy for the aftercontraction. A model is presented that shows how the Kohnstamm phenomenon is generated and controlled. This systematic study of the control principles of the Kohnstamm phenomenon sheds important new light on the classical distinction between involuntary and voluntary movement.

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## Contents

## List of figures

## List of tables

## Chapter 1. The Kohnstamm phenomenon: an introduction

**The Kohnstamm phenomenon refers to the observation that if one pushes hard outward against a fixed surface with the back of the hand for approximately 30 s and then ceases, an abduction of the arm will occur, accompanied by a feeling that the movement is involuntary and the arm lighter than usual. A full review of the published literature reveals that central, peripheral and hybrid theories of the Kohnstamm phenomenon have been advanced. The role of afferent signalling in establishing and controlling this involuntary aftercontraction has been of great interest, yet many questions remain unanswered. Afferent signals may be irrelevant if purely central theories are correct. Alternatively, according to peripheral accounts, unusual afferent signalling may actually drive the involuntary aftercontraction. Hybrid theories suggest afferent signals control the aftercontraction via negative position feedback control or positive force feedback control. Contrasts with voluntary movement have often been made, particularly with respect to the subjective experience of the aftercontraction and the question of whether involuntary movements can be brought under voluntary control. The Kohnstamm phenomenon has been studied because it provides a novel tool to explore sensorimotor physiology. In addition, it may clarify the nature of voluntariness by allowing comparisons between voluntary and involuntary movements. It retains enduring scientific interest because it offers a strange example of a prolonged, co-ordinated action that just happens, contrasting with the intuition that we voluntarily control our own actions. Indeed, the Kohnstamm phenomenon raises questions of automaticity versus autonomy that remain central to the neuroscientific study of human nature.**

### 1.1. Description of Kohnstamm phenomenon and literature

#### 1.1.1. What is the Kohnstamm phenomenon?

The Kohnstamm phenomenon (Fig. 1.1.), as originally described, refers to the observation that if one pushes hard outward against a fixed surface with the back of the hand for approximately 30 s and then ceases, an abduction of the arm will occur, accompanied by a feeling that the movement is involuntary and the arm lighter than usual (Kohnstamm, 1915; Salmon, 1915). When pre-screening is not used, the Kohnstamm phenomenon is reported in about 75% of healthy participants (Adamson & McDonagh, 2004; Duclos, Roll, Kavounoudias, & Roll, 2007; Hagbarth & Nordin, 1998; Ivanenko, Wright, Gurfinkel, Horak, & Cordo, 2006). It is not known why some individuals do not display the effect, although general anxiety towards the experimental environment is likely a factor (Craske & Craske, 1985). Researchers have noted large individual differences in how easily the aftercontraction can be elicited, and when it is, differences in movement speed and amplitude (Adamson & McDonagh, 2004; Kohnstamm, 1915; Salmon, 1916, 1925). Early work claimed that the Kohnstamm phenomenon displays uniformity across sessions in healthy individuals (Allen, 1937), though this has not been verified statistically.

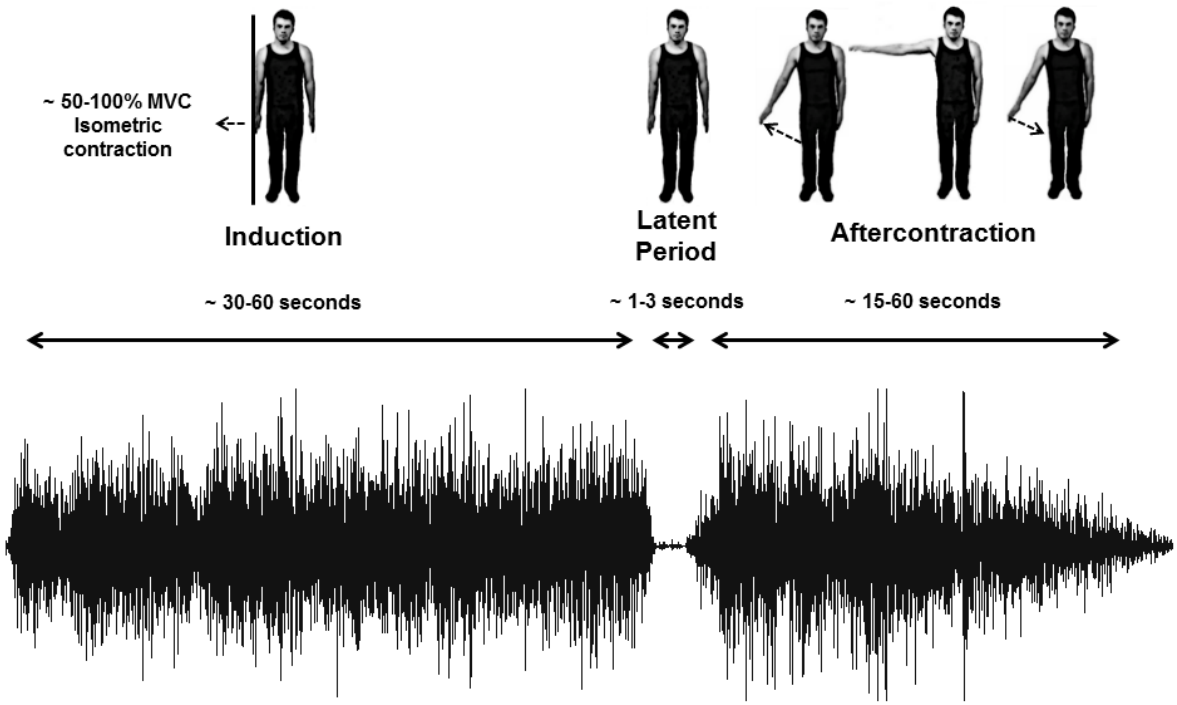


**Figure 1.1.** ***The Kohnstamm phenomenon****: basic kinematics, average duration and a typical EMG trace from the deltoid muscle.*

While most studies utilise the deltoid muscle (Adamson & McDonagh, 2004; Fessard & Tournay, 1949; Kohnstamm, 1915; Pinkhof, 1922; Salmon, 1915, 1916), it has always been known that the Kohnstamm phenomenon can be easily demonstrated in many muscles including flexors and extensors of the arm, wrist, ankle, knee, hip and also the neck muscles (Allen & O'Donoghue, 1927; Csiky, 1915; Forbes, Baird, & Hopkins, 1926). Indeed, it has been suggested that an aftercontraction can be elicited from any skeletal muscle providing a suitable induction exists (Forbes et al., 1926) and early work documented the aftercontractions in 20 different muscles within the same individual (Matthaei, 1924b). However, it was also reported that the Kohnstamm phenomenon is hardest to produce in the muscles of the hand (Matthaei, 1924b). Recently, it has been found that aftercontractions emerge more clearly in proximal joint muscles compared to the muscles of distal parts of the limb (Gregory, Morgan, & Proske, 1988; Gurfinkel, Levik, & Lebedev, 1989). Traditionally the Kohnstamm phenomenon is studied in the context of a single muscle. Co-contraction of antagonistic muscles such as the biceps and triceps does not produce any aftercontraction (Gilhodes, Gurfinkel, &

Roll, 1992). However, with specific complex movements of the axial muscles, aftercontraction activity is found simultaneously in antagonistic muscles (Ghafouri, Thullier, Gurfinkel, & Lestienne, 1998). Pushing the legs together for extended periods of time can produce involuntary air stepping (Selionov et al., 2013; Selionov, Ivanenko, Solopova, & Gurfinkel, 2009), demonstrating that complex muscle synergies can be recruited.

In all previous studies, the aftercontraction is elicited via an isometric muscle contraction. This can be achieved by pushing against a solid surface (Kohnstamm, 1915) or holding a fixed amount of weight stationary out from the body (e.g. Sapirstein, Herman, and Wallace 1937). Even small amounts of force, requiring just 10% of the muscle's maximum voluntary contraction (MVC), maintained for 10 s, are adequate in some individuals (Allen & O'Donoghue, 1927). However, to induce a robust effect across participants most paradigms involve 50-100% MVC for durations of 30-60 s. It is possible to generate the effect with the muscle at a variety of lengths during the induction (Forbes et al., 1926; Hagbarth & Nordin, 1998).

After cessation of the voluntary contraction there is a latent period. The muscle is not active and the limb is stationary (Gurfinkel et al., 1989; Kozhina, Person, Popov, Smetanin, & Shlikov, 1996). The duration of this period varies across participants, but on average lasts 1-3 s (Csiky, 1915; Kozhina et al., 1996; Meigal, Lupandin, & Hanninen, 1996; Parkinson & McDonagh, 2006; Pinkhof, 1922; Sapirstein et al., 1937). Typically, participants are instructed to relax to trigger the aftercontraction (Sapirstein, Herman, and Wallace 1937; Mathis, Gurfinkel, and Struppler 1996; Ghafouri et al. 1998). However, it is unknown what signals are necessary to trigger the aftercontraction beyond the cessation of the voluntary contraction. Instruction to relax may result in smaller aftercontractions relative to maintaining normal posture (Hick, 1953). However, this observation has not been statistically verified.

The aftercontraction phase of the Kohnstamm phenomenon causes a movement of the limb in the direction of the induction force. In the deltoid it is routinely reported that in many individuals the arm abducts to the maximum 90° (Adamson & McDonagh, 2004; Kohnstamm, 1915; Salmon, 1916). There is high variability across protocols, but typically the aftercontraction duration is in the range of 10-60 s (Sapirstein, Herman, and Wallace 1937; Gurfinkel, Levik, and Lebedev 1989; Parkinson, McDonagh, and Vidyasagar 2009), though in one experiment

postural effects were detected for up to 14 minutes (Duclos, Roll, Kavounoudias, & Roll, 2004). The end of the aftercontraction is poorly defined. With some participants (Matthaei 1924b; Sapirstein, Herman, and Wallace 1937) or protocols (Craske & Craske, 1985; Forbes et al., 1926) it naturally takes on an oscillatory character. However, in most cases the arm is brought down from a statically abducted position either by instruction or by the voluntary decision to adopt a new posture. Subjective feeling of lightness may be the best way to gauge the true duration of the aftercontraction (Cratty & Duffy, 1969).

### 1.1.2. Why study the Kohnstamm phenomenon?

The Kohnstamm phenomenon has been reported in the literature for 100 years. It has likely been known about for much longer (Pereira, 1925a) and may be considered a folk illusion (Barker & Rice, 2012). General interest in the phenomenon is due to the ease with which the effect can be demonstrated, the accompanying strange sensation, the surprised reaction it evokes in those experiencing it for the first time, and the associated pleasure that comes from both its performance and the passing of 'secret' knowledge in a social context (Barker & Rice, 2012). However, the Kohnstamm phenomenon is not merely a parlour trick. Early researchers understood the physiological and psychological insights that could be gained from its study. It was central to resolving a long-standing debate about the possibility of muscle contractions without action currents (Forbes et al., 1926; Pereira, 1925a; Pinkhof, 1922; Salmon, 1925; Salomonson, 1921; Schwartz, 1924; Schwartz & Meyer, 1921). After years of sporadic study, scientific interest in the Kohnstamm phenomenon began to increase from the late 1980s to the present day. However, many questions remain regarding its cognitive control. Advances in the understanding of motor control (Bizzi, Accornero, Chapple, & Hogan, 1984; Marsden, Merton, & Morton, 1976a) and the neurocognitive basis of the sense of agency (Blakemore & Frith, 2003; Haggard, 2008; Shergill, Bays, Frith, & Wolpert, 2003; Wolpert & Kawato, 1998), mean there is now a strong theoretical context in which to interpret findings from Kohnstamm experiments. The phenomenon's status as something of an isolated oddity should not prevent vigorous study. Researchers have long drawn the analogy with visual illusions (Fessard & Tournay, 1949; Salmon, 1916, 1925), themselves once considered just games, but now recognised as a key source of knowledge about the mechanisms of visual perception. Similarly,

the Kohnstamm phenomenon may provide important insights into the fundamental nature of voluntary and involuntary movement control.

Comparison between voluntary and involuntary movements is clearly an important tool to study volition. Involuntary movements provide a novel way to dissect these questions, but are usually difficult to study. Isolating the motor commands of reflexes, and determining how they contribute to action awareness is difficult, because of their rapid onset, short duration and close interaction with afferent signals (Ghosh and Haggard, 2014). The Kohnstamm phenomenon does not suffer from this problem. It is the speed of a slow voluntary movement, meaning that it can be perturbed, and the physiological consequences recorded. The quality of being physically indistinguishable from a voluntary movement, yet subjectively entirely different, makes the Kohnstamm phenomenon an attractive tool to study how these two components of movement are linked. The results of such experiments will elucidate both voluntary and involuntary movement. They may also help to explain where the Kohnstamm phenomenon fits within the range of reflexive, postural and voluntary motor control. Furthermore, by contrasting voluntary motor control and Kohnstamm movements, important questions about the inhibition of existing movements can be addressed.

### 1.1.3. Previous Literature

The Kohnstamm phenomenon has also been referred to as the *Katatonusversuch* (Kohnstamm, 1915), *after movement* (Csiky, 1915), *residual contraction* (Pinkhof, 1922), *Salmon-Kohnstamm phenomenon* (Henriques & Lindhard, 1921), *automatic movement* (Salmon, 1925), *automatic contraction* (Pereira, 1925a), *involuntary contraction* (Forbes et al., 1926), *post-contraction* (Allen, 1937) and *aftercontraction* (Sapirstein, Herman, and Wallace 1937). Literature for the following review was obtained by searching Pubmed and Web of Science using the above search terms. Once all listed studies had been found, additional papers were located by examining the reference lists of all papers. For the purposes of clarity, in this review the term Kohnstamm phenomenon will be used to refer to the entire effect, while individual stages will be referred to as Induction, Latent period and Aftercontraction. Papers are only included in the table if they are peer reviewed, present original research data, and focus on involuntary aftercontraction.

***Table 1.1. All previous original research on the Kohnstamm phenomenon***. *Papers are listed in chronological order. AC = aftercontraction.*

| Reference | Techniques used | n | % had AC | Muscles studied | Induction method | Induction strength | Induction duration | Latent period | Size of AC | Duration of AC | Subjective reports | Key findings |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| (Salmon, 1915) | Observation only. | No report | No report | Lateral deltoid, bicep, thigh, anterior flexion of trunk, neck extensors | Push hard outwards against experimenters arms or hard surface | No report | No report | No report | No report | No report | Lightness, surprise | 1) First report of the AC, which is found in most participants. AC size not strongly dependent on induction strength/duration.<br>2) Easier to elicit in emotionally reactive people.<br>3) AC is stronger in patients with hysteria, absent in schizophrenia, more pronounced in Parkinsons disease, present in Tabes Dorsalis, absent in hemiplegia. |
| (Kohnstamm, 1915) | Faradic stimulation. | No report | No report | Lateral deltoid and leg muscles (no specific details) | Pressing backs of hands against wall with high tension | No report | 5-60 s | No report | up to 120° | No report | Mysterious force, strange, flying | 1) Independent discovery of phenomenon. Size of AC depends on the individual and duration of push.<br>2) Faradic stimulation does not produce AC.<br>3) Diminished in cases of Tabes Dorsalis, lacking in people with negativistic personality type, very strong in hypnotised people. |
| (Rothmann, 1915) | Observation only. | No report | No report | Lateral deltoid, pectoralis, wrist extensors, neck muscles | Pressing backs of hands against wall with high tension | No report | 5-60 s | No report | No report | No report | Surprise, involuntary, automatic. | 1) AC restricted to the extensor muscles.<br>2) Found in Tabes Dorsalis, absent in Hemiplegia, absent in patient with Cerebellar damage. |
| (Csiky, 1915) | Observation only. | No report | No report | Lateral deltoid, extensors and flexors of arms and legs. | Pressing backs of hands against wall with high tension | No report | 30-60 s | 2-3 s | No report | 12-15 s | Strange feeling, involuntary. | 1) First to time and define separate phases of Kohnstamm phenomenon (induction, latent period, AC).<br>2) AC found in both flexor and extensor muscles.<br>3) AC found in some participants after 1min of intense faradic stimulation. |
| (Salmon, 1916) | Observation only. | No report | No report | Lateral deltoid, knee, arm and neck extensors | Push hard outwards against experimenters arms or hard surface | No report | No report | No report | No report | No report | Lightness, flying | 1) AC more common in emotional people. AC is stronger in patients with hysteria, absent in dementia, more pronounced in Parkinsons disease, present in Tabes Dorsalis, absent in hemiplegia.<br>2) No clear relationships between tendon reflex strength and AC across participants. |
| (Salomonson, 1921) | EMG (string galvanometer). | No report | No report | Lateral deltoid, hand extensors | Isometric contraction of deltoid against rigid surface | max effort | 60 s | No report | No report | 1-10 s | Arm drawn upwards without, or even against will. | 1) AC less pronounced in old and apathetic or subjects with early dementia.<br>2) No electrical activity in muscle detected during AC. |
| (Danielopolu, Radovici, & Carniol, 1921) | Kinematics (no methodology), injection of caffeine to muscle. | No report | No report | Lateral deltoid, biceps, back, neck trunk and leg muscles. | Hold heavy weight or push hard | No report | 10-15 s | No report | No report | No report | No report | 1) AC exists for all voluntary muscles, contraction must be isometric**.**<br>2) AC highly diminished with repeated inductions (fatigue).<br>3) Absent AC (deltoid and bicep) in 1 patient with myasthenia gravis, 4 patients with cachexia, but occurred after injection of caffeine. |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|
| (Henriques & Lindhard, 1921) | EMG (string galvanometer), faradization of muscle. | No report | No report | Lateral deltoid | Pushing against solid surface, leaning with body weight. | No report | No report | No report | > 45° | No report | No report | 1) Muscle activity at all stages of the Kohnstamm phenomenon (one trace shown, not clear).<br>2) Leaning with body weight (supposedly no contraction) produced 45 ° AC. Not present if a cushion used.<br>3) Faradization (1 min) produced small AC. |
| (Schwartz & Meyer, 1921) | EMG (string galvanometer; no traces shown). | No report | No report | Lateral deltoid | Push against solid surface | max effort | 10-12 s | No report | 90° | No report | Surprise, foreign force independent of will | 1) Electrical activity in muscle present throughout AC (even when arm stationary at 90 deg).<br>2) Similar to activity seen during voluntary action. |
| (Pinkhof, 1921) | EMG (string galvanometer), kinematics (air tyre surrounding body). | 4 | No report | Lateral deltoid, bicep, wrist extensors | Push against solid object or hold weight | 5 kg | 60 s | 2 s | No report | up to 30 s | Like passive movement, flying, weightless, like in water, slight pressure on underside of arm | 1) Action currents present during AC for biceps & deltoid, all cases (20 cases from 4 participants).<br>2) Action currents during AC same intensity and frequency as those of voluntary movements.<br>3) Muscle is silent during latent period (1-2 s) |
| (Pinkhof, 1922) | EMG (string galvanometer), kinematics (air tyre surrounding body), electrical stimulation. | 4 | No report | Lateral deltoid, bicep, wrist extensors | Push against solid object or hold weight | 5 kg | 60 s | 2 s | No report | up to 30 s | Like passive movement, flying, weightless, like in water, slight pressure on underside of arm | 1) Reflexes (from electrical stimulation) produced after inductions were similar to those during voluntary contraction.<br>2) Re-reported the results of earlier paper (Pinkhof, 1921). |
| (Matthaei, 1924b) | Spring to measure weight of arm during AC, Faradization. | > 40 | 100% | Deltoid, biceps, triceps, hand extensors, quadriceps, psoas, gluteus, hamstrings, hip. | Pushing outward on padded surface, weights for other muscles. | up to 5 kg | 10-120 s | < 1-10 s | up to 90° | 30-60 s | Lightness, passivity, pulled upwards, moves by itself, flight, like a dream. Heaviness at end. | 1) AC can be induced in any skeletal muscle, rarely in the hand. AC manifest in direction of contraction of muscle, not direction of force.<br>2) Size of AC (distance moved by arm) depended on intensity/duration of induction.<br>3) Alcohol ingestion increases AC size, injecting novocaine in shoulder removes subjective feeling of lightness, but AC unaffected |
| (Matthaei, 1924a) | Early form of strain gauge | 28 | No report | Biceps | Holding suspended weight with arm bent. | 0.5-5 kg | 5-120 s | No report | No report | No report | Lightness | 1) Found a logarithmic relationship between induction intensity and size of subjective force overestimation, indicated via voluntary movement of other arm.<br>2) Magnitude of error does not depend on the voluntary hand. |
| (Pereira, 1925a) | EMG (string galvanometer; cathode amplification). | No report | No report | Lateral deltoid | Hard push against wall | No report | 60 s | No report | ~ 90° | No report | No report | 1) Electrical muscle activity not detected when arm reached max position during AC and was stationary. Seen only during movement. Obstruction and voluntary inhibition caused action currents to stop, but muscle was still contracting.<br>2) Rapid voluntary contraction, immediately after induction prevented AC. |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|
| (Salmon, 1925). | Observation, Faradic stimulation. | No report | No report | Lateral deltoid | Resisting the force exerted on the arms by experimenter. | No report | No report | No report | No report | No report | Feeling of automaticity, limb lighter, flying | 1) AC more pronounced in emotional subjects, subjects with hysteria and subjects gifted with a very vivid imagination (sometimes produced by just mental imagery).<br>2) Faradic stimulation produced only very weak AC.<br>3) Decreased AC in 2 patients with Tabes Dorsalis, decreased AC on affected side in 2 patients with hemiplegia. |
| (Verzár & Kovács, 1925) | EMG (string galvanometer; steel needle electrodes). | 15 | 93.33% | Biceps | Hold weight with bent arm (90° angle relative to the upper arm) | 5 kg | 60-90 s | No report | Up to 120° | No report | No report | 1) Action currents during AC with 10-20% fewer waves per second than during voluntary movement (no way to exactly match velocity).<br>2) Muscle cooling (ice pack 15 mins) produced ~20% reduction in waves/s during AC and voluntary movement. |
| (Forbes et al., 1926) | EMG (string galvanometer, Kinematics (kymograph). | 7 | 86% | Lateral deltoid, biceps, pectoralis, triceps, wrist flexors, hip, knee, neck. | Seated, push outwards. | ~100% MVC (effort) | 20-25 s (also 60 s) | No report | No report | Up to 25 s | Surprise | 1) EMG signal present throughout AC, similar to matched voluntary movements.<br>2) Obstruction of arm during latent period abolished AC, but obstruction during AC did not reduce muscle activity (arm held in place at obstacle).<br>3) Inhibition of arm possible without use of antagonist muscle, easier at start of movement. |
| (Allen & O'Donoghue, 1927) | Kinematics (protractor). | 4 | No report | Lateral Deltoid & leg muscles | Wire and pulleys, arm away from body, standing | 0.55 - 4.55 kg | 10 s | No report | Up to 100° | No report | Lightness, rise of its own accord, no volition. | 1) Size of AC increases (logarithmically) with induction strength at fixed duration.<br>2) Fatigue with repeated inductions. Augmentation if a 20 min rest was included.<br>3) Other arm fatigue causes reduction in AC, and then augmentation with rest. |
| (Laignel-Lavastine, Chevalier, & Vie, 1927) | Torque device for measuring induction force (no details). | No report | No report | Lateral deltoid | Push against solid surface | 4 kg | 120 s | 0-5 s | 45 to 120° | 9-45 s | No report | 1) AC Abolished in general paralysis caused by syphilis (4 cases), multiple sclerosis (2 cases), early dementia (2 cases) and paranoid dementia (1 case), very decreased for the affected side of hemiplegic patients.<br>2) Very extended AC duration of in Parkinson's disease (10 cases), melancholia (3 cases), myxedema (2 cases), psychiatric patients (hysteria, phobia, schizophrenics, addicts). |
| (Salmon, 1929) | Observation only. | No report | 100% | Lateral deltoid, bicep (arm flexor), knee extensors (quadriceps), neck extensors | Push hard against solid surface | No report | 20-30 s | 1-2 s | No report | No report | Feeling that the arm is lighter than normal, flies | 1) More pronounced AC in Hysteria patients and patients with Parkinsons or morphine addiction.<br>2) Reduced in Hemiplegia, Early dementia, Tabes Dorsalis.<br>3) Latency increases with longer inductions. |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|
| (Sapirstein, Herman, & Wallace, 1936) | Kinematics (Kymograph), administration of drugs. | 60 | No report | Hip flexion | Supporting suspended weight | 6 kg | 15 s | No report | No report | No report | No report | 1) Leg AC markedly reduced after 2 gm sodium bromide (often abolished, despite knee jerk being normal).<br>2) Caffeine (0.15 g) found to increase size of AC. Very effective at offsetting suppression by sodium bromide.<br>3) Other drugs (chloral hydrate, strychnine & barbital) found to have lesser effect |
| (Sapirstein et al., 1937) | Kinematics (Kymograph), administration of drugs. | No report | No report | Hip flexion | Supporting suspended weight | 1-6 kg | 10-25 s | up to 3 s | No report | 3-40 s | No report | 1) Increased strength and duration of induction produces bigger AC.<br>2) Dorsiflexion of the foot increased the size of hip AC. Abducting ipsilateral arm with 2 kg weight caused increase in leg AC. Contralateral arm usually produced decrease, but sometimes produced an increase.<br>3) AC can be prevented by exerting a voluntary force in the other direction at the point of relaxation. If movement is restrained by experimenter at relaxation AC is delayed. |
| (Allen, 1937) | Kinematics (protractor). | 5 | No report | Lateral deltoid & leg muscles | Wire and pulleys, arm away from body, standing. | 0.55 - 4.55 kg | 10-15 s | No report | Up to 75° | No report | Involuntary, detachment, lightness, floating, weight loss. | 1) Bigger and longer induction increases AC size.<br>2) Fatigue reduces AC size.<br>3) Right leg contractions during right arm induction, reduced size of right arm AC. |
| (Holway, Crolius, Pratt, & Zigler, 1937) | Kinematics (protractor), adjustable weight balance. | 3 | No report | Lateral deltoid | Push outwards against weighted balance | 0.02 - 6.4 kg | 15 s | No report | up to 122.8° | No report | No report | 1) Size of AC found to be a power function of force during induction (wide range of forces). |
| (Sapirstein, Herman, & Wechsler, 1938) | Kinematics (Kymograph), administration of drugs. | > 20 | No report | Hip flexion | Supporting suspended weight | 3-6 kg | 15 s | No report | No report | No report | No report | 1) Normal AC found in 10 Tabes Dorsalis patients, small AC found in 2. No correlation between severity of condition and size of AC.<br>2) Prolonged AC in Parkinson's disease, jerky in single case of cerebellar damage.<br>3) AC reduced in hemiplegia on affected side (spinal reflexes hyper-sensitive). |
| (Wells, 1944) | Observation only. | No report | No report | Lateral deltoid, knee extensors | Push outwards against solid surface | No report | 60-120 s | No report | No report | No report | No report | 1) During bilateral AC, turning head to right, or turning eyes strongly to left, or shining strong light into eyes from left, increases AC of right arm and diminishes or abolishes on the left.<br>2) Forceful downward eye rotation or backward tilting of the head increases AC. Opposite (i.e. upward eye rotation etc.) reduces AC.<br>3) Similar pattern observed in knee extensor muscles. |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|
| (Sapirstein 1948) | Kinematics (Kymograph), administration of drugs. | No report | No report | Hip flexion | Supporting suspended weight | 3-6 kg | 16 s | No report | No report | No report | No report | 1) AC is absent in affective psychosis, severe depression, manic depression. AC absent in 3 cases of depression - appeared after electro-shock treatment.<br>2) AC normal in schizophrenia, providing there was no accompanying emotional disturbance.<br>3) Lack of AC linked to anxiety in patients with OCD, phobias and anxious hysteria. |
| (Zigler et al. 1948) | Kinematics (Protractor). | 4 | No report | Lateral deltoid | Pull on cord holding suspended weight | 0.8 - 3.2 kg | 7.5-30 s | No report | No report | No report | No report | 1) Across a range of strength and durations of inductions, size of AC rapidly increased with successive trials and then gradually decreased with fatigue. |
| (Fessard & Tournay, 1949) | EMG (single traces, needle electrodes), kinematics (photo-electric instruments). | 4 | No report | Lateral deltoid, pectoralis. | Arm ~20° abducted push outward. | No report | 5-120 s | 2.7-6.3 s | Up to 70° | 3.5-37 s | Surprise | 1) Duration and amplitude of aftercontraction depend on induction duration of induction.<br>2) Matched voluntary actions show similar EMG. Voluntary movement on top of AC does not abolish AC. Muscular atrophy patient showed same unusual EMG pattern during AC and voluntary movement.<br>3) Adducting (inhibition) does not abolish the Kohnstamm, there are up to 6 spontaneous recoveries. |
| (Paillard, 1951) | Kinematics (mechanogram, potentiometric sliders system). | No report | No report | Lateral deltoid | Pushing outward on solid surface | max effort | 5-30 s | No report | up to 80° | No report | No report | 1) Bilateral AC was smaller (~ 25 deg) than unilateral (~80 deg).<br>2) If AC is prevented in one arm at start of bilateral AC, the other arm rises to the normal angle (~80 deg).<br>3) Fast voluntary upward movement of right arm causes temporary inhibition of a left AC (stronger if a 2 kg weight held). Final arm angle similar to normal AC, after plateau. |
| (Hick, 1953) | Spring to measure force. | 14 | No report | Lateral deltoid | Pushing outwards against spring | up to 3.63 kg | 15 s | No report | No report | No report | No report | 1) Cognitive distractor task (write name backwards) produced bigger AC effect than baseline.<br>2) Voluntary movements (“produce this force”) could be superimposed on top of AC.<br>3) Instruction to maintain 0 force induced more AC force then instruction to relax after induction. |
| (Sapirstein, 1960) | Kinematics and EMG (no data shown). | > 200 | No report | Knee extension, Hip flexion, lateral deltoid | Supporting suspended weight | 7.26 kg | 20 s | No report | No report | No report | No report | 1) Of 200 patients at psychiatric hospital, AC appearance pre-empted improvement, AC loss pre-empted decline in mental health.<br>2) Patients with depression rarely had AC. 17/19 depressed patients had AC only after electro-shock therapy.<br>3) Association between negative emotions and lack of AC. Outward anger did not reduce AC. |
| (Cratty & Duffy, 1969) | Subjective reporting of effect. | 39 | 86% | Lateral deltoid | Standing in constructed doorframe | 100% effort | 5-20 s | No report | No report | Mean 14 s | Arm felt lighter than normal | 1) Duration of Kohnstamm (defined by self-report of subjective feeling of lightness) was not correlated with strength of other aftereffects (e.g. position errors). |

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| (Howard & Anstis, 1974) | Moveable trolley to indicate head position with hands. | 12 | No report | Neck | Resisting suspended weight | 95 gm | 10 min | No report | up to 24° | No report | No report | 1) Pointing accuracy to head position did not differ from baseline during neck AC.<br>2) Pointing accuracy to head position after head turning showed bias to direction of turn (postural persistence). |
| (Craske & Craske, 1985) | Kinematics (receiving microphone). | 55 | No report | Deltoid, triceps, gluteus. | Push against solid surface (various postures) | Max effort (exp. 1), moderate (exp. 2 & 3) | 30 s | No report | 36.35° (median) | median 219.65 s | Surprise, lightness, floating, move of own accord, without decision or intention. | 1) AC has an oscillatory quality (5.5 median no. cycles)<br>2) Simultaneous AC in shoulder and forearm produce oscillations of same frequency (16/20). In phase (6/15), rest in 180° or 90° phase.<br>3) Oscillations could be transferred to an un-induced limb by silently naming the limb. |
| (Craske & Craske, 1986) | Kinematics (receiving microphone). | 52 | No report | Deltoid | Push against solid surface (various postures) | 50% MVC | 30 s | No report | Exp. 1: 9.9°; Exp3: 34.15° | No report | No report | 1) Oscillatory AC can be transferred from inducted arm to other arm by naming the limb.<br>2) Oscillations in right and left arm interact when inductions are in different planes.<br>3) AC (34.15°) can be induced by motor imagery. |
| (Gurfinkel et al., 1989) | EMG, kinematics (mechanogram), vibration, electric stimulation. | 7 | No report | Calf, quadriceps, hand extensors, lateral deltoid, trunk. | Lift weights against gravity | 2-5 kg | 30-60 s | No report | > 30° | 40-50 s | Lightness | 1) Induction with distal muscle sometimes switched to proximal muscle AC. Also is produced by muscle vibration (up to 20mins later).<br>2) Deltoid AC larger in standing versus sitting subjects (even larger if standing on toes).<br>3) Electrical stimulation failed to produce AC. |
| (Gilhodes et al., 1992) | EMG, kinematics, vibration, electronically controlled eye mask. | 14 | 71.43% | Biceps and triceps. | Seated, push against static restraint (arm bent at 95°) | 4-5 kg | 30 s | No report | No report | > 60 s | No report | 1) In darkness eyes opening and closing had no effect, but in diffuse light opening and closing correlated with switch back and forth between muscles (bicep/triceps).<br>2) Muscle switching occurred for both bicep and triceps inductions. Not if co-contracted.<br>3) Same effect achieved via vibration. |
| (Mathis et al., 1996) | EMG, kinematics (potentiometer), TMS, vibration. | 7 | No report | Lateral deltoid | Seated, arm abducted (10-20°) push outwards against counter weight. | 4-6 kg | 40-60 s | No report | 20-72° | No report | No report | 1) MEP size correlated with background EMG level for AC and matched voluntary movements. MEP amplitude, gain, latency and dynamics did not differ. Similar results for vibration induced movement.<br>2) Found bigger MEPs for rising EMG (i.e. muscle shortening) compared to falling EMG in 20% of Vol trials and 30% of AC trials. |
| (Kozhina et al., 1996) | Single motor unit recording (intramuscular needle electrodes), EMG, kinematics (goniometer). | 4 | No report | Lateral deltoid, triceps & anterior tibialis. | Pulling up on handle or pushing out against elastic band. | 50-70% MVC | 40 s | 1.4 s | 30-40° | ~ 10 s | No report | 1) Mean firing rate of motor units significantly lower during AC (12 pps) compared to matched voluntary movements (14 pps).<br>2) Other properties (e.g. spike amplitude) did not differ.<br>3) Firing rate increased with movement. Very low firing rate if movement prevented before AC developed. |

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| (Meigal et al., 1996) | EMG, heating and cooling of entire body | 6 | No report | Biceps and triceps | Flexion of elbow against sold plate | 70% MVC | 60 s | 2-3 s | No report | 1-6 min | No report | 1) Cold air exposure (+5 °C), increased EMG (%MVC) during AC, relative to room temperature (+22 °C). Hot air exposure (+75 °C) decreased AC EMG and duration.<br>2) AC sometimes spontaneous transferred from biceps to triceps. |
| (Hagbarth & Nordin, 1998) | EMG, kinematics, muscle cooling/ heating, vibration. | 14 | 71.43% | Lateral deltoid | Pushing upwards against solid surface, arms at 90° | 0-100% max effort | ~20 s | No report | ~ 10° | ~ 10 s | Lightness, involuntary. | 1) Omission of steps of muscle conditioning procedure (from animal literature to maximise post-contraction afferent discharge) reduced size of AC.<br>2) Warming muscle produced significant decrease in AC size. Cooling produced trend towards increase in AC size.<br>3) AC from vibration same as from contraction. |
| (Ghafouri et al., 1998) | Kinematics (scapula: 3D optical motion analysis), EMG. | 10 | 60% | Trapezius pars descendens & latissimus dorsalis. | Produce isometric contraction against weight attached in shoulder bag. | 8 kg | 360 s | No report | No report | 50-60 s | No report | 1) Greater EMG during standing than sitting AC. Different activity in the two muscles.<br>2) Different direction of spiral unrolling motion of scapula in standing (clockwise) and sitting (anticlockwise).<br>3) Opening eyes after induction triggered AC switch from traps to lats in standing, but not sitting condition. |
| (Brice & McDonagh, 2001) | Force, Kinematics (goniometer). | 6 | No report | Lateral deltoid & leg muscles | Arm 30° abducted, push outward, standing. | 20-100% MVC | 15-75 s | No report | Up to 92° | No report | No report | 1) Threshold induction duration is required to produce AC. Beyond this, magnitude of AC proportional to force generated during induction. |
| (Lemon, Price, & McDonagh, 2003) | EMG, strain gauge, tilt table. | 9 | No report | Lateral deltoid | Pushing outwards against strain gauge. | 60% MVC | 60 s | No report | No report | No report | No report | 1) Mean AC EMG decreased almost linearly from 46.6% MVC when upright to 12.7% MVC when supine. |
| (Adamson & McDonagh, 2004) | Strain gauge, Kinematics (goniometer), EMG, cuffing wrist. | 9 | ~70% | Lateral deltoid | Arm 15-20° abducted push outward, standing. | 100% effort, dropped to 60% by end | 60 s | 1-5 s | Up to 70° | ~ 60 s | No report | 1) AC EMG (%MVC), when arm obstructed, is linearly dependent on joint angle.<br>2) EMG on downward adduction is linearly dependent on position, but lower.<br>3) Changes in EMG not dependent on cutaneous input. |
| (Duclos et al., 2004) | Force, centre of pressure recordings, electrical stimulation. | 14 | No report | Neck muscles (splenius, trapezius, obliques). | Pushing head against differently positioned pads | 50% MVC | 30 s | No report | No report | up to 14 mins. | No report | 1) Immediate, long lasting whole body leaning, specific to muscle contracted.<br>2) Did not occur after electrical stimulation of muscle. |
| (Ivanenko et al., 2006) | Kinematics (Motion tracking cameras), strain gauge for induction. | 21 | 75% | Trunk | Resist a rotational torque applied at the pelvis | 40 Nm (rotational torque) | 30 s | No report | ~ 5° | Up to 40 s | No report | 1) Trunk AC produced curved deviations (10%) in voluntary walking in the direction of induction contraction.<br>2) Did not occur when stepping on the spot. |

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| (Parkinson & McDonagh, 2006) | Kinematics (goniometer), EMG, pivot lever arm with moveable counter-weight. | 10 | No report | Anterior deltoid | Shoulder flexion (40°) seated, pushing upwards on solid surface. | 60% MVC | 60 s | 2-5 s | up to 90° | ~ 60 s | Lightness, movement due to external force. | 1) AC EMG (% of induction) linearly decreased at every arm angle with increased assistive counter-weight (decreased load: 100 - 0%). |
| (Duclos et al., 2007) | fMRI, EMG, vibration. | 11 | No report | Wrist extensors | Push upwards (wrist 10° extended) against solid surface, supine. | 50% MVC | 30 s | No report | Up to 30° | 50 s | No report | 1) AC associated with activity in primary sensory and motor cortices, premotor cortex, anterior and posterior cingulate, parietal regions, insula and vermis of cerebellum.<br>2) Supplementary motor area (BA6) active during voluntary movement, not AC. Cerebellar vermis more active during AC.<br>3) Activation during AC similar to during TVR. |
| (Parkinson et al., 2009) | fMRI, kinematics, EMG (outside scanner). | 11 | No report | Anterior deltoid | Shoulder flexion, pulling upwards on rope attached to body, lying supine. | 100% MVC (effort) | 60 s | 1-2 s | 11.54 cm disp. | ~ 30 s | No report | 1) Widespread cortical and sub-cortical activation during AC (motor cortex, pre- central gyrus, superior parietal, caudate, thalamus, cerebellum).<br>2) Greater activity in supplementary motor area and anterior cingulate during AC than voluntary movement.<br>3) Greater activity in putamen during voluntary movement than during AC |
| (Selionov et al., 2009) | EMG, kinematics (potentiometers, elastic chord to measure force). | 18 | 88.89% | Hip flexor and leg extensor muscles | Supine, legs supported. One leg pushing forward, the other back against each other. | 50% MVC | 30 s | No report | No report | 5-60 s | No report | 1) Observed rhythmic air stepping (forward motion) activity in both legs for about 15 s after induction.<br>2) EMG showed AC in multiple muscles.<br>3) Maximal frequency and amplitude of the hip and knee joint movements occurred after 3–7 cycles. |
| (Meigal & Pis'mennyi, 2009) | EMG, heating and cooling of entire body | 102 | 82% | Lateral deltoid and biceps | Pushing outwards against belt and flexion of elbow against table underside | 50% MVC | 60 s | No report | No report | mean = 60 s, max > 5 mins | No report | 1) Body heating reduced the duration of the biceps AC. Cooling increased biceps AC EMG (% MVC).<br>2) Hot air exposure produced a trend towards increased AC EMG (%MVC) in deltoid. Cooling had no effect.<br>3) 76% of participants had long AC (arm held horizontal), 10 % had rapid AC (arm rose and fell in 30 s), 8% showed oscillatory AC, 8% no AC. |
| (Selionov et al., 2013) | EMG, kinematics (potentiometers, elastic chord to measure force). | 47 (22 controls, 25 patients) | 50% of controls, 4% of patients | Hip flexor and leg extensor muscles | Supine, legs supported. One leg pushing forward, the other back against each other). | 50% MVC | 30 s | No report | No report | 5-60 s | No report | 1) AC air stepping found in 50% of controls, but only 1/25 Parkinsons patients (did not appear after dopaminergic treatment). |

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| (Ghosh, Rothwell, & Haggard, 2014) | EMG, kinematics (LEDs & 60fps camera), TMS (single pulse). | 39 | ~ 70% | Lateral deltoid | Push outwards against solid surface, arms slightly abducted (15°) | 40-60% MVC | 40-60 s | No report | up to 90° | No report | Sense of resistance when voluntarily adducting during AC | 1) TMS to primary motor cortex during AC induces silent period in agonist muscle. Silent period has same latency and duration as during voluntary movement.<br>2) Voluntarily inhibition of AC; bring arm down, then additional ACs without use of antagonist.<br>3) Voluntary inhibition (adduction) associated with stronger subjective feeling of resistance than when no AC present. |
| (Brun et al., 2015) | EMG, kinematics, strain gauge, vibration. | 21 | ~70% | Biceps | Pulling upwards on handle | 40% MVC | 35 s | 1-2 s | ~ 30° | ~ 10 s | No report | 1) Velocity of bicep AC adjusts towards velocity of a passive movement of other arm.<br>2) Velocity of bicep AC adjusts towards increasing velocity of a simulated movement of other arm (increasing vibration frequency: 25-75Hz). |

### 1.1.4. Summary of table

The table identifies 56 original research papers. The most prolific decade for research was the 1920s (15 papers), there was then a steady decline until the 1980s when interest began to increase. The table includes 37 papers written in English, 10 in French, 7 in German, 1 in Italian and 1 in Dutch. The most prolific authors are Victor Gurfinkel (7 papers: 1989-2013), Martin McDonagh (5 papers: 2001-2009), Milton Sapirstein (5 papers: 1936-1960) and Albert Salmon (4 papers: 1915-1929). Research was published from the USA (11 papers), France (9), UK (7), Italy (7), Germany (5), Canada (5), Russia (4), Netherlands (3), Hungary (2), Denmark (1), Switzerland (1), and Sweden (1).

Numbers of participants were not typically reported prior to the 1950s. It is difficult to estimate the mean number of participants included in subsequent studies because some experiments used pre-screening, whilst others did not. Likewise the prevalence of the aftercontraction is skewed by pre-screening, but appears to be 70-80% of healthy participants. Kinematic recording was used in 34 experiments, EMG in 27 experiments, fMRI in 2 experiments and TMS in 2 experiments. The most commonly studied muscle is the deltoid, which was used in 41/56 papers. A variety of methods have been used to induce the aftercontraction, but they all involve isometric contractions and an attempt to maintain a constant force, either against gravity (holding weight) or a fixed surface (pushing). A standard Kohnstamm induction is 40-100% MVC for 20-60 s. Only one study (Kozhina et al., 1996) appears to have reported accurate mean data for the latent period between the end of induction and the onset of aftercontraction. Others report a range, with the general consensus being that the mean is 1-3 s. Little can be concluded about the size and duration of the aftercontraction owing to the wide range of methodologies used and muscles studied. Reports of the mean size and duration of the aftercontraction are surprisingly rare, perhaps because many studies used more than one induction protocol. However, it can be noted that aftercontractions of the deltoid can induce involuntary movements of up to 90°, using a variety of inductions. The typical duration of the aftercontraction appears to be 10-60 s. The percentage of this time involving a moving versus stationary limb varies considerably across individuals. Key findings are discussed below.

## 1.2. Research questions

### 1.2.1. What is happening at the muscle during the Kohnstamm phenomenon?

The muscle itself is the logical starting point for an exploration of the causes of the Kohnstamm phenomenon. Initial work concerned a wholly muscular origin (but see Rothmann, 1915; Salmon, 1915, 1916). Csiky (1915) was the first to time and formally describe the individual phases of the Kohntsamm phenomenon. He noted a close analogy with the optical afterimage. Both were considered by him to be caused by fatigue of the peripheral apparatus. Supporting this muscular theory, high levels of electrical stimulation of the muscle could apparently induce an aftercontraction (Csiky, 1915). However, this was not replicated (Duclos et al., 2004; Gurfinkel et al., 1989; Kohnstamm, 1915; Matthaei, 1924b) and it is likely that the original finding was due to the participants voluntarily contracting against the direction of the powerful shocks (Zigler, 1944). With the availability of the string galvanometer, it became possible to measure innervation of the muscle. Early attempts showed a lack of EMG activity during the aftercontraction (Salomonson, 1921), suggesting muscle tone was maintained without central innervation (Salomonson, 1921). Kohnstamm's (1915) own theory was that the aftercontraction depended on the muscle taking on a new equilibrium point during the 'hard push' and then trying to return to that point. He speculated that muscle tone was normally maintained in this local manner and that it was an inhibition of the voluntary movement signal that actually allowed the arm to move. However, this 'holding back' of the arm is fundamentally incompatible with the characteristic latent period of 2-3 s (Csiky, 1915). Further experiments showed EMG activity during the aftercontraction (Henriques & Lindhard, 1921; Pinkhof, 1921, 1922; Schwartz & Meyer, 1921; Verzár & Kovács, 1925). There was a debate as to whether these were products of the movement itself (Pereira, 1925a, 1925b) or true central innervation (Salmon, 1925), but this was elegantly resolved by showing that they persisted even when the involuntarily rising arm was obstructed (Forbes et al., 1926). Later, modern electromyographic (EMG) recording convincingly showed central motor drive during aftercontraction (Fessard & Tournay, 1949), allowing purely muscular theories to be abandoned.

Central innervation does not preclude changes in the muscle from being the origin of the aftercontraction. This is the basis of the muscle thixotropy hypothesis (Gregory et al., 1988; Hagbarth & Nordin, 1998). Here, the key factor in generating

the Kohnstamm phenomenon is changes in the stiffness and slackness of fusimotor fibres. The theory states that the Kohnstamm phenomenon occurs for the following reasons: 1) Kohnstamm induction is static and muscle length is short (relative to start of aftercontraction), 2) during induction contraction, stable actin and myosin cross bridges form in intrafusal muscle fibers, 3) relaxation causes arm to be brought back to a longer muscle length, 4) stable cross bridges in intrafusal fibers remain, maintaining them in a state of relative shortness, compared to their state following alternative contraction histories (e.g. isotonic), 5) relative shortness in intrafusal muscle fibers causes muscle spindles to be stretched and to send afferent signals. 6). Spindle signalling causes muscular contraction via established motor mechanisms, such as spinal and other stretch reflexes. Hagbarth and Nordin (1998) modified a muscular conditioning sequence (used in animals to enhance resting spindle discharge) to act as a Kohnstamm induction. The sequence involved: 1) participants first holding both arms slightly abducted, 2) actively lifting up their arms against two solid stands and forcefully pressing (max effort) for 5-10 s, 3) relaxing while the experimenter held them up for 4-8 s, and 4) having their arms passively adducted by experimenter to the start position. On each trial the full procedure was performed on one arm, while on the other arm one of the steps would be systematically omitted. The procedure was found to produce a small aftercontraction with a mean angular displacement of 8°. Omitting any of the steps produced a significant decline in the amount of angular displacement, suggesting the aftercontraction was largest when a procedure was used that maximised the maintenance of shortness and stiffness in the fusimotor fibres. So, for example, omitting the step that involved passive holding of the muscle at maximum abduction for 4-8 s, purportedly reduced the aftercontraction because it reduced the gradual formation of stable cross-bridges. Replacing the slow, passive adduction with a fast movement purportedly reduced the aftercontraction because it disrupted the existing stable cross-bridges. The aftercontractions were much smaller than typically seen during a deltoid Kohnstamm (Adamson & McDonagh, 2004; Brice & McDonagh, 2001; Fessard & Tournay, 1949; Laignel-Lavastine et al., 1927; Matthaei, 1924b; Paillard, 1951; Pereira, 1925a; Schwartz & Meyer, 1921; Verzár & Kovács, 1925). Thus, subjectively imperceptible voluntary movements may have contributed to the effect (knowledge of the complexity of the induction may have set up an expectation of movement size). However, Hagbarth and Nordin (1998) also found that heating

the muscle by 3-4°C significantly decreased aftercontraction size, while cooling by the same amount produced a trend towards an increase. This result is commensurate with the thixotropy hypothesis. Muscle temperature may increase (heating) or decrease (cooling) the effects of Brownian motion on the weak physico-chemical bonds that form the actin-myosin cross-bridges (Edwards et al., 1972; Lakie, Walsh, & Wright, 1984, 1986; Sekihara et al., 2007). Indeed, significant whole-body heating and cooling effects on the size of the EMG response during aftercontraction (Meigal et al., 1996) were reported. Muscle cooling was also reported to reduce the frequency of muscle activity during the aftercontraction (Verzár & Kovács, 1925). Interestingly, recent evidence suggests the effects of heating and cooling on the Kohnstamm phenomenon may be more complex. Aftercontraction in the biceps was significantly increased by whole body cooling, and tended to decrease with whole body heating (Meigal & Pis'mennyi, 2009). Conversely, in the deltoid muscle, whole body cooling had no effect, while heating resulted in a larger aftercontraction.

Thixotropic changes might be epiphenomenal. Whether the muscle itself is the origin of the Kohnstamm phenomenon depends on the spindle discharge being high enough to generate a sufficiently strong and sustained 'reflex response'. In the cat, resting discharge of 60% of muscle spindles has been found to be significantly increased for up to 15 minutes following electrically induced contraction (Hutton, Smith, & Eldred, 1973). Similar results have been obtained following isometric contraction (Suzuki & Hutton, 1976). There is also some supporting microneurographic evidence in humans. Short periods of isometric contraction of the ankle (5 s) produce 65% increases in spindle firing rates, lasting up to 52 s (Wilson, Gandevia, & Burke, 1995). Other human research is less commensurate with the animal work, finding that fewer than 15% of primary spindles show any post-contraction sensory discharge, and that this discharge never exceeds 40 s in duration (Ribot-Ciscar, Rossi-Durand, & Roll, 1998; Ribot-Ciscar, Tardy-Gervet, Vedel, & Roll, 1991). Increased spindle firing rates are abolished by stretching of the muscle (Wilson et al., 1995). Observations involving obstructing the aftercontraction (Forbes et al., 1926), adducting against the aftercontraction (Fessard & Tournay, 1949; Ghosh et al., 2014), and tapping the tendon during aftercontraction (Gurfinkel et al., 1989), suggest that introducing stretch to the muscle does not eliminate the Kohnstamm phenomenon. However, this has not been properly tested. Finally, the

deltoid aftercontraction was observed to be still present after novocaine (20 cc., 1% solution) was injected into the muscle (Matthaei, 1924b). The extent of the afferent block was not established so the interpretation is limited. Indeed, many questions remain regarding what afferent signals reach the brain during the Kohnstamm phenomenon, and whether these afferent signals are sufficient to drive the efferent motor command that lifts the arm.

### 1.2.2. What sensory signals are coming to the brain?

Other, non-muscular afferent signals interacting with the central nervous system may explain the origin of the Kohnstamm phenomenon. Cutaneous signals from the dorsum of the arm during induction were proposed as a cause (Henriques & Lindhard, 1921), but can be dismissed due to numerous experiments using suspended weights to elicit the isometric contraction and subsequent aftercontraction (Allen, 1937; Allen & O'Donoghue, 1927; Ghafouri et al., 1998; Pinkhof, 1922; Sapirstein et al., 1937). Afferent signals from the muscle spindles have received more support (Forbes et al., 1926; Matthaei, 1924b; Pinkhof, 1922; Schwartz, 1924; Schwartz & Meyer, 1921; Zigler, 1944). Theoretically, this afferent signal would drive the aftercontraction by: a) establishing central adaptations during the induction, b) altering continuous reflex loops with central regions during the aftercontraction, or c) a combination of both. Evidence for the role of afferent signals in the Kohnstamm phenomenon comes from its similarity to the Tonic vibration reflex (TVR). The TVR is induced by vibrating the muscle tendon at 80-100Hz for around 30 s, causing the activation of muscle spindles (Duclos et al., 2007; Gilhodes et al., 1992; Mathis et al., 1996). This produces an involuntary contraction of the muscle, resulting in a similar kinematic and EMG profile to the Kohnstamm phenomenon (Gilhodes et al., 1992; Mathis et al., 1996), along with overlapping activations in the cortex (Duclos et al., 2007), and the elicitation of comparable descriptions of the subjective experience (Hagbarth & Nordin, 1998). If the Kohnstamm phenomenon and TVR are the same phenomenon, it would follow that afferent signals from muscle spindles are the common origin (although signals from Golgi tendon organs could not be completely dismissed). However, there have been no experiments attempting to dissociate the Kohnstamm phenomenon and TVR. Establishing if this afferent signal is necessary for the Kohnstamm phenomenon, though important,

does not reveal what central mechanisms in the spinal cord or brain may underlie the generation of the aftercontraction.

Determining what afferent signals reach the cortex during the aftercontraction can be tested via position sense of the limb. It is known that isometric contractions and changes attributed to muscle thixotropy alter position sense (Tsay, Savage, Allen, & Proske, 2014). However, it has also been found that sustained, isometric contractions do not reduce pointing accuracy during a voluntary movement (Heide & Molbech, 1973), although they do reduce the participant's confidence in their responses. Moreover, while postural persistence (turning the head to the right for 10 minutes) produces a bias in position sense, this was not found after inducing a neck turning aftercontraction (Howard & Anstis, 1974). Indeed, positional after-effects have been reported to be unrelated to the Kohnstamm phenomenon in terms of how their duration varies across individuals (Cratty & Duffy, 1969). Thus, there is some evidence that afferent signals from the involuntarily contracting muscle are processed in the cortex not as purely peripheral sensory events, but as corollaries of voluntary action.

To determine what sensory signals reach the brain during the Kohnstamm phenomenon it is especially informative to explore how sensory inputs affect the aftercontraction. Contractions from other muscles in the body can alter the aftercontraction. Concurrent voluntary dorsiflexion of the foot and weighted ipsilateral arm inductions have been seen to increase the size of hip aftercontractions (Sapirstein et al., 1937). Paillard (1951) reported that bilateral aftercontractions of the lateral deltoid were smaller than those that were unilateral. EMG was not recorded in any of these studies, making it impossible to know if the activity of the agonist muscle was constant across conditions. However, recent studies have found that despite matched inductions (forces and duration), sitting and lying supine are associated with significantly reduced aftercontraction of the deltoid muscle relative to standing (Ghafouri et al., 1998; Lemon et al., 2003). These findings could all be explained by efference-related changes in central regions.

Contrastingly, a few notable experiments have employed purely sensory perturbations. Building on the surprising finding that the aftercontraction sometimes transfers from one muscle to another (Craske & Craske, 1985, 1986; Gurfinkel et al., 1989), it has been found that this switching can be triggered by visual input. By having participants position their arm so that both extension and flexion was

possible, it was demonstrated that under diffuse light conditions (but not darkness) opening and closing the eyes led to the aftercontraction switching from the biceps to the triceps and vice versa in 10/14 participants tested (Gilhodes et al., 1992). The effect was also shown in the same participants for the TVR. EMG recordings showed that switching was not due to muscle activity during induction. Further work has confirmed visually induced switching in other muscle groups (Ghafouri et al., 1998). Integration of ascending sensory signals may occur in tonigenic sub-cortical structures such as the reticular formation (Gurfinkel et al., 1989), which is known to be strongly activated by visual input (Mori, Nishimura, & Aoki, 1980). However, cortical accounts cannot be ruled out. The basis of these remarkable effects is not fully understood. Such results may appear like auto-suggestion or experimenter effects. However, spontaneous muscle switching has been independently replicated (Meigal et al., 1996). Further, shining strong light into participant's eyes from the left has been shown to reduce a right arm aftercontraction (during bilateral aftercontractions), while shining light from the right reduces the left arm aftercontraction (Wells, 1944).

It is not clear how afferent input from the muscle influences the aftercontraction. Proprioceptive input in the form of tendon vibration applied to the ipsilateral arm can increase the velocity of a contralateral aftercontraction (Brun et al., 2015). Additionally, reducing the weight of the arm using a counterweight was found to reduce EMG during the aftercontraction (Parkinson & McDonagh, 2006). This effect may be due to reduced afferent discharge from Golgi tendon organs (GTO) or lower spindle firing due to reduced arm velocity. On that view, the control of the Kohnstamm movement would involve a putative positive feedback loop linking GTO discharge to α motor neuron drive, or the established negative feedback loop linking spindle discharge to α motor neuron drive. The most direct way to determine the effects of afferent input on the Kohnstamm generator is via physical obstruction of the involuntarily rising arm. An early report involving single traces obtained by a string galvanometer suggested that obstruction does not end the aftercontraction or reduce central innervation (Forbes et al., 1926). However, these experiments could not determine if the afferent input had a significant effect on the muscle activity. A more recent experiment demonstrated that EMG during the aftercontraction is proportional to the angle of the rising arm (Adamson & McDonagh, 2004). Here, the arm was obstructed at 15, 35, 55 and 70° of abduction. Mean EMG at contact with

obstacle increased across these positions, differing significantly between 15 and 70°. Single traces also appeared to show that at the point of contact with the obstacle the EMG remained constant, but this was not statistically tested. The results suggest that afferent position signals from the contracting muscle set the level of drive from the Kohnstamm generator, creating a position-control feedback system. However, without statistical comparisons of EMG in the time domain, locked to obstruction, it is unclear how strong of an effect afferent signals have on the Kohnstamm generator.

### 1.2.3. What is changing in the brain?

A key question regarding both the mechanisms behind the Kohnstamm phenomenon and its relevance to voluntary action is the extent to which changes can be detected in the brain. Subcortical (Foix & Thevenard, 1923; Rothmann, 1915) and cortical (Salmon, 1915, 1916, 1925; Sapirstein et al., 1936, 1937, 1938) theories have been advanced. Early cortical explanations involved either a persistence of the voluntary movement, akin to a kinaesthetic after-image (Salmon, 1916, 1925), or changes in the excitatory state of the motor cortex (Sapirstein et al., 1937, 1938). It was observed that the aftercontraction was diminished, but present, in patients with Tabes dorsalis (Kohnstamm, 1915; Rothmann, 1915; Salmon, 1916, 1925), a condition resulting from untreated syphilis, which caused demyelination of proprioceptive pathways. Sapirstein, Herman, and Wechsler (1938) studied twelve tabetic patients, all of whom lacked basic proprioception and showed no knee jerk response to a tendon tap. A normal aftercontraction was observed in 10 of the patients and there was no correlation between symptom severity and aftercontraction size (but see Salmon, 1929, for evidence of absence). The authors also examined 7 patients with Parkinson's and found that they all exhibited strong, prolonged aftercontractions, and that in some cases tremors were visibly reduced during the movement. This extended duration was noted by earlier authors (Laignel-Lavastine et al., 1927; Salmon, 1916, 1929; but see Selionov et al., 2013 for evidence of no aftercontraction in Parkinson's when multiple muscles are involved). Amongst patients with hemiplegia, they found that while that the spinal reflexes were hypersensitive on the affected side of the body, aftercontractions were markedly reduced. Others noted this reduction (Rothmann, 1915; Salmon, 1916, 1925). However, it could be that these patients could not produce adequate voluntary induction contractions (Salmon, 1929). Finally, a single case of abnormal cerebellar

development was studied and it was noted that the aftercontraction was strong, but unusually jerky in character. Together, the results suggest that Kohnstamm generation is cortical, and that it is modified by sub-cortical structures in a similar fashion to voluntary movement.

Other evidence purporting to demonstrate a cortical origin is harder to interpret. Bromides (2 gm sodium bromide) were found to reduce the size of the aftercontraction, while other drugs that are known to have less effect on cortical function had no effect (Sapirstein et al., 1936). The effect of bromides was found to be ameliorated by caffeine (Sapirstein et al., 1936), which, along with alcohol has been reported to increase the aftercontraction (Danielopolu et al., 1921; Forbes et al., 1926; Matthaei, 1924b). However, without adequate control experiments and EMG recordings, it is impossible to know if the drugs had a direct effect on the aftercontraction.

Similarly, there is a notable consensus amongst authors that personality traits such as positivity and emotional reactivity were correlated with large aftercontraction, while negativity and low reactivity were associated with smaller aftercontraction (Kohnstamm, 1915; Laignel-Lavastine et al., 1927; Salmon, 1925, 1929; Sapirstein, 1948, 1960; Sapirstein et al., 1937). Indeed, Sapirstein (1948; 1960) employed the aftercontraction as a diagnostic tool within the field of psychiatry, testing hundreds of individuals, and observing that this relationship between traits and the aftercontraction persisted when they were amplified into the psychiatric range. The appearance of the aftercontraction predicted the recovery of patients, while its disappearance predicted periods of worsening mental health. Unfortunately, without physiological recordings it is impossible to discount task compliance as the significant variable. There have been no modern experiments on the topic.

Historically, direct attempts to show a cortical origin were confined to animal experiments. Sustained stimulation of the monkey motor cortex produced prolonged contractions of the muscle, but these innervations could not be distinguished from those during seizures (Sapirstein, 1941). However, recent fMRI work in humans has confirmed the involvement of the cortex in the Kohnstamm phenomenon. Duclos et al. (2007) had participants first experience a small wrist aftercontraction, and then a TVR, involving the extensor muscle tendon at the wrist level. In the scanner these movements were compared to rest and voluntary movements. No significant differences were found between the aftercontraction and TVR. Both activated an

extensive network of regions including primary sensory and motor cortices, premotor cortex, cingulate cortex, inferior and superior parietal cortex, insula and the vermis of the cerebellum. In the contrasts between aftercontraction and voluntary movement, the aftercontraction was associated with greater activity in bilateral cerebellar vermis, right premotor cortex, cingulate cortex, supramarginal gyrus, and the thalamus. Voluntary movement involved significantly higher activity in the left supplementary motor area, primary sensory and motor cortices, posterior parietal cortex and insular.

The finding that the Kohnstamm phenomenon is associated with activity throughout the cortex has been replicated (Parkinson et al., 2009). Both studies found the anterior cingulate cortex showed prominent activity during the aftercontraction. This could be due to the region's well-documented role in error monitoring (Carter et al., 1998; Taylor, Stern, & Gehring, 2007) or a more direct involvement in generating a movement command (Ball et al., 1999; Paus, 2001), perhaps via the modulation of postural centres in the brainstem (Takakusaki, Saitoh, Harada, & Kashiwayanagi, 2004). Both studies found high levels of activity in the parietal lobes, cerebellum, primary motor cortex and premotor regions (Duclos et al., 2007; Parkinson et al., 2009). The supplementary motor area, which is a key structure in goal-directed movement programming (Geyer, Matelli, Luppino, & Zilles, 2000; Tanji, 1996), was either only active during voluntary movement (Duclos et al., 2007), or active to the same degree across aftercontraction and voluntary movement (Parkinson et al., 2009). The cortex is clearly involved in the Kohnstamm phenomenon. However, activity in the cortex could be epiphenomenal, rather than a direct reflection of the Kohnstamm generator itself. For example, it could reflect sensory feedback from the moving limb, or even mental imagery triggered by the unusual experience (Decety, 1996).

More direct evidence comes from the effects of attention, mental imagery and visual input. Inductions involving isometric contractions of the elbow and shoulder can produce aftercontractions in the ipsilateral hip and knee (Craske & Craske, 1985). The effect also worked in the other direction and involved having participants name the non-induction limb repeatedly and silently at the point of relaxation. It was confirmed that this effect of attention could induce transfer of aftercontraction from one arm to the other (Craske & Craske, 1986). Intriguingly, it was also found that imagining pushing outwards for 60 s could also result in an aftercontraction of the shoulder. The above experiments did not involve verification of transfer by EMG and

featured a reasonable degree of unexplained spontaneous arm movements, indicative of an expectation effect. However, the previously cited experiments showing that visual input can induce muscle switching (Ghafouri et al., 1998; Gilhodes et al., 1992) do not suffer from this limitation. These experiments indicate that, regardless of the origin of the aftercontraction, output to the muscle must first pass through the cortex. This has been confirmed. Applying transcortical magnetic stimulation to the primary motor cortex during the aftercontraction induces a silent period in the contracting agonist muscle (Ghosh, Rothwell, and Haggard, 2014). The silent period did not differ in terms of latency or duration from that obtained during a matched voluntary movement. They were > 100 ms, which is an established indicator of cortical inhibition (Chen, Lozano, & Ashby, 1999; Fuhr, Agostino, & Hallett, 1991; Terao & Ugawa, 2002). In sum, there is now good evidence that the aftercontraction is driven by output from the primary motor cortex. However, many questions remain regarding cortical involvement in the Kohnstamm phenomenon, with comparisons voluntary movement being particularly informative.

### 1.2.4. What is the relationship between this involuntary movement and voluntary control?

Kinematically the aftercontraction is identical to a slow voluntary movement. Similarly, the EMG signal is comparable to a voluntary movement of similar size and speed (Fessard & Tournay, 1949; Forbes et al., 1926; Schwartz, 1924; Schwartz & Meyer, 1921). There is also evidence that the entire motor system shows the same level of excitability during both forms of movement. Mathis et al. (1996) applied 8-10 Transcranial Magnetic Stimulation (TMS) pulses (ISI = 8 s) to the left motor cortex during right deltoid aftercontractions and matched voluntary movements in seven healthy participants. They found that, despite the maximum abduction being lower in the aftercontraction compared to the voluntary movement (22 vs. 27°), the EMG did not significantly differ (57 vs. 45 mV). Importantly, there was no significant difference in the mean amplitude of Motor Evoked Potentials (MEP) elicited by the TMS (aftercontraction = 1.3, Voluntary = 1 mV). In both conditions MEP size correlated with background EMG level, and there was no difference in the gain, latency, or dynamics of the MEPs across conditions. Interestingly, an additional benefit of rising EMG (i.e. abduction, muscle shortening) compared to falling EMG was found in 20% of voluntary trials and 30% of aftercontraction trials. These findings are

complemented by the already cited imaging work which found no significant difference in the activity in the primary motor cortex during aftercontraction and matched voluntary movements (Duclos et al., 2007; Parkinson et al., 2009).

However, work using intramuscular needle electrodes does not fully support this account. Kozhina et al. (1996) recorded single motor unit activity from the deltoid and triceps muscle in four participants during aftercontraction and matched voluntary movements. The standard latent period of muscle silence was seen after the Kohnstamm induction (triceps = 1.4, deltoid = 1.5 s), followed by a 1-2 s when the firing rate increased, before remaining constant for the rest of the aftercontraction. Standard deviation of spike rate did not differ across voluntary movements and aftercontraction. Additionally, EMG recordings from the antagonist muscle (bicep) during tricep contractions did not differ. However, the mean firing rate of motor units was significantly lower during aftercontraction (12 pps) compared to voluntary movements (14 pps), despite the velocity and amplitude of the voluntary movements never exceeding that seen during aftercontraction. Thus, while the motor cortex and descending pathways do not differ in terms of gross excitability across aftercontraction and matched voluntary movements (Mathis et al., 1996), this does not preclude subtle differences in the state of motoneurons. It may be that the aftercontraction involves adaptations in motoneurons, which allow the same movement to be achieved with a lower firing rate (Kozhina et al., 1996).

Central to understanding involuntary and voluntary motor control is determining how the two forms of movement interact. The Kohnstamm phenomenon may feel subjectively like it is uncontrollable, yet the arm can be easily brought under voluntary control by the participant (Kohnstamm, 1915). Small voluntary movements in the direction of the aftercontraction may actually aid the appearance of the phenomenon (Salmon, 1916), although the precise timing of this effect has not been investigated. The aftercontraction does not prevent simultaneous voluntary movements of the same muscle (Fessard & Tournay, 1949; Hick, 1953; Shea, Shebilske, Kohl, & Guadagnoli, 1991), with voluntary movements apparently superimposed over the involuntary one (Hick, 1953). Furthermore, hip aftercontractions have been shown to dramatically alter the attempts of blindfolded participants to walk in a straight line (Ivanenko et al., 2006). The effect was always in the direction of the aftercontraction and disappeared when participants stepped in place on a treadmill, suggesting specificity in the movement programs affected.

However, the above experiments have limited interpretability, since the observed behaviour does not separate the involuntary and voluntary contributions to the movement. Other voluntary movements have been found to have an inhibitory effect on the aftercontraction. Rapid voluntary movements during the latent period can prevent the aftercontraction from emerging (Duclos et al., 2004; Hutton, Kaiya, Suzuki, & Watanabe, 1987). Paillard (1951) noted that sudden voluntary upwards movements of one arm cause transient inhibition of an aftercontraction occurring in the other arm. These effects may be due to a form of 'resetting' of the sensorimotor system caused by the voluntary movement or a form of top-down motor inhibition of the developing aftercontraction. Alternatively, the contralateral movement may just superimpose a postural adjustment on the other arm in addition to the aftercontraction.

The possibility of voluntarily stopping the aftercontraction has always been known about (Kohnstamm, 1915). Early reports indicated that it was easily possible to stop the aftercontraction during the latent period (Forbes et al., 1926; Pinkhof, 1922). Indeed, inhibition of one arm during latent period apparently does not affect the aftercontraction in the other arm (Paillard, 1951). Voluntarily stopping the arm and holding it stationary during the involuntary movement is possible, though reportedly difficult (Forbes et al., 1926). Recordings using string galvanometery suggested that the antagonist muscle was not always necessary to stop the involuntary movement (Forbes et al., 1926). However, those authors also showed data where antagonist muscle was active during stopping. Lack of averaging and statistical testing means that this issue remained unresolved. Actively adducting the arm against an abducting aftercontraction does not appear to extinguish the phenomenon (Fessard & Tournay, 1949), with the effect that the arm sometimes begins to rise again once it has been brought back to the start position. These findings suggest an intriguing possibility: that voluntary inhibitory commands can modify involuntary movements.

Ghosh et al. (2014) verified these observations. Following an aftercontraction of the lateral deltoid, participants were randomly instructed 'gently bring the arm back down and actively keep it down'. They did this without the use of the antagonist muscle (pectoralis). After 'holding' the arm down for 1-3 s, it spontaneously rose, albeit with reduced EMG relative to the first aftercontraction. This suggests something akin to a 'negative motor command' can be sent to oppose the upward

drive from the Kohnstamm generator. Such commands may originate from 'negative motor areas' upstream of the primary motor cortex. Several cortical areas have been reported to cause slowing and cessation of movement when directly stimulated (Filevich, Kühn, and Haggard 2012; Brown and Sherrington 1912). This putative negative motor command appears not to permanently override the generator. Exactly how this command integrates with the Kohnstamm generator is not known. However, this finding, combined with evidence that aftercontraction is driven by output from the primary motor cortex, provides an important basis for establishing the direct comparison between voluntary and involuntary movement. It also constrains theories on the control principles underlying the Kohnstamm phenomenon.

### 1.2.5. Control principles underlying the Kohnstamm phenomenon

**Table 1.2. Theories of the control principles of the aftercontraction**

| Theory name | Control principle |
|---|---|
| Persistence of motor activity | Ballistic, feedforward control. Kohnstamm motor command during aftercontraction is not modulated by afferent feedback. |
| Negative position feedback | Kohnstamm motor command depends on the discrepancy between a central specification of a muscle equilibrium point, and muscle spindle input specifying the disparity between current arm position and the equilibrium value. Equilibrium value may move over time, defining a "virtual trajectory". |
| Positive force feedback | Kohnstamm motor command depends on a positive feedback loop between a central excitatory drive and Golgi tendon organ afferent firing rates. |

The control principles underlying the Kohnstamm phenomenon have been investigated by systematically varying the induction contraction. Duration (Fessard & Tournay, 1949; Matthaei, 1924b) and amplitude (Allen, 1937; Allen & O'Donoghue, 1927; Holway et al., 1937; Matthaei, 1924b) of the induction contraction are positively correlated with the amplitude of the aftercontraction in terms of the angular displacement of the limb. This holds for durations up to ~2 minutes, when the aftercontraction begins to decrease due to fatigue (Salmon, 1929). Attempts were made to characterise this relationship in terms of a log function (Allen &

O'Donoghue, 1927) and power function (Holway et al., 1937). However, these efforts were based on inadequate samples and were confounded by the fact that repeating many Kohnstamms within a short space of time may initially produce reinforcement, resulting in increased aftercontraction size (Sapirstein et al., 1937) and then fatigue, resulting in decreased aftercontraction size (Danielopolu et al., 1921; Sapirstein et al., 1937; Zigler et al., 1948). Other authors have observed possible augmentation effects resulting from performing multiple Kohnstamms, interspersed with 20 minute rests (Allen & O'Donoghue, 1927), rendering the possibility of obtaining simple laws for aftercontraction size unlikely. A more recent attempt, using a larger sample size and modern recording equipment, found that once the duration of the induction reaches a certain threshold (~45 s) the size of the aftercontraction is related to the size of the muscular contraction (Brice & McDonagh, 2001), with for example 60 s of 30% deltoid MVC producing 50° of angular displacement of the arm, and 70% producing 92° on average.

#### 1.2.5.1. Persistence of motor activity

The above evidence can be explained by the Kohnstamm generator being a persistence of the voluntary command (Salmon, 1925; Sapirstein et al., 1937). This theory (see table 1.2) is consistent with reports of aftercontractions in patients with deafferentation due to Tabes dorsalis, but reduced aftercontractions in patients with hemiplegia (Kohnstamm, 1915; Rothmann, 1915; Salmon, 1925; Sapirstein et al., 1938). Indeed, it also seems consistent with reports that muscle length during induction does not seem important (Forbes et al., 1926; Hagbarth & Nordin, 1998). On such an account, any modulation in the structure of the inducing contraction would be expected to be present in the aftercontraction. Previous literature on varying the induction gives little indication of the control principles of the Kohnstamm generator. There have been no studies where the induction contraction is systematically varied, whilst controlling for the total amount of muscle activity.

A number of findings disagree with ballistic, feedforward control. Firstly, it is difficult to reconcile the latent period of several seconds with a simple replaying of the motor command (Csiky, 1915; Kozhina et al., 1996; Salmon, 1929). If the Kohnstamm represents perseveration of a voluntary motor command, why is there a delay before perseveration starts? Early suggestions that the latent period is actually the time taken to release an unspecified inhibitory control (Kohnstamm, 1915), are

not supported by the subjective sensation of simply relaxing. This contrasts with the sensation of active inhibition when participants voluntarily stop the aftercontraction (Forbes et al., 1926; Ghosh et al., 2014). Furthermore, theories of persistence of excitation within the motor cortex (Sapirstein et al., 1937), are not supported by the finding that the size of cortical evoked potentials is small and proportional to EMG during the latent period (Mathis et al., 1996). It is also hard to reconcile this theory with unidirectional leg inductions producing complex patterns of movement (Selionov et al., 2009), and sensory input causing muscle switching (Ghafouri et al., 1998; Gilhodes et al., 1992). However, the theory is yet to be fully discounted via a direct test.

#### 1.2.5.2. Negative position feedback

Once the aftercontraction contraction has begun, muscle activity could be controlled via negative position feedback from muscle afferents (Table 1.2). It is known that there exists a tight coupling between the arm angle during the aftercontraction and EMG (Adamson & McDonagh, 2004). Indeed, such positional theories are consistent with a peripheral origin of the Kohnstamm phenomenon, whereby the induction phase would lead to some change in a peripheral signal that drives motor circuits. One model views the Kohnstamm phenomenon as a form of proportional-integral-derivative (PID) control, similar to equilibrium point control (Feldman, 1986; Bizzi et al., 1992), proposed for both stretch reflexes and voluntary actions. For such control, a central motor signal setting the equilibrium point of the muscle would result in a follow-up servo contraction of the muscle, causing a movement towards that position. Alternatively, the equilibrium point might move gradually over time, defining a virtual trajectory (Bizzi et al., 1984; Hogan, 1985). Here, increased aftercontraction from longer and more powerful induction contractions would be explained by greater peripheral adaptation. A virtual trajectory account seems broadly consistent with the existing electrophysiological evidence of increasing muscular activity with movement (Adamson & McDonagh, 2004; Fessard & Tournay, 1949; Kozhina et al., 1996). Involvement of the motor cortex (Duclos et al., 2007; Ghosh et al., 2014; Parkinson et al., 2009) would be interpreted as being a proportional response to the ‘abnormal’ afferent inflow, existing within normal transcortical control loops. Here, silence in the muscle during the latent period (Kozhina et al., 1996), must be the time required for a sufficiently uniform afferent

volley to reach the cortex so that an efferent response is triggered. An obvious way to test the position control theory is to determine how physical obstruction of the aftercontraction affects motor output. Existing experiments using this technique suggest that obstruction does not abolish the aftercontraction (Adamson & McDonagh, 2004; Forbes et al., 1926). However, neither experiment examined the time-course of the EMG across participants in response to the obstruction. Thus, position control models of the Kohnstamm phenomenon also cannot be discounted.

#### 1.2.5.3. Positive force feedback

Force feedback could underlie the Kohnstamm phenomenon (Table 1.2). Based on work showing that EMG was lower during supine than during standing aftercontractions it was hypothesised that positive force feedback could be a critical control principle (Lemon et al., 2003). Parkinson and McDonagh (2006) tested this principle by manipulating the weight of the nine participant's arms during a shoulder Kohnstamm in the frontal plane. Arm weight was systematically reduced (100, 75, 50, 25, 0%) via the use of a moveable counter-weight on a lever attached to the arm. Across conditions, participants induced the aftercontraction by pushing upwards with a force of 60% of their maximum for 1 minute. It was found that mean aftercontraction EMG (as a percentage of voluntary induction EMG) was reduced across every arm angle as the weight of the arm was reduced. At a given arm angle (70°) EMG was significantly higher in the 100% arm weight (normal arm weight) condition than in the 50, 25 and 0% arm weight conditions. This was interpreted as evidence of positive feedback. As GTO signal increased throughout the abduction (due to increased muscle torque), motor efference also increased via a putative peripheral-central feedback loop. However, the design and analysis of the experiment limit interpretations. Firstly, the counter-weight was attached throughout the induction, latent period and aftercontraction. Afferent signals during the first two stages could establish central adaptations, which underlie the EMG reductions observed. Secondly, it is perhaps problematic that all EMG values during the aftercontraction were referenced to the mean EMG during induction rather than an independent maximum contraction. This analysis may have been performed to control for the fact that trial order was not randomised across conditions. However, the assumption of a linear relationship between induction size and aftercontraction has numerous caveats (Brice & McDonagh, 2001; Salmon, 1925). It would have

been preferable to first verify that the inductions did not differ across conditions and then look for changes in the aftercontraction EMG as a percentage of MVC. Finally, velocity of arm movements was not reported, so no inferences can be made about shoulder torque or spindle firing rate across conditions.

The Kohnstamm phenomenon may represent an adaptation within tonogenic structures, which functionally overlap with central pattern generators involved in repetitive actions such as walking (Craske & Craske, 1986; Selionov et al., 2009; Waters & Morris, 1972). Complex interactions occur between muscle groups (Craske & Craske, 1985), while sensory input can interact with the aftercontraction in surprising and divergent ways (Brun et al., 2015; Forbes et al., 1926; Ghafouri et al., 1998). As such, it may be necessary to consider hybrid models to explain the Kohnstamm phenomenon. However, it is important to first discount the simple accounts already postulated.

### 1.2.6. Subjective experience of involuntary movement

Perhaps the most striking, yet least studied, feature of the Kohnstamm is that while the movement looks the same as a slow voluntary contraction, it feels very different for the person to whom it is actually happening (Fessard & Tournay, 1949). Participants often report feeling surprised when their limb begins to move (Craske & Craske, 1985; Forbes et al., 1926), and state that the limb is floating (Craske & Craske, 1985; Salmon, 1915), either of its own accord (Craske & Craske, 1985) or via some ‘hidden force’ (Kohnstamm, 1915). Another, often vivid sensation is that the limb feels much lighter than normal (Craske & Craske, 1985; Cratty & Duffy, 1969; Gurfinkel et al., 1989; Hagbarth & Nordin, 1998; Kohnstamm, 1915). Indeed, it has been argued that the subjective feeling of lightness is the best way to gauge the duration of the aftercontraction (Cratty & Duffy, 1969). In the latter study, participants continuously reported whether their arm felt lighter or heavier than normal, reporting that the arm felt lighter for an average of 14 s. However, most subjective findings in the literature are the author’s ad-hoc recollections of participant’s self-reported phenomenology or spontaneous commentary, with few attempts to fully catalogue participant’s experiences in an unbiased manner.

There have been some attempts to quantify the feeling of lightness. Matthaei, (1924a) instructed participants to maintain an equal upward force on two springs. After inducing an aftercontraction on one arm it was found that the length of the

spring held by this arm was much longer than the spring held by the non-aftercontraction arm. This was interpreted as evidence that the perception of arm weight is reduced during the Kohnstamm phenomenon. However, such findings are confounded by the addition of the voluntary movement on top of the aftercontraction. Indeed, this problem also affects studies finding that inducing an aftercontraction causes both isometric and isotonic voluntary forces to be significantly larger than intended (Hutton, Enoka, & Suzuki, 1984; Hutton et al., 1987; Knight, Marmon, & Poojari, 2008; Shea et al., 1991). Such results may reflect peripheral adaptations to the induction contraction. However, the already cited work on intact position sense during aftercontraction does not support this hypothesis (Cratty & Duffy, 1969; Heide & Molbech, 1973; Howard & Anstis, 1974).

Most relevant to the subjective experience of the Kohnstamm phenomenon is the already cited experiment on inhibition. Ghosh et al., (2014) examined the subjective experience of (n = 21) participants as they lowered their arms during an aftercontraction, and compared this to the feeling of lowering the arm without an aftercontraction (n = 10). In the latter condition the arm was first held in the abducted position at shoulder level for 1 minute. The authors also tested the same effect in five participants who did not experience an aftercontraction after the Kohnstamm induction. Here the arm was first passively raised before being lowered voluntarily, allowing a test of the hypothesis that any subjective effects were simply a by-product of the isometric contraction. Across three trials in each condition, participants rated the sense of resistance on a scale from 0-50. It was found that the strongest sense of resistance was felt during the downward movement with aftercontraction. In participants with no visible aftercontraction, the ratings did not differ between the conditions. Participants were also asked to rate this sensation of resistance on a scale from 0-5 according to how much it resembled a series of descriptions. Sixteen of the 21 participants gave strongest agreement to the feeling that the falling arm was pressing against a soft air balloon. In the no aftercontraction (baseline) condition no participant strongly agreed with this statement, instead reporting ‘no sensation’. This was also the case for the five participants who had no aftercontraction. Thus, the sensation of resistance arose as a result of the interaction between the Kohnstamm generator and normal sensory inflow from the moving limb. One explanation is that the upward lift from the Kohnstamm generator was not perceived as self-generated. If the Kohnstamm generator does not produce efference copies of

the movement command, than there would be nothing to cancel against the sensory inflow, resulting in a miss-attribution of a resistance to overcome (Blakemore & Frith, 2003). This could be the case more generally during the aftercontraction and account for its unique subjective characteristics. However, it is also possible that the ratings of resistance were influenced by the feeling of effort required to inhibit the aftercontraction, or the fact that the upward drive from the Kohnstamm generator rendered the downward movement less fluent than normal.

### 1.3. Knowledge gaps and the present thesis

#### 1.3.1. Knowledge gaps

Regarding changes to the muscle during the Kohnstamm phenomenon, one central unresolved issue is how the muscle afferents contribute to the phenomenon. It is not known if the hypothesised thixotropic changes occur during the standard Kohnstamm induction. It is also not known if thixotropic changes alter the sensitivity or firing rate of muscle afferents during the induction phase, nor whether such peripheral changes drive the aftercontraction. Only indirect evidence exists to support this claim. Ideally, such questions would be addressed by microneurographic recording from identified afferents across the individual phases of the Kohnstamm phenomenon, though the difficulties of microneurographic techniques may make this unfeasible. A novel approach to whether the sensitivity of the spindle response is increased in the Kohnstamm phenomenon would be to examine stretch reflexes in the aftercontraction and matched voluntary movements. If spindle sensitivity is abnormally high then one would predict that stretch responses should be significantly larger during the aftercontraction. The problem can also be explored via position sense, which is derived from muscle spindle firing rates. Existing studies suggest that position sense is normal during the aftercontraction (Howard & Anstis, 1974). However, further evidence is required before this can be asserted with confidence.

It is also unclear if sensory signals contribute to the generation of the Kohnstamm more generally. Putting aside peripheral accounts of the phenomenon, the question of what sensory signals are necessary for an aftercontraction to occur remains unresolved. Aside from the already cited literature involving Tabes dorsalis (Sapirstein et al., 1938), there have been no studies involving individuals with afferent damage. As such, it is unclear if normal afferent signalling during the induction is necessary for the aftercontraction to develop. On a related note, it is not

clear whether changes in sensory stimulation during the induction in healthy individuals have effects on the aftercontraction. All reports in the literature involve an isometric contraction to induce the Kohnstamm phenomenon. Yet surprisingly, there are no recorded attempts to produce the aftercontraction from non-isometric contractions.

Afferent inflow from the muscle does not appear to abolish the aftercontraction once it has been initiated (Adamson & McDonagh, 2004; Forbes et al., 1926). However, it is still not clear if the afferent input definitively influences the efferent input to the muscle. Moreover, it is not known how this afferent input affects the Kohnstamm generator. It could be that the generator is permanently altered by such input, or perhaps only transiently affected.

Further, it is not clear if separate Kohnstamm generators exist in each hemisphere. Studies have shown interactions between the arms (Brun et al., 2015; Craske & Craske, 1986; Paillard, 1951). Neuroimaging work points to the activation of a widespread bilateral sensorimotor network during aftercontraction (Duclos et al., 2007; Parkinson et al., 2009). However, there have been no attempts to electrophysiologically dissociate the control of each arm during bilateral aftercontraction.

Voluntary motor commands can counteract the aftercontraction (Fessard & Tournay, 1949; Ghosh et al., 2014). However, it is unknown how precisely these negative motor commands can oppose the aftercontraction. Inhibition may be an all or nothing process, which, once initiated always causes the arm to fall. Alternatively, inhibition may be able to precisely balance the quantity of aftercontraction, resulting in a static arm. It also remains unclear whether voluntary inhibition acts directly on the generator, or acts at some downstream level, merely inhibiting the motor expression of the generator output. The fact that the Kohnstamm may resume after inhibition seems consistent with the latter account. It is also unknown if these negative motor commands have a bilateral effect, as is common with studies examining the inhibition of voluntary action.

There is no satisfactory computational account of the Kohnstamm phenomenon. Two related issues lie at the heart of developing such an account. Firstly, it is not known what control principles link the induction phase to the subsequent aftercontraction. Existing studies have focused on the amplitude of the voluntary contraction without manipulating its control regime or varying its efferent

and afferent patterns. Secondly, it is not known what control principles underlie the aftercontraction once it has begun. Ballistic, feedforward control theories, though unlikely have not been adequately dismissed. Simple position feedback control has also not been discounted. Positive force feedback control is supported by a single experiment (Parkinson & McDonagh, 2006), which, though important, has a number of features that limit the generalizability of the finding.

Mechanisms behind the subjective 'strangeness' of the Kohnstamm phenomenon are poorly understood. No previous researchers have conducted questionnaires to try and build a taxonomy of these reported feelings. As such, it is unclear if the reports in the literature accurately reflect the full range of sensations associated with the aftercontraction. The involuntary nature of the movement and associated feeling of lightness are clearly central. A tentative computational account has been put forward to explain these sensations. Reports of resistance while inhibiting the aftercontraction suggested that the Kohnstamm generator does not produce efference copies to cancel against the sensory inflow (Ghosh et al., 2014). However, it is not known if this is truly the case or just a by-product of the inhibitory command and/or unusual adduction.

This thesis attempts to fill some of these knowledge gaps, and thus cast new light on both the Kohnstamm phenomenon in particular, and on voluntary and involuntary movement more generally.

### 1.3.2. Outline of the experimental part of the thesis.

A series of experiments were conducted to address the knowledge gaps present in the literature.

### Chapter 2. How does activity during the induction phase influence the Kohnstamm aftercontraction?

In the first experiment, we manipulated the control (Position vs. Force) and variability (Fixed vs. Varying) of the voluntary induction contraction and recorded the effect on the aftercontraction. This experiment addressed the control principles underlying the Kohnstamm phenomenon. It also determined whether a fixed proprioceptive signal (isometric contraction) was necessary for the aftercontraction to develop and whether the aftercontraction could be considered a simple persistence of the voluntary command.

**Chapter 3. Physical obstacles reveal mechanisms of afferent feedback and subjective awareness in unilateral and bilateral aftercontractions.**

The next two experiments explored the use of physical obstruction during the aftercontraction. Random obstruction of a unilateral aftercontraction was compared to an unobstructed condition to determine if afferent input affects the efferent input to the muscle. Subjective ratings of force were compared to voluntary and passive movements to test the 'lack of efference copy' hypothesis. In the second experiment, one arm was randomly obstructed for 2 s during a bilateral aftercontraction. This experiment addressed if bilateral control was possible, determined if the afferent input permanently altered the output from the generators, examined spindle sensitivity via stretch reflexes and assessed the 'lack of efference copy' hypothesis via an implicit force matching task.

**Chapter 4. Perturbations applied during horizontal aftercontraction suggest negative-position feedback control in the Kohnstamm phenomenon**

An experiment investigated the control principles underlying the aftercontraction. The effects of adding resistive and assistive perturbations to a horizontal aftercontraction were tested via the use of a single joint manipulandum and compared to matched voluntary movements. This allowed a direct test of the positive force feedback theory. It also revealed the extent to which the control principles underlying the Kohnstamm phenomenon were the same as those during voluntary movement.

**Chapter 5. Voluntary motor commands reveal awareness and control of involuntary movement**

Finally, an experiment was conducted to explore voluntary inhibition of the aftercontraction. Here, participants were randomly instructed to inhibit unilateral and bilateral aftercontractions for 2 s and then 'release' the inhibition. The experiment investigated whether negative motor commands could perfectly counter the Kohnstamm generator, whether they had a permanent effect on the generator and whether they were bilateral or unilateral. A subjective task again tested the hypothesis that the Kohnstamm generator does not produce efference copies. A

questionnaire allowed us to draw important comparisons between the subjective sensations associated with the Kohnstamm phenomenon and the accompanying physiological recordings. Finally, voluntary replication movements allowed us to test position sense during the Kohnstamm phenomenon.

**Chapter 6: Summary and general conclusions**

The thesis ends with a summary of the results, and a tentative model of the processes contributing to the Kohnstamm phenomenon.

## Chapter 2. How does activity during induction phase influence the Kohnstamm aftercontraction?

**Few studies have explored the control principles behind the Kohnstamm phenomenon. Longer and more powerful induction contractions cause larger aftercontractions, possibly because they more strongly recruit a "Kohnstamm generator". All previous studies used isometric contractions, leading to theoretical accounts based on muscle thixotropy. Alternatively, the aftercontraction may be due to a persistence of motor command activity in the cortex. We tested these theories by varying the input and feedback control during induction contraction and measuring the effect on the resulting aftercontraction. In a Fixed Force (FF) condition participants induced the Kohnstamm by pushing on a strain gauge with their arm abducted at an angle of 80°. Visual feedback was given and they had to maintain a constant level of force (50% MVC). In a Varying Force (VF) condition the induction task involved tracking a sinusoidal target force (varying by +/- 7.5 % of max. force, with a mean of 50%). In a Fixed Position (FP) condition participants held a weight with the arm 80° abducted, requiring 50% MVC to do so. Visual feedback allowed them to maintain constant arm position. In the Varying Position (VP) condition, participants continually moved the arm to track a sinusoidal target position (1 Hz, mean = 80 +/- 10°). Surprisingly, we found no significant differences in the aftercontraction EMG or the size of the involuntary movement (final arm angle) across all four induction conditions. Bayesian statistical analysis suggested this lack of difference was genuine, rather than a lack of power. We also found time-varying motor commands during induction were not followed by similar time-varying patterns in the aftercontraction. The results suggest that models of the Kohnstamm phenomenon based on muscle thixotropy or a persistence of the motor command are inadequate. They are consistent with the view of a Kohnstamm generator acting as a low frequency integrator of sensorimotor signals occurring during the induction.**

### 2.1. Introduction

Understanding how prior activity influences the functioning of the motor system is a central question in cognitive neuroscience. In the case of reflexes, the cause is external and much has been learned about the motor system by exploring how afferent inflow modifies efferent output. Reflexes tend to be rapid and short-lasting in their influence. At the other end of this spectrum is motor learning. Experiments exploring how motor skills are acquired and modified demonstrate how effortful voluntary action becomes more automatic and invariant over time. Less is known about adaptations in the motor system that exist between these two extremes, occurring at timescales on the order of seconds to minutes.

The Kohnstamm phenomenon, whereby a sustained contraction of a muscle, produces, upon relaxation, a sustained, involuntary aftercontraction of the same muscle (Kohnstamm, 1915; Salmon, 1916), offers a means to address these

medium duration adaptations and the underlying control principles. The Kohnstamm phenomenon apparently occurs in all skeletal muscles (Forbes et al., 1926; Matthaei, 1924b) and may represent an adaptation within the postural control system (Fessard and Tournay, 1949; Gurfinkel, Levik, and Lebedev, 1989; Ghafouri et al., 1998; Adamson and McDonagh, 2004; Duclos et al., 2004). Like other involuntary movements (Moraitis & Ghosh, 2014) key to understanding the phenomenon is determining the characteristics of the 'Kohnstamm generator' (De Havas, Ghosh, Gomi, & Haggard, 2015; Ghosh et al., 2014). It is well established that by providing the Kohnstamm generator with more input, in the form of longer and more powerful induction contractions, there is a resulting increase in output in the form of larger aftercontractions (Allen, 1937; Allen & O'Donoghue, 1927; Brice & McDonagh, 2001; Fessard & Tournay, 1949; Matthaei, 1924b). Such experiments reveal a basic 'dose-response' type of relation in the generator, but say little about the functional control principles of the generator itself.

Peripheral, central and hybrid theories of the Kohnstamm generator have been proposed. Purely peripheral theories of the Kohnstamm generator argue that the aftercontraction results from increased afferent discharge from muscle spindles (Hagbarth & Nordin, 1998; Hutton et al., 1987). One influential theory posits that this is due to muscle thixotropy (Gregory et al., 1988; Hagbarth & Nordin, 1998). The theory states that the Kohnstamm phenomenon occurs for the following reasons: 1) Kohnstamm induction is static and muscle length is short (relative to start of aftercontraction), 2) during induction contraction, stable actin and myosin cross-bridges form in intrafusal muscle fibers, 3) relaxation causes arm to be brought back to a longer muscle length, 4) stable cross-bridges in intrafusal fiber remain and mean that it is in a state of relative shortness, 5) relative shortness in intrafusal muscle fibers causes muscle spindles to be stretched and to fire, resulting in muscular contraction via spinal reflexes. Hagbarth and Nordin (1998) modified a muscular conditioning sequence (used in animals to enhance resting spindle discharge) to act as a Kohnstamm induction. The sequence involved: 1) participants first holding both arms slightly abducted, 2) actively lifting up their arms against two solid stands and forcefully pressing (max. effort) for 5-10 s, 3) relaxing while the experimenter held them up for 4-8 s, and 4) having their arms passively adducted by the experimenter to the start position. Removing any of these successive steps reduced the size of the

subsequent aftercontraction, which was taken as evidence in support of the thixotropy account. Further, heating and cooling the muscle affected the size of the aftercontraction in a way broadly predicted by the thixotropy account (Hagbarth & Nordin, 1998; Meigal et al., 1996; Meigal & Pis'mennyi, 2009). However, whether the formation of *stable* cross-bridges during the Kohnstamm induction is necessary for the aftercontraction to occur has not been tested.

Another account proposes that the Kohnstamm generator is central rather than peripheral. This view suggests that activity in the motor cortex persists after the cessation of the induction contraction (Sapirstein et al., 1937, 1938). Another account argues that the Kohnstamm generator is a "kinaesthetic afterimage" (Salmon, 1916, 1925), which in modern terms might equate to a reactivation of the motor programs involved in the voluntary induction contraction. Functional imaging and Transcranial Magnetic Stimulation (TMS) evidence point to cortical involvement in the phenomenon, including the primary motor cortex (Duclos et al., 2007; Ghosh et al., 2014; Mathis et al., 1996; Parkinson et al., 2009). It is not known if motor areas of the cortex form part of the Kohnstamm generator itself, or simply form part of the output pathway for a generator housed elsewhere. In general, accounts based on a persistence of the voluntary command have not been discounted. Crucially, persistence accounts imply continuity between the induction phase and the aftercontraction phase. In particular, any systematic variations in the induction should persist into in the aftercontraction. This prediction has not been tested, with the exception of the basic dose-response relation mentioned above.

Hybrid accounts have been proposed, emphasising a central adaptation that interacts with signals from muscle spindles (Duclos et al., 2004, 2007; Gilhodes et al., 1992) or Golgi tendon organs (Parkinson & McDonagh, 2006). These afferents primarily convey muscle length and muscle force information, respectively. The Kohnstamm generator is indeed sensitive to afferent input once the aftercontraction has begun (De Havas et al., 2015). However, it is not known if afferent input contributes to activating the Kohnstamm generator *during* the induction. Much of work in this area assumes that the Kohnstamm generator overlaps with the generator for the Tendon vibration reflex (TVR). Identical involuntary motor responses have been reported following 30 s tendon vibration, and following similar isometric contractions (Gilhodes et al., 1992), and both interventions also activate

the same sensorimotor regions of the cortex (Duclos et al., 2007). Discharge from muscle spindle primary endings strongly increases during an isometric contraction or a vibratory stimulation (Edin & Vallbo, 1990). For isometric contractions during the Kohnstamm induction this is due to co-activation of α-γ motoneurons (Edin & Vallbo, 1990; Vallbo, 1974). This proprioceptive signal could cause a central adaptation within brain areas signalling muscle length, essentially setting up a new-equilibrium point for the muscle, which would in turn trigger the aftercontraction (Duclos et al., 2004). Alternatively, the Kohnstamm generator may be sensitive to input relating to sensation of force (Parkinson & McDonagh, 2006).

The functioning of muscle spindles during the Kohnstam induction is worth briefly considering. Type Ia muscle spindles are most sensitive to the rate of change of muscle stretch, while type II spindles are sensitive to the steady level of tension. During isometric contractions they behave differently, with Ia afferent firing rates being high during the initiation of the contraction and then reducing to a lower level, and type II afferent firing rates being more constant (Fitz-Ritson, 1984; Matthews, 1964). Spindle firing rates increase (relative to passive stretch) due to fusimotor activation which maintains the tension on the cell (Taylor, Butler, & Gandevia, 2000; Vallbo, 1970a). This alpha-gamma co-activation means that under isometric conditions spindle firing rates are broadly proportional to the strength of contraction, with even small changes in force (1% MVC) producing measurable increases in firing rates in many afferents (Wilson, Gandevia, & Burke, 1997). During a sustained contraction spindle firing rate may decrease, due to adaptation from repetitive firing or because of changes (stiffness, temperature, chemical) in the muscle itself (Taylor et al., 2000). It has been found that after 30 s of an isometric contraction, firing rate in 72 % of spindle afferents declined progressively, while the remainder maintained a constant firing rate (Macefield et al., 1991). However, in this experiment spindles were not classified into type Ia or II. The decline was relatively small (66% of initial firing rate) and could reflect the fact that firing rates were referenced to the initial ramp phase of the contraction, which would particularly drive Ia afferents. Under conditions of slow, loaded lengthening and shortening of the muscle a more complex afferent firing pattern may be observed, with higher rates during muscle lengthening and lower rates during shortening (Al-Falahe, Nagaoka, & Vallbo, 1990; Burke, Hagbarth, & Löfstedt, 1978; Jones, Wessberg, & Vallbo, 2001). To our knowledge,

the effects of sustaining such a contraction have not been measured via microneurography.

Because both force and position change are present in the induction of the aftercontraction, the traditional method of invoking the Kohnstamm cannot clearly distinguish between the possible roles of these two afferent signals. In all previous studies, the aftercontraction is elicited via an isometric muscle contraction, pushing against a solid surface (Kohnstamm, 1915) or holding a fixed weight (e.g. Sapirstein, Herman, and Wallace 1937). If either force or position information is the key input for activating the Kohnstamm generator, one would expect that contrasting these forms of regulation would have an effect of the size of the aftercontraction.

Regardless of whether the Kohnstamm generator is activated by efferent, spindle or Golgi tendon organ (GTO) input (or a combination), the signal must be integrated over time to produce the adaptation. Previous experiments have emphasised that a continuous contraction is necessary to elicit the aftercontraction. Variations in sensorimotor activity during the induction phase have not been explored. Both central persistence and peripheral adaptation accounts make clear predictions about the effects of varying the induction period. If the Kohnstamm reflects persistence of a central motor command, then any variation in motor output during the induction phase should produce the same pattern of variation continuing into the aftercontraction phase. Alternatively, the aftercontraction might be abolished, because it remains an open question whether an induction with a substantially varying intensity can produce an aftercontraction at all. Indeed, rapid voluntary movements have been shown to abolish the aftercontraction (Duclos et al., 2004; Hutton et al., 1987). According to peripheral accounts, the effects of varying sensorimotor signals during the induction phase should depend on what parameters are varied, and what remain constant. For example, if the Kohnstamm phenomenon represents an adaptation of spindle signalling, such as the stabilisation of cross-bridges predicted by the thixotropy account, then varying muscle length during the induction phase should reduce or abolish the phenomenon.

Finally, any effect of varying signals during the induction phase could be informative about the dynamics of the generator. In particular, if variation of the induction has no influence on the aftercontraction, then an integration stage must precede the recruitment of the Kohnstamm generator, and this integrator must have

a time constant at least equal to the frequency of the variations in the induction phase. Manipulating variability of input and feedback control, whilst holding contraction duration and mean contraction strength constant, will therefore reveal a great deal about the Kohnstamm generator. As such, we employed a 2x2 experimental design to test the effect of variability of signalling during the induction phase (Fixed vs. Varying) and the parameter used for visual feedback control in the induction phase (Position vs. Force) on the size and character of the aftercontraction.

### 2.2. Methods

#### 2.2.1. Equipment

Electromyography (EMG) was recorded from bipolar, surface electrodes placed over the middle of both lateral deltoid muscles (agonist), parallel to the orientation of the muscle fibres. Electrodes were also placed on both pectoralis (antagonist) muscles (n = 7). EMG signals were sampled at 2000 Hz and amplified using variable gain (MME-3132, Nihon Kohden, Tokyo, Japan). Force was measured by a strain gauge (FGP-20, Nidec-Shimpo, Kyoto, Japan) mounted to a vertical pole via adjustable clamps. Participants wore wrist splints to remove the possibility of wrist extensor muscles contributing to force generation. Arm position was determined via the use of four infrared reflective markers positioned at the shoulder and forearm of each arm. Marker position was detected via four motion tracking cameras (Oqus300, Qualisys AB, Gothenburg, Sweden) with a sampling rate of 200 Hz. Across all trial types participants held a hollow aluminium handle (length = 18.8 cm; diameter = 3.9 cm). During position trials, strip weights were attached evenly to each end of the handle. During force trials, the handle was pushed against the strain gauge via a screwed-in, custom-made, plastic holder (14 x 7.5 x 6 cm) with a v-shaped recess (10 x 5.5 x 6 cm). Visual feedback of force and position was controlled by Cogent Graphics in MATLAB (2007b). It was displayed on a 23 inch LCD screen (1280x1024 pixels, 60 Hz refresh rate) located 1.5 m in front of participants. Force signal was low-pass filtered at 10 Hz before being displayed. Position signal was not filtered. Visual feedback signals were also output at 60 Hz with a D/A converter (USB-1208FS, Measurement Computing, MA, USA) for the purposes of recording. Analogue signals (Force, EMG, and visual feedback) were all

sampled at 2000 Hz and recorded, along with arm position data (200 Hz), via the software used to control motion tracking (QTM, Qualisys AB, Gothenburg, Sweden).

### 2.2.2. Participants

Eleven participants took part in the experiment (2 female; Age: mean = 30.18, SD = 4.85). Two participants were excluded because they did not display the Kohnstamm phenomenon, leaving 9 participants in the analysis (1 female; Age: mean = 30.11, SD = 3.91). The experiment was 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 local ethical committee approval.

### 2.2.3. Procedure

Participants first completed a 5 s maximum voluntary contraction (MVC) of all muscles from which EMG was to be recorded. In the case of the lateral deltoid, this was performed by pushing upwards with the handle on the strain gauge, with a straightened arm at an angular displacement of 80° (relative to the midline). Maximum force was recorded in kilograms. Participants completed two brief ~20 s trials to practice tracking Varying Position (holding handle only, no weight) and Varying Force (mean = 10% of max. force). They then rested for 3 minutes before beginning the experiment.

Participants completed 8 unilateral trials across 4 conditions (2 trials per condition, 1 per arm). In all conditions, participants completed 30 s of contractions of the lateral deltoid muscle with the arm in an abducted position (induction phase). This was followed by a tone instructing them to relax the arm and release the handle. The arm then returned to the participant's side. They were told to remain relaxed and not prevent any aftercontraction movement that occurred. Only the induction phase varied across conditions. In the Fixed Force (FF) condition participants pushed upwards with the handle during the induction. The strain gauge was positioned such that this force would require the arm to maintain a constant angular displacement of 80°. They maintained a constant force of 50% Maximum Voluntary Force (MVF) via an isometric contraction of the lateral deltoid and visual feedback consisting of a flat target force level and a dot showing current force level. In the Varying Force (VF) condition participants had to vary their isometric contraction strength. Current force was again displayed as dot, but here the target force was a sinusoid (1 Hz) centred

on 50% MVF (+/- 7.5% MVF). This value was determined from pilot studies indicating that larger variability in force compromised performance accuracy. In the Fixed Position (FP) condition participants held the handle at an angle of 80°. Attached to the handle were weights equal to 50% MVF. They kept the contraction isometric via position feedback on the screen, showing the target arm angle as a flat line and the current arm angle as a moving dot. Finally, in the Varying Position (VP) condition, participants held the weight as before, but had to continuously move their arm by varying a near-isotonic contraction (slowly alternating between concentric and eccentric contractions). Current position was again displayed as a dot on the screen, while target force corresponded to a sinusoid (1 Hz) centred on 80° of angular displacement (+/- 10°). This value was selected to ensure that the movement was large yet minimised changes in shoulder torque (maintain near-isotonic conditions) and fell within the range of motion where the lateral deltoid is the primary agonist.

There was a 3 minute rest between trials. Trials alternated between the right and left arm. Trial order was pseudo-randomised (such that trials belonging to each condition were evenly distributed) and counter-balanced across participants. In the case of one participant a single trial had to be repeated because the participant failed to remember to release the handle after the induction. The experiment lasted approximately 1 hour.

### 2.2.4. Analysis

EMG data from the agonist and antagonist was bandpass filtered (10 to 500 Hz), rectified and then smoothed using a low pass filter (4 Hz). Arm angle was determined by calculating the angle between a line connecting the two reflective markers and the vertical. EMG and force signals were normalised for each participant to recordings made during a maximum force contraction (100% MVF/MVC; Fig. 2.1.). The first and last 3 s of the 30 s induction period were excluded from the analysis. The remaining 24 s window was then used to calculate the mean and standard deviation of arm position, agonist EMG and antagonist EMG in each trial. For FF and VF conditions, the mean and standard deviation of force was also calculated during the same time window. Maximum aftercontraction arm angle was calculated by taking the peak arm position after the instruction to release the handle on every trial. Peak aftercontraction EMG was calculated by taking the

max. value of the smoothed agonist EMG after the release instruction. Latency was calculated by taking the time difference between the point of relaxation (minimum arm angle after release instruction) and the point in time when the arm angle began to continuously increase.

To determine if variability of input or feedback control had an effect on any of these variables, input variability (Fixed vs. Varying) by feedback control (Position vs. Force) repeated measures ANOVAs were conducted for each DV across participants (Fig. 2.2). We also verified whether the effects of induction phase parameters (fixed vs. varying input; force vs. position feedback control) remained even after accounting for variations in mean induction arm position. To do this, we fitted an additional ANCOVA model with 'difference of induction arm position between Position and Force feedback conditions' as a covariate.

Fixed effects (collapsed across conditions and participants) and random effects (average for each participant in each condition) correlations were also computed to determine if there was a relationship between latency and max. aftercontraction arm angle.

The final position of the arm in a Kohnstamm phenomenon is an important and established indicator of the strength of the Kohnstamm generator (Allen, 1937; Allen & O'Donoghue, 1927; Brice & McDonagh, 2001; Hagbarth & Nordin, 1998; Holway et al., 1937; Paillard, 1951; Sapirstein et al., 1937; Zigler et al., 1948). To determine whether effects of input variability and feedback control on final arm position represented evidence for or against the null hypothesis of no effect of induction method, we used Bayesian analysis (Dienes; 2008). With Bayesian analysis there is no requirement to correct for multiple comparisons (Dienes, 2011). Instead, direct comparisons between conditions were performed, based on the size of the obtained effect, the variability in the data and a posterior distribution. The analysis assumes the parameter estimate is normally distributed with known variance. However, in a *t*-test the variance is only estimated. Since the degrees of freedom were < 30 (in all our analyses df = 8), the assumption of known variance was not good enough. Therefore, for each comparison the standard error was corrected by multiplying it by 1 + 20/(df x df), as it produces a good approximation to *t*, over-correcting by a small amount (Berry, 1996; Dienes, 2014). We used the final arm angle as a suitable dependent variable to define the estimated size of the

Kohnstamm effect. We defined the posterior distribution in the following manner: 1) lower bound was always 0°, since this is the minimum effect size; 2) upper bounds were selected from 5 to 90°. The normal range of the lateral deltoid muscle is from 0 to 90°. It is common for the Kohnstamm phenomenon to produce aftercontractions where the arm rises to 90° (Adamson & McDonagh, 2004; De Havas et al., 2015; Fessard & Tournay, 1949; Kohnstamm, 1915; Paillard, 1951; Parkinson & McDonagh, 2006; Salmon, 1925; Sapirstein et al., 1937). Thus, if the aftercontraction occurred in one condition and did not occur in another condition, we could reasonably expect the maximum effect size to approach 90°. Bayesian analysis depends on an estimate of the prior probability. No previous study has investigated aftercontractions using varying input inductions (VP and VF conditions). Thixotropy accounts (Hagbarth & Nordin, 1998) predict that isometric inductions might be followed by an aftercontraction, but near-isotonic inductions should not, due to the lack of muscle conditions to produce stable actin-myosin cross-bridges. These clearly contrasting predictions imply that the contrast between fixed and varying input should produce differences in maximal arm position approaching 90°.

We had less clear predictions about how the control parameter selected for feedback control (force or position) would influence the final arm position. Previous experiments observed aftercontractions using suspended weights (e.g. Sapirstein, Herman, and Wallace 1937) and force regulation (Parkinson & McDonagh, 2006). However, in the case of suspended weight, no previous experiment had provided visual feedback on arm position. It is therefore uncertain how big of a difference in arm position there could be between position feedback and force feedback. We therefore elected to plot Bayes factors for all maximum plausible effects (see Fig. 2.3.). Bayes factors were calculated by taking the likelihood of the obtained data given the theory, divided by the likelihood of obtained data given the null. A Bayes factor below 0.3 is traditionally considered good support for the null hypothesis, while a Bayes factor above 3 is traditionally considered good support for the alternative hypothesis (Dienes, 2014)

Frequency analysis was performed to determine if time-varying motor commands during the induction would persist during the aftercontraction. A first analysis window of 12 s was selected from the middle of the induction period. A second analysis window was selected, starting 1 s after the release instruction, and

again lasting for 12 s (Fig. 2.4a.). Agonist EMG (%MVC) from these time windows was Fourier transformed. Mean amplitude of the 1 Hz spectral peak (i.e., the frequency of the varying visual entrainment signal) was calculated during the induction and during the aftercontraction. A Contraction type (Induction vs. Aftercontraction) by Variability of input (Fixed vs. Varying) by Feedback control (Position vs. Force) repeated measures ANOVA was calculated. This procedure was repeated at 2 Hz, to determine if any effects manifested as harmonics.

## 2.3. Results

### 2.3.1. Aftercontraction size did not differ across conditions

Maximum aftercontraction arm angle was highly uniform across conditions (Fig. 2.2g.). There was no significant main effect of Variability of input on max. angular displacement of the arm during aftercontraction ($F(1,8) = 1.834$, $p = 0.213$). There was also no significant main effect of Feedback control ($F(1,8) = 1.036$, $p = 0.339$) and no Variability of input x Feedback control interaction ($F(1,8) = 2.921$, $p = 0.126$). Peak EMG (% MVC) during the aftercontraction was also similar across conditions (Fig. 2.2e.). There was no significant main effect of Variability of input on peak agonist EMG during aftercontraction ($F(1,8) = 0.034$, $p = 0.858$). There was also no main effect of Feedback control ($F(1,8) = 2.354$, $p = 0.163$) and no significant Variability of input x Feedback control interaction ($F(1,8) = 2.254$, $p = 0.172$). Participants showed a range of mean aftercontraction sizes, but these individual differences were apparently unaffected by experimental manipulations (Fig. 2.2f & 2.2h).

Additionally, we investigated whether the control principle during induction influenced the final aftercontraction arm position, by correlating max. arm angle in the two fixed conditions (average of FP and FF conditions) and max. arm angle in the varying input conditions (average of VP and VF conditions). This showed a strong correlation (Fig. 2.2f: $r = 0.965$, $n = 9$, $p < 0.001$), suggesting a common underlying process. We also found a strong positive correlation across participants in maximum arm angle in the aftercontraction, between conditions where position was the controlled parameter during induction (average of FP and VP conditions) and conditions where force was the controlled parameter (average of FF and VF conditions): Fig. 2.2h. $r = 0.934$, $n = 9$, $p < 0.001$).

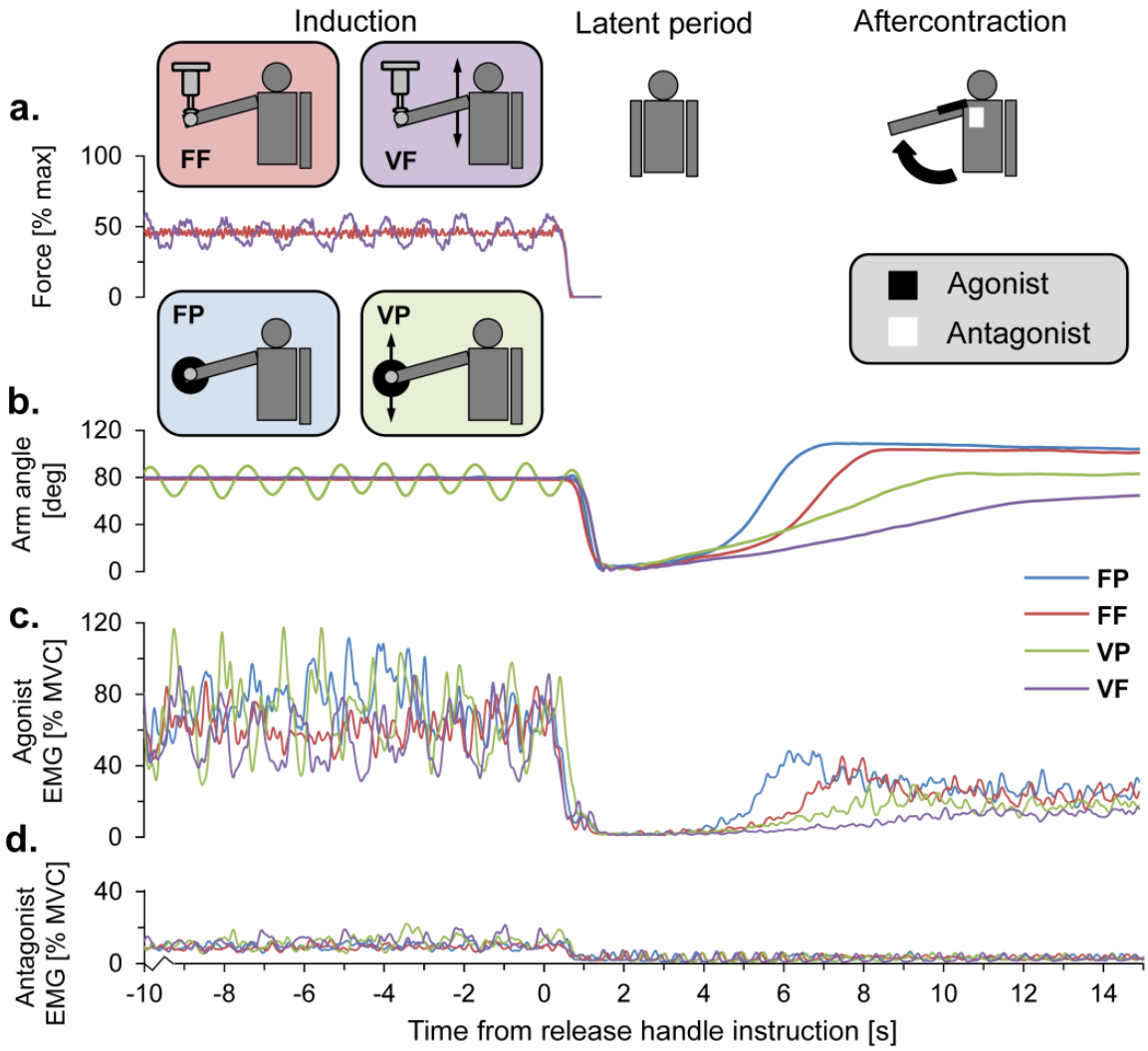



***Figure 2.1. Individual trials.*** *A single trial from each condition from a single representative participant. Note that only the last 10 s of the inductions are shown. The top panel (a) shows force in the FF and VF condition. Note that mean force level is matched. After the release instruction force immediately drops to 0. Shown alongside are schematics of each condition (see main text for details) and the location of the electrodes which recorded from the agonist (lateral deltoid) and the antagonist (pectoralis) muscle. Arm angle is shown for all four conditions (b). Note that during the induction mean arm angle was matched between FP and VP conditions. After the release instruction the arm is relaxed and falls, before starting to involuntarily abduct after a short latent period. EMG (filtered, rectified, smoothed and normalized to MVC) is shown for the agonist (c) and antagonist muscle (d). Note that variability of agonist EMG differs between the conditions during the induction, but that the mean level is the same.*

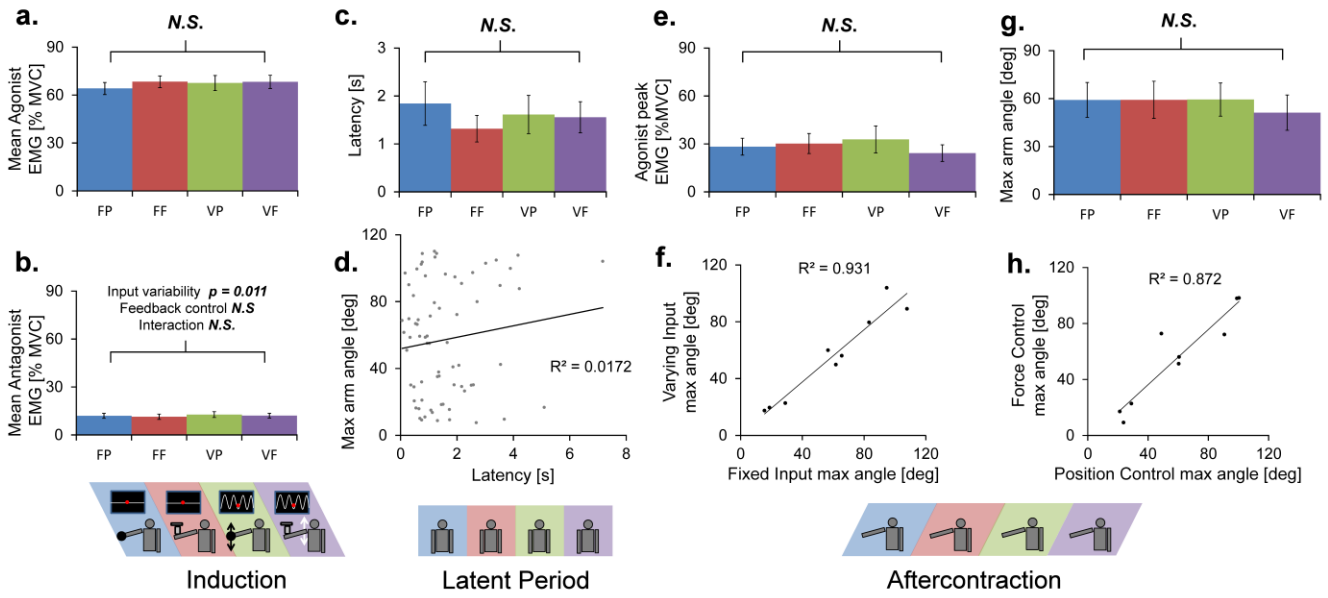



***Figure 2.2. The effect of variability of input and feedback control on the Kohnstamm phenomenon.*** *Mean agonist EMG was matched across conditions (a). Antagonist EMG was low across conditions, but slightly higher in varying input conditions (b). There was no difference in latency across conditions (c). Latency showed a high degree of variability across trials and participants, but did not have any association to the size of the subsequent aftercontraction (d). Peak aftercontraction agonist EMG (e) and max. aftercontraction arm angle (g) did not differ across conditions. Participants showed a high degree of consistency of aftercontraction size across experimental manipulations of input variability (f) and feedback control (h).*

### 2.3.2. Latency duration did not differ across conditions and did not correlate with aftercontraction size

The latency of involuntary movement (time from relaxation of arms to start of movement) has been identified as a key feature of the Kohnstamm phenomenon (Csiky, 1915; Fessard & Tournay, 1949; Kozhina et al., 1996; Matthaei, 1924b; Pinkhof, 1922). A wide range of durations have been reported, ranging from < 2 s (Kozhina et al., 1996; Parkinson et al., 2009; Salmon, 1929) to up to 10 s (Matthaei, 1924b). These discrepancies could reflect different states of the Kohnstamm generator, resulting from differences across studies in the induction protocol. However, this question has not previously been directly addressed. We found that latency of involuntary movement was similar across conditions (Fig. 2.2c.). There was no significant main effect of variability of input on latency ($F(1,8) = 0.00038$, $p = 0.985$), no significant main effect of feedback control ($F(1,8) = 1.535$, $p = 0.25$) and no significant interaction ($F(1,8) = 1.503$, $p = 0.255$). Since previous studies suggested an association between the onset latency of the aftercontraction and its size (Fessard & Tournay, 1949), we additionally correlated aftercontraction latency and maximum arm angle (Fig. 2.2d), but found no clear relation ($r = 0.131$, $n = 72$, $p = 0.272$). This analysis used the trial, rather than the participant, as unit of observation. Therefore, we repeated the analysis after averaging across trials within

participant, and using only the participant as a unit of observation. We again found no significant correlation between aftercontraction onset latency and maximum arm angle ($r = 0.267$, $n = 9$, $p = 0.488$).

### 2.3.3. Mean agonist EMG was matched during induction

Importantly, mean agonist EMG (% MVC) during the induction was similar across conditions (Fig. 2.2a.). There was no significant main effect of Variability of input (Fixed vs. Varying) on mean agonist EMG (% MVC) during the induction ($F(1,8) = 1.282$, $p = 0.29$). There was also no significant main effect of Feedback control ($F(1,8) = 0.757$, $p = 0.41$) and no significant Variability of input x Feedback control interaction ($F(1,8) = 1.441$, $p = 0.264$).

### 2.3.4. Mean force was matched during induction for force feedback conditions

There was no significant difference between the FF condition and VF condition in terms of the mean force exerted on the strain gauge (% max. force) during the induction (50.92 [SD = 2.93] vs. 50.31 [SD = 2.59]; $t(8) = 1.363$, $p = 0.21$). As expected, mean variability (SD) of force exerted on the strain gauge (% max. force) during the induction was much higher during the VF than the FF condition (8.33 [SD = 1.86] vs. 2.19 [SD = 0.55]; $t(8) = 10.171$, $p < 0.001$).

### 2.3.5. Differences in mean arm position during induction did not suppress possible differences between conditions

Mean arm position during induction differed slightly across conditions (FP = 79.97° [SD = 0.39°], FF = 78.53° [SD = 1.35°], VP = 79.48° [SD = 1.22°], VF = 78.75° [SD = 1.22°]). Mean arm angle was 1.09° [SD = 0.9°] higher in conditions where induction involved feedback of position, compared to when feedback involved feedback of force. This may reflect the fact that upward arm movement was prevented in the FF and VF conditions, but free in the FP and VP conditions. This manifested as a significant main effect of feedback control parameter on mean arm angle during induction ($F(1,8) = 13.076$, $p = 0.007$). There was no significant main effect of fixed versus varying input on arm angle during induction ($F(1,8) = 0.446$, $p = 0.523$) and no significant Variability of input x Feedback control interaction ($F(1,8) = 2.952$, $p = 0.124$). To determine if the difference in induction arm angle affected the size of the aftercontraction across participants, this difference (mean angle in position feedback conditions minus mean angle in force feedback conditions) was included as a covariate. Once again there was no significant main effect of Variability

of input (F(1,7) = 0.203, p = 0.666), nor Feedback control (F(1,7) = 1.687, p = 0.235) on maximum arm angle during aftercontraction, nor was there any significant interaction (F(1,7) = 1.506, p = 0.259). Thus, the slight difference in mean arm position during induction was judged irrelevant.

As expected mean variability (SD) of the induction arm angle was high for the condition where the task required movement (VP = 7.49° [SD = 0.99°]), and low for the others (FP = 0.52° [SD = 0.14°], FF = 0.47° [SD = 0.22°], VF = 0.44° [SD = 0.17°]). This manifested as a significant main effect of Variability of input (F(1,8) = 412.249, p < 0.001), a significant main effect of Feedback control (F(1,8) = 348.756, p < 0.001) and a significant interaction (F(1,8) = 502.67, p < 0.001). Planned comparisons showed that this was entirely due to the VP condition, which had significantly more variability of induction arm angle than the FP condition (t(8) = 6.974,p < 0.001), the FF condition (t(8) = 7.02,p < 0.001) and VF condition (t(8) = 7.058, p < 0.001). Other conditions did not differ significantly from one another, indicating the task was performed correctly.

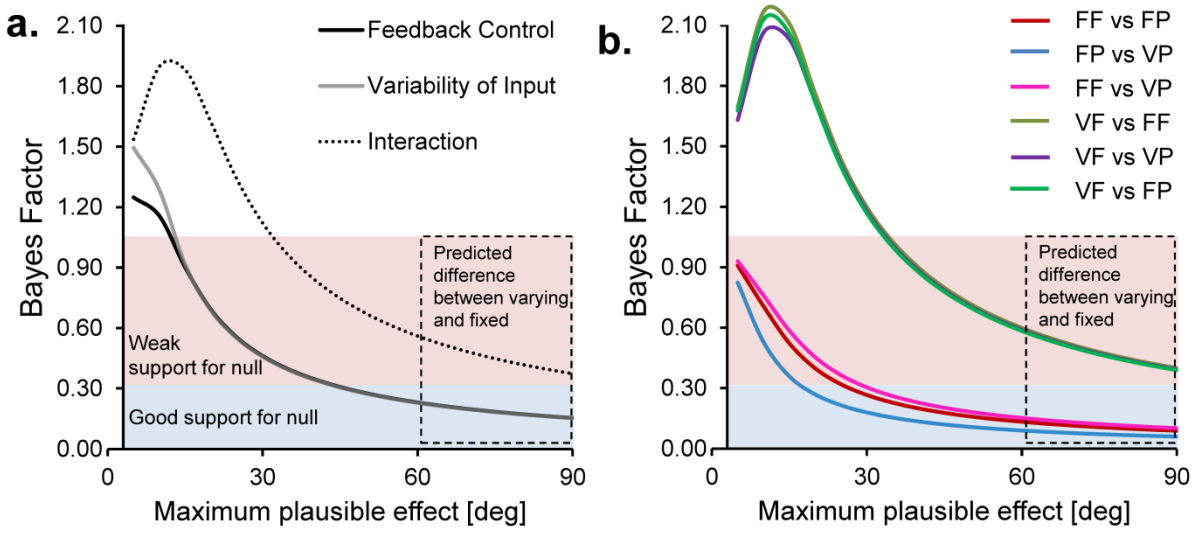



***Figure 2.3. A Bayesian analysis showing support for null hypotheses.*** *The plots show Bayes factors obtained from our comparisons of interest across a range of maximum plausible effect sizes of each main effect and interaction in our experimental design. Previous knowledge about the Kohnstamm phenomenon suggested that if fixed input was a requirement for the aftercontraction to occur, then the maximum plausible effect should approach 90°. This means that for the main effect of variability of induction, we found good support for the null hypothesis (dashed box; a). Likewise when comparing individual trials (b), there was good support for the null hypothesis when the VF condition was not involved, indicating that the lack of difference between the FP, VP and FF conditions was genuine and did not reflect a lack of statistical power (dashed box; b). For the main effect of Feedback control principle and the interaction, the literature allows less clear predictions, but these favoured modest rather than large effect sizes. It is reasonable to think since an aftercontraction was expected (at least in the fixed conditions), the maximum plausible effect would be modest. Therefore, the null hypothesis was less strongly supported (see main text).*

### 2.3.6. Bayesian analyses: confirmation of no induction effect

Bayesian analysis found good support (Bayes factor < 0.3; Dienes, 2014) for the null hypothesis when directly comparing varying and fixed input (Fig. 2.3a). Existing results, based on a thixotropy account of the Kohnstamm phenomenon (Hagbarth & Nordin, 1998), predicted that fixed and varying input should produce differences in final arm position of up to 90°. Using this criterion we found good support for the null hypothesis of equal arm angles following fixed and varying induction protocols (Fig. 2.3a; dashed box).

We also used Bayesian analysis to investigate possible effects of the control principle (Force vs. Position) during the induction. Previous research does not make clear predictions about the plausible effect size for control principle during the induction. Early studies found large aftercontractions using both handheld weights (i.e., Position control) and force regulation tasks (Adamson & McDonagh, 2004; Brice & McDonagh, 2001; Kohnstamm, 1915; Matthaei, 1924b; Parkinson & McDonagh, 2006; Pinkhof, 1922; Sapirstein et al., 1937), so large differences between force and position control principles during induction were not expected. Thus the null hypothesis was not strongly supported (area outside dashed box; Fig. 2.3a). Likewise, the null was weakly supported in the case of the interaction (Fig. 2.3a). Individual comparisons between conditions showed that strong support for the null for all pairwise comparisons except for those comparisons involving the VF condition, in which case support for the null was weak (Fig. 2.3b). However, other direct comparisons resulted in good support for the null hypothesis (dashed box; Fig. 2.3b). It is therefore highly probable that the FP, VP and FF induction conditions activate the Kohnstamm generator in a qualitatively and quantitatively similar fashion.

### 2.3.7. Frequency analysis shows no persistence of motor command

Persistence of the induction motor command in the output of the Kohnstamm generator should produce a similar frequency spectrum in aftercontraction as in induction (Fig. 2.4). There was a clear spike at 1 Hz during the induction in the varying input conditions. However, this did not lead to any corresponding peak during the aftercontraction (Fig. 2.4c). This manifested as a significant main effect of Contraction type ($F(1,8) = 112.934$, $p < 0.001$) on 1 Hz peak amplitude, a significant main effect of Variability of input ($F(1,8) = 95.373$, $p < 0.001$), but no significant main

effect of Feedback control ($F(1,8) = 2.165$, $p = 0.179$). There was a significant Contraction type x Variability of input interaction ($F(1,8) = 95.660$, $p < 0.001$), but no significant Contraction type x Feedback control interaction ($F(1,8) = 1.711$, $p = 0.227$), no significant Variability of input x Feedback control interaction ($F(1,8) = 2.079$, $p = 0.187$) and no significant Contraction type x Variability of input x Feedback control interaction ($F(1,8) = 1.794$, $p = 0.217$). To explore the significant interaction, individual 2 x 2 ANOVAs were performed for the induction contraction and aftercontraction. For the induction contraction there was a significant main effect of Variability of input ($F(1,8) = 96.542$, $p < 0.001$) on 1 Hz peak amplitude, but no significant main effect of Feedback control ($F(1,8) = 1.941$, $p = 0.201$) and no significant Variability of input x Feedback control interaction ($F(1,8) = 1.954$, $p = 0.2$). For the aftercontraction there was no significant main effect of Variability of input ($F(1,8) = 0.548$, $p = 0.48$) on 1 Hz peak amplitude, no significant main effect of Feedback control ($F(1,8) = 0.00005$, $p = 0.998$) and no significant Variability of input x Feedback control interaction ($F(1,8) = 0.837$, $p = 0.387$).

We also investigated the amplitude at 2 Hz (Fig. 2.4.). Induction amplitude of EMG (% MVC) at 2 Hz frequency differed across conditions (FP = 1.75 [SD = 0.74], FF = 1.44 [SD = 0.74], VP = 4.69 [SD = 1.99], VF = 2.54 [SD = 0.75]). However, aftercontraction amplitude of EMG at 2 Hz frequency was relatively similar across conditions (FP = 0.39 [SD = 0.25], FF = 0.42 [SD = 0.36], VP = 0.37 [SD = 0.26], VF = 0.38 [SD = 0.30]). This manifested as a significant main effect of Contraction type ($F(1,8) = 145.511$, $p < 0.001$ ), Variability of input ($F(1,8) = 51.415$, $p < 0.001$) and Feedback control ($F(1,8) = 11.769$, $p = 0.009$) on 2 Hz peak amplitude. There was also a significant Contraction type x Variability of input interaction ($F(1,8) = 44.997$, $p < 0.001$ ) and a significant Contraction type x Feedback control interaction ($F(1,8) = 11.356$, $p = 0.01$), but no significant Variability of input x Feedback control interaction ($F(1,8) = 3.300$, $p = 0.107$) and no significant Contraction type x Variability of input x Feedback control interaction ($F(1,8) = 3.892$, $p = 0.084$). Separate 2x2 ANOVAS were again performed. During induction there was a significant main effect of Variability of input ($F(1,8) = 48.697$, $p < 0.001$), a significant main effect of Feedback control ($F(1,8) = 11.972$, $p = 0.009$), but no interaction ($F(1,8) = 3.602$, $p = 0.094$). During the aftercontraction there was no significant main effect of Variability of input

(F(1,8) = 0.45, p = 0.521), Feedback control (F(1,8) = 0.118, p = 0.74) or Variability of input x Feedback control interaction (F(1,8) = 0.033, p = 0.861).

We therefore found no evidence for a persistence of the time-varying features of the induction motor command in the aftercontraction motor output.

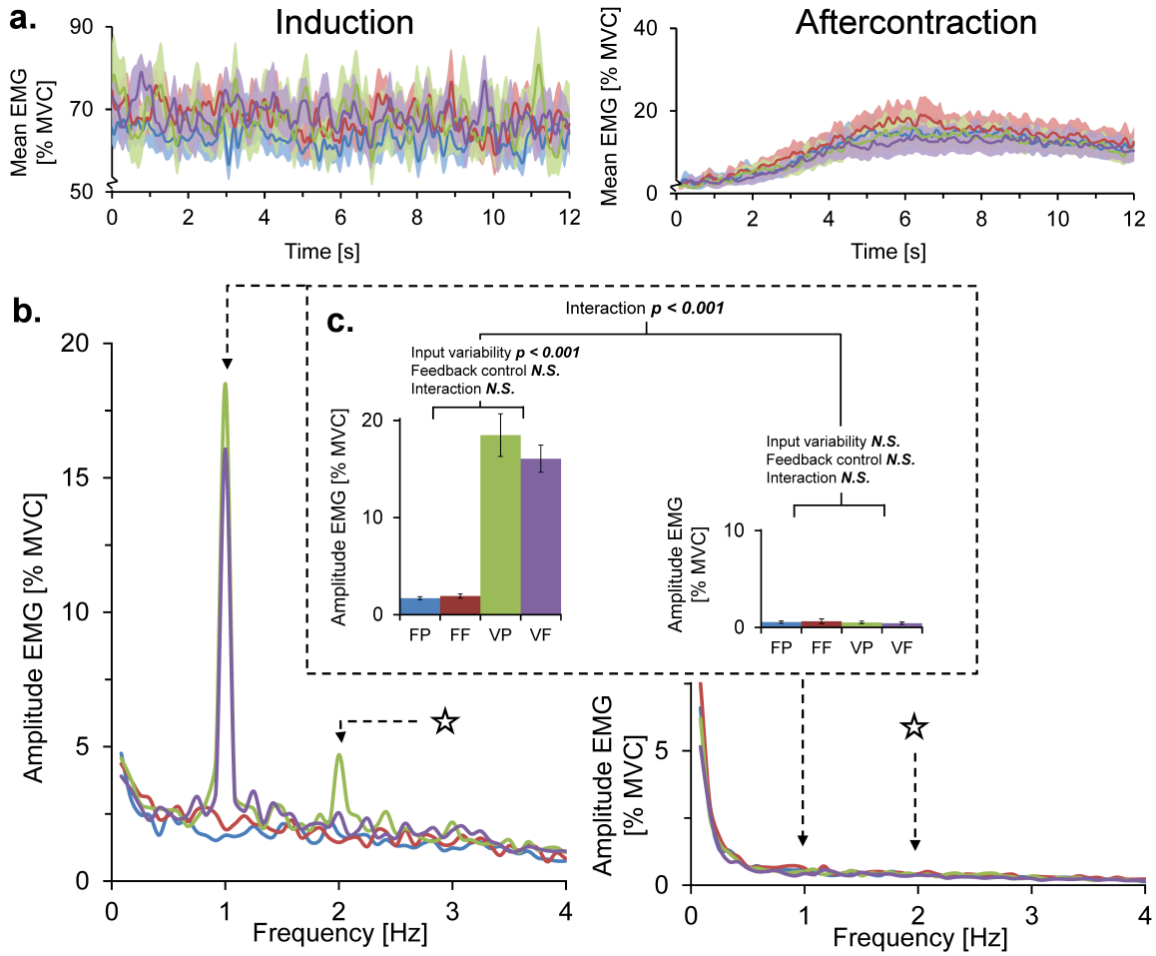



***Figure 2.4. Frequency analysis of agonist EMG during induction and aftercontraction.*** *Frequency analysis was performed during a 12 s window during the induction and aftercontraction. The mean EMG during these windows is shown (a), but Fourier transformation was performed on each trial separately, before averaging across the conditions for each participant (b). As expected, the induction contraction showed a large spike at 1 Hz in the varying input conditions (VP and VF). This was not present during the aftercontraction. Amplitude at 1 Hz did not differ across conditions during the aftercontraction (c). This was also true at 2 Hz (☆).*

## 2.4. Discussion

Surprisingly, we found no difference in the size of the aftercontraction across conditions. All participants produced a measurable aftercontraction in every condition and there was no trend in the data towards any main effects or interactions. Bayesian analysis showed that, given the prior knowledge about how the

Kohnstamm phenomenon is generated, there was a high probability that this similarity across conditions was genuine, rather than merely reflecting a lack of statistical power. Of particular interest was the finding that a near-isotonic, varying contraction produced an aftercontraction of the same size as an isometric contraction. We found no evidence of the structure of the varying induction contraction persisting during the subsequent aftercontraction and no evidence of a correlation between the duration of the latent period and the size of the subsequent aftercontraction.

### 2.4.1. Predictions of the thixotropy account not supported

The thixotropy account of the Kohnstamm phenomenon (Hagbarth & Nordin, 1998) would predict that the Varying Position condition should produce either no aftercontraction or a significantly reduced aftercontraction. According to that theory, the Kohnstamm induction causes stable cross-bridges to form in extrafusal and intrafusal muscle fibres during a prolonged isometric contraction. This stiffness in the intrafusal fibers causes a contraction of muscle spindles after the induction has finished, resulting in an increased spindle firing rate. This triggers the involuntary movement via the usual reflex pathways (Hagbarth & Nordin, 1998). However, during the Varying Position condition the muscle length was continuously varying over a range of 20° of angular displacement. So while we cannot know the exact state of cross-bridge formation, we assume that it would be markedly reduced. Thus, our finding of a large aftercontraction in the Varying Position condition, which did not differ in size from those produced in the other conditions, suggests that muscle thixotropy is not driving the Kohnstamm generator. This claim is strengthened by the fact that our Fixed Force condition, which should have maximised the formation of stable cross-bridges and was highly similar to the inductions used in previous studies (Hagbarth & Nordin, 1998), did not produce a larger aftercontraction than the other conditions.

Previous support for thixotropic accounts came from experiments where the aftercontractions were much smaller (< 8° of angular displacement) than is typically reported in the Kohnstamm literature (Hagbarth & Nordin, 1998). Varying the muscle conditioning procedure presumably induced central changes, which may have led to small differences in the state of the Kohnstamm generator. Indeed, muscle heating and cooling effects, which in the past had been taken as support for the thixotropy

account, have been shown to be reversed across muscle groups (Meigal & Pis'mennyi, 2009), suggesting a central rather than a peripheral effect.

Our finding does not rule out sustained afferent discharge of a different, non-thixotropic origin from contributing to the Kohnstamm phenomenon. Direct recordings from animals and humans suggest there is sustained spindle discharge after isometric contractions (Ribot-Ciscar et al., 1991; Suzuki & Hutton, 1976; Wilson et al., 1995). However, it is not known if this is causative in the Kohnstamm phenomenon. Indirect evidence suggests otherwise. Signalling from muscle spindles contributes strongly to position sense (Kuehn, De Havas, Silkoset, Gomi, & Haggard, 2015; Matthews, 1933; Uwe Proske & Gandevia, 2009; Stuart, Mosher, Gerlach, & Reinking, 1970; Windhorst, 2008), which has been found to be normal during Kohnstamm movements (Heide & Molbech, 1973; Howard & Anstis, 1974). Further, stretch reflexes have been found to be slightly smaller during aftercontractions compared to matched voluntary movements (De Havas et al., 2015). If sustained afferent discharge was driving the Kohnstamm phenomenon, then position sense would be biased in the direction of movement and stretch reflex responses would be large. Such accounts also seem incompatible with the long latent period. If afferent discharge was driving the movement, it should occur within 100 ms of relaxing the arm, since this is the typical duration of the spindle-driven transcortical reflex. However, we observed an average latent period of 1.58 s, consistent with previous reports (Csiky, 1915; Gurfinkel et al., 1989; Kozhina et al., 1996; Matthaei, 1924b; Parkinson & McDonagh, 2006; Pinkhof, 1922; Salmon, 1929). It is possible that afferent discharge has to reach a threshold before movement is triggered. If this were true, then greater activity in this putative peripheral 'Kohnstamm generator' might reduce the time taken to reach this threshold. However, we showed, apparently for the first time, that there was no relationship between the duration of the latent period and the size of the subsequent aftercontraction.

### 2.4.2. Persistence of motor command accounts not supported

Some accounts of Kohnstamm generator invoke activity in the motor cortex that persists after the cessation of the induction contraction (Sapirstein et al., 1937, 1938). This theory is also difficult to reconcile with our findings regarding the latent period. Again, if this theory was correct one would expect to see a negative correlation between the duration of the latent period and the size of the

aftercontraction. Another theory is that the Kohnstamm generator is a reactivation of the motor programs involved in the voluntary induction contraction (Salmon, 1916, 1925). The motor programs involved in generating the induction contraction differed greatly across our 4 conditions. During the induction we observed strong EMG amplitude at 1 Hz in the varying input conditions. We also observed a 2 Hz harmonic. Neither component was present during the aftercontraction. In fact, these components of the EMG frequency spectrum were uniform across the different induction conditions. Thus, while the motor programs differed during the induction, the output of the Kohnstamm generator showed no evidence for their persistence or reactivation. This finding could suggest a separation between the regions where the voluntary motor command originates and the regions that constitute the Kohnstamm generator. Functional imaging and Transcranial Magnetic Stimulation (TMS) studies have shown the primary motor cortex is active during the aftercontraction (Duclos et al., 2007; Ghosh et al., 2014; Mathis et al., 1996; Parkinson et al., 2009). The results of the present study suggest that the primary motor cortex is more likely to be an output region of the generator, rather than housing the Kohnstamm generator itself.

### 2.4.3. The Kohnstamm generator is a low frequency integrator

The lack of any difference between the varying and fixed input conditions suggests that the Kohnstamm generator integrates the inducing signal at a frequency of ≤ 1 Hz. The Kohnstamm generator must integrate input in a continuous fashion, as evidenced by the finding that the duration of the induction positively correlates with the size of the aftercontraction (Brice & McDonagh, 2001; Fessard & Tournay, 1949; Matthaei, 1924b). If the integration window was less than 1 s (i.e., the time constant > 1 Hz), then over the course of a 30 s induction the Kohnstamm generator would be significantly less activated in the varying induction conditions. This fits with the hypothesised link between the Kohnstamm generator and normal postural control (Fessard and Tournay, 1949; Gurfinkel, Levik, and Lebedev, 1989; Ghafouri et al., 1998; Adamson and McDonagh, 2004; Duclos et al., 2004). Postural maintenance and modulation occurs at a lower frequency than voluntary movement. It has been suggested that the Kohnstamm phenomenon represents an amplification of the normal involuntary postural drive, which supplies tonic motor efference (De Havas et al., 2015; Gurfinkel et al., 1989). Thus, the current finding lends support to

this framework and helps to situate the Kohnstamm phenomenon in relation to postural motor control more generally.

It remains unclear what signal the Kohnstamm generator is integrating. We did not find a significant difference between inductions involving position and force feedback control. However, previous literature does not make strong predictions about how strong a difference could be expected. If our null result proves genuine, it suggests that the Kohnstamm generator is insensitive to the task. There are important cognitive differences between position and force control, with accompanying differences in the cortical activation pattern within sensorimotor regions (Noble, Eng, & Boyd, 2013; Ogawa, Inui, & Sugio, 2006). Broadly speaking, the generator could be driven by efferent or afferent signals occurring during the induction. Efferent signal accounts naturally associate with the idea of a persisting motor command, yet we indicated above two key predictions of efferent persistence models which were not supported by our data.

Afferent input could be from muscle spindles and/or GTO. Resolving this issue will require experiments where afferent signal is blocked from the active muscle. Determining if muscle spindles or GTO supply the crucial signal will require first establishing if the TVR and Kohnstamm phenomenon are the same. Previous experiments have shown that the Kohnstamm generator can seemingly be 'reactivated' by the application of a small amount of muscle vibration (Gurfinkel et al., 1989). Muscle vibration is thought to affect primarily spindle signals, rather than GTO signals. If this effect is indeed large and replicable, then it could exclude GTO signals as the input that recruits the Kohnstamm generator. On the other hand, the decrease in aftercontraction with muscle load suggests some modulatory role for GTOs (Parkinson & McDonagh, 2006).

## Chapter 3. Physical obstacles reveal mechanisms of afferent feedback and subjective awareness in unilateral and bilateral aftercontractions.

**Involuntary movements share much of the motor control circuitry used for voluntary movement, yet the two can be easily distinguished. The Kohnstamm phenomenon (where a sustained, hard push produces subsequent involuntary arm raising) is a useful experimental model for exploring differences between voluntary and involuntary movement. Both central and peripheral accounts have been proposed, but little is known regarding how the putative Kohnstamm generator responds to afferent input. We addressed this by obstructing the involuntary upward movement of the arm. Obstruction prevented the rising EMG pattern that characterizes the Kohnstamm. Importantly, once the obstruction was removed, the EMG signal resumed its former increase, suggesting a generator that persists despite peripheral input. When only one arm was obstructed during bilateral involuntary movements, only the EMG signal from the obstructed arm showed the effect. Upon release of the obstacle, the obstructed arm reached the same position and EMG level as the unobstructed arm. Comparison to matched voluntary movements revealed a preserved stretch response when a Kohnstamm movement first contacts an obstacle, and also an overestimation of the perceived contact force. Our findings support a hybrid central and peripheral account of the Kohnstamm phenomenon. The strange subjective experience of this involuntary movement is consistent with the view that movement awareness depends strongly on efference copies, but that the Kohnstamm generator does not produces efference copies.**

### 3.1. Introduction

Ludwig Wittgenstein famously asked “What is left over if I subtract the fact that my arm goes up from the fact that I raise my arm?”(Wittgenstein, 2009). The voluntary command to raise one’s arm is so tightly coupled to the feeling of the arm rising that the two often appear indistinguishable. However, this familiar phenomenology belies the complexity of the motor control hierarchy recruited in even simple voluntary actions. Multiple involuntary processes are required to translate a high level goal into the specific patterns of muscle activity that characterize the initiation, maintenance and cessation of movement (Scepkowski and Cronin-Golomb, 2003; Fowler, Griffiths, and de Groat, 2008; Scott, 2012). Yet the detailed implementation of a voluntary action remains outside conscious awareness: one feels *entirely* in control of a process which, in fact, is merely *initiated* voluntarily. In contrast, when the cause of body movement is external, as when one’s arm is lifted by another person, the event is unambiguously felt as external. Most models of action control suggest that the critical difference between a voluntary action and a passive movement is the presence or absence respectively of an efference copy of the motor command. When sensory information from the moving

arm can be cancelled by an efference copy, the action is perceived as voluntary (Blakemore, Goodbody, and Wolpert, 1998).

Another established distinction in motor control contrasts voluntary movements to reflexes. Reflexes are stereotyped, rapid responses to a specific afferent signal (Kimura, Haggard, and Gomi, 2006). Although not initiated voluntarily, they are modulated by task and voluntary set (Overduin et al., 2012). The awareness of reflexive movements has rarely been studied. Isolating the motor commands of these movements, and determining how they contribute to action awareness is difficult, because of their rapid onset, short duration and close interaction with afferent signals (Ghosh and Haggard, 2014).

Here, we use the Kohnstamm phenomenon (Kohnstamm, 1915) as a convenient experimental model for comparing reflex and voluntary movement, and thus for isolating the specific elements of motor awareness that depend on voluntary control. In the Kohnstamm phenomenon, a strong, sustained, isometric muscle contraction produces, upon relaxation, a sustained aftercontraction in the same muscle. In a classic, party-trick version, participants press outwards with the back of the hands against a doorframe for around 1 minute. Stepping forward away from the doorframe and relaxing the arm muscles is followed by the arms involuntary rising, or 'levitating'. The movement differs from other postural reflexes such as stretch in two ways: it is slow and prolonged, and it is largely confined to a single muscle (Duclos et al., 2004). Crucially, while the involuntary movement produced by the aftercontraction falls within the same temporal and force range as voluntary movement, it feels subjectively very different. The movement is surprising (Forbes, 1926; Craske and Craske, 1985), with the arm feeling lighter than normal (Kohnstamm, 1915; Cratty and Duffy, 1969; Craske and Craske, 1985; Gurfinkel, Levik, and Lebedev, 1989; Hagbarth and Nordin, 1998), as if it is floating (Craske & Craske, 1985; Salmon, 1915), either of its own accord (Craske and Craske, 1985) or via some 'hidden force' (Kohnstamm, 1915).

The Kohnstamm phenomenon has been interpreted as a result of neural adaptation within a postural control system (Gurfinkel, Levik, and Lebedev, 1989; Ghafouri et al., 1998; Duclos et al., 2004; Parkinson and McDonagh, 2006; Duclos et al., 2007). The postural control system is thought to maintain a reference value of motor activity against external perturbation or voluntary movement (Massion, 1992; Adamson and McDonagh, 2004). This implies an ability to adjust to transient afferent

input, before returning to the desired level of motor output. In normal circumstances, many movements include both a postural and a voluntary goal-directed component. These two components are controlled by quite different mechanisms, but may nevertheless be experienced as a single event (Gurfinkel, Levik, and Lebedev, 1989; Ghafouri et al., 1998; Ghosh and Haggard, 2014). In contrast, in the Kohnstamm aftercontraction, a postural component is experienced in isolation, without any voluntary component.

The mechanisms behind the Kohnstamm phenomenon are poorly understood. On one, peripheralist, view, the Kohnstamm generator is driven by a sustained afferent discharge (Gregory, Morgan, and Proske, 1988; Hagbarth and Nordin, 1998; Duclos et al., 2004). Consistent with this view, microneurographic recordings showed increased spindle firing rates following isometric contractions (Ribot-Ciscar et al., 1991; Wilson, Gandevia, and Burke, 1995; Ribot-Ciscar, Rossi-Durand, and Roll, 1998). Muscle thixotropy may result in fusimotor fibres continuing to stretch the spindles after the end of voluntary contraction (Hagbarth and Nordin, 1998). This would in turn generate an aftercontraction via spinal or supraspinal reflexes (Hutton, Smith, and Eldred, 1973; Smith, Hutton, and Eldred, 1974; Durkovic, 1976; Gregory, Morgan, and Proske, 1986; Hagbarth and Nordin, 1998). Indeed, involuntary movement similar to the Kohnstamm can be generated from sustained mechanical vibration applied to a single muscle (Duclos et al., 2007; Gilhodes et al., 1992). Further, vibration-induced and Kohnstamm movements produce a similar pattern of brain activity (Duclos et al., 2007).

Alternatively, the Kohnstamm phenomenon may be caused by a central adaptation. It has been proposed that the Kohnstamm generator is a persistence of the inducing voluntary contraction (Salmon, 1915, 1916, 1925), possibly reflecting changes in the excitatory state of the motor cortex (Sapirstein, Herman, and Wallace, 1936; Sapirstein, Herman, and Wechsler, 1938). Indeed, it has been reported that it is possible to induce the Kohnstamm phenomenon via sustained motor mental imagery (Craske & Craske, 1986). Recent neuroimaging work supports the central adaptation account. Aftercontractions were associated with widespread cortical activations resembling those seen during voluntary movement (Duclos et al., 2007; Parkinson, McDonagh, and Vidyasagar, 2009). Further, applying transcortical magnetic stimulation to the motor cortex during the aftercontraction induces a silent period in the contracting deltoid muscle (Ghosh, Rothwell, and Haggard, 2014). The

silent period did not differ in terms of latency or duration from that obtained during a matched voluntary movement. This suggests that that the motor cortex can be considered part of the Kohnstamm generator.

The Kohnstamm generator may therefore be activated by either peripheral, or central sources, or a hybrid of both. Establishing whether the Kohnstamm generator is altered by sensory inputs may clarify this question. Specifically, a purely central, feedforward generator should be unaffected by peripheral sensory input. A purely peripheral mechanism could, potentially, be entirely reset by a novel peripheral input, stopping the Kohnstamm contraction entirely. Here, we obstruct the rising arm to determine if sensorimotor feedback forms part of the Kohnstamm control circuitry. Because this obstruction has clear perceptual correlates, it can be used to quantify the subjective experience of the aftercontraction. The response to a physical obstruction has proved important in understanding neural mechanisms of central pattern generation (CPG), as in control of stepping behaviour (Duysens and Van de Crommert, 1998; McVea and Pearson, 2006; McVea and Pearson, 2007). However, this approach has rarely been applied to involuntary movements.

Visual and proprioceptive input from the other arm can affect aftercontractions under specific conditions (Brun et al., 2015; Gilhodes et al., 1992). However, only two studies have previously investigated the interaction between aftercontractions and sensory input from physical obstruction. Forbes (1926) reported that contacting an obstacle does not abolish the aftercontraction. Adamson and McDonagh (2004) reported that blocking the rising arm resulted in a constant EMG whose amplitude was proportional to the arm angle at the time of the block. However, these studies did not address how this sensory information regarding obstruction might affect the Kohnstamm generator. Specifically, they did not investigate how the muscle activity changed over time in response to contacting the obstacle, relative to a matched, unobstructed aftercontraction. Further, they did not attempt to quantify the subjective experience of encountering obstruction during Kohnstamm aftercontraction. Finally, they did not address whether obstruction had a lasting or transient effect on muscle activity, nor whether the effects were unilateral or bilateral. Thus, several questions remain about the sensorimotor organization of the Kohnstamm aftercontraction, and in particular about the effects of sensory input from obstruction.

We have therefore conducted two experiments to address the following research questions: 1) Does the Kohnstamm generator rely solely on central

feedforward control or is it modulated sensorimotor feedback? 2) Does one Kohnstamm generator drive aftercontractions in both sides of the body, or does a separate generator exist for each side 3) Is the sensory response of the muscle the same as during voluntary movement? 4) Are the forces from movements produced by the generator perceived differently to voluntary movements? Experiment 1 assessed the effects of random and unexpected obstruction of a unilateral Kohnstamm on EMG. Perception of force relative to voluntary and passive movements was explicitly reported. Experiment 2 assessed the effects of obstructing one arm during a bilateral Kohnstamm and then removing this obstacle. Perception of contact force, relative to voluntary movements, was again investigated, this time via an implicit force matching task.

## 3.2. Experiment 1

### 3.2.1. Methods

#### 3.2.1.1. Equipment

The setup is schematically shown in figure 3.2. Electromyography (EMG) was recorded from bipolar, surface electrodes placed over the middle of the lateral deltoid, parallel to the orientation of the muscle fibers. The electrodes were connected to a 1902 amplifier (Cambridge electronic design), which was controlled via custom Labview scripts (sample rate = 2000 Hz, gain = 1000, 50 Hz notch filter). Pilot studies showed that small changes in posture across trials could lead to large differences in the arm position during aftercontraction. To ensure that the arm was completely stopped on all obstruction trials, a rigid steel rod (length = 20 cm, diameter 1 cm) instrumented with strain gauges was used to obstruct movements. The gauges were connected to amplifiers (low pass filter = 10 kHz, high pass filter = DC, 50 Hz notch filter). However, the strain gauges were calibrated offline, so that the force exerted at a known location on the rod could be calculated. A camera was used to continuously record the force rod so that the position of every arm contact could be coded. Kinematics were recorded via a second video camera (60 fps) and two 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 during shoulder abductions.

### 3.2.1.2. Participants

In total 23 participants (14 female, mean age = 23.8 years old) 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 = 3), or 3) displayed an aftercontraction that did not produce sufficient arm movement to contact the obstacle (n = 3). This left 16 participants (9 female, mean age = 23.6 years old) in the final analysis. 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 local ethical committee approval.

### 3.2.1.3. Procedure

Before the experiment began, participants were instructed to perform a brief maximal isometric voluntary contraction (MVC) of the lateral deltoid muscle by pushing outwards against a wall for 5 s. They were told that from that point on they should aim to reproduce approximately 70% MVC for all subsequent isometric contractions. In line with previous studies of the Kohnstamm phenomenon (Craske and Craske, 1985; Duclos et al., 2007; Ghosh, Rothwell, and Haggard, 2014), we chose to use this subjective criterion of induction force to maximize the likelihood of getting reliable aftercontractions. EMG was monitored online to ensure participants were complying with this level of effort throughout the remainder of the task. A schematic of the entire experiment is shown in figure 3.1. Participants were familiarized with a scale for subjective rating of forces. Participants were told that throughout the experiment they would be using a linear scale from 0-100 to report the amount of force they were experiencing. The experimenter then demonstrated the meaning of the numerical scale by passively lifting the participant's arm against the force rod in order to achieve an announced level of force. Thus, participants learned that an experienced force of 12 N was labelled 33 on the scale, 23 N was labelled 66, and 35N corresponded to 100 on the scale. They were further told that a value of 0 corresponded to no force at all. This procedure aimed to instruct participants in rating a set of equispaced force levels. In practice, there were small variations, because the reading from the strain gauges depended not only on the actual force applied, but also on the location of the contact along the rod. Thus, the

actual force applied during instruction was known only after subsequent calibration taking the position of force application into account.

At the start of each Kohnstamm trial, participants were instructed to stand upright with their palms facing inwards, and their arms relaxed and by their sides. The only object that participants could see was an LED placed at eye level on the opposite wall. The LED was controlled by the experimenter, and was used to trigger the different phases of each trial. The first LED onset signaled participants to begin a continuous, unimanual, isometric contraction of the lateral deltoid at 70% MVC. After 30 s the LED signaled participants to stop pushing, step forward and relax. An aftercontraction of the lateral deltoid then occurred causing the arm to abduct. During 'No Obstruction' trials (Fig. 3.2a) the arm was allowed to rise unimpeded and participants were simply instructed to stay relaxed and let the arm rise and fall whenever it felt natural to do so. In the obstruction trials (Fig. 3.2b) the arm was blocked by the instrumented rod after around 20° of abduction. After ~1 s of contact, a further LED signal instructed participants to report the amount of force they were experiencing using the 0-100 scale. Participants were naïve to whether the obstacle was going to be present or not in any trial, and trial order was randomized.

Kohnstamm trials alternated between the left and right arm. Participants completed 6-9 trials (Mn = 7.44, SD = 1.26), comprising at least 2 no obstruction trials, and at least 4 obstruction trials (Fig. 3.1.). The number of trials could vary because sometimes the arm did not abduct far enough to reach the obstacle. In these instances the trials were repeated. After every Kohnstamm there was a 3 minute rest. Following rest, participants engaged in blocks of 4 Voluntary and Passive trials (in randomized order). These trials were systematically alternated with Kohnstamm conditions, rather than tested in a separate block. We reasoned that alternation would help to prevent long-lasting motor post-effects (Duclos et al., 2004; Hutton et al., 1987). Voluntary trials consisted of the experimenter giving the participant a number on the force scale. The numbers were drawn from 4 distributions centered on 10, 25, 50 and 75 (one from each per block). Participants then had to abduct their arm and push against the force rod with the amount of force they thought corresponded to the number they had been given, based on their previous learning of the scale. The experimenter recorded when the participant felt they had generated the correct amount of force with a button press. On Passive trials the experimenter lifted the participant's arm against the force rod, attempting to

achieve one of four pre-set levels of force (~4, 9, 18, & 26 N), designed to correspond to ratings of 10, 25, 50, & 75 respectively on the previously-learned numerical scale. As before, the experimenter's passive force generation could only be approximately accurate, because the experimenter monitored a raw force signal, and the actual force was known only after offline calibration, taking into account the position of the participant's hand along the force rod. The analysis used the actual, calibrated force levels for each participant. Once the experimenter achieved the target force level, the LED was switched on, and participants verbally reported the current force level, as a rating between 0 and 100. All participants completed 3 blocks of Voluntary trials and 3 blocks of Passive trials (counterbalanced). The experiment lasted approximately 2 hours.

#### 3.2.1.4. Analysis

Kinematics analysis was performed by determining the angle between the horizontal and two LEDs, placed on the shoulder and forearm using ImageJ (Schneider, Rasband, and Eliceiri, 2012). EMG was band pass filtered (10-500 Hz) and rectified. For display purposes the rectified EMG was smoothed with a 4 Hz low pass filter (Fig. 3.3). On obstruction trials, the point in time when the participant made contact with the obstacle was determined from the strain gauges mounted in the obstacle. Four 250 ms bins were created either side of this time point. The mean EMG in each bin across all obstruction trials was then calculated for every participant. Next, using the kinematics data, the angular displacement for the obstacle on every obstruction trial was determined individually for each participant. The mean was then calculated and this was taken as the point in space and time where the obstacle would have appeared on the no obstruction trials. This was performed to account for small variations in the position of the obstacle relative to the participant across trials. Although the obstacle was in a fixed location, minor postural changes meant that the precise angle of the arm when contacting the obstacle could vary across trials. Again four 250 ms bins were created either side of this time point. The mean EMG from each bin across all no obstruction trials was then calculated for every participant. Because the EMG generally increased linearly during this time, a linear trend was fitted to quantify the change in EMG over time, using the standard coefficients -3, -1, 1, 3 for the 4 successive bins prior to the contact, and again for the 4 bins after contact. Contrast coefficients were calculated by multiplying mean

EMG signal in the four 250 ms bins by the standard coefficients. The average EMG trend value could then be calculated for each participant in the two 1 s windows of interest in each of the two conditions. A 2x2 within subjects ANOVA with the variables Time (before contact point vs. after contact point) and Condition (obstruction vs. no obstruction) was then performed on the trend values to assess if contact with the obstacle altered the EMG pattern. Any trial where the participant's arm did not reach the obstacle (obstruction trials), or the corresponding point in no obstruction trials, was excluded from the above analysis.

To calculate the force between the participant's arm and the obstacle, we took into account the position along the steel rod that the participant's forearm made contact on every trial. An analysis window of 500 ms was selected and the mean force within this time-bin was calculated for every trial. In the Kohnstamm and Passive conditions this bin was directly after the onset of the button press/light which instructed participants to report their force ratings. In the Voluntary condition the 500 ms bin was centered on the onset of the button press/light onset to ensure that the analysis corresponded to the point in time where participants felt they had achieved the correct level of force. For every trial the subjective rating of force was divided by the actual force, to produce a value indicating the perceptual intensity per unit of physical force. These values were then averaged across conditions for each participant. Statistical analysis was then performed via a one-way within subjects ANOVA.

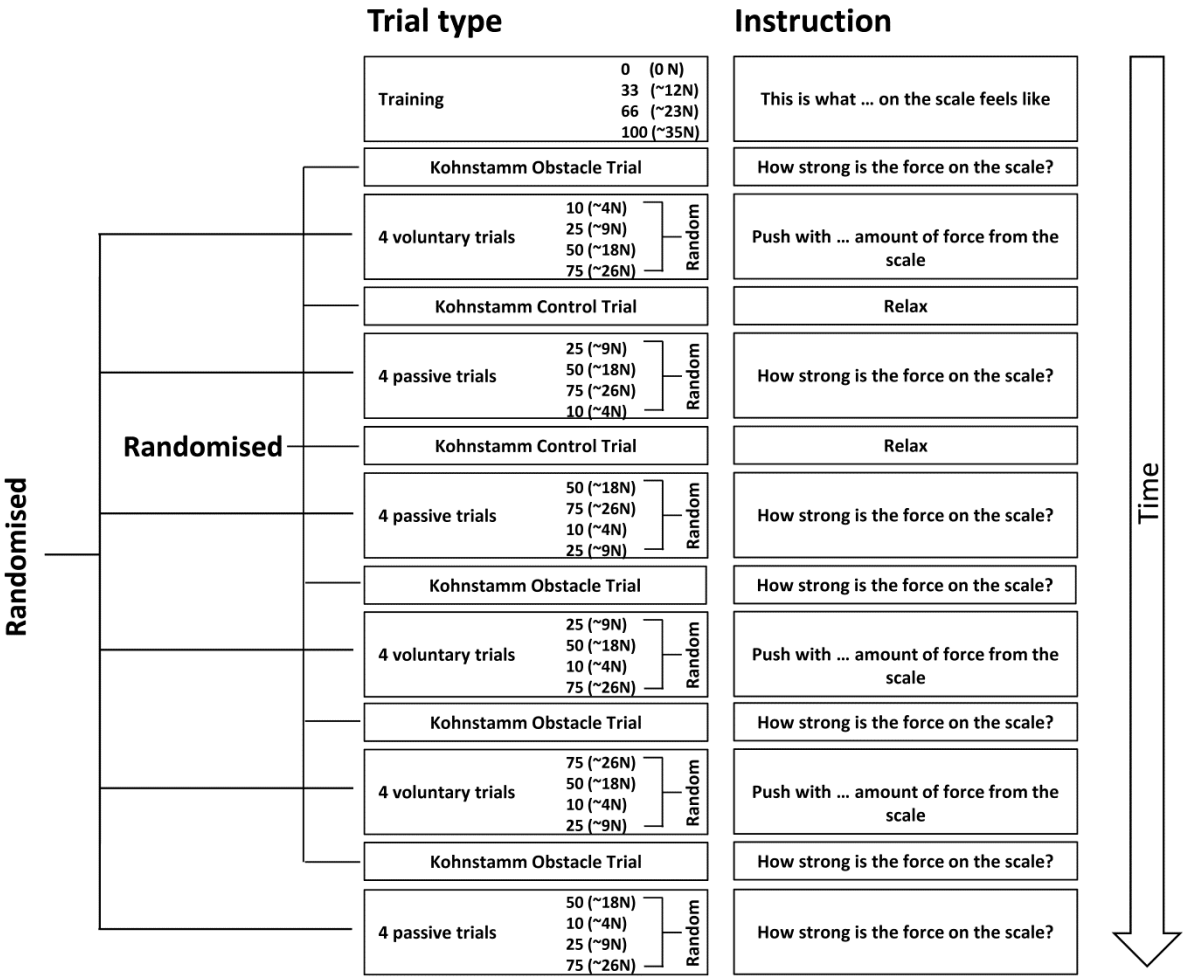



**Figure 3.1**. ***A schematic of experiment 1 showing the order in which the trials were experienced and the specific instructions given to the participants***. *Training was always completed first, followed by a Kohnstamm trial. The order of Kohnstamm trial types was randomized and counterbalanced across participants. Next were blocks of either Voluntary or Passive Movement trials, which were separately randomized and counterbalanced. Within each block of Voluntary or Passive trials there was always one trial at each force level. The specific order was randomized.*

### 3.2.2. Results

#### 3.2.2.1. Obstruction reduces linear trend of EMG relative to an unobstructed Kohnstamm

As can be seen from figure 3.3, contact with the obstacle reduced the linear trend of EMG activity relative to an unobstructed Kohnstamm. The ANOVA based on linear trend analysis showed a significant main effect of Time ($F(1, 15) = 6.5$, $p = 0.02$), a significant main effect of Condition ($F(1, 15) = 5.75$, $p = 0.03$) and significant Time x Condition interaction ($F(1, 15) = 8.85$, $p = 0.01$). Post hoc t-tests showed a significant decrease in the linear trend of EMG during the 1000 ms after contact with

the obstacle, relative to before the obstacle, in the obstruction condition only (t (15) = 3.67, p = 0.002). There was no significant change in the linear trend of the EMG in the no obstruction condition (t (15) = -0.39 , p = 0.7).

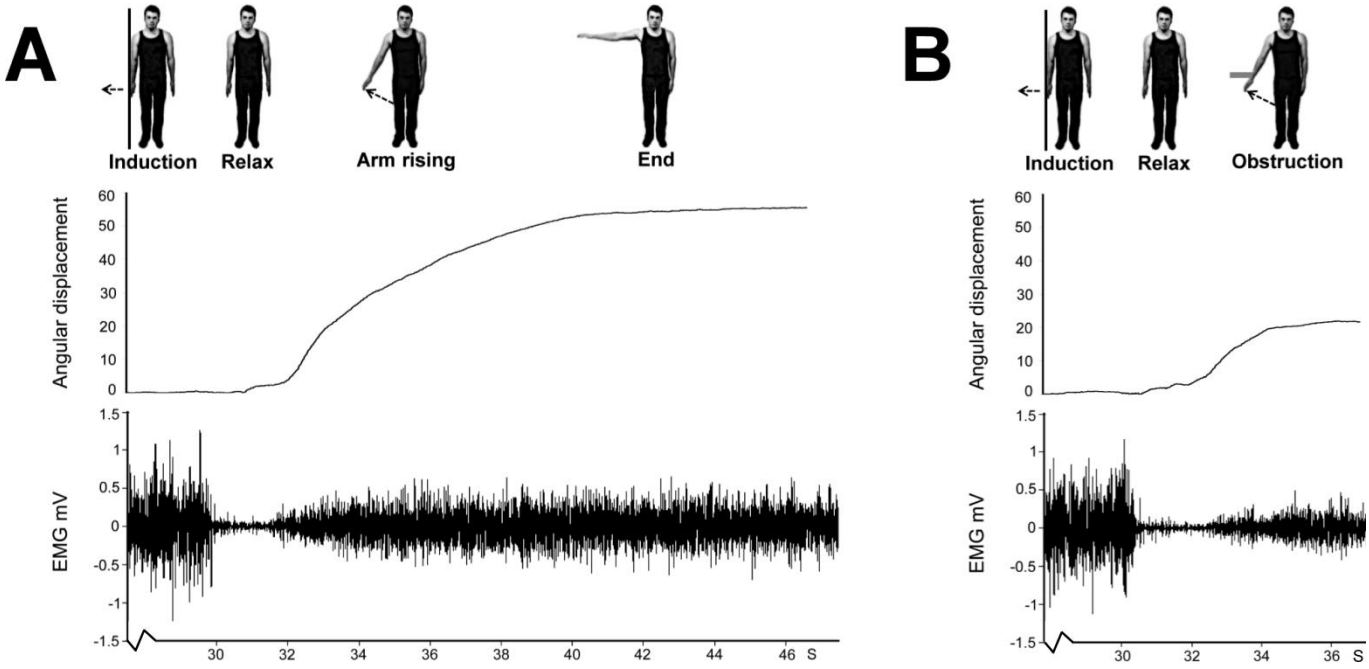



**Figure 3.2**. ***Schematic for experiment 1 showing arm displacement and EMG from a representative no obstruction (A) and obstruction (B) trial***. *Note that only the last ~2 s of the 30 s isometric induction contraction are shown for both trials. This is followed by relaxation of the muscle which lasted ~1.5 s in this participant. The aftercontraction then began, accompanied by involuntary movement. In the no obstruction trial (A) the arm rose unimpeded. In the obstruction trial (B) an obstacle stopped the arm at ~ 20°.*

### 3.2.2.2. Kohnstamm forces are rated as higher than passive and voluntary forces

In the Kohnstamm condition, the mean subjective rating of force divided by actual force was 20.67 (SD=20.68), whereas in the Passive condition it was 3.64 (SD = 1.7) and in the Voluntary condition it was 3.81 (SD = 2.12; Table 3.1). A significant effect of condition was found (F (1,15) = 10.5, p = 0.005, Greenhouse-Geisser corrected). Post hoc t-tests revealed that experienced force was significantly higher in the Kohnstamm condition compared to the Passive condition (t(15) = 3.33, p < 0.05, Bonferroni corrected) and Voluntary condition (t (15)= 3.17, p < 0.05, Bonferroni corrected). There was no significant difference between the Passive and Voluntary conditions.

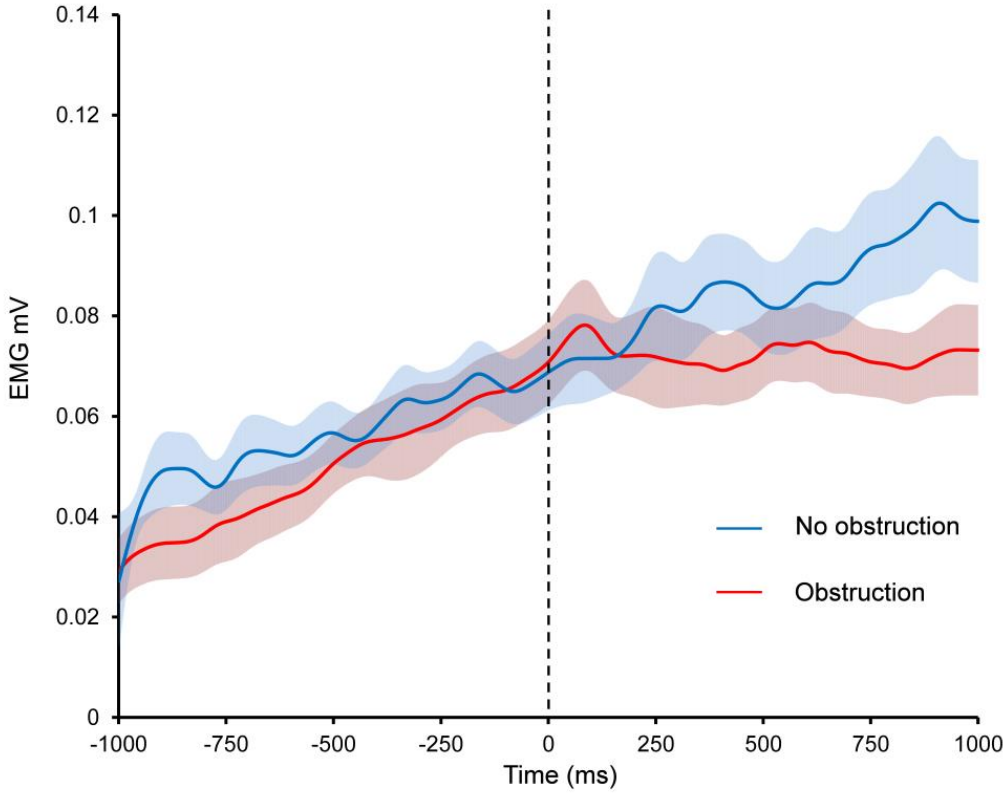
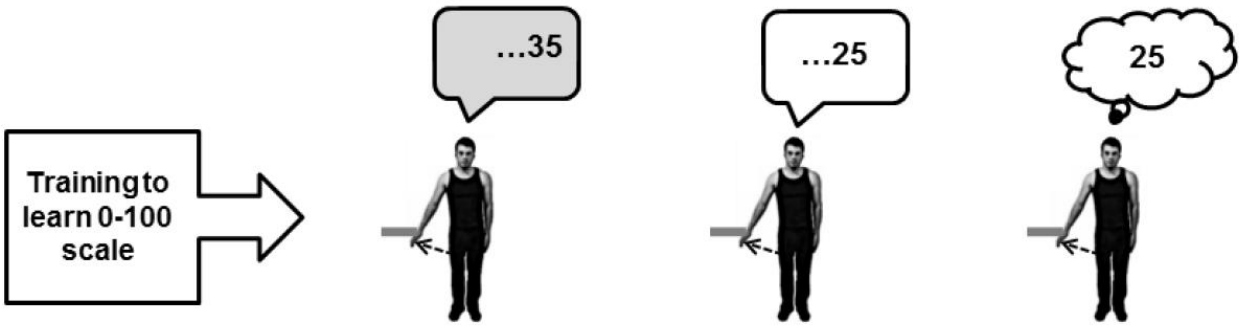



**Figure 3.3**. ***The effect of obstruction on EMG during Kohnstamm***. *Dashed line indicates time of obstruction in obstruction condition and time when obstruction would have occurred in the no obstruction condition. Error bars show SEM.*

**Table 3.1**: ***Rating of force divided by actual force for Kohnstamm, Passive and Voluntary movements.***




| Rating/Force | Kohnstamm | Passive | Voluntary |
|---|---|---|---|
| Mean | 20.67 | 3.64 | 3.81 |
| SD | 20.68 | 1.70 | 2.12 |

### 3.2.3. Discussion

Obstructing a Kohnstamm aftercontraction with an obstacle produced a clear plateau in the agonist EMG signal. A single EMG trace from a single participant in an earlier paper shows, but does not quantify, a similar phenomenon (Forbes, 1926). Later work examined the effect of stopping the Kohnstamm at different arm angles (Adamson & McDonagh, 2004), but (a) did not include an unobstructed condition, and (b) focused on the EMG level at each angle of arm abduction, rather than how contacting an obstacle affects EMG in the time domain. By comparing obstruction and no obstruction trials, we showed for the first time that it is the obstruction, and associated afferent input, that causes the change in EMG signal. However, two important questions remain. First, is this influence permanent, or does it end when the obstacle is removed. Second, how does peripheral sensory information interact with the Kohnstamm generator? These questions are addressed in Experiment 2.

Kohnstamm forces were rated as being subjectively stronger than voluntary and passive forces applied to the same area of the forearm. Overestimation of force during Kohnstamm could reflect lack of an efference copy to cancel against the sensory consequences of the action (Blakemore, Goodbody, and Wolpert, 1998). Efference copy is often invoked to explain the relative underestimation of voluntary compared to passive forces (Shergill et al., 2003). Interestingly, however, we did not reproduce this result in our dataset. Thus a lack of efference cannot fully explain the results of experiment 1 (see general discussion for a consideration of involuntary and passive movements). However, the range of forces in the Kohnstamm condition could not easily be matched to the other conditions. Therefore, the subjective perception results from experiment 1 remain rather tentative. The explicit reporting of force could also encourage participants to respond to the overall ‘strangeness’ of the Kohnstamm, meaning the overestimation of force could be postdictive. As such, an implicit force reproduction task was used in experiment 2.

## 3.3. Experiment 2

### 3.3.1. Methods

#### 3.3.1.1. Equipment

EMG was recorded in the same manner as experiment 1 simultaneously from the left and right lateral deltoid muscles. 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. Unlike Experiment 1, in this experiment it was necessary to have an obstacle that could be applied randomly to each arm in an alternate fashion. Thus the fixed obstacle previously used was inappropriate. Obstacle contact force was recorded using a strain gauge (Mecmesin Advanced Force Gauge) fitted with a flat circular metal disc (diameter = 2 cm). The strain gauge was placed inside a wooden casing that could be braced against the experimenter who stood against a solid surface. Data was acquired in the same manner as experiment 1. A webcam was used to record the session and participants were again fitted with LEDs. Participants also wore earplugs to avoid any sound cues from the experimenter or apparatus regarding the repositioning of the obstacle from one arm to the other.

#### 3.3.1.2. Participants

Inclusion criteria were the same as for experiment 1. In total 18 participants (7 female, mean age = 24.5 years old) were recruited. Of these, 6 were excluded from the final analysis for the following reasons: 1) voluntarily withdrew from the experiment (n=1), 2) did not display an aftercontraction (n=1), never displayed an aftercontraction large enough to produce 20° of angular displacement (n=4). This final exclusion criterion was necessary as the unobstructed arm needed to be capable of rising above the point in space where the obstacle was applied (~15°) for the analysis to be meaningful. This left 12 participants in the final analysis (3 female, mean age = 25.2 years old). None of these participants had participated in experiment 1.

#### 3.3.1.3. Procedure

The participant's MVC was established as before, and they were once again instructed to push with 70% MVC to induce a Kohnstamm effect. Kohnstamm trials were the same as in experiment 1, with the important difference that this time participants pushed outwards with *both* arms. Participants were simply instructed to allow any arm movements that might follow the induction process. As the aftercontraction began, the experimenter obstructed one arm after ~15° of angular displacement using the braced strain gauge applied to the dorsal forearm just above the wrist. The other arm was free to rise unobstructed (Fig. 3.4). Based on pilot experiments, it was hypothesized that removing the obstacle after a short duration

would result in the arm continuing to rise involuntarily. This would require an increase in EMG. The obstacle was thus removed after ~2 s allowing the obstructed arm to rise. Participants knew that one arm would be obstructed on each trial, but were unaware which it would be. They were instructed to remember the force with which their arm had hit the obstacle. Once both arms had ceased moving, participants were told to bring their arms back to the start position and relax. The experimenter then verified that the arm was completely stationary and all signs of the aftercontraction had ended. After 1 minute participants were told to reproduce the force with which they had just hit the obstacle via a voluntary movement. Unlike experiment 1, here participants had not been told about any subjective force scale. The obstacle was in the same position as during the aftercontraction.

After a 2 minute rest, participants then completed a voluntary trial. On these trials participants were instructed to raise both their arms at the same speed as during the Kohnstamm trials. Once again the experimenter would obstruct one of the arms for 2 s at ~15° of angular displacement and then release it. The other arm was free to rise unobstructed. Again participants were naïve to which arm would be obstructed. Once both arms had stopped moving the experimenter instructed the participant to bring them down. As before, they were instructed to remember the force with which they hit the obstacle and after 1 minute reproduce that force.

Participants completed 4-6 Kohnstamm trials (Mn = 5.08, SD = 0.67) and a matched number of Voluntary trials. Trial number varied because sometimes it was necessary to repeat a trial where the arms did not rise past ~15° of angular displacement. The obstructed arm was independently randomized for the Kohnstamm and Voluntary trials to minimize any expectation on the part of the participant. During post-test questioning all participants stated that they could not guess in advance which arm would be obstructed. The experiment lasted ~2 hours.

#### 3.3.1.4. Analysis

EMG analysis centered on the contact with the obstacle, as experiment 1. The detection of contact with the obstacle was based on the signal from the strain gauge. Statistical analysis was broadly as in experiment 1. The factor of Time (before contact point vs. after contact point) was included in the ANOVA. We also included a

factor of Arm to distinguish between the arm that did contact the obstacle on each trial, and the other arm that did not.

Unlike experiment 1, the obstacle was removed after ~2 s, and the arm released. The effects of releasing were investigated in the same way as the effects of contacting the obstacle: resampling of EMG into time bins, linear trend analysis and ANOVA were performed as for the onset of contact. Smoothing (4 Hz) was performed as before only for the purposes of displaying the data (Fig. 3.5). In the case of the release-locked analysis, data is shown for 2 s after the release (statistical analysis performed on 1 s, split into 4 bins). The additional 1 s of data was included to determine whether the EMG in the obstructed arm reached the same level as the unobstructed arm. A direct comparison via t-test was performed on the final 250 ms bin across both arms.

We specifically investigated EMG transients just after contact with the obstacle to measure possible stretch reflexes. An analysis window of 60-160 ms post-contact was used, as this is thought to correspond to long loop transcortical reflexes (Conrad and Meyer-Lohmann, 1980; Matthews, 1991). Since EMG increases during the Kohnstamm, any reflex would be superimposed on an underlying Kohnstamm pattern. We therefore used a special procedure to estimate reflex amplitude despite absence of a stable baseline. EMG from the obstructed arm was extrapolated from before the contact with the obstacle (-800 ms – 0 ms; linear regression) forwards in time beyond the contact with the obstacle. The actual EMG within the reflex analysis window (60-160 ms post contact) was then subtracted from this extrapolated signal within the same time window. This was performed for all Kohnstamm and Voluntary trials, and the mean stretch reflex amplitude was calculated in each participant. To determine whether a stretch response was present, a one sample t-test against 0 was performed in each condition. Differences across conditions were determined via a within subjects t-test.

We also investigated the detailed pattern of EMG during the obstacle phase at the level of single trials, to determine how afferent input from the obstacle affected the putative Kohnstamm generator. The previous linear trend analysis was insensitive to whether the EMG signal was truly flat during contact with the obstacle or just appeared that way due to averaging. We examined the first derivative of the rectified and smoothed EMG signal for both arms to quantify positive and negative signal change at the level of the individual trial (Julkunen et al., 2013). The positive

and negative area under the curve (AUC) of the first derivative was calculated during several time windows for each individual trial, and divided by the duration of each window. The time windows of interest were: when the muscle was at rest (1000 ms window at start of the trial, prior to the induction and aftercontraction), immediately before contact with obstacle (500 ms window), during entire contact with the obstacle (~750 ms, first 250 ms excluded due to possible stretch responses), and immediately after release of the obstacle (500 ms window).

Signals from the strain gauge were analyzed to determine force perception and reproduction. The force with which the participant made contact with the obstacle was calculated by taking the amplitude of the first peak in the signal post-contact (Fig. 3.9b). This was done to ensure the analysis matched the instruction for the participants to remember the initial contact force. Contact force was defined as the first peak in the signal from the strain gauge. We chose this approach to make our experiment commensurate with previous studies of sensory suppression which used discrete taps (Shergill et al., 2003). This was performed in four conditions: for all Kohnstamm trials, Voluntary trials and subsequent reproduction of forces on Kohnstamm and Voluntary trials. The mean contact force in each condition was analyzed with 2x2 within subjects ANOVA with the variables force type (initial force vs. force reproduction) and movement condition (Voluntary vs. Kohnstamm).

Video data was analyzed using ImageJ (Schneider, Rasband, and Eliceiri, 2012) from 11 participants to determine: 1) angular displacement of the obstructed arm when it contacted the obstacle on Kohnstamm trials, Voluntary trials and force reproduction trials, 2) the maximum angle of both arms during Kohnstamm trials, and 3) effect of the obstacle on the angle of participant's trunk (posture). Data was lost for 1 participant due to recording equipment failure.

### 3.3.2. Results

#### 3.3.2.1. Effect of obstructing one arm on EMG in the other

During Kohnstamm, obstructing one arm caused EMG amplitude in that arm to change from its usual rising pattern (Fig 3.5.) in the same manner as was seen in experiment 1. However, there was no such change in the unobstructed arm. This manifested as a significant main effect of Arm ($F(1,11) = 8.02$, $p = 0.02$), a significant main effect of Time ($F(1,11) = 12.88$, $p < 0.01$) and a significant Arm x Time interaction ($F(1,11) = 8.59$, $p = 0.01$). Planned comparisons revealed that during

Kohnstamm the obstacle produced a significant change in the linear trend of the EMG signal from the obstructed arm ($t(11) = 4.04$, $p < 0.01$). There was no significant change in EMG acquired simultaneously from the unobstructed arm ($t(11) = 0.81$, $p = 0.43$).

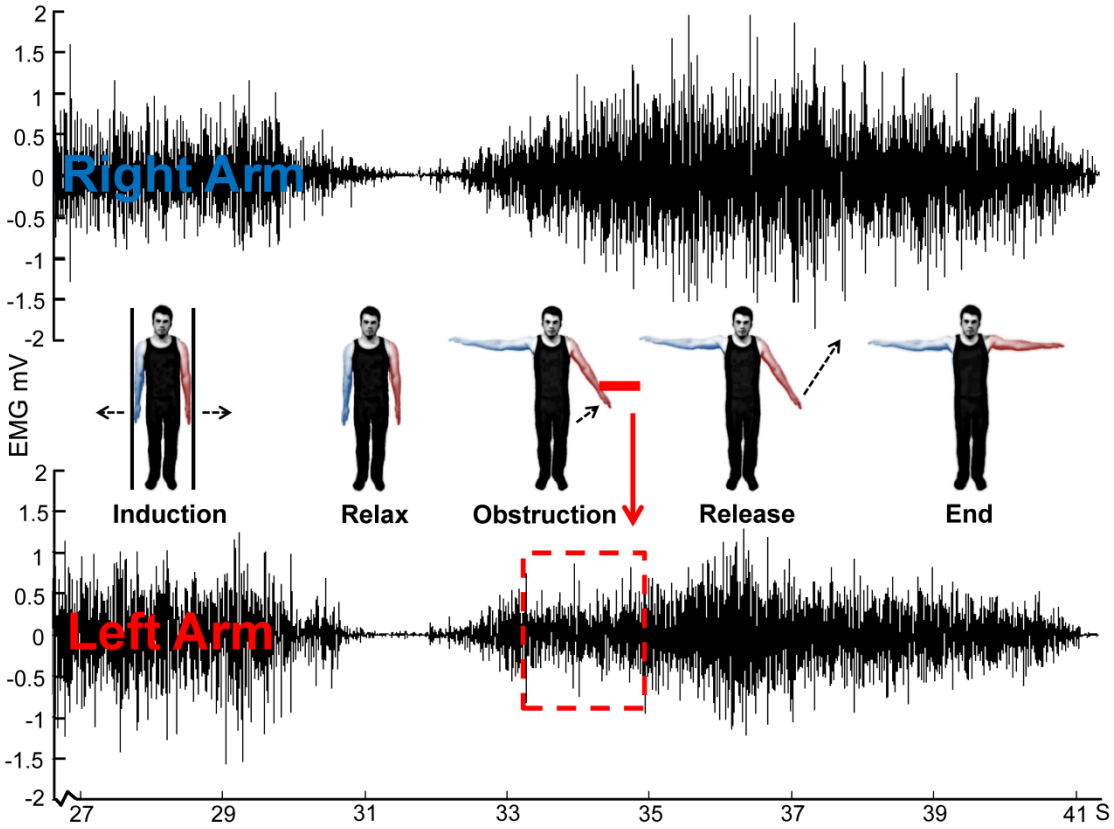



**Figure 3.4**. ***Schematic for experiment 2 showing EMG of obstructed left arm and unobstructed right arm from a single representative trial***. *Note that only the last ~3 s of the 30 s isometric induction contraction is shown.*

### 3.3.2.2. EMG increases following obstacle removal

As can be seen from figure 3.5, the removal of obstruction during Kohnstamm was accompanied by an immediate increase in the linear trend of EMG from the previously obstructed arm. ANOVA showed a significant Time x Arm interaction ($F(1,11) = 6.09$, $p = 0.031$), and no main effects of Arm or Time. Simple effects t-tests were used to investigate this interaction. We found that during Kohnstamm there was a significant increase in the linear trend of the obstructed arm EMG after release from the obstacle ($t(11) = -3.23$, $p < 0.01$). In contrast, t-tests revealed no significant effect of the obstacle release on the arm that was not blocked by the obstacle ($t(11) = 1.82$, $p = 0.096$).

During Kohnstamm the EMG of the obstructed arm continued to increase after unblocking. There was no significant difference between the final EMG of the obstructed arm (mean = 1.11 mV, SD = 0.06 mV) and unobstructed arm (mean = 1.11 mV, SD = 0.06 mV; t(11)= 0.48, p=0.64). Indeed, there was no significant difference between the maximum angular displacement of the obstructed arm (mean = 39.5°, SD = 19.76°) and unobstructed arm (mean = 39.83°, SD = 21.6°; t(10)= 0.31, p=0.76) on Kohnstamm trials.

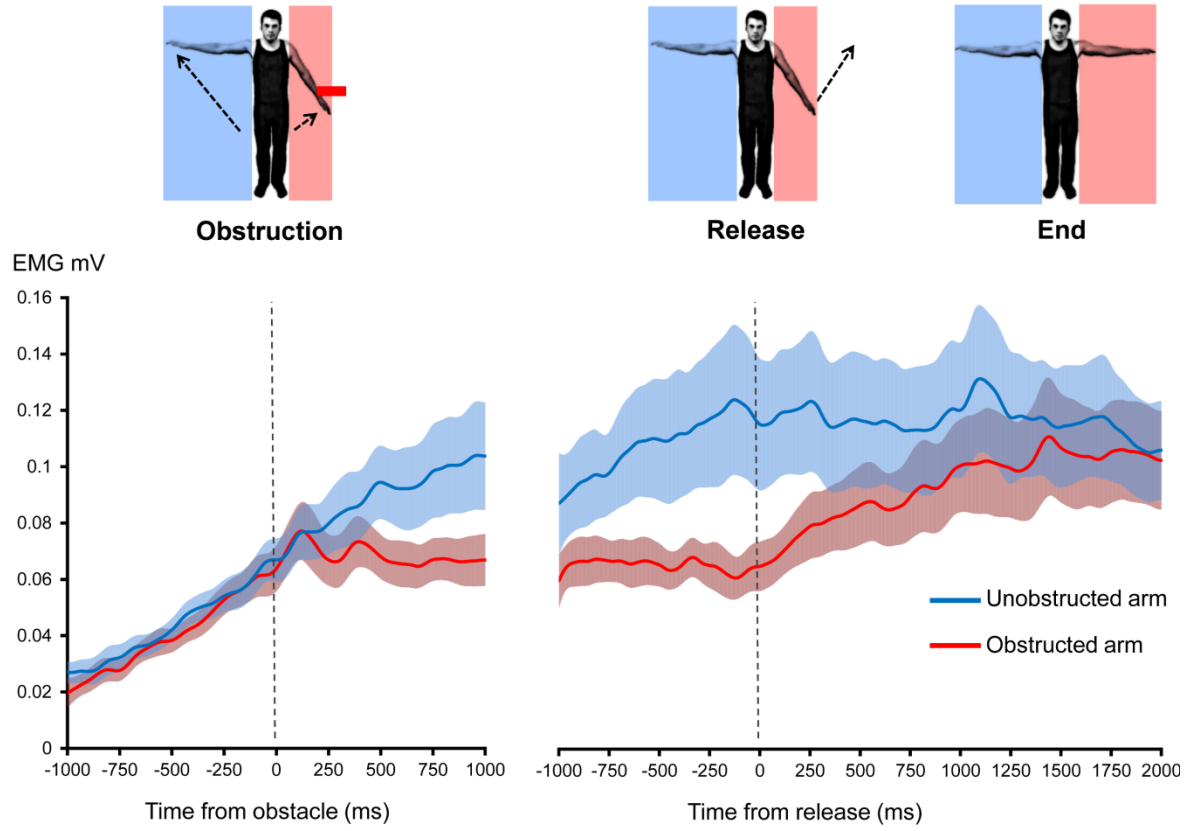



**Figure 3.5**. ***Effects of introduction and removal of an obstacle on both the unobstructed and obstructed arm during bilateral Kohnstamm.*** *Error bars show SEM.*

### 3.3.2.3. Stretch reflex response is preserved during Kohnstamm

A significant, transient increase in obstructed arm EMG (Fig. 3.6.) was found in *both* the Kohnstamm (t(11)=2.7, p = 0.02) and Voluntary movement (t(11) = 2.52, p = 0.03) conditions after contacting the obstacle (60-160 ms post contact). However, the magnitude of this increase did not significantly differ *across* Kohnstamm and Voluntary movement conditions (t(11) = -0.81, p = 0.43).

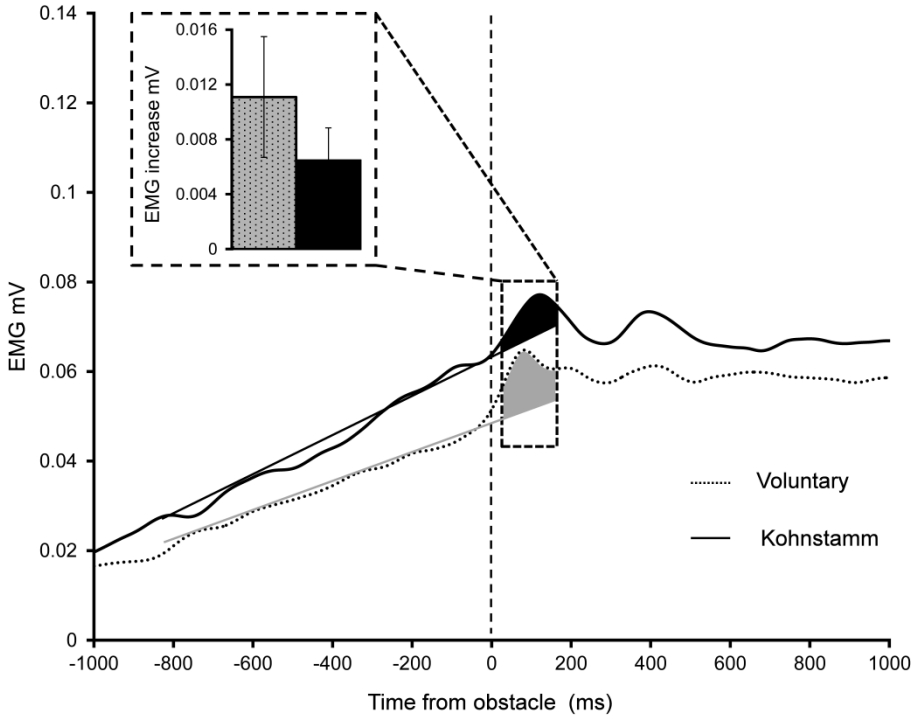



**Figure 3.6**. ***Increase in EMG 60-160 ms post-contact with obstacle during Voluntary and Kohnstamm movements****. Insert shows the mean increase in EMG relative to a trend line fitted to pre-contact EMG on every trial. Trend line is show for illustrative purposes.*

### 3.3.2.4. EMG during Kohnstamm obstruction: plateau or oscillation?

Inspection of grand average EMG gives the impression that the EMG is flat during contact with the obstacle on Kohnstamm trials. However, inspection of individual trials suggested an oscillating pattern (Fig. 3.7.), with periodic increase and decrease of EMG throughout the obstacle contact phase. Because these oscillations were poorly time-locked to contact with the obstacle, they produced a flat EMG trace after averaging. To characterize this oscillatory pattern, we computed the signed positive and negative areas under the EMG first derivative. On Kohnstamm trials both positive ($t(12) = 8.77$, $p<0.001$) and negative ($t(12) = 9.51$, $p<0.001$) EMG signal change were significantly higher during obstruction than when the muscle was at rest (Fig. 3.8.). Positive EMG signal change remained stable from before contact to during contact with the obstacle ($t(12) = 0.10$, $p=0.92$). Contrastingly, negative signal change significantly increased ($t(12) = 6.48$, $p<0.001$) after obstruction compared to immediately before. This suggests strong downward adjustment in EMG triggered by contacting the obstacle. On Kohnstamm trials, when the arm is released from obstruction a significant reduction in negative signal change ($t(12) =$

4.04, p<0.01) and a trend towards increased positive signal change (t(12) = 2.20, p = 0.05) was found, relative to during contact phase.

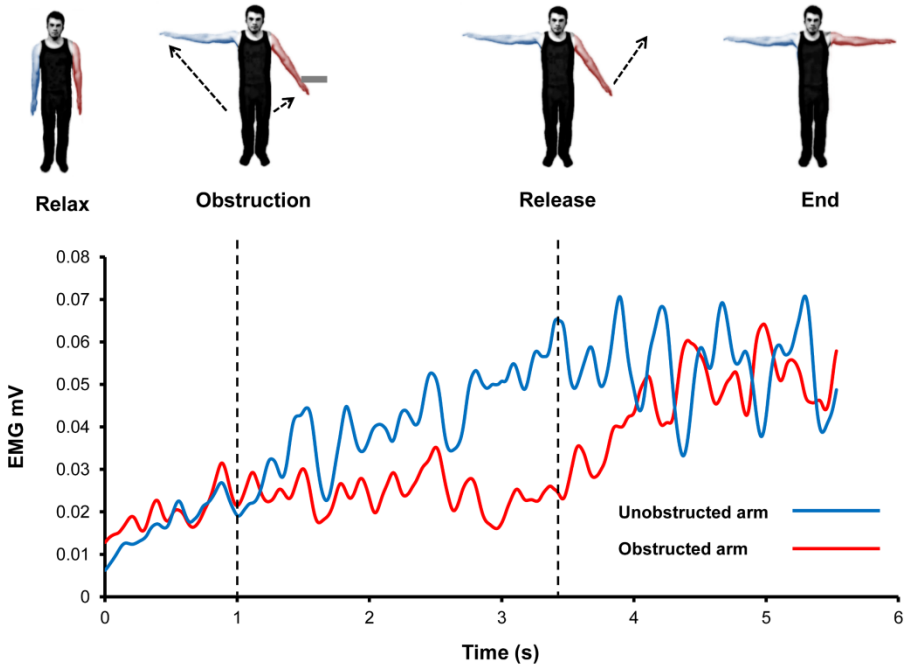



**Figure 3.7.** ***Rectified and smoothed EMG from both arms from a single representative trial (illustrates the signal oscillation during contact with obstacle)***

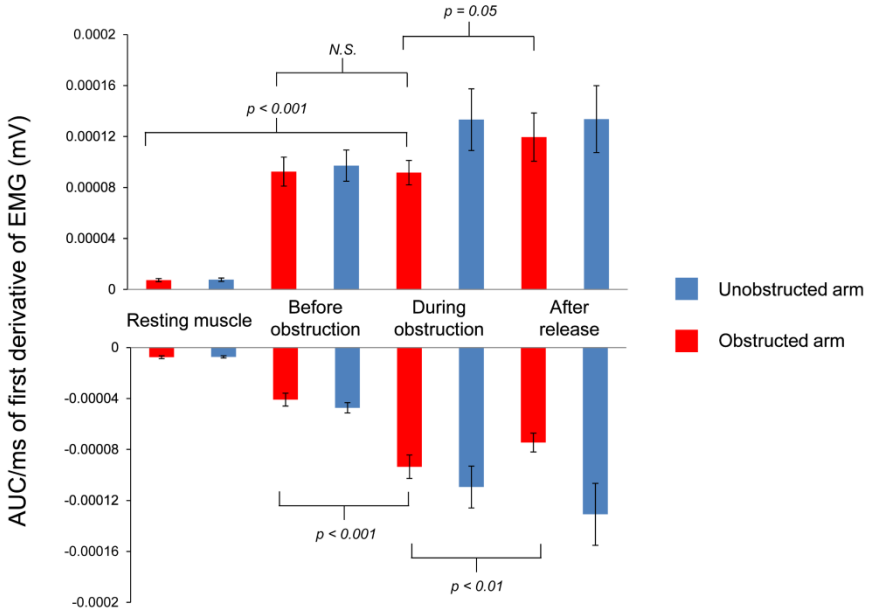



**Figure 3.8.** ***Group average positive and negative AUC of first derivative of EMG for both Obstructed arm and Unobstructed arm****. Resting muscle refers to 1000 ms window at the start of the trial, before the Kohnstamm induction. Before obstruction refers to a 500 ms window immediately prior to contact with obstacle. During obstruction refers to a window that includes the entire time in contact with the obstacle (~1750 ms), excluding the first 250 ms (stretch response). After release is a 500 ms window immediately after obstacle has been removed. All AUC calculations are adjusted for the number of samples in each window.*

### 3.3.2.5. Kohnstamm forces are perceived as being stronger than voluntary forces

Figure 3.9a shows participants' attempts to voluntarily reproduce a Kohnstamm contact force. The reproductions were stronger than the initial Kohnstamm force (6.54 N (SD = 3.91) vs. 5.68 N (SD = 4.19)). However, when asked to reproduce Voluntary forces, they reproduced weaker forces than the initial force (7.03 N (SD = 5.09) vs. 7.47 N (SD = 5.04)). A 2x2 ANOVA with factors of movement condition (Voluntary, Kohnstamm) and force type (initial force, force reproduction) showed a significant Type x Condition interaction ($F(1,11) = 5.72$, $p = 0.04$; Fig. 3.9c). There was no main effect of force type ($F(1,11) = 0.1$, $p = 0.76$) or movement condition ($F(1,11) = 2.04$, $p = 0.18$). Post-hoc t-tests to explore the interaction did not find any significant pairwise differences between conditions, showing that the interaction was based on a difference of differences.

It is possible that the differences between Kohnstamm and Voluntary trials may result from differences in arm position or body posture. For this reason video data from all trial types was examined. Mean angular displacement of the obstructed arm at contact with the obstacle did not differ between Kohnstamm trials (mean = 15.03°, SD = 4.3°), Voluntary trials (mean = 15.17°, SD = 4.14°), Kohnstamm reproduction trials (mean =15.12 °, SD = 4.48°) or Voluntary reproduction trials (mean = 15.96°, SD = 4.11°). Contact with the obstacle produced small but significant changes in the angle of the participant's trunk towards the obstacle. This was true for both Kohnstamm trials (mean = 0.76°, SD = 1.12°, $t(10) = 2.25$, $p < 0.05$) and Voluntary trials (mean = 0.64°, SD = 0.86°, $t(10) = 2.47$, $p < 0.05$). However, there was no significant difference between the conditions.

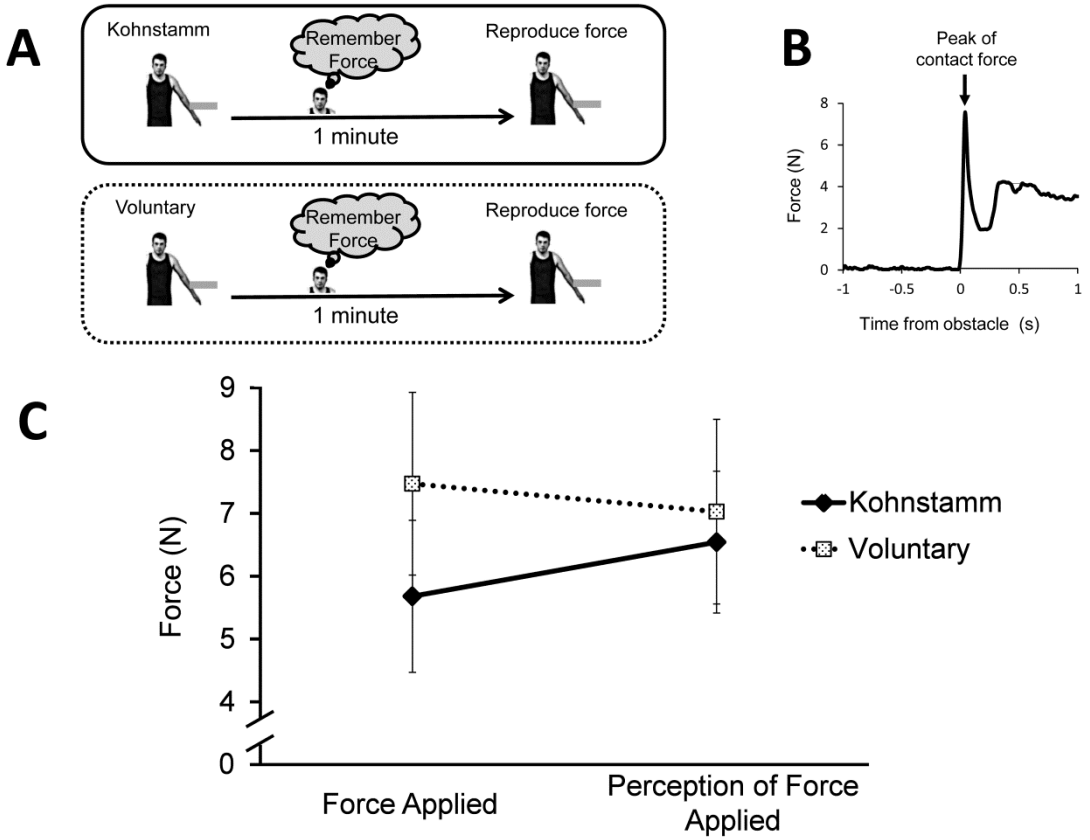



**Figure 3.9**. ***Force of initial Kohnstamm and Voluntary movements and subsequent Voluntary reproductions after 1 minute. A.*** *In both conditions the movement generated a force and participant's had to remember the force and then reproduce it via a voluntary movement.* ***B.*** *Force levels were defined based on the maximum amplitude of the first peak after contact with the stain gauge (shown is the initial force applied during a representative Kohnstamm trial ).* ***C.*** *There was significant interaction between force applied and the perception of that force across Kohnstamm and voluntary conditions (F(1,11) = 5.72, p = 0.04).*

### 3.3.3. Discussion

During a bilateral Kohnstamm, unilateral obstruction resulted in a plateau of the obstructed arm EMG, but had no effect on the unobstructed arm EMG. This suggests there are separate Kohnstamm generators for each arm, and moreover that each generator processes its own arm-specific sensory feedback. Experiment 1 and previous studies (Forbes, 1926; Adamson and McDonagh, 2004) could not resolve whether sensory inputs permanently reset the output of the Kohnstamm generator to a new stable level, or merely temporarily gated the generator output while the obstacle was in place. The results of experiment 2 clearly support the latter view. Removal of the obstacle caused the EMG signal to increase. Post-release EMG resumed the increasing trend seen prior to obstruction. Moreover, the

obstructed arm reached a similar final level of EMG and angular displacement to the unobstructed arm.

EMG signals from the obstructed arm showed that contact with the obstacle produced an oscillating EMG pattern. Taking the first derivative of the EMG signal across the trial revealed that while obstruction is associated with an increase in negative signal change, positive signal change remained constant. These results show that the afferent input does not set the output of the Kohnstamm generator to a lower level. Rather, our data suggests that the generator continues to *specify* a steadily increasing EMG level. At the same time, afferent input associated with obstacle contact triggers an intermittent decrease in EMG. The combination of continuous, efferent-driven EMG increase and repeated, afferent-driven EMG decrease could explain the oscillating EGM patterns that we observed.

A significant, transient increase in EMG, consistent with a transcortical long loop reflex (Conrad and Meyer-Lohmann, 1980; Matthews, 1991), was found after both Kohnstamm and voluntary contact with the obstacle. This putative stretch response did not significantly differ in size between Kohnstamm and voluntary conditions, suggesting that the Kohnstamm induction does not alter the excitability of either the afferent spindle-driven or efferent arms of the long-loop reflex.

Finally our force reproduction task showed that Kohnstamm forces are perceived as stronger than equivalent voluntary forces. This is consistent with the possibility that Kohnstamm generators do not send an efference copy to the neural centers thought to underlie awareness of self-produced force (Blakemore, Goodbody, and Wolpert, 1998; Shergill et al., 2003).

### 3.4. General discussion

We physically obstructed the Kohnstamm aftercontraction by blocking arm movement with an obstacle. This resulted in a halt to the gradually increasing EMG signal that characterizes the Kohnstamm phenomenon. Experiment 1 found this for unimanual aftercontractions, where the occurrence of an obstruction was unpredictable. A similar result was found in experiment 2 for bilateral aftercontractions, when the obstruction could be unpredictably supplied to either arm. Contact with the obstacle was associated with a transient stretch response in the activity of the muscle, which was similar in magnitude to that seen during

matched voluntary movements. Removal of the obstacle caused the EMG signal to resume the characteristic increase found with aftercontractions. This increase resumed the linear trend seen prior to the introduction of an obstacle. Moreover, the obstructed arm reached a similar final level of EMG and angular displacement to the unobstructed arm, albeit with a 2 s delay due to the obstacle. Analysis of individual trials showed that the change in the EMG signal during obstruction was an oscillation with repeated negative corrections preventing the gradual rise of EMG that characterized the Kohnstamm. During bilateral aftercontractions, the unobstructed arm was unaffected by the obstacle applied to the other arm. In both experiments Kohnstamm forces were overestimated relative to voluntary forces.

### 3.4.1. Central models of Kohnstamm generation

Purely ballistic, central feedforward models of the Kohnstamm phenomenon have been proposed based on persistence of the inducing voluntary motor command (Salmon, 1915, 1916, 1925) or cortical excitation (Sapirstein, Herman, and Wallace, 1936; Sapirstein, Herman, and Wechsler, 1938). These purely central models seem inconsistent with our finding of afferent-triggered changes in EMG.

### 3.4.2. Peripheral models of Kohnstamm Generation

The Kohnstamm drive could come entirely from peripheral signals. On this view, the induction phase would lead to some change in a peripheral signal that drives motor circuits. One model views the Kohnstamm phenomenon as a form of proportional-integral-derivative (PID) control, similar to equilibrium point control (Feldman, 1986; Bizzi et al., 1992), proposed for both stretch reflexes and voluntary actions. For such control, a central motor signal setting the equilibrium point of the muscle would result in a follow-up servo contraction of the muscle, causing a movement towards that position. However, in these simple, linear equilibrium-point models, the EMG signal would be greatest at the start of the movement, when the muscle is far from its desired length, and would then decrease. In fact, we found that EMG increases as the arm moves, consistent with previous reports.

Alternatively, the equilibrium point might move gradually over time, defining a virtual trajectory (Bizzi et al., 1984; Hogan, 1985). On these models, the EMG level after release of an obstacle should be higher than before the obstacle was applied, and higher than the EMG level at the same point on unobstructed trials. The

equilibrium point would shift farther ahead of the actual limb position during any period of obstruction, leading to an increased force on release. This pattern was not found in our data.

One influential peripheral account holds that spindle response properties are altered following prolonged isometric contraction during the induction phase (Hagbarth and Nordin, 1998). On this view, Kohnstamm induction might cause a high number of stable cross-bridges to form between actin and myosin in intrafusal fibers. The persistence of these cross-bridges maintains stiffness in the intrafusal fibers leading to excitation of primary spindle endings (Proske, Morgan, and Gregory, 1993), which in turn feeds back to motor regions causing the EMG to increase (Gregory, Morgan, and Proske, 1988; Hagbarth and Nordin, 1998; Duclos et al., 2004).

Indeed, it has been reported that such muscle thixotropy leads to a shift in the perceived position of the elbow joint in the same direction as a previous isometric contraction (Tsay et al., 2014). Perhaps a combination of this sensory change and equilibrium point control explains the Kohnstamm phenomenon. The thixotropy account predicts that Kohnstamm induction should produce a *perceptual* illusion of the shoulder being abducted. However, to produce a *movement* of the shoulder, the equilibrium point of the muscle must also shift, and by an amount greater than the altered sensory signal. The equilibrium point account has been discussed above. However, the experience of the Kohnstamm seems less like a perceptual illusion of position sense than a veridical perception of an unexplained movement. Indeed, previous studies suggest that position sense is normal during Kohnstamm phenomenon (Howard and Anstis, 1974). In addition, we have shown the equilibrium point accounts cannot readily explain the full features of the Kohnstamm EMG pattern. It therefore remains unclear whether such peripheral mechanisms can fully account for the Kohnstamm phenomenon.

We attempted to measure the transient stretch response due to obstruction during the Kohnstamm phenomenon, apparently for the first time. The timescale of the stretch response was comparable to the transcortical long loop reflex (Conrad and Meyer-Lohmann, 1980; Matthews, 1991). Existing peripheral accounts of the Kohnstamm phenomenon posit high spindle sensitivity and/or increased spindle discharge (Gregory, Morgan, and Proske, 1988; Hagbarth and Nordin, 1998; Duclos et al., 2004) during the aftercontraction. We found that the stretch response was

actually slightly (though non-significantly) *smaller* on Kohnstamm movements compared to matched voluntary movements. The state of the muscle spindles in both our Kohnstamm and voluntary movement conditions could not be measured directly. However, our results seem incompatible with peripheral accounts of the Kohnstamm phenomenon that are based on increased excitability.

### 3.4.3. Hybrid models

Our data supports previous claims that both central and peripheral signals contribute to aftercontractions. Our results show that sensory feedback can modulate the putative Kohnstamm generator, but that some aspects of the drive remain independent of sensory input (Parkinson and McDonagh, 2006). Obstructing a movement, as in our data, would trigger simultaneous afferent signals from muscle spindle, skin and tendon receptors, *inter alia*. One model gives force sensing, perhaps from Golgi tendon organs, a key role in the Kohnstamm, by suggesting a positive feedback loop between muscle force and Kohnstamm generator (Parkinson and McDonagh, 2006). However, the effects of release from obstruction seem inconsistent with this model. When an obstacle is removed, there is a sudden decrease in the load on the muscle, (Marsden, Merton, and Morton, 1976), causing a decrease in tendon organ firing. A positive force feedback model would therefore predict a decrease in EMG, at least transiently. Instead, we observed an immediate *increase* in EMG following muscle unloading, and a return to the preceding EMG pattern. We suggest that the immediate resumption of EMG increase on obstacle release must reflect a persistent central drive from the Kohnstamm generator, rather than a feedback loop involving the periphery.

Some models have suggested that Kohnstamm induction causes central excitatory changes within the brain regions responsible for generating muscle tone, and that these changes decay over time (Craske and Craske, 1986; Ghafouri et al., 1998; Gurfinkel et al., 2006). Thus, the 'normal' role of the Kohnstamm generator would be to provide output that achieves and maintains stable muscle lengths, and thus body posture (Fessard and Tournay, 1949; Gurfinkel, Levik, and Lebedev, 1989; Ghafouri et al., 1998; Adamson and McDonagh, 2004; Duclos et al., 2004). Postural control requires peripheral input and central compensatory commands to achieve the desired posture in response to changes in the environment (Cordo & Nashner, 1982). Since the processes for *maintaining* current posture are generally

slow and sustained, it follows that the system would return to an underlying pattern of motor output once the afferent input returned to normal levels. This is consistent with the pattern of results we observed, and with a hybrid model of the Kohnstamm phenomenon. In the case of the Kohnstamm phenomenon, output from the generator is much higher than normal, due to the induction period. The present results indicate that the output from this generator can be gated by afferent signals. We observed that, at the level of individual trials, the EMG signal shows an oscillation during contact with the obstacle. The EMG continually increases, but is then repeatedly reset to a lower level while contact continues. This produces a reduced mean level of activity over time. When contact with the obstacle is ended, the gate is reopened, and EMG again rises. We found that the EMG and angular displacement of the obstructed arm reached the same final levels as the unobstructed arm. EMG increase after obstacle removal was also much more rapid than the 1-3 s it takes for the aftercontraction to begin after the relaxation of the arm (Csiky, 1915; Pinkhof, 1922). These findings indicate that the Kohnstamm generator is not suspended during obstacle contact. Rather, it continues to generate motor commands, but these commands are repeatedly corrected by a circuit driven by afferent input. This could be achieved by a high level generator outputting to a low-level sensorimotor control circuit, which in turn outputs to the muscle. Afferent input would have a suppressive effect on this lower-level circuit, but no effect on the highest level command generator (Fig. 3.10.). Interestingly, two studies reported that voluntary movements immediately after the induction could reduce aftercontractions (Duclos et al., 2004; Hutton et al., 1987), suggesting that the sensorimotor processes underlying the Kohnstamm movement can be partly reset by voluntary commands.

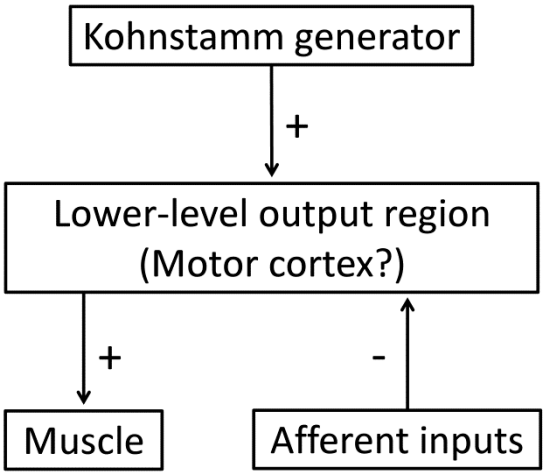



**Figure 3.10**. ***A hybrid model of the Kohnstamm circuit.*** *Note that afferent input has a suppressive effect on the motor commands output from the lower-level motor region, but there is no afferent feedback to the generator itself. See text for further details.*

### 3.4.4. Laterality

Our results indicate independence of the Kohnstamm generators that control each arm. Obstructing one arm after a bilateral induction did not significantly affect the Kohnstamm phenomenon in the other arm. Theoretically, this could also be achieved by a single generator outputting to separate lower level areas, which receive separate afferent input. Nevertheless, this unilateral organization suggests that the EMG effects seen in experiment 1 and 2 are unlikely to reflect a voluntary reaction to contacting the obstacle. Voluntary reactions, particularly fast inhibitory reactions, are generally organized bilaterally (Coxon, Stinear, and Byblow, 2007; Garbarini et al., 2012; Mattia et al., 2012). Previous functional magnetic resonance imaging (fMRI) studies have found wide ranging bilateral activity in sensorimotor and cerebellar regions during Kohnstamm aftercontraction (Duclos et al., 2007; Parkinson, McDonagh, and Vidyasagar, 2009). This suggested that the Kohnstamm generators are not completely separate. However, these inferences are based on correlational neuroimaging data, and cannot distinguish between the generator itself and correlated epiphenomenal activations. Our results indicate that ipsilateral brain activations in these studies may not be output from the Kohnstamm generator to the muscle. Instead it could reflect normal sensorimotor feedback, or some epiphenomenal activation. Previous studies of bilateral aftercontractions reported that the pattern of oscillation in one arm influenced the other (Craske & Craske, 1986), just as in bimanual voluntary movements. Further, proprioceptive input from the ipsilateral arm can influence the velocity of a contralateral aftercontraction (Brun

et al., 2015). Further work is required to characterize the effect of contralateral input on the Kohnstamm movement.

### 3.4.5. Subjective experience during the Kohnstamm phenomenon

Voluntary and involuntary movement may be physically identical, yet subjectively feel very different. The enduring scientific interest in the Kohnstamm phenomenon may relate to the strange feelings it produces (Forbes, 1926; Craske and Craske, 1985). Like other examples of 'voluntariness' and 'involuntariness', these experiences often elude experimental measurement. We developed novel, quantitative and implicit measures of subjective experience during Kohnstamm phenomena, based on the perceived contact force when movement encounters an obstacle. We found that Kohnstamm forces were overestimated relative to voluntary forces in both experiments. This overestimation of Kohnstamm forces is consistent with the view that the Kohnstamm generator does not send the efference copies used to cancel against the sensory consequences of the action (Blakemore, Goodbody, and Wolpert, 1998). The precise origin of efference copies remains controversial. However, several studies suggest efference copies underlying perceptual attenuation of self-generated events originate at a relatively high level of the action control hierarchy, upstream of the primary motor cortex (Haggard and Whitford, 2004; Voss et al., 2006). Neuroimaging studies of the Kohnstamm phenomenon showed activation in primary motor areas during aftercontractions (Duclos et al., 2007), but, interestingly, did not show significant activations of the medial frontal regions hypothesized to generate the efferent signals that contribute to action awareness (Fried et al., 1991; Haggard and Magno, 1999; Haggard and Whitford, 2004; Haggard, 2011).

Lack of efference copy might suggest that the Kohnstamm phenomenon should feel similar to passive movements. However, Kohnstamm and passive movements are easily distinguishable. In fact, our participants rated Kohnstamm forces as being stronger than passive movements in experiment 1, though this result should be interpreted with caution, as we were unable to precisely match the force ranges for the two conditions. In addition, the sensory signals reaching the brain differ profoundly between passive movement and the Kohnstamm phenomenon. In passive movement, there is a strong additional external input not present in the Kohnstamm case, from the experimenter's handling of the participant's passive arm.

It remains to be determined whether the other reported phenomena associated with Kohnstamm movement, such as the lightness of the arm, result from the absence of these additional inputs or some other more fundamental difference between passive and Kohnstamm movement.

### 3.5. Conclusion

In conclusion, the Kohnstamm phenomenon is modulated by peripheral sensory input. Our results are not consistent with the view that the Kohnstamm generator is a simple PID controller, in which a single peripheral signal, such as muscle position or force is driven to a target level by a sensory feedback loop. Rather, the Kohnstamm phenomenon depends on an apparently central generator, whose output is temporarily gated, or limited by the sensory signals produced during contact with the obstacle. Further, the Kohnstamm generator is hemispherically lateralized, and presumably located contralateral to the moving limb. The Kohnstamm generator appears not to transmit efference copies to the brain centers responsible for action awareness, thus explaining some of the strange sensations associated with the phenomenon. Our results fit within a framework that views the Kohnstamm phenomenon as a by-product of adaptations within a complex postural control system. In particular, postural control often requires motor drive to be maintained over long periods while cognitive control capacity is directed towards other tasks. Interestingly, this drive can persist when the peripheral environment changes. Our results also shed important light on the nature of voluntary and involuntary movement control. We show that movements that are involuntary can nevertheless be well-organized, persistent, and environment-sensitive. Despite all the sophisticated information-processing that modulates Kohnstamm after-contractions, they nevertheless *feel* completely different from voluntary actions. Our results highlight that awareness of action involves a complex interplay between central commands and peripheral signals. The interactions between these signals may occur at multiple levels of the motor hierarchy. Most importantly, our results suggest that some specific central generator circuits produce an experience of voluntariness, while others, like the Kohnstamm generator, do not – irrespective of the specific peripheral circuits they engage. Future research might usefully focus on identifying those key features that cause some central motor generators, but not others, to trigger an experience of voluntariness.

## Chapter 4. Perturbations applied to a horizontal aftercontraction suggest negative-position feedback control in the Kohnstamm phenomenon

**The Kohnstamm phenomenon provides a unique opportunity to study the control of an involuntary movement. Previous theories argued the aftercontraction is caused by a small central excitation and force-dependent positive feedback from Golgi tendon organs. Experimental investigations of this theory showed changes in Kohnstamm EMG when external forces were applied to the limb. However, those studies used vertical aftercontractions, in which muscle load varies continuously due to gravity, meaning that effects of gravity and of perturbing forces are superimposed. We investigated a horizontal Kohnstamm movement, and applied perturbing forces with a maniuplandum. Participants induced an aftercontraction of the posterior deltoid muscle via a 30 s voluntary isometric contraction (70% MVC). In the No perturbation control condition the arm moved freely. In the Resistive condition, a continuous torque (0.5 Nm; 250 ms ramp) was applied in the *opposite* direction to the movement once the aftercontraction reached 20° of angular displacement. In the Assistive condition the same torque was applied in the *same* direction as the movement. Velocity matched voluntary movements were also completed for all conditions. Resistive perturbations produced a small, transient increase in agonist EMG, in both Kohnstamm and voluntary movements, while assistive perturbations produced a small, transient decrease. Agonist EMG was higher during Kohnstamm movements than voluntary movements having the same velocity. When we controlled for differences in agonist activity, we found that EMG responses to perturbation were significantly smaller during Kohnstamm movements than during voluntary movements. The results do not support a force-dependent positive feedback model. Instead, the aftercontraction appears to involve a negative feedback loop between a central adaptation, specifying a muscle equilibrium point, and spindle input specifying the disparity between current arm position and the equilibrium value. Crucially, this peripheral feedback loop runs at lower gain in Kohnstamm than in voluntary movements. This implies that the generation of the Kohnstamm aftercontraction may be largely central, rather than peripheral.**

### 4.1. Introduction

Several studies have investigated how voluntary motor control interacts with reflex control. In these studies, the reflex is considered as a brief transient perturbation superimposed on a slower, ongoing voluntary movement. When an assistive or resistive perturbation is applied to a voluntary movement, reflex control compensates for the change in load by attempting to restore the initial, voluntary trajectory (Marsden, Merton, & Morton, 1975; Marsden et al., 1976a; Marsden, Merton, & Morton, 1977). Such reflexive control is multi-level, involving several circuits including the sensorimotor cortex. Reflexive control has some features in common with involuntary movement, and other features resembling voluntary movement (Pruszynski, Kurtzer, & Scott, 2011). It is modified by high-level features such as task and context, as well as more low-level features such as the muscle

length, velocity of movement and level of motor drive (Crevecoeur, Kurtzer, Bourke, & Scott, 2013; Rothwell, Traub, & Marsden, 1980; Scott, 2012). Such perturbation studies have been highly informative not only with respect to reflexive control but the organisation of voluntary movement itself.

However, it is difficult to study *involuntary* movements in the same manner. Most involuntary movements are too rapid to be explored via superimposition of additional perturbations and measurement of reflexive responses. It may be difficult to separate the involuntary movement being studied from further involuntary movements evoked by the perturbation. The Kohnstamm phenomenon avoids these issues, and provides a unique opportunity to study the organisation of control of an involuntary movement, because the involuntary movement lasts long enough for the effects of perturbations to be assessed. In the classic version of the Kohnstamm phenomenon, participants push outwards with a straight arm against a solid surface for around a minute, using the lateral deltoid muscle. If they then relax, the arm lifts up of its own accord without any voluntary command on the part of the participant (Kohnstamm, 1915; Salmon, 1916). The involuntary aftercontraction of the deltoid is slow and sustained, and is kinematically and electromyographically comparable to voluntary movements (Fessard & Tournay, 1949; Mathis et al., 1996; Pinkhof, 1922; Verzár & Kovács, 1925). The phenomenon is more pronounced in proximal than distal muscles (Forbes et al., 1926; Gilhodes et al., 1992; Matthaei, 1924b), leading some to view it as an adaptation within the postural control system (Fessard and Tournay, 1949; Gurfinkel, Levik, and Lebedev, 1989; Ghafouri et al., 1998; Adamson and McDonagh, 2004; Duclos et al., 2004).

There are two related questions regarding the control principles underlying the Kohnstamm phenomenon. The first is the origin of adaptation. According to a peripheral account, changes in the state of the muscle spindles during the induction cause the involuntary arm-lift movement to be triggered via the usual reflex pathways. There is indirect evidence that altered spindle function due to thixotropic changes occurs in the active muscle during the induction (Gregory et al., 1988; Hagbarth & Nordin, 1998; Meigal et al., 1996; Meigal & Pis'mennyi, 2009). However, it is not clear if such alterations actually drive the involuntary movement, or are more epiphenomenal. The peripheral thixotropy hypothesis assumes that induction causes spindle hypersensitivity, presumably enhancing reflex contractions. However, a previous study found that stretch reflexes elicited by hitting a rigid obstacle were

actually weaker during Kohnstamm movements than during matched voluntary movements, contrary to this prediction (De Havas et al., 2015).

Alternatively, the adaptation that occurs during Kohnstamm induction may be central. Spinal (Matthaei, 1924b; Pinkhof, 1922; Schwartz, 1924; Schwartz & Meyer, 1921; Zigler, 1944), sub-cortical (Foix & Thevenard, 1923; Rothmann, 1915) and cortical (Salmon, 1915, 1916, 1925; Sapirstein et al., 1936, 1937, 1938) loci have all been proposed. Interestingly, the Kohnstamm phenomenon is associated with a similar pattern of activation across sensorimotor regions of the cerebral cortex as during voluntary movements (Duclos et al., 2007; Parkinson et al., 2009), consistent with a cortical locus. TMS studies suggest that muscular activity during Kohnstamm movements passes through the contralateral primary motor cortex (Ghosh et al., 2014; Mathis et al., 1996). Several studies found that visual input can cause the involuntary contraction to switch muscles, consistent with a central but not a peripheral locus (Ghafouri et al., 1998; Gilhodes et al., 1992; Gurfinkel et al., 1989). Some early theories proposed that the involuntary movement represented a persistence of the voluntary motor command used during induction (Sapirstein et al., 1937, 1938), or an exact replaying of the voluntary movement (Salmon, 1916, 1925). Beyond this, however, there has been little work on the control principles underlying the Kohnstamm phenomenon.

A second key question concerns the role of sensory feedback during the Kohnstamm phenomenon. Once the involuntary aftercontraction has begun, muscle activity could be driven by negative position feedback from muscle afferents. For example, a central motor signal might set an equilibrium point for the muscle, resulting in a follow-up servo contraction in the adapted muscle, causing a movement of the arm. Crucially, many equilibrium point accounts suggest that the equilibrium moves gradually towards the target over time, defining a virtual trajectory (Bizzi et al., 1984; Hogan, 1985). By these accounts, EMG should be proportional to the lead of the virtual over the actual position. Indeed, EMG tends to increase over time during Kohnstamm movements (Fessard & Tournay, 1949; Ghosh et al., 2014) and arm position and EMG are tightly coupled during the involuntary aftercontraction (Adamson & McDonagh, 2004). According to negative-position feedback control theories, one would expect obstruction of Kohnstamm movement to produce an increase in EMG, since the virtual trajectory would develop an increasing lead over the actual arm position, causing an increased follow-up servo command. In fact,

EMG rapidly plateaus when a Kohnstamm movement is obstructed, contrary to the prediction of negative feedback models (De Havas et al., 2015). Instead, the effects of obstructing a Kohnstamm movement resembled an afferent gating of the Kohnstamm motor command, akin to the afferent resetting of central pattern generators reported in the animal literature (Guertin, Angel, Perreault, & McCrea, 1995; Perreault, Enriquez-Denton, & Hultborn, 1999; Schomburg, Petersen, Barajon, & Hultborn, 1998; Stecina, Quevedo, & McCrea, 2005). When the arm is actually moving, negative-position feedback control may operate in a similar manner as during voluntary movements.

An alternative control principle for Kohnstamm movements is based on positive force feedback. On this view, the initial force generated during the aftercontraction produces further force, which feeds into a feedback circuit and prolongs the aftercontraction. Evidence for this comes from studies that reduced the effective weight of the arm using an arrangement based on a fulcrum and counterweight. This produced a systematic decrease in EMG throughout the Kohnstamm movement (Parkinson & McDonagh, 2006). A physiological model suggested that the Kohnstamm generator may consist of a positive feedback loop between a modest central excitation and the afferent load signal from Golgi tendon organs (Parkinson & McDonagh, 2006). On this account, the increase in EMG observed throughout the Kohnstamm aftercontraction is caused by the increased shoulder torque when the arm is moving through the gravitational field. The arm is effectively a pendulum. Therefore, as the arm rises, the downward force of gravity results in a strong firing from Golgi tendon organs (GTO). This would lead to strong positive feedback, increasing central motor drive and producing a continual increase in EMG. Though GTO signals are classically known to have an inhibitory effect on movement, excitatory influences are also possible (Donelan & Pearson, 2004; Duysens, Clarac, & Cruse, 2000). If positive feedback does indeed form the basis of the Kohnstamm phenomenon, it would lead to the fascinating prospect that two fundamentally different control mechanisms can be employed by the central nervous system to achieve movement. Some movements, such as most voluntary and involuntary aiming and orienting movements, would be based on a negative-position feedback control principle, while other movements, such as the Kohnstamm, would be based on positive-force feedback control principles.

Here we attempt to discriminate between these two accounts of the Kohnstamm by analysing the response to perturbations. Both negative-position feedback, and positive-force feedback predict an initial increase in EMG in response to a resistive perturbation, and a decrease in EMG in response to an assistive perturbation (decreased load on the muscle). However, the positive-force feedback model would predict that this change should be large and sustained; negative-position feedback would predict a tulip shape to the response. The tulip shape refers to a pattern of results, whereby loading the muscle during a contraction causes an initial increase in EMG followed by a partial return to baseline, while unloading cuases a decrease followed by a partial return (Marsden et al., 1977). Thus, if the positive-force feedback model is correct, when extra resistive load is suddenly added to an aftercontracting arm, GTO firing rate will increase, leading to higher efferent output and higher EMG for the remainder of the involuntary movement, relative to the same movement without the added load. However, if the negative-position feedback model is correct, it is the muscle spindle signals that are important. Increased load stretches the muscle, signalling that the actual position of the arm differs from the position specified by virtual trajectory. This produces an increase in efferent signal until the spindle signal indicates that the actual limb position has caught up with the virtual trajectory.

With the classic vertical Kohnstamm the load on the arm continually increases due to gravity, as the arm rises. According to force-feedback theories, it is this increasing gravitational load which causes the steep increase in Kohnstamm EMG. However, if the Kohnstamm movement is performed in the horizontal plane then the effects of gravity are removed. GTO firing rate would be more uniform across the range of motion of the limb. As such, if EMG still showed a steep increase during the aftercontraction, then positive-force feedback would be an unlikely explanation for the Kohnstamm phenomenon.

In addition, a horizontal Kohnstamm arrangement would allow a clearer investigation of the responses to perturbing forces during aftercontraction, because the confounding effects of gravitational forces are absent. Since both force-feedback and position-feedback accounts may include a central adaptation, only by systematic perturbation can they be fully dissociated. We tested these theories via resistive and assistive perturbations applied to a horizontal Kohnstamm on a single-joint manipulandum. This set-up gave us a number of advantages over previous

experiments. Firstly, the horizontal Kohnstamm meant there was no gravitational change throughout the movement. Secondly, the use of a manipulandum meant we could precisely apply both increased and decreased loading on the muscle at a specific point during the involuntary movement, and measure the effects on EMG and arm velocity. Since the control principles behind voluntary movements are well-understood by comparison, comparing Kohnstamm and voluntary movements is a meaningful way to assess the validity of competing models of the Kohnstamm phenomenon. The use of the manipulandum meant that, unlike previous experiments, we could give precise visual feedback to enhance voluntary replication accuracy and record precise velocity and position data during the movements to determine if there were any inherent differences between the two forms of movement.

### 4.2. Methods

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 approval of the local NTT BRL ethical committee. No adverse events occurred during the experiment.

#### 4.2.1. Equipment

Electromyography (EMG) was recorded from bipolar, surface electrodes (Ag-AgCl disposable electrode, GE Healthcare Japan, Tokyo, Japan) placed over the middle of the right posterior deltoid, parallel to the orientation of the muscle fibres. In a subset of participants, additional electrodes were also placed on the right pectoralis (n = 11) and the right triceps long head (n = 9). The electrodes were connected to an amplifier (MME-3116, Nihon Kohden, Tokyo, Japan), which was controlled via custom scripts (sample rate = 4000 Hz). A fully adjustable chair (height, rotation, pitch) was used so that all participants could have their right arm positioned comfortably on a single-joint manipulandum (Custom-made single joint manipulandum; max torque 6.8 Nm); motor command at 2000 Hz). The angle of the manipulandum was calculated via a rotary encoder (resolution of 0.0055°) and output (D/A converted) at 2000 Hz. Torque was measured with a 6-axis force sensor (UFS-3012A25, Nitta, Osaka, Japan). The manipulandum was controlled via custom MATLAB (2007b) scripts. The manipulandum had a strip of wood (60 x 10 x 2 cm) clamped at a right angle with an upwards pointing handle at one end. This was to

support the participant's forearm and was fully adjustable. A custom-built, rigid pushing surface was clamped to the manipulandum. It was adjustable so that the participant's elbow could comfortably push against it. On the opposite side of the manipulandum to the participant was a strain gauge (UFS-3012A15, Nitta, Osaka, Japan) mounted to a moveable, rigid beam and located at a distance of 0.5 m from the rotation centre of the manipulandum. This was positioned such that the strain gauge would register the amount of torque being generated by the participant during isometric Kohnstamm inductions. This information was relayed to participants via an oscilloscope (TDS2004C, Tektronix, Inc., Oregon, USA) positioned at eye level in front of them. Participants wore goggles with a cardboard cone (length = 45 cm) to prevent them from seeing their arm. Unwanted movement of the right arm was prevented via two adjustable straps (forearm and upper arm). A flat screen monitor (19 inch LCD, 800x600 pixels, 60 Hz refresh rate) was positioned in front of the participants to provide visual feedback of arm position during the learning phase of voluntary replication movements. Visual feedback was controlled by Cogent Graphics (John Romaya, Wellcome Trust Centre for Neuroimaging and Institute of Cognitive Neuroscience development team, UCL) in MATLAB (2007b). Analogue signals (EMG, position, toque during induction and aftercontraction) were sampled and stored at 4000 Hz via custom-made software (MATLAB, 2007b). Experimental set up can be seen in Fig. 4.1.

### 4.2.2. Participants

A total of 39 participants were recruited (13 female; age: mean = 31.62, SD = 5.34). It is known that the Kohnstamm phenomenon is present in the lateral deltoid for about 75% of healthy participants (Adamson & McDonagh, 2004; Duclos et al., 2007; Hagbarth & Nordin, 1998; Ivanenko et al., 2006). We screened participants using this muscle as it is the most widely reported in the literature. Fourteen participants did not show any signs of the Kohnstamm phenomenon and were excluded, leaving 25 participants (Female = 8; age: mean = 32.32, SD = 5.47). No previous study has examined the posterior deltoid muscle. Four of our participants did not display the Kohnstamm phenomenon in the posterior deltoid and were excluded, leaving 21 participants (Female = 7; age: mean = 32.48, SD = 5.14). Of these, we found that resistive perturbations completely arrested Kohnstamm arm

movements in six participants. Since we were specifically studying the control principles during Kohnstamm arm movement following perturbations, these participants were excluded from the main analysis, leaving 15 participants (Female = 4; age: mean = 32.27, SD = 5.56). It is possible that the six participants in whom our perturbations arrested the Kohnstamm completely represent a subgroup with relatively low Kohnstamm gains.

### 4.2.3. Procedure

Participants were seated throughout the experiment wearing googles that prevented any view of their right arm. The chair was adjusted such that participants right arm rested on the manipulandum, with their elbow bent at 90°, their shoulder above the centre of rotation and their arm horizontal to the ground. Unwanted movement was prevented with straps attached to the forearm and upper arm. The handle rested between their thumb and forefinger. They were instructed not to grip, as this could induce differences in the contraction of the forearm across conditions. Tilt and rotation of the chair was adjusted until the participant's arm remained in the start position when relaxed. This was essential to prevent any movement occurring as a result of the tension on the shoulder or the release of antagonist contraction.

Participants completed a 5 s, 100% MVC isometric contraction of the posterior deltoid muscle (agonist) in the home position, by pushing outwards against the rigid elbow support. The oscilloscope was then set to display 70% of this value as the target force level during Kohnstamm inductions. If EMG data was being recorded from the pectoralis (antagonist) and triceps long head muscle, 5 s, 100% MVC isometric contractions were also recorded for these muscles.

A tone signalled the start of each Kohnstamm trial. Participants maintained a 70% MVC isometric contraction of the lateral deltoid by pushing outwards against the rigid surface. Target force and actual force were displayed continuously on the oscilloscope. After 30 s a tone signalled that they should stop pushing and relax. As soon as the force level reached zero, the experimenter rotated the rigid surface with the attached strain gauge, allowing the arm to move freely. This was easily achieved before any involuntary movement, owing to the latent period of muscle silence which is known to occur in the Kohnstamm phenomenon and last 1-3 s (Csiky, 1915; Kozhina et al., 1996; Parkinson & McDonagh, 2006; Pinkhof, 1922). An aftercontraction of the posterior deltoid then occurred causing an involuntary

movement of the arm. The shoulder was free to rotate 100°. Participants were instructed to remain relaxed and not attempt to move the arm voluntarily.

In the No perturbation control condition the arm was allowed to move freely. However, in the Resistive condition a constant torque of 0.5 Nm was applied at the shoulder in the opposite direction to movement once arm reached 20° of angular displacement. In the Assistive condition the same size of perturbation was applied in same direction as the movement (Fig. 4.1.). A ramp was used in both cases, such that the applied torque increased linearly over a duration of 250 ms. This ensured arm movement was smooth. It was important the perturbation was not felt as rigid obstacle, as this could induce the kind of 'afferent resetting' seen in previous studies (De Havas et al., 2015). There were 6 trials, 2 for each of the three conditions (No perturbation control condition, Assistive perturbation condition, Resistive perturbation condition). Trial order was ABCCBA, counterbalanced across participants. Participants were always naïve to trial type. The Kohnstamm phenomenon is associated with a high degree of variability (Brice & McDonagh, 2001; Hagbarth & Nordin, 1998; Salmon, 1916, 1925). We therefore repeated trials where no clear Kohnstamm movement was detected. Because of these occasional repetitions, the actual number of trials undertaken by each participant was therefore slightly higher than the intended number of 6 (Mean = 6.67, SD = 0.98). If trials had to be repeated, efforts were made to maintain the randomisation process by re-adjusting trial order, so that the mean position of trials within the order of the experiment did not differ across conditions. Average position of trials did not significantly differ across conditions: No perturbation control (Mean = 3.77, SD = 0.42) vs. Resistive (Mean = 4.23, SD = 1.45) vs. Assistive perturbations (Mean = 3.53, SD = 1.03; $F(2, 28) = 2.479$, $p = 0.102$). After every trial there was a rest period of 7 minutes to minimise fatigue and long-lasting motor post-effects (Duclos et al., 2004; Hutton et al., 1987).

The trajectory of the two Kohnstamm No perturbation control trials was then used to create voluntary replication trials. One of the No perturbation control trials was randomly selected. Participants heard a tone signalling that the movement was to begin in 3s. They then saw the trajectory of the Kohnstamm control trial represented on the screen as a moving dot. They replicated the previous involuntary movement with a voluntary contraction of the posterior deltoid. Position of the arm was displayed continuously as a line of hollow circles. Participants completed 10 practice trials, followed by 12 trials in which no visual feedback was given. As with

the Kohnstamm trials, these voluntary trials were either No perturbation control, Resistive or Assistive (4 per condition; randomised). Perturbations were applied in exactly the same manner. Participants were not told about the perturbations and simply instructed to complete each movement. Interposed with these voluntary trials were occasional trials in which visual feedback was given to ensure replication accuracy was maintained. Trials with visual feedback were not analysed. This entire process was then repeated for the replication of the second Kohnstamm control trial, resulting in a total of 24 voluntary movement trials. The experiment lasted ~2.5 hours.

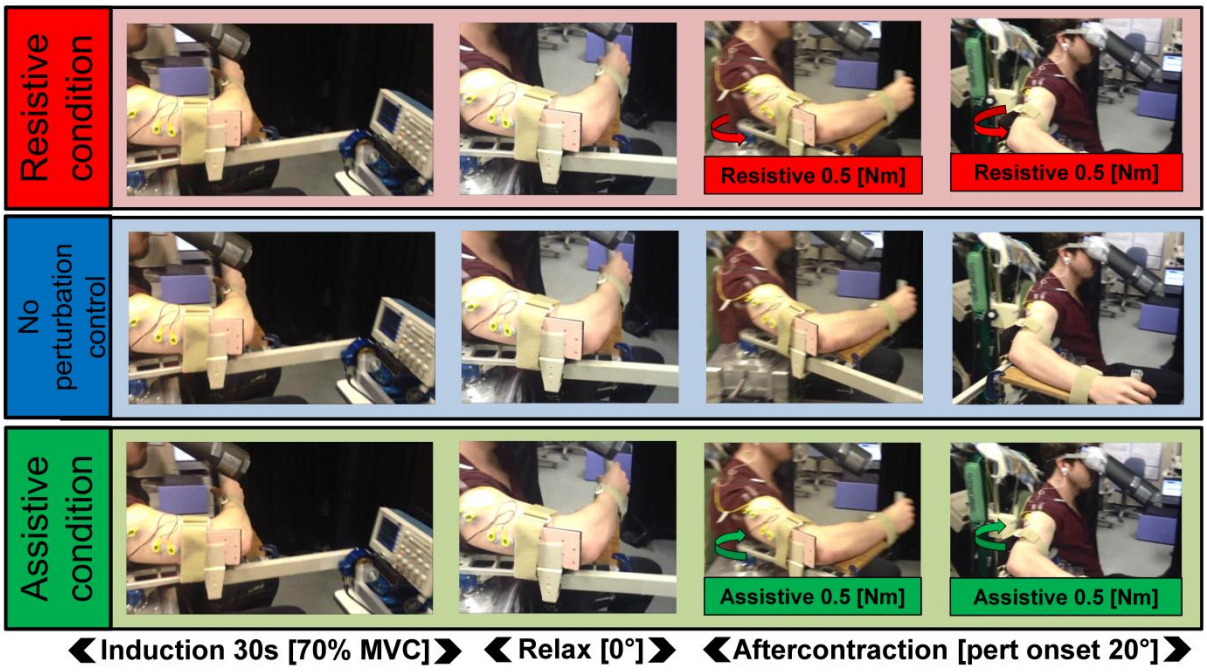



**Figure 4.1. Schematic of the task.** *In all three Kohnstamm conditions, participants maintained a constant isometric contraction of the right posterior deltoid for 30 s (70% MVC). They then relaxed and the aftercontraction began. In the Resistive condition, a torque was applied by the manipulandum motor in the opposite direction to the movement (0.5 Nm; 250 ms linear ramp) once the angular displacement of the arm reached 20°. In the Assistive condition, a torque was applied by the manipulandum motor in the same direction as the movement (0.5 Nm; 250 ms linear ramp) once the angular displacement of the arm reached 20°. In the No perturbation control condition, no torque was applied.*

### 4.2.4. Analysis

Kohnstamm trials were only included in the analysis if the arm continued to move for at least 500 ms after the perturbation. Examples of trials from each condition can be seen in figure 4.2. If arm movement stopped within this window the trial was repeated (*see Procedure*). If the perturbation again stopped the arm movement we did not continue to repeat trials indefinitely, because and the total number of trials is constrained by fatigue (Allen, 1937; Allen & O'Donoghue, 1927;

Danielopolu et al., 1921; Zigler et al., 1948). Of the fifteen participants included in the main analysis, two participants had one Resistive condition trial missing for this reason. Further, equipment failure led to one participant having one Resistive condition trial removed, and another participant having one Assistive condition trial removed. Angular velocity was computed by calculating differences between angles at current and previous frames, then low-pass filtered at 80 Hz. For the voluntary replication movements, 6 trials (2 per condition) were selected for each participant. The three trials (1 per condition) that had the closest pre-perturbation velocity were selected for each of the two Kohnstamm No perturbation control trials. This was done by calculating mean SSE from the voluntary trial velocity compared to the Kohnstamm control trial velocity, between 10 and 20° of angular displacement.

The strain gauge signal was low-pass filtered at 30 Hz. EMG was band-pass filtered (10-500 Hz) and rectified before being smoothed (4 Hz). A 1 s window was selected for the purposes of displaying the data, centred on the onset of the perturbation. For the agonist muscle, two alternative forms of normalisation were used. The first involved normalising to each participant's MVC (EMG as % MVC across the three conditions). This standard form of normalisation was also used for the antagonist muscle (pectoralis) and triceps long head muscle. Since background level of agonist EMG is known to influence the size of reflex responses (Matthews, 1986; Toft, Sinkjaer, & Andreassen, 1989), an alternative normalisation was also used. Each participant's Resistive and Assistive condition agonist EMG was normalized to their No perturbation control condition agonist EMG (% EMG change from baseline across two conditions). This was performed separately for Kohnstamm and voluntary movements.

Mean agonist EMG, antagonist EMG, triceps long head EMG, torque, angular displacement and velocity were calculated during an analysis window of 200-400 ms post-perturbation. 2 (Movement type: Kohnstamm vs. Voluntary) x 3 (Condition: No perturbation control vs. Resistive vs. Assistive) within subjects ANOVA were conducted.

To explore whether arm movement began to return to a virtual trajectory, an additional 400-500 ms time window was selected for the analysis of movement velocity. Because the characteristic feature of reflex responses is a near-linear increase or decrease in velocity during the selected time period, linear regression lines were fitted to each participant's individual mean velocity data in this time

window, separately for each perturbation condition and for Voluntary vs. Kohnstamm movements. Mean slope values were compared via the same 2x3 within subjects ANOVA.

For the 'EMG % of no perturbation control' normalisation, a 2 (Movement type: Kohnstamm vs. Voluntary) x 2 (Condition: Resistive vs. Assistive) within subjects ANOVA was conducted on the agonist EMG data, based on mean values during the same time window (200-400 ms post-perturbation).

### 4.3. Results

#### 4.3.1. "Tulip" responses during involuntary and voluntary movements

During Kohnstamm and voluntary movements, the resistive perturbation produced an increase in agonist EMG, while the assistive perturbation produced a decrease, compared to the No perturbation control condition (Fig. 4.3.a,b,c). This manifested as a significant main effect of Condition ($F(2,28) = 10.349$ , $p < 0.001$). There was no Movement type x Condition interaction ($F(2,28) = 0.676$, $p = 0.517$), indicating that this 'tulip' response did not differ as a function of whether the movement was due to the Kohnstamm phenomenon or was voluntary.

#### 4.3.2. Opposite pattern of movement velocity 400-500 ms post-perturbation in Kohnstamm movements compared to Voluntary movements

During Voluntary movements, velocity initially increased in the Assistive condition and decreased in the Resistive condition. These changes then reversed direction after around 400 ms, showing decrease in the Assistive condition and increase in the Resistive condition (Fig. 4.3.e). This reversal may indicate a voluntary compensation for the perturbation, to 'catch up' with an intended movement trajectory. Interestingly, this reversal did not occur during Kohnstamm movements (Fig. 4.3.d). This suggests that such voluntary compensatory movements were absent. Fitting linear trend lines to individual participant averages during this time window showed that the difference was statistically significant (Fig. 4.3.g). The mean value of these slopes did not differ in magnitude across movement types ($F(1,14) = 0.033$ , $p = 0.859$). Similarly, there was no main effect of Condition ($F(2,28) = 0.204$, $p = 0.702$ ; Greenhouse-Geisser corrected). However, there was a significant Movement type x Condition interaction ($F(2,28) = 21.621$, $p < 0.001$). To explore this interaction, one-way ANOVAS were conducted. There was a significant difference

across perturbation conditions in both Kohnstamm ($F(2,28) = 8.426$, $p = 0.00137$) and Voluntary movements ($F(2,28) = 13.077$, $p = < 0.001$). Planned comparisons showed that during Kohnstamm movements, Resistive condition velocity decreased relative to the No perturbation control condition ($t(14) = -2.420$, $p = 0.03$), while Assistive condition velocity *increased* relative to control ($t(14) = 2.162$, $p = 0.048$). In stark contrast, for Voluntary movements, Resistive condition velocity *increased* relative to No perturbation control ($t(14) = 3.54$, $p = 0.003$), while Assistive condition *decreased* relative to control ($t(14) = -2.499$, $p = 0.026$). There was no significant difference in the mean slope of No perturbation control condition velocity across movement types ($t(14) = -0.47$, $p = 0.963$).

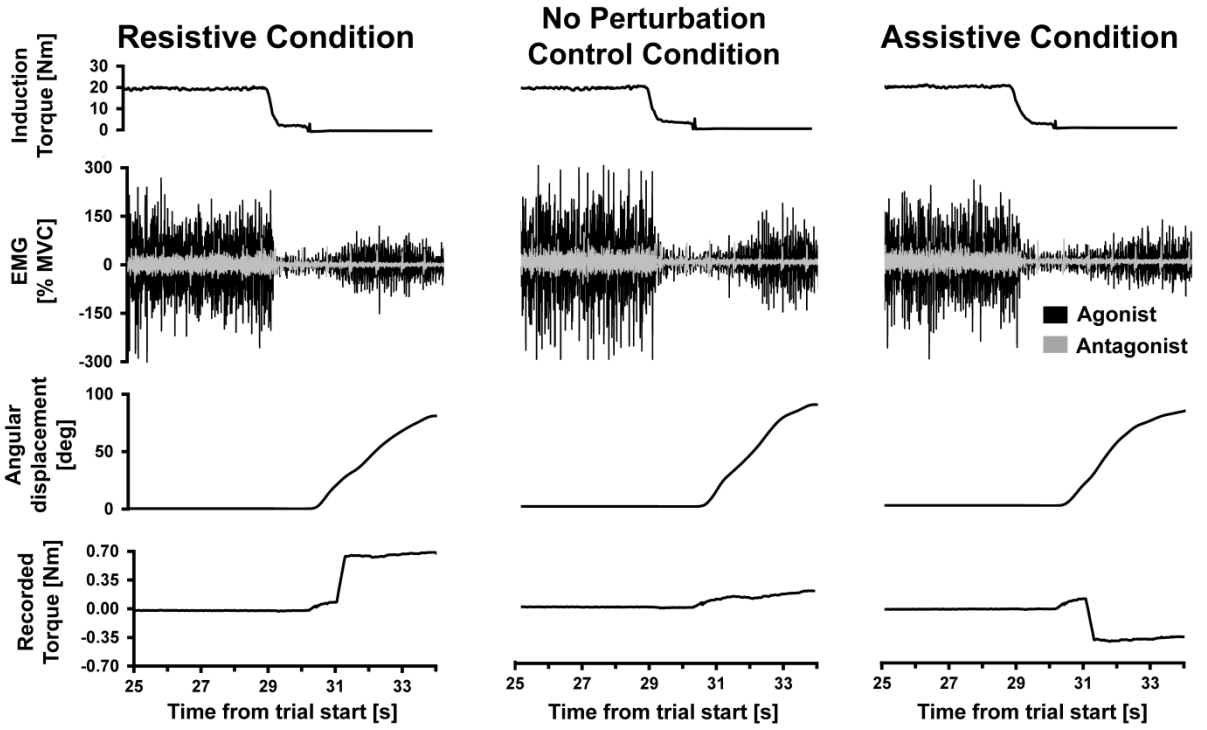



**Figure 4.2. Single trial data.** *Data from three Kohnstamm movement trials from a single representative participant, belonging to the Resistive, No perturbation control and Assistive conditions. Last 5 s of the 30 s isometric induction contraction is shown, followed by a brief latent period of ~1 s and then the aftercontraction. Note that induction torque was equivalent across trials. During aftercontraction agonist (posterior deltoid) EMG increased in amplitude as angular displacement of the shoulder increased. Antagonist (pectoralis) EMG was flat throughout the aftercontraction (regular spikes shown were from heart beat artefact). In the Resistive condition trial, a torque was applied in the opposite direction to the movement once angular displacement reached 20° (0.5 Nm; 250 ms linear ramp). In the Assistive condition trial, a torque was applied in the same direction as the movement once angular displacement reached 20° (0.5 Nm; 250 ms linear ramp). The lower traces show the torque recorded at the shoulder manipulandum: note the abrupt changes in torque due to the perturbations.*

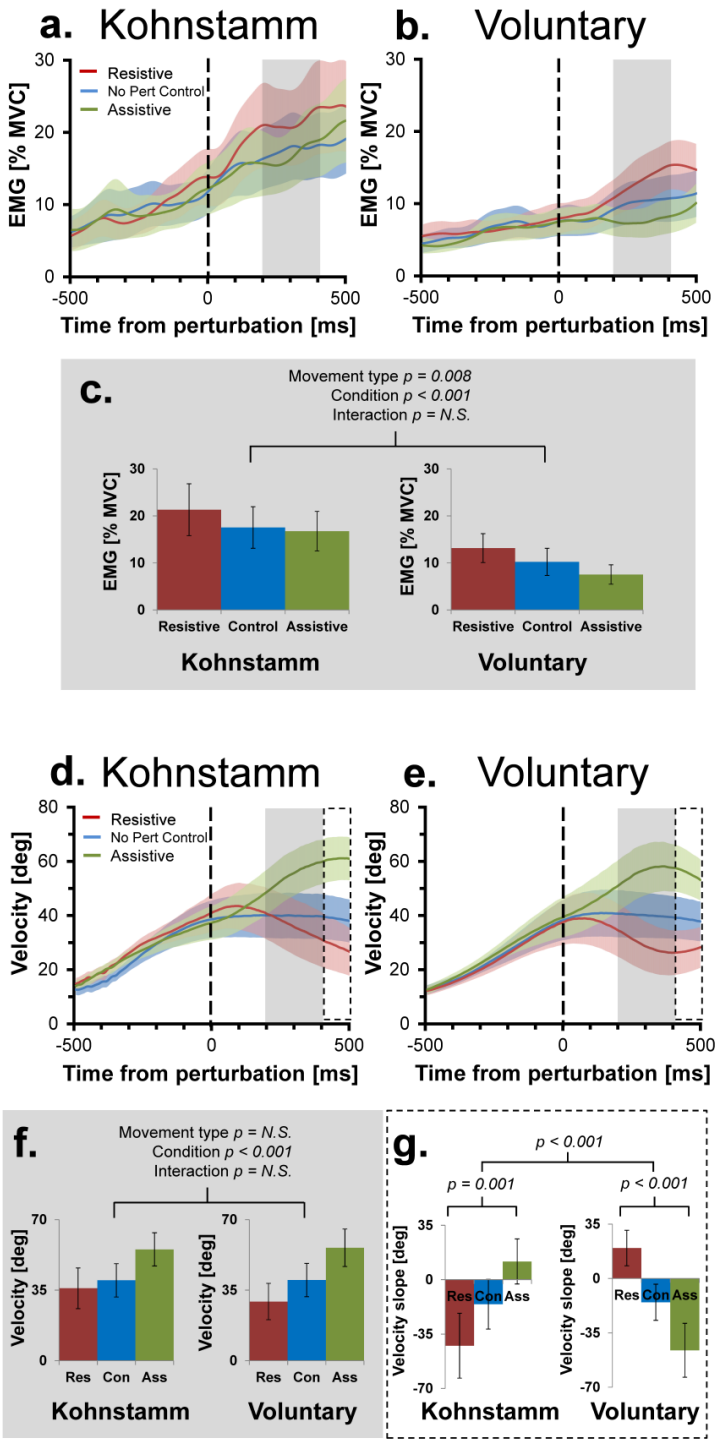



**Figure 4.3. Mean smoothed agonist EMG and velocity of movement across movement types and conditions.** *Agonist smoothed group EMG (% MVC) across conditions, from 500 ms prior to onset of perturbation to 500 ms post-perturbation, for Kohnstamm (a) and Voluntary movements (b). Mean EMG 200-400 ms post-perturbation (c). There was significantly higher EMG during Kohnstamm movements than Voluntary movements during this time window. EMG increased in the* Resistive *condition and decreased in the Assistive condition, relative to the No perturbation control condition. This change in EMG was significant across the two types of movement. Velocity of angular displacement during the same time window for Kohnstamm (d) and Voluntary movements (e). Mean velocity 200-400 ms post-perturbation (f). Velocity decreased in the Resistive condition and increased in the Assistive condition, relative to the No perturbation control condition (200-400 ms post-perturbation). This change in velocity was significant across the two types of movement. Mean slope of velocity across participants (400-500 ms post-perturbation) showed the opposite pattern of results when comparing Kohnstamm to Voluntary movements (g).*

### 4.3.3. Tulip response *smaller* during Kohnstamm than Voluntary after controlling for baseline EMG

High yet variable background EMG levels during Kohnstamm could have hidden genuine differences in reflex responses between movement types. The analysis was therefore repeated on agonist EMG after normalization to individual participants' EMG in the same time window of the No perturbation control condition, instead of the conventional normalisation to MVC (Fig. 4.5). 2x2 ANOVA showed that mean EMG did not significantly differ across Movement types ($F(1,14) = 0.242$, $p = 0.630$). The perturbation still decreased EMG in the Assistive condition and increased it in the Resistive condition, as evidenced by a significant main effect of Condition ($F(1,14) = 31.535$ , $p < 0.001$). Importantly, the magnitude of this difference was larger for Voluntary movements, as evidenced by a significant Movement type by Condition interaction ($F(1,14) = 6.146$, $p = 0.027$). Planned comparisons showed that Resistive condition EMG was higher than Assistive condition EMG for Kohnstamm movements ($t(14) = 2.54$, $p = 0.024$) and for Voluntary movements ($t(14) = 5.641$, $p < 0.001$), suggesting a 'tulip' response in both cases. However, the interaction arose because Kohnstamm responses were weaker than Voluntary responses after this normalisation procedure.

### 4.3.4. Increased Kohnstamm EMG not explained by behavioural differences or activity of other muscles

There was a significant main effect of Movement type ($F(1,14) = 9.377$ , $p = 0.008$) on agonist EMG. Across conditions, agonist EMG was higher during Kohnstamm movements compared to matched Voluntary movements (Fig. 4.3.a,b,c). This was not explained by differences in recorded torque during the same time window. During Kohnstamm movements, mean Resistive condition torque was 0.58 Nm (SD = 0.04 Nm), compared to 0.07 Nm (SD = 0.05 Nm) during No perturbation control condition and -0.44 Nm (SD = 0.04 Nm) during the Assistive condition. During Voluntary movements, mean Resistive condition torque was 0.63 Nm (SD = 0.11 Nm), compared to 0.13 Nm (SD = 0.10 Nm) during No perturbation control and -0.38 Nm (SD = 0.10 Nm) during the Assistive condition. This manifested as a significant main effect of Condition ($F(2,28) = 18765.987$, $p < 0.001$), but no

main effect of Movement type ($F(1,14) = 3.634$, $p = 0.077$) and no interaction ($F(2,28) = 0.264$, $p = 0.694$; Greenhouse-Geisser corrected). Angular displacement showed the same pattern of results in this time window. Mean arm angle during Kohnstamm movements was 32.02° (SD = 10.43°) for the Resistive condition, 31.71° (SD = 9.16°) for the No perturbation control condition, and 33.62° (SD = 8.93°) for the Assistive condition. During Voluntary movements, it was 30.39° (SD = 10.92°) for Resistive, 31.87° (SD = 9.73°) for No perturbation control, and 34.27° (SD = 10.37°) for the Assistive condition. There was a significant main effect of Condition ($F(2,28) = 6.2$, $p = 0.0059$), but importantly, no main effect of Movement type ($F(1,14) = 0.96$, $p = 0.761$) or interaction ($F(2,28) = 1.667$, $p = 0.215$). For velocity of movement, there was a main effect of Condition ($F(2,28) = 26.924$, $p < 0.001$) with Resistive perturbations reducing velocity and Assistive perturbations increasing velocity, as predicted (Fig. 4.3.d,e,f). However, again there was no main effect of Movement type ($F(1,14) = 0.304$, $p = 0.59$) and no interaction ($F(2,28) = 2.038$, $p = 0.149$).

Higher agonist activity in Kohnstamm conditions could be due to differences in the state of antagonist muscle. However, recordings from the pectoralis showed that EMG was low and even flat across all trial types (Fig. 4.4.a,b,c). In the time window of interest there was no main effect of Movement type on antagonist EMG ($F(1,10) = 0.114$, $p = 0.742$). There was also no main effect of Condition ($F(2,20) = 0.245$, $p = 0.785$) or Movement type x Condition interaction ($F(2,20) = 2.782$, $p = 0.112$).

Lower agonist activity during Voluntary movements could reflect contributions of other synergist muscles to the voluntary movement. This hypothesis predicts higher activity in the triceps long head muscle during Voluntary movements than during Kohnstamm movements. In fact we observed a trend in the opposite direction ($F(1,8) = 4.777$, $p = 0.060$; Fig. 4.4.d,e,f). In this muscle there was also a main effect of Condition in the same direction as for the agonist muscle ($F(2,16) = 6.739$, $p = 0.0075$). Once again there was no Movement type x Condition interaction ($F(2,16) = 0.498$, $p = 0.617$).

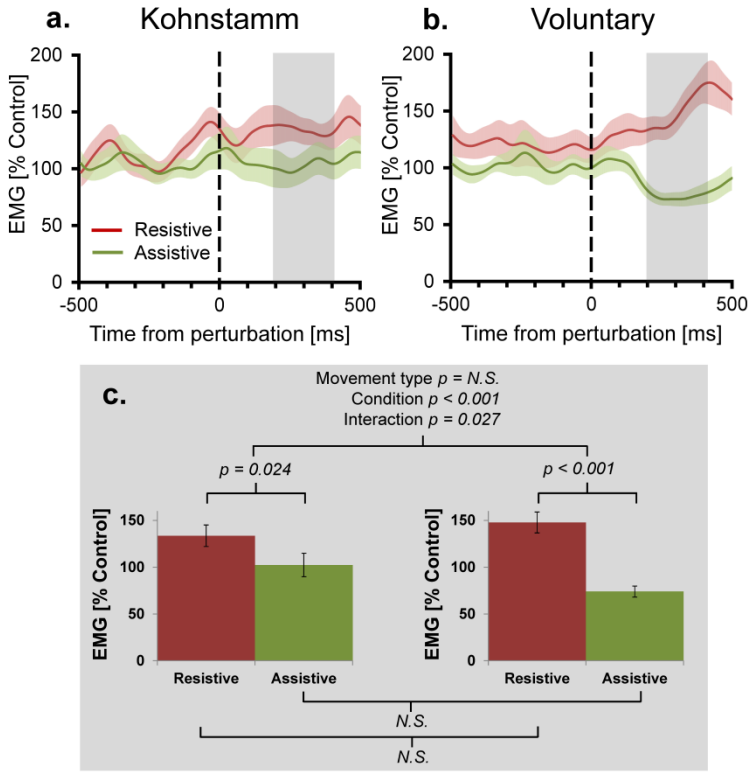



**Figure 4.4. Mean agonist EMG in Resistive and Assistive conditions normalised to No perturbation control condition.** *Agonist smoothed group EMG (% No perturbation control condition) across conditions, from 500 ms prior to onset of perturbation to 500 ms post-perturbation, for Kohnstamm (a) and Voluntary movements (b). Note larger difference between conditions for Voluntary movements. Mean EMG (% No perturbation control condition) 200-400 ms post-perturbation (c). There was no difference in overall EMG level across movement types. There was larger EMG in the Resistive condition than in the Assistive condition across movement types. This difference was significantly larger for Voluntary movements than Kohnstamm movements.*

### 4.3.5. Potential biases due to inclusion/exclusion criteria

The above analyses excluded participants whose arm was stopped by the resistive perturbation. This might bias our sample towards participants with stronger Kohnstamm effects. To investigate whether this bias could influence our conclusions, we repeated the previous analyses including all participants (n = 21), but with the Resistive condition omitted. The effect of the assistive perturbation was as before. When agonist EMG was normalised to MVC, Kohnstamm EMG was higher than Voluntary EMG, and the Assistive condition EMG was lower than the No perturbation control condition EMG (Kohnstamm: 12.69% (SD = 15.19%) vs. 13.40% (SD = 15.84%); Voluntary 6.00% (SD = 7.18%) vs. 8.32% (SD = 9.91%)), as predicted. This manifested as a significant main effect of Movement type ($F(1,20) = 9.643$, $p = 0.0056$), a significant main effect of Condition ($F(1,20) = 4.990$, $p = 0.037$), but no

significant interaction (F(1,20) = 1.972, p = 0.176). When agonist EMG in the Assistive condition was normalised to the No perturbation control condition, there was again a trend towards Voluntary movement EMG being lower than Kohnstamm EMG (71% (SD = 25.26%) vs. 96.25% (SD = 42.32%); (t(20) = -2.0035, p = 0.0589).

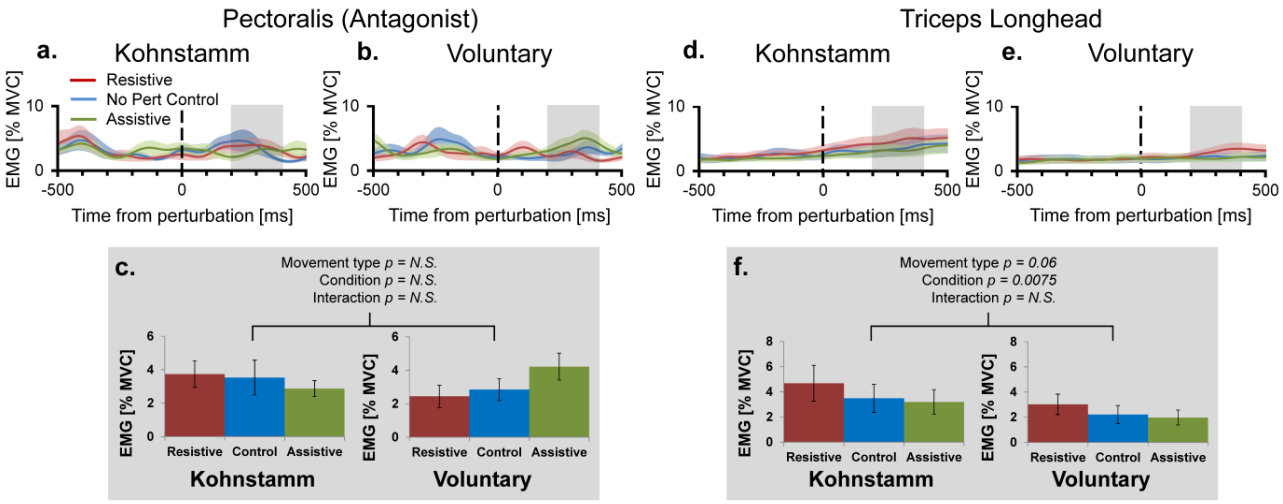



**Figure 4.5. Mean smoothed antagonist and triceps long head EMG across movement types and conditions.** *Smoothed group (n = 11) antagonist EMG (% MVC) across conditions, from 500 ms prior to onset of perturbation to 500 ms post-perturbation, for Kohnstamm (a) and Voluntary movements (b). Mean antagonist EMG (200-400 ms post-perturbation) showed there were no differences across movement types and conditions (c). Smoothed group (n = 9) triceps long head EMG (% MVC) across conditions for Kohnstamm (d) and Voluntary movements (e). Mean triceps long head EMG (200-400 ms post-perturbation) showed that this muscle was not more active during Voluntary than Kohnstamm movements (f). There was a trend in the other direction. EMG increased in the Resistive condition and decreased in the Assistive condition, relative to the No perturbation control condition. This change in EMG was significant across the two types of movement.*

## 4.4. Discussion

Increased loading on the muscle during Kohnstamm aftercontraction produced an increase in EMG and a decrease in velocity. Decreasing the loading produced a decrease in EMG and an increase in velocity. The size of this response did not differ from responses to perturbation during matched voluntary movements, when EMG levels were expressed relative to MVC. Overall EMG in the absence of perturbation was higher during aftercontractions compared to kinematically matched voluntary movements. This higher EMG level was not explained by differences in the movement of the arm, differential activation of antagonist muscles or differential activation of triceps long head across Kohnstamm and Voluntary movements. When we controlled for this high level of agonist muscle activity in Kohnstamm contractions by appropriate normalisation, the size of the perturbation response was smaller during Kohnstamm movements than during Voluntary movements.

The positive force-feedback hypothesis was not supported by our data. According to this account, increasing loads should generate high forces at the GTO, leading to higher afferent firing rates, further activating the Kohnstamm generator. Decreasing load has the opposite effect. By design, positive feedback loops rapidly multiply the effects of an input. Positive force feedback therefore predicts that *resistive* perturbations should produce a large, sustained or explosive increase in EMG, while *assistive* perturbation should produce a large and sustained decrease (Parkinson & McDonagh, 2006). However, we found a small increase in the case of resistive perturbation and a small, transient decrease in the case of assistive perturbation. Indeed, mean agonist EMG in the Assistive condition actually began to rise above the No perturbation control condition EMG at ~300 ms post-perturbation (Fig. 4.3.a), though this rise was not statistically significant. This 'tulip' response to the perturbations did not differ significantly in size to that found during matched voluntary movements when normalised to MVC, but was actually smaller when normalised to the No perturbation control condition. EMG in the Kohnstamm No perturbation control condition was similar to previous experiments. During a vertical Kohnstamm, agonist EMG was previously reported to be around 22% of MVC at a joint angle of 35° (Adamson & McDonagh, 2004). In the present experiment, at an angle of 31°, it was 18% MVC. It is not possible to know the precise shoulder torque profile of an individual without imaging or anatomical modelling. However, the use of a manipulandum and horizontal Kohnstamm meant we could be confident that the loading on the muscle was low throughout the natural range of motion. Crucially, by removing the effects of gravity with a horizontal arrangement, we can provide a clear test of the force feedback model by avoiding the additional, substantial and time-varying forces generated by the effects of gravity on the rising arm in the classical Kohnstamm arrangement. Thus, taken together, we found no evidence for the explosive, increasing, sustained response to a resistive perturbation that is the hallmark of positive force feedback control, but instead found small, transient increases. It seems unlikely that positive-load feedback can underlie the Kohnstamm generator.

Our results are reminiscent of the 'tulips' observed in studies of loading and unloading the muscle during *voluntary* movement (Marsden et al., 1975, 1976a, 1977). For such control, a central motor signal setting the equilibrium point of the

muscle would result in a follow-up servo contraction of the muscle, causing a movement towards that position. This movement compensating for the perturbation is self-terminating, through negative position feedback, ending when the original target position is re-attained. The equilibrium point might move gradually over time to achieve voluntary movements, defining a virtual trajectory (Bizzi et al., 1984; Hogan, 1985). When the muscle is stretched by a resistive perturbation, increased spindle firing causes a further contraction of the muscle, resulting in an increase in EMG. The efferent signal continues to increase until the spindle signal indicates that arm is returning to the virtual trajectory. Conversely, when the existing stretch on the muscle decreases due to the unloading, caused by an assistive perturbation, there is a *transient* decrease in efferent output until the spindle signal indicates that the position of the arm is returning to the virtual trajectory. Our results indicate that similar control underlies the Kohnstamm phenomenon, suggesting shared neuroanatomical pathways. Kohnstamm movements activate similar sensorimotor areas of the cortex to voluntary movement (Duclos et al., 2007; Parkinson et al., 2009). Cortical silent period studies have suggested that the Kohnstamm generator transmits outputs via the motor cortex (Ghosh et al., 2014).

Decreasing the load on the muscle using a counterweight reduced EMG throughout the Kohnstamm movement (Parkinson and McDonagh 2006). This was taken as evidence of positive-force feedback in the Kohnstamm phenomenon. However, these results could be explained by negative-position feedback, with the counterweight amounting to a succession of assistive perturbations. When the arm rises during a vertical Kohnstamm, the load on the muscle increases steeply due to gravity acting on the mass of the arm. A counterweight decreases this load. The magnitude of this decrease gets larger with increasing joint angle (Parkinson & McDonagh, 2006). Thus, the observed decreases in EMG could be due to a spindle signal, indicating the disparity between actual arm position and a “virtual trajectory”. This disparity would increase as the Kohnstamm movement progresses, resulting in a reduction in EMG. However, dissociating these position and force theories is complicated by the constantly varying load in all conditions due to gravity.

We also found differences between Kohnstamm and voluntary movements. When controlling for the level of baseline EMG, the perturbation response was larger during Voluntary movements. This seems consistent with the widespread report that

reflexive responses can be modified by contextual factors (Rothwell et al., 1980). Also, given the time windows used in our analysis, we cannot exclude a *voluntary* response to the perturbation for Voluntary movements, though this should have been absent for Kohnstamm movements. This could explain the late velocity reversals that we found 400 ms after perturbation. Participants may have voluntarily adjusted their arm trajectory to match the voluntary task goal of replicating the learned trajectory.

The transcortical long-loop reflex is said to operate over a time window from 60-120 ms. It contains two distinct components which sum linearly (Pruszynski et al., 2011). One component shares features with voluntary movement, such as task dependency and sensitivity to goals. It is difficult to relate our EMG responses directly to the long-loop reflex response. In particular, our experiment necessitated a long, isotonic ramp-like movement, on which we superimposed a relatively long perturbation. In contrast, many long-loop studies used rapid perturbations (Marsden, Merton, & Morton, 1976b; Marsden et al., 1977). It should also be noted that many of the classic experiments in this field use small muscles, small movements and proportionally large changes in muscle load. In contrast, we studied a large muscle making a large movement, and measured the response to relatively small changes in muscle load. In addition, our Kohnstamm movements were quite variable in velocity, both across and between participants. Thus, although our EMG responses to perturbation of Kohnstamm movements were found in a time window later than the classic long-loop time window, we suggest that our results could reflect a long-loop mechanism, because the *effective* time of perturbation may be relatively delayed in our experiment.

The small response to perturbation during Kohnstamm movements compared to voluntary movements provides indirect evidence against a peripheral origin of the aftercontraction. In particular, it was proposed that the Kohnstamm contraction might be a consequence of sustained afferent discharge from muscle spindles, possibly due to thixotropic changes occurring during the induction phase (Gregory et al., 1988; Hagbarth & Nordin, 1998). These accounts predict higher spindle sensitivity in Kohnstamm movements compared to voluntary movements, and therefore stronger EMG responses to the stretch induced by resistive perturbations. This theory equally predicts a strong decrease in EMG following assistive perturbations. Neither prediction was fulfilled: responses to perturbations were smaller during Kohnstamm

than during Voluntary movements, when the background EMG level during movement was taken into account. Sensitization of spindle afferents is therefore unlikely to underlie the Kohnstamm phenomenon. This result is consistent with other investigations of the Kohnstamm movement. For example, signals from muscle spindles contribute strongly to position sense (Matthews 1933; Stuart et al. 1970; Windhorst 2008; Proske and Gandevia 2009; Kuehn et al. 2015), which has been found to be normal during Kohnstamm movements (Heide & Molbech, 1973; Howard & Anstis, 1974). Moreover, a previous study of EMG responses to contacting a physical obstacle found no difference between Kohnstamm and voluntary movements (De Havas et al., 2015).

An alternative view of the Kohnstamm phenomenon proposes that the induction phase leads to adaptation of a central motor command generator. Importantly, this adaptation is unlikely to be a simple persistence of the induction voluntary motor command (Sapirstein et al., 1937, 1938) or an exact replaying of a voluntary movement (Salmon, 1916, 1925) Both those models imply a similarity between Kohnstamm and voluntary movements, but we found several differences. Instead, our results are consistent with the idea that the induction phase leads to adaptation of a central mechanism that sets the gain of a negative feedback loop driven by muscle spindle signals. Central adaptations could be spinal (Matthaei, 1924b; Pinkhof, 1922; Schwartz, 1924; Schwartz & Meyer, 1921; Zigler, 1944), subcortical (Foix & Thevenard, 1923; Rothmann, 1915) or cortical (Salmon, 1915, 1916, 1925; Sapirstein et al., 1936, 1937, 1938). Dissociating between these accounts is difficult since all these regions are actively involved in producing both Kohnstamm and voluntary movements. As a suggestion for future research, we believe that studies of patients with focal lesions might reveal the locus of this central gain.

Kohnstamm EMG was significantly stronger than matched voluntary EMG. Our setup ruled out peripheral explanations for this increase, while our experimental setup ruled out artefactual explanations based on interactions with gravity. This high EMG could not be explained by differences in recorded torque, arm position, velocity, antagonist activity or the contribution of other arm muscles. Lateral deltoid Kohnstamm movements were reported to produce the same level of EMG as larger voluntary movements of the same muscle (Mathis et al., 1996). The direction of this

effect is consistent with our findings: we found higher levels of posterior deltoid EMG occurred during a Kohnstamm contraction than during a kinematically matched voluntary contraction. This difference in EMG levels may seem paradoxical given the reported almost-linear relationship between force and EMG at a given deltoid muscle length (Calvert & Chapman, 1977; Hashemi, Morin, Mousavi, & Hashtrudi-Zaad, 2013; Lawrence & De Luca, 1983; Ringelberg, 1985). One possible explanation is a fundamental difference in how the Kohnstamm generator produces muscle force compared to how the voluntary system achieves the same task (Kozhina et al., 1996). For example, the Kohnstamm motor command could be more diffuse and less targeted than the voluntary motor command, recruiting a spatially wider and less optimal range of motor units. The surprising report that Kohnstamm contractions can 'jump' from one muscle to another may be consistent with this view of a diffuse motor command (Craske and Craske 1986; Gurfinkel, Levik, and Lebedev 1989; Gilhodes, Gurfinkel, and Roll 1992; Meigal, Lupandin, and Hanninen 1996; Ghafouri et al. 1998). Importantly, however, we found little evidence that the Kohnstamm motor command spread to the antagonist muscle.

In conclusion, by negating the effects of gravity via the use of a horizontal manipulandum, it was possible to show that the Kohnstamm generator is not a force-dependent positive feedback loop. Instead, it appears to involve a negative feedback loop between a central adaptation, specifying a muscle equilibrium point, and spindle input specifying the disparity between current arm position and the equilibrium value. We propose that this circuit includes two gains, one on the afferent input to the putative Kohnstamm generator, and one located either in the generator itself or on the efferent arm. The reduced response to perturbations for Kohnstamm movements compared to voluntary movements suggests that the Kohnstamm generator has a limited ability to adjust to environmental changes once an aftercontraction has begun, implying a low afferent gain. The high background EMG during the Kohnstamm suggests a strong efferent gain, perhaps reflecting a relationship between the Kohnstamm generator and the circuits that ensure high levels of tonic activity in postural muscles. However, we could not directly measure this efferent gain and the high EMG may be caused by low level factors such as changes in the cellular properties of the motoneurons. Our results do not favour purely peripheral accounts of the Kohnstamm phenomenon, but rather suggest central adaptation of a peripheral-central feedback circuit, reminiscent of the classical long loop.

## Chapter 5. Voluntary motor commands reveal awareness and control of involuntary movement

**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, EMG showed that agonist EMG plateaued during this voluntary inhibition, and resumed its previous increase thereafter. There was no discernible antagonist activation. Thus, a "negative motor command" appeared to temporarily counter the involuntary motor drive, without directly affecting the Kohnstamm generator. In a further condition to test the specificity of the negative motor command, bilateral Kohnstamm movements were induced, and voluntary inhibition was instructed for one arm at random. The results suggested negative motor commands responsible for inhibition are initially broad, affecting both arms, and then become focused. Finally, a psychophysical investigation into the experience of this inhibition found that the aftercontraction was significantly overestimated, relative to voluntary contractions of similar intensity. 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.**

### 5.1. 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 usually brief. 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 (Kohnstamm 1915; Forbes 1926; Craske and Craske 1985).

There is evidence for central (Duclos et al., 2007; Ghosh & Haggard, 2014) 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 et al., 1998; Gilhodes et al., 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. 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), and reflects adaptation of a postural control system (Duclos et al., 2004; Gurfinkel et al., 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, 1925a), 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. 5.1.). 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, may 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.

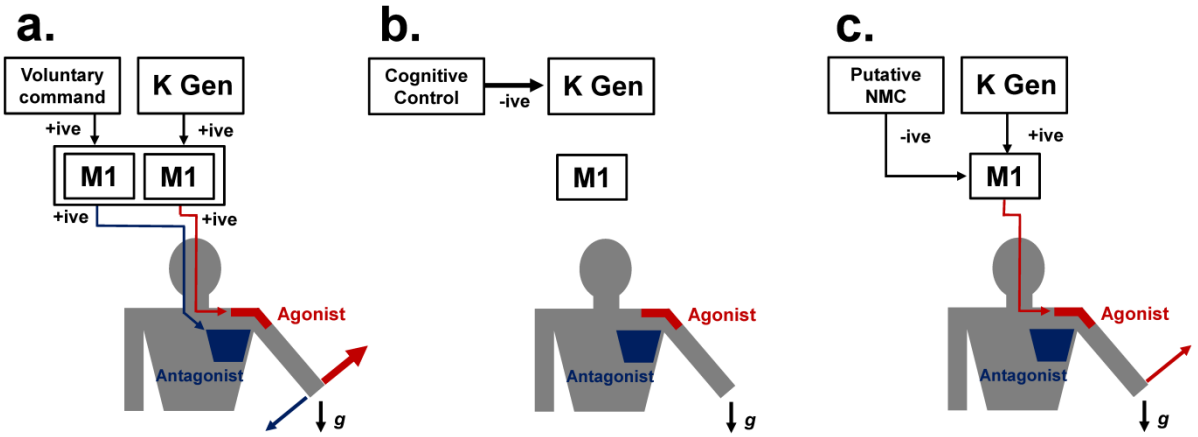



***Figure 5.1. Possible mechanisms for aftercontraction inhibition.*** *Theoretically the arm could be stopped from moving by activation of the antagonist muscle (a). Motor drive to the muscle could be cut by cognitive control circuits 'switching off' the Kohnstamm generator (b). If this was total the 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). 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 a by-product of the downward movement made to counteract the Kohnstamm lift, or it could reflect a lack of efference copies from the generator, to cancel against the sensory inflow from the arm (Blakemore & Frith, 2003; Blakemore, Goodbody, et al., 1998; Blakemore, Wolpert, & Frith, 1998; Frith, Blakemore, & Wolpert, 2000; Shergill et al., 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; Mathis et al., 1996; Pinkhof, 1922).

Here we investigate whether voluntary inhibition acts directly on the Kohnstamm generator to reduce its activation, or merely blocks the link between the generator and the motor apparatus. Previous studies found that after inhibitory adduction of the aftercontraction, subsequent abductive movement of the arm showed reduced EMG (Fessard & Tournay, 1949; Ghosh et al., 2014). This could reflect inhibition reducing the activity of the Kohnstamm generator, or it could simply reflect the decay due to passage of time between the initial and subsequent aftercontractions. Second, to investigate the tuning of voluntary inhibitory control, we measured inhibition of one arm during bilateral Kohnstamm movements. Finally, we measured the perceptual experience of the involuntary aftercontraction during the period when voluntary inhibition holds the arm static, in order to investigate the putative role of efference copies.

## 5.2. Methods

### 5.2.1. 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 et al., 1996; Marsden et al., 1976b), and could suffice to check whether any major recruitment of the antagonist is involved in voluntary inhibition. 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.

### 5.2.2. 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).

### 5.2.3. 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. 5.5A). 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. 5.5A&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. 5.5B). 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 et al., 1921; Parkinson & McDonagh, 2006; Zigler et al., 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 5.1). They rated each statement from -3 (strongly disagree) to 3 (strongly agree) on a 7-point Likert scale.

### 5.2.4. Analysis

Kinematics analysis was performed by determining the angle between the two body-mounted LEDs over time using IMAGEJ (Schneider et al., 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. 5.5A). 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. 5.5C; 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. 5.5B&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. 5.5C; 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. 5.5C; right scatter plot). This was termed the *perceived aftercontraction* (Fig. 5.5D). The *actual aftercontraction* was calculated from the mean EMG during the Kohnstamm inhibition period (0.5 – 2 s post instruction to inhibit; Fig 5.5B&D). *Perceived aftercontraction* was compared to *actual aftercontraction* across participants via a paired sample t-test (Fig. 5.5D).

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.

## 5.3. Results

### 5.3.1. 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. 5.2). Mean antagonist EMG was 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 ($F(1,3) = 0.035$, $p = 0.864$).

Importantly, the inhibition condition showed a reduced agonist EMG trend relative to the control condition (Fig. 5.3). 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 the kinematics, there was a trend towards *offset* response time being faster than *onset* response time (Mean = 496, SD = 240 vs. Mean = 674, SD = 227 ms; $t(13) = 2.16$, $p = 0.05$). 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; $t(13) = 8.04$, $p < 0.001$. 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, 1924b). 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.

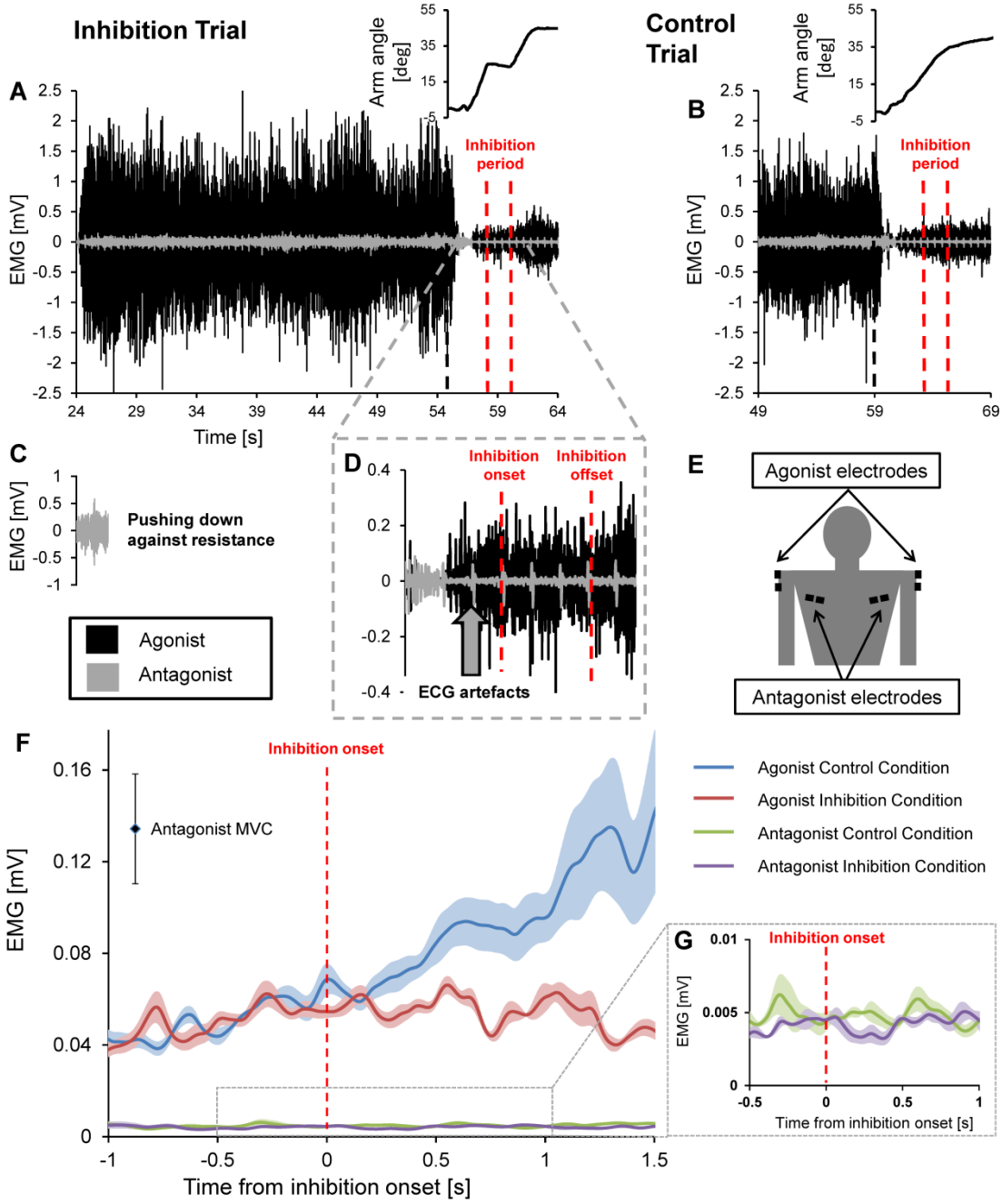



**Figure 5.2.** ***The effect of inhibiting a unilateral Kohnstamm aftercontraction****. Agonist and antagonist EMG and kinematics from a single representative participant during a right arm unilateral inhibition (A) and control (B) trial. Note that antagonist activity was always much lower across both 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.*

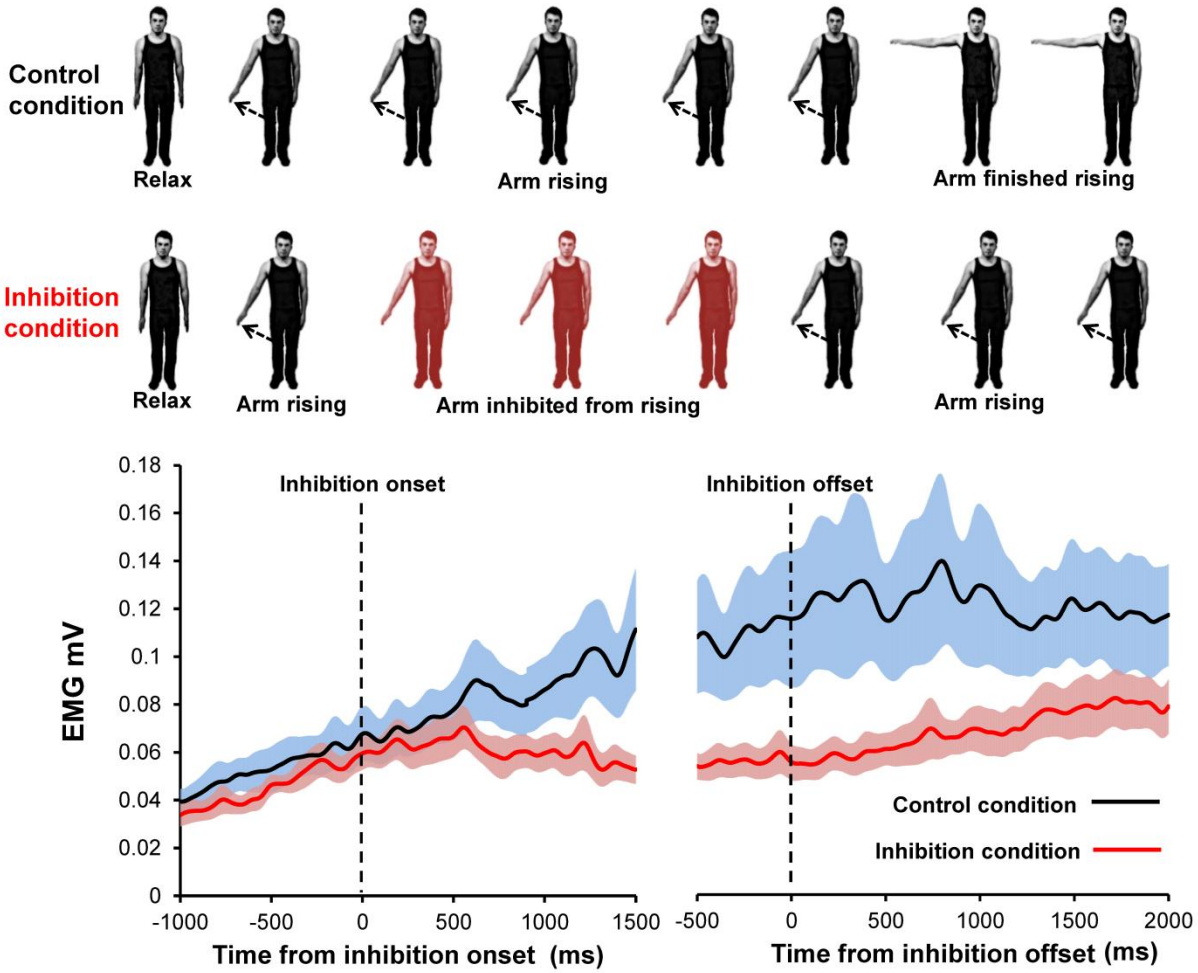



**Figure 5.3.** ***The effect of inhibiting and releasing inhibition of a unilateral Kohnstamm aftercontraction on rectified, smoothed deltoid EMG across participants****. Dashed lines show the time of the onset of the inhibition instruction and offset of inhibition instruction. Error bars show SEM.*

### 5.3.2. No bias in position sense during aftercontraction

Participants were asked to voluntarily replicate the final arm position of Kohnstamm trials in the absence of visual information. There was no significant difference in reproduced final arm angle between Kohnstamm control trials and voluntary control trials, both for unilateral (50.12° vs. 51.53°; $t(13) = 0.52$, $p = 0.61$) and bilateral movements (44.37° vs. 48.37°; $t(13) = 1.33$, $p = 0.21$). This suggests there is no bias in position sense during Kohnstamm movement. However, it should be noted that demonstrating such an effect was not the primary aim of the experiment.

### 5.3.3. 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. 5.4). 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 ($F(1,13) = 0.05$, $p = 0.83$).

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 ($t(13) = 2.12$, $p = 0.054$).

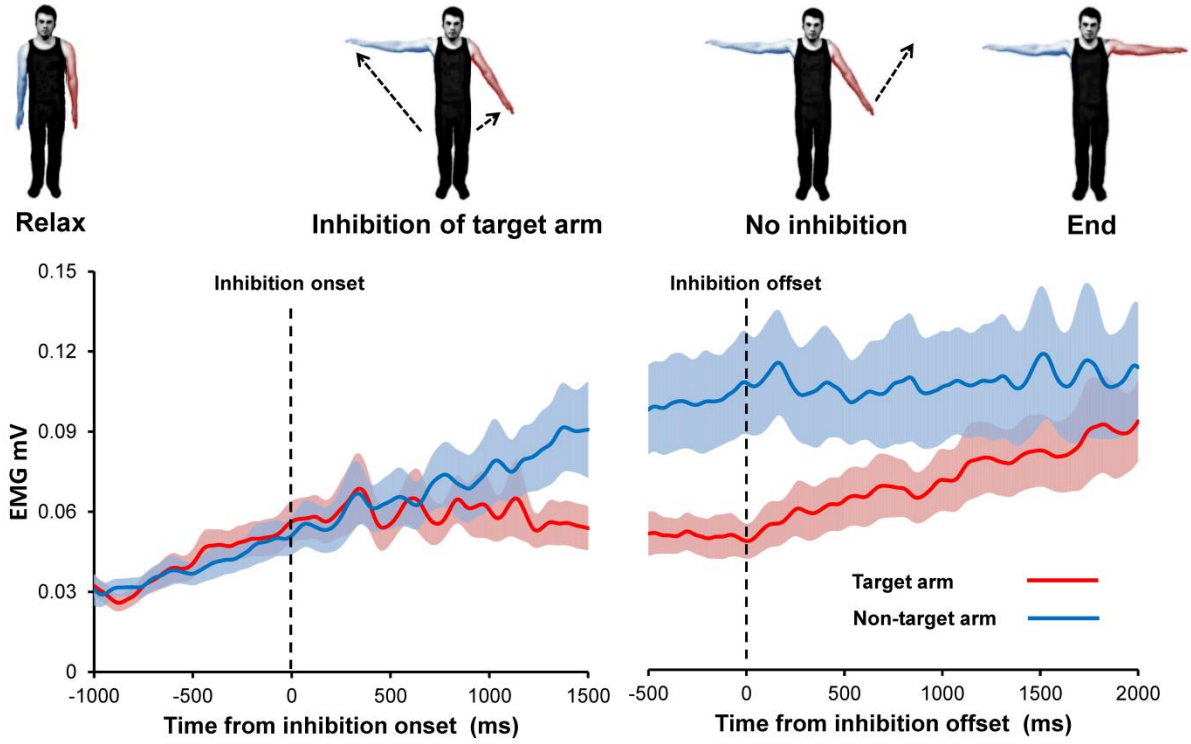



**Figure 5.4.** ***The effect of inhibiting, and releasing inhibition, of a single 'target' arm during bilateral Kohnstamm aftercontraction on rectified, smoothed deltoid EMG.*** *Dashed lines show time of inhibition onset and offset. Note the continued increase in EMG for the non-target arm, together with plateauing EMG in the target arm, beginning approximately 500 ms after the instruction to inhibit. Error bars show SEM.*

### 5.3.4. 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). 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). 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). 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. 5.6), perhaps explaining why they did not cause any change in the EMG trend for the non-inhibited arm overall.

### 5.3.5. Involuntary aftercontraction is overestimated

Participants could perceive the aftercontraction caused by the Kohnstamm generator. The involuntary aftercontraction was perceived as being able to support a weight of 8.61 N (SD = 6.55 N) during the inhibition period (Fig. 5.5B & C). For the participants who successfully performed the weight estimation task (n = 12; Fig. 5.5A & C), this represented an overestimate of the actual EMG level during Kohnstamm inhibition. The perceived aftercontraction was perceptually equivalent to a higher EMG level than was actually present (Mean = 0.0972 mV, SD = 0.0465 mV vs. Mean = 0.0528 mV, SD = 0.0232 mV; t(11) = 4.20, p = 0.0015). That is, participants appeared to experience the aftercontraction as almost twice as strong as an equivalent voluntary contraction (Fig. 5.5D).

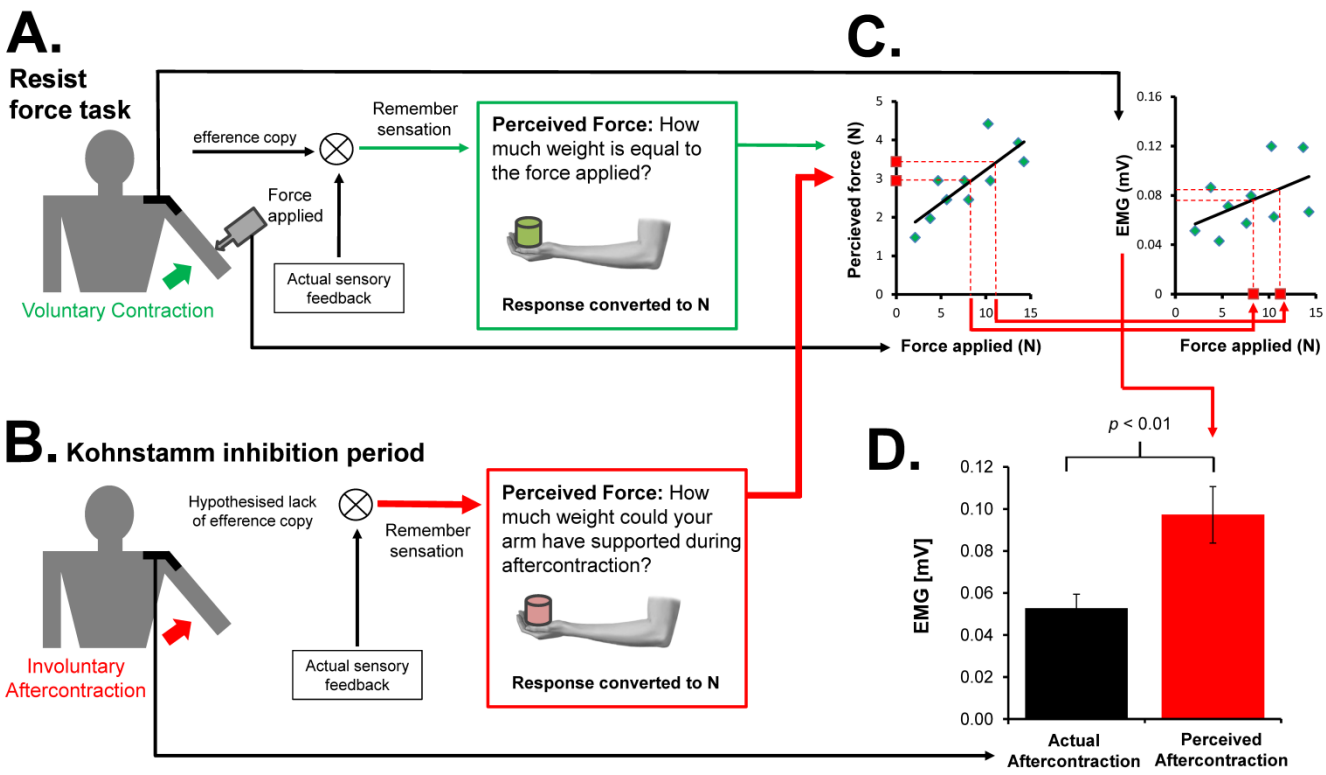



**Figure 5.5**. ***Subjective awareness of the involuntary aftercontraction during voluntary inhibition***. *The methods for estimating perceived force are shown for Voluntary trials (A), and Kohnstamm trials (B), along with the hypothesised difference in sensorimotor attenuation. Results from one illustrative 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. 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).*

### 5.3.6. 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 5.1. 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 |

## 5.4. 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. When participants stopped inhibiting, EMG resumed its previous pattern of increase. 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 'transient bilateral cessations of movement' on some trials. There were significantly more transient bilateral cessations of movement during Kohnstamm than during matched voluntary movements.

The present findings replicate and extend previous reports that the Kohnstamm phenomenon can be voluntarily inhibited (Fessard & Tournay, 1949; Forbes et al., 1926; Ghosh et al., 2014; Pereira, 1925a). Contrary to an earlier report (Pereira, 1925a), inhibition does not involve an absence of agonist activity. Theoretically, inhibition could be achieved by voluntarily contracting the antagonist, cognitive control signals supressing the Kohnstamm generator, or some form of negative motor command (Fig. 5.1.). 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 are forced to postulate a novel neural signal, the "negative motor command" to explain the data (Fig. 5.1C). Several cortical areas have been reported to cause slowing and cessation of movement when directly stimulated (Brown & Sherrington, 1912; Filevich et al., 2012b). The negative motor command could be implemented as an 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).

Our experiment revealed several novel findings regarding putative 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, 1924b; Sapirstein et al., 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”, yet this was not found.

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 et al., 1936, 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 hemisphere, and is consistent with earlier reports (De Havas et al., 2015).

The use of bilateral Kohnstamm and matched voluntary movements allowed us to compare inhibition across these two conditions. 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 et al., 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). Corticomotor excitability related to a task irrelevant leg muscle has been found to be reduced when behaviourally non-selective stopping of the hand was required. However, when behaviourally selective stopping was required, there was no mean leg suppression (Majid, Cai, George, Verbruggen, & Aron, 2012). It has been suggested that 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). Performance in 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.

The basal ganglia may play a key role in the suppression of movement. It has been found that greater striatal activation at the time of selective voluntary movement stopping correlates with greater behavioural selectivity (Majid, Cai, Corey-Bloom, & Aron, 2013). We speculate that the Kohnstamm phenomenon involves decreased coupling between frontal motor regions and the basal ganglia, resulting in a less selective stop signal, compared to during voluntary movement. Interestingly, aftercontractions have been found to be of abnormally long duration in patients with Parkinson's disease (Laignel-Lavastine et al., 1927; Salmon, 1915, 1916, 1925, 1929; Sapirstein et al., 1938), perhaps reflecting an impaired ability to end the involuntary movement via inhibition.

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. 5.6.). 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.

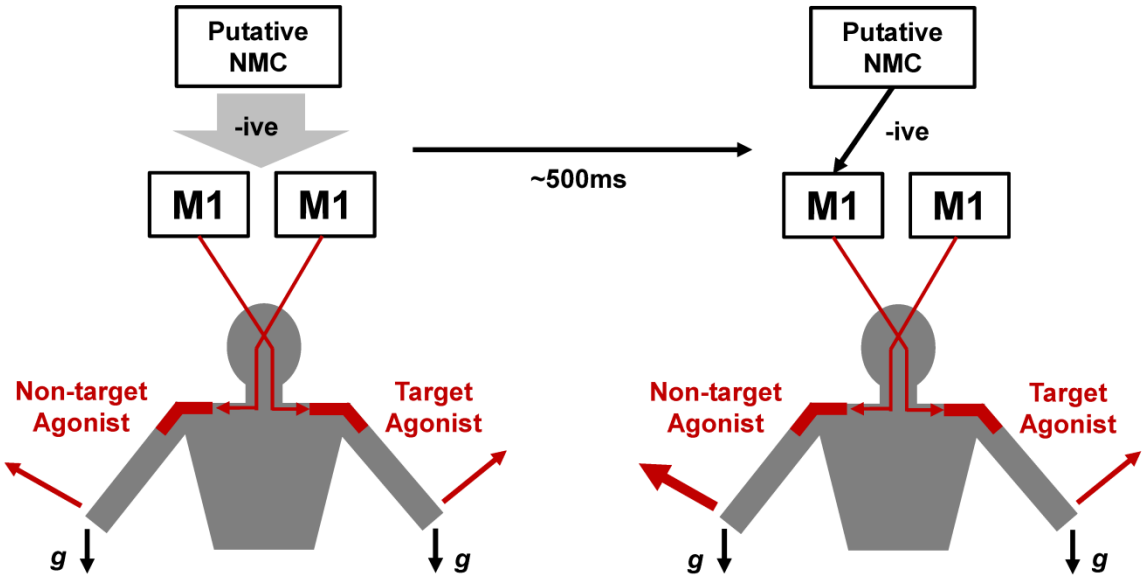



***Figure 5.6. A schematic showing dynamics of putative negative motor commands during bilateral aftercontractions****. Our results suggest that putative negative motor commands have an initially broad focus, but are quickly refined. This explains why both arms sometimes stopped moving, but within ~500ms 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 found that final arm position during voluntary replication movements did not differ from those of Kohnstamm movements. This suggests that there was no bias in position sense during the aftercontraction. A lack of biased position sense suggests 'normal' afferent signalling. Signalling from muscle spindles contributes strongly to position sense (Kuehn et al., 2015; Matthews, 1933; Uwe Proske & Gandevia, 2009; Stuart et al., 1970; Windhorst, 2008), which has previously been found to be normal during Kohnstamm movements (Heide & Molbech, 1973; Howard & Anstis, 1974).

We also found implicit evidence regarding the experience of involuntary movements. Participants estimated 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, 1924b). 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. Thus nothing could be cancelled against the sensory inflow leading 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.

## Chapter 6. General discussion

This thesis has focussed on the Kohnstamm phenomenon. A review of the scientific literature showed a long and sustained interest in this class of involuntary movement, leading to a number of mechanistic accounts of its origin. In contrast, there have been relatively few discussions of how the control of the Kohnstamm differs from the control of voluntary movement. These questions were addressed in a series of experimental studies.

An experiment presented in Chapter 2 revealed that manipulating variability of the input (Fixed vs. Varying) and the form of visual feedback control (Force vs. Position) during the Kohnstamm induction had no effect on the size of the subsequent aftercontraction. These manipulations did not alter the maximum position of the involuntarily rising arm, nor the peak EMG recorded from agonist muscle during the aftercontraction. They also had no effect on the latency of the involuntary movement. Further, the latency of an involuntary movement did not correlate with its eventual magnitude (max. arm angle). Of particular interest was the finding that a near-isotonic contraction (VP condition) can induce an aftercontraction of the same size as isometric inductions, used in all previous experiments. Finally, no evidence was found that time-varying features of the induction motor command persist in the aftercontraction motor output. These findings show that peripheral thixotropic changes and central persistence of the motor command are unlikely to be the mechanisms behind the Kohnstamm phenomenon. They suggest that the Kohnstamm generator is a low-frequency integrator.

Two experiments in chapter 3 showed that afferent signals can have a dramatic effect on the output from the Kohnstamm generator. Obstructing the involuntarily rising arm caused an apparent plateau in the rising agonist EMG signal. This was found for both unilateral and bilateral aftercontractions. When bilateral aftercontractions were elicited, obstructing one arm had no effect on the agonist EMG of the non-obstructed arm, indicating a degree of separation in Kohnstamm generators controlling each arm. Hitting an obstacle induced a stretch response for both Kohnstamm and matched voluntary movements. The size of this stretch-related increase in EMG was slightly, though not significantly, smaller for Kohnstamm movements. This is indirect evidence against increased spindle sensitivity causing

the aftercontraction. Analysis of individual trials showed that the apparent EMG plateau during contact with the obstacle was due to averaging. Contact with the obstacle produced a slow oscillation of EMG, with a continuous negative modulation of the increasing signal. When the obstacle was removed the arm immediately began to involuntarily rise and resumed the previous pattern of increasing agonist EMG. The obstructed arm reached the same final angle as the non-obstructed arm. These findings suggest that afferent input gates the output from the Kohnstamm generator, without affecting the state of the generator itself. Across two experiments subjective estimates of force were larger for Kohnstamm movements compared to matched voluntary movements. This finding is consistent with the hypothesis that the Kohnstamm generator does not provide an efference copy signal.

Chapter 4 presented an experiment where resistive and assistive perturbations were applied to a horizontal Kohnstamm movement and matched voluntary movements. The use of horizontal movement negated the effects of gravity, which created varying loading on the muscle in previous experiments. We found that both Kohnstamm and voluntary movements produced perturbation-dependent changes in agonist EMG, which were consistent with negative-position feedback. Agonist EMG in the no perturbation control condition was higher during Kohnstamm movements than voluntary movements. This higher EMG was not explained by differences in the position or velocity of the arm. Nor was it explained by differences in the antagonist muscle or other synergist muscles. When this high EMG was controlled for, by normalising to the no perturbation control condition, the size of the perturbation response was smaller during Kohnstamm movements than during voluntary movements. For voluntary movements, at 400ms post-perturbation the velocity of the arm in the resistive condition reversed and began to increase. In the assistive condition it reversed and began to decrease. This intentional return to a ‘virtual trajectory’ did not occur in Kohnstamm movements. The results do not support previous positive-force feedback models of the Kohnstamm phenomenon. Instead, they support a negative feedback loop between a central adaptation, specifying a muscle equilibrium point, and spindle input specifying the disparity between current arm position and the equilibrium value. Reduced response to perturbations, suggest the Kohnstamm generator has a limited ability to adjust to environmental changes, implying a low afferent gain. High background EMG

suggests a strong efferent gain (though low level changes in motoneurons cannot be discounted), perhaps reflecting a relationship between the Kohnstamm generator and the circuits that ensure high levels of tonic activity in postural muscles.

Finally, an experiment in chapter 5 demonstrated that participants could voluntarily inhibit the aftercontraction. Participants achieved this by modulating agonist muscle activity, rather than by antagonist contraction. Thus, a “negative motor command” appeared to precisely counter the involuntary motor drive, causing the arm to remain stationary. When they ‘released’ this inhibitory control, the arm immediately began to involuntarily rise, reaching the same arm angle as an uninhibited aftercontraction. This suggests the negative motor command does not affect the Kohnstamm generator directly. In a further condition to test the specificity of the negative motor command, bilateral Kohnstamm movements were induced, and voluntary inhibition was instructed for one arm at random. The results suggested negative motor commands responsible for inhibition are initially broad, affecting both arms. Focussing inhibition on a target arm appeared to be difficult and/or time-consuming. Finally, a psychophysical investigation into the experience of this inhibition found that the upward drive due to the Kohnstamm generator was significantly overestimated, relative to voluntary contractions of similar intensity. This finding is consistent with the hypothesis that the Kohnstamm generator does not provide an efference copy signal.

### 6.1. Peripheral theories are not supported

Purely peripheral theories of the Kohnstamm generator argue that the aftercontraction is caused by increased afferent discharge from muscle spindles (Hagbarth & Nordin, 1998; Hutton et al., 1987). One influential theory posits that this is due to muscle thixotropy (Gregory et al., 1988; Hagbarth & Nordin, 1998). According to that theory, the Kohnstamm induction causes stable cross-bridges to form in extrafusal and intrafusal muscle fibres during a prolonged isometric contraction. Stiffness in the intrafusal fibers causes a contraction of muscle spindles after the induction has finished, resulting in an increased spindle firing rate. This triggers the involuntary movement via the usual reflex pathways (Hagbarth & Nordin, 1998). We found evidence against this theory using a variety of approaches. Hitting an obstacle produced a slightly smaller stretch response during Kohnstamm

movements compared to matched voluntary movements (chapter 3). If, as predicted by peripheral theories, the induction caused muscle spindles to be sensitised, then we should have seen a much larger stretch response during Kohnstamm movements. Likewise, the response to a perturbation should be large. However, we observed that EMG changes in response to perturbations were actually smaller during Kohnstamm movements compared to matched voluntary movements (chapter 4). Signalling from muscle spindles contributes strongly to position sense (Matthews, 1933; Uwe Proske & Gandevia, 2009; Stuart et al., 1970; Windhorst, 2008), and this has been found to be normal during Kohnstamm movements (Heide & Molbech, 1973; Howard & Anstis, 1974). In support of this, we found no bias in arm angle when participants matched an aftercontraction using a voluntary movement (chapter 5).

Our strongest evidence against the thixotropy account is presented in chapter 2. Here we found that an oscillating (1 Hz), near-isotonic contraction produced a large aftercontraction, with the same size as those produced by standard isometric inductions. During this induction the agonist muscle continuously changed length as the arm moved between 70° and 90° of abduction. This would have prevented the formation of stable actin and myosin cross-bridges. According to the thixotropy account this induction should not, therefore, have resulted in an aftercontraction.

More generally, peripheral accounts cannot readily explain the latent period of 1-3 s before the aftercontraction (Csiky, 1915; Gurfinkel et al., 1989; Kozhina et al., 1996; Matthaei, 1924b; Parkinson & McDonagh, 2006; Pinkhof, 1922; Salmon, 1929). If afferent discharge was driving the movement, it should occur within 100 ms of relaxing the arm, since this is the typical duration of the spindle-driven transcortical reflex. It is possible that afferent discharge has to reach a threshold before movement is triggered. If this were true, then greater activity in this putative peripheral ‘Kohnstamm generator’ should reduce the time taken to reach this threshold. In chapter 2, we systematically measured and analysed the latent period, seemingly for the first time. We showed no relationship between the duration of the latent period and the size of the subsequent aftercontraction (chapter 2). Taken together, it seems that changes in muscle spindles alone cannot be responsible for the aftercontraction.

### 6.2. Theories of a persistence of motor command are not supported

Purely central accounts of the Kohnstamm phenomenon have been advanced. This view suggests that activity in the motor cortex persists after the cessation of the induction contraction (Sapirstein et al., 1937, 1938). Another account argues that the Kohnstamm generator is a “kinaesthetic afterimage” (Salmon, 1915, 1916, 1925), which in modern terms might equate to a reactivation of the motor programs involved in the voluntary induction contraction. Such theories argue the aftercontraction is ballistic, involving only feedforward control. However, we found that output from the Kohnstamm generator could be gated by afferent signals (chapter 3). Feedforward models predict that EMG will continue to increase when the arm is in contact with a physical obstacle. However, we observed a plateau followed by a resumed increase once the obstacle was removed. This indicates that the Kohnstamm phenomenon could be strongly modulated by afferent input, so cannot be purely feedforward. We also observed afferent modulation of the aftercontraction in response to assistive and resistive perturbations (chapter 4), again showing that a purely central account of the Kohnstamm phenomenon is inadequate. If the aftercontraction is a reactivation of the motor programs of induction, then strong, time-varying features of induction should also be preserved. We found no evidence for this (chapter 2). Thus, it seems unlikely that a reactivation of motor programs or a persistence of voluntary motor command activity can explain the Kohnstamm phenomenon.

Though recent publications have asserted that a hybrid model of the Kohnstamm phenomenon is correct (Adamson & McDonagh, 2004; Brun et al., 2015; Duclos et al., 2004, 2007), purely central theories had actually not been directly tested until now. As such, the experiments presented in this thesis are important in establishing that purely central accounts are inadequate. Central regions are clearly involved in the Kohnstamm phenomenon (Duclos et al., 2007; Ghosh et al., 2014; Mathis et al., 1996; Parkinson et al., 2009). The finding that time-varying features of the induction motor command do not persist in the aftercontraction motor output is also important in constraining these hybrid theories. This also applies to the finding that bilateral Kohnstamm generators show a degree of separation.

### 6.3. Separation of bilateral Kohnstamm generators

One early paper reported that bilateral aftercontractions were dramatically smaller than unilateral aftercontractions (Paillard, 1951). However, lack of statistical testing means that this could merely reflect the known variability of the Kohnstamm phenomenon. Across multiple experiments, we observed no difference in the final arm angle of bilateral or unilateral aftercontractions (chapters 3 and 5). We found that during bilateral aftercontractions obstructing one arm had no effect on the agonist EMG signal from the non-obstructed arm (chapter 3). We also observed that voluntary inhibition of one arm had no lasting effect on the non-inhibited arm EMG (chapter 5). Taken together, these results suggest a profound difference between voluntary and Kohnstamm movements. Simultaneous performance of two *voluntary* actions with different hands is difficult, and tends to produce coupling and entrainment effects (Franz & Ramachandran, 1998; Kelso, Southard, & Goodman, 1979a, 1979b; Shea, Boyle, & Kovacs, 2012). These are often attributed to the low bandwidth of a central process: intentional actions tend to occur one at a time, and in series. In contrast, two Kohnstamm movements for each limb can proceed independently, apparently without strong coupling, and apparently without bandwidth limitations. This strongly suggests that the Kohnstamm movement recruits independent generator processes in each hemisphere. In contrast, the Kohnstamm movement does not recruit the same cognitive mechanisms as intentional action, such as the prefrontal cortex.

We did observe that on many trials there was a transient (< 500 ms) slowing of the non-inhibited arm. However, this was likely due to the difficulty of focussing the "negative motor command" for voluntary inhibition, rather than any feature of the Kohnstamm generator. These findings show that there is a degree of separation in bilateral Kohnstamm generators. Nevertheless, other studies have found interactions between the arms (Allen & O'Donoghue, 1927; Brun et al., 2015; Craske & Craske, 1986). These might merely reflect differences between the arms in the strength of the induction, rather than any true interaction.

Reports that the aftercontraction can switch from one arm to another (Craske & Craske, 1986) were also not verified via EMG, but seem related to the well-established finding of muscle switching during the aftercontraction (Ghafouri et al., 1998; Gilhodes et al., 1992; Gurfinkel et al., 1989; Meigal et al., 1996). Across our

experiments we did not observe any evidence of spontaneous muscle switching. The mechanisms of this muscle switching clearly warrant further study (see *outstanding questions* below). It may be that output from the Kohnstamm generator passes through regions where sensory input can induce a switch in the output muscle (Gilhodes et al., 1992). Nevertheless, until such a switch occurs, Kohnstamm generators controlling different muscles apparently retain independent control. A recent study involving aftercontractions of the bicep contradicts this finding (Brun et al., 2015). Here, passive movement of the other arm and implied movement from vibration were found to increase the velocity of the involuntarily moving arm. Further work is needed to determine if interactions of this sort only occur when both limbs are moving, and if they are sensitive to posture and the specific muscles used.

### 6.4. Subjective 'strangeness' of Kohnstamm phenomenon due to lack of efference copy

Voluntary and involuntary movement may be physically identical, yet they feel very different. The enduring scientific interest in the Kohnstamm phenomenon may relate to the strange feelings it produces (Craske & Craske, 1985; Csiky, 1915; Forbes et al., 1926; Kohnstamm, 1915; Matthaei, 1924b). Many have noted that during the aftercontraction the arm feels lighter than normal (Allen & O'Donoghue, 1927; Craske & Craske, 1985; Cratty & Duffy, 1969; Gurfinkel et al., 1989; Matthaei, 1924b; Parkinson & McDonagh, 2006; Salmon, 1915), seems to fly or float upwards (Craske & Craske, 1985; Kohnstamm, 1915; Pinkhof, 1922; Salmon, 1916, 1929), causing surprise (Fessard & Tournay, 1949; Rothmann, 1915; Salmon, 1915; Schwartz & Meyer, 1921) and a strong sense that the movement is automatic and involuntary (Allen, 1937; Allen & O'Donoghue, 1927; Parkinson & McDonagh, 2006; Rothmann, 1915; Salmon, 1925; Salomonson, 1921; Schwartz & Meyer, 1921). However, these reports have not been systematically tested. In Chapter 5, we quantified these various elements via questionnaire. Participants agreed with previous subjective descriptions of the Kohnstamm. They also agreed that the movement felt like it had an *external* cause. Moreover, they confirmed other elements of the experience not previously reported. Namely, that the 'strange' experience was limited to the moving limb, and that the movement seemed smoother than normal movements. They also found the experience interesting and pleasant.

Like other examples of “voluntariness” and “involuntariness,” these experiences often elude experimental measurement. One previous experiment attempted to measure the subjective feeling of lightness by having participants pull upwards on a spring using a voluntary movement to match the force they felt was being exerted by an aftercontraction in the other arm (Matthaei, 1924a). It was reported that stronger aftercontractions were associated with a larger overestimation of the force. However, the methodology meant that it was hard to exclude voluntary movements on both arms. There was also no statistical testing. We found that Kohnstamm forces were significantly overestimated relative to voluntary forces in response to contacting physical obstacles (chapter 3). This was found for both verbal ratings of force and implicit force replication. We also found that during voluntary inhibition, participants estimated that the ‘floating’, stationary arm could support surprisingly high weights (chapter 5). This agrees with reports of a sensation of resistance as participants adducted voluntarily against the aftercontraction (Ghosh et al., 2014). We found that the Kohnstamm drive was perceptually overestimated relative to equivalent voluntary drive. These robust overestimations of Kohnstamm forces are consistent with the view that the Kohnstamm generator does not send efference copies that may be used to cancel the predictable sensory consequences of *voluntary* action (Blakemore & Frith, 2003; Blakemore, Goodbody, et al., 1998). Several studies suggest efference copies underlying perceptual attenuation of self-generated events originate at a relatively high level of the action control hierarchy, upstream of the primary motor cortex (Haggard & Whitford, 2004; Voss et al., 2006).

Neuroimaging studies of the Kohnstamm phenomenon showed activation in primary motor areas during aftercontractions (Duclos et al., 2007), but, interestingly, did not show significant activations of the medial frontal regions hypothesized to generate the efferent signals that contribute to action awareness (Fried et al., 1991; Haggard, 2011; Haggard & Magno, 1999; Haggard & Whitford, 2004).

## 6.5. What do experiments on Kohnstamm phenomenon reveal about motor control?

The Kohnstamm phenomenon apparently occurs in all skeletal muscles (Forbes et al., 1926; Matthaei, 1924b) and may represent an adaptation within the

postural control system (Fessard and Tournay, 1949; Gurfinkel, Levik, and Lebedev, 1989; Ghafouri et al., 1998; Adamson and McDonagh, 2004; Duclos et al., 2004). We found that the Kohnstamm generator is a low-frequency integrator (chapter 2). Postural maintenance and modulation occurs at a lower frequency than voluntary movement. It has been suggested that the Kohnstamm phenomenon represents an amplification of the normal involuntary postural drive, which supplies tonic motor efference (De Havas et al., 2015; Gurfinkel et al., 1989). If this is the case, then the findings in this thesis provide insights regarding the control principles of the postural system. Across two sets of experiments (chapter 3 and 5) we showed that input (afferent and central) to the system can temporarily limit tonic motor efference, without permanently altering the state of the generator. This may explain how the postural system automatically achieves switching between extended periods of fixed posture and brief periods of movement. The apparent afferent resetting of oscillations in motor efference during contact with an obstacle (chapter 3) may be related to the afferent resetting of central pattern generators reported in the animal literature (Guertin et al., 1995; Perreault et al., 1999; Schomburg et al., 1998; Stecina et al., 2005). There is increasing evidence for central pattern generators in humans (Duysens & Van de Crommert, 1998) and recent experiments have shown that Kohnstamm inductions can cause involuntary air-stepping (Selionov et al., 2009). Indeed, it has long been speculated that the Kohnstamm phenomenon may be related to repetitive actions such as locomotion (Craske & Craske, 1986; Sapirstein et al., 1937, 1938). Our finding, that a rhythmic, near-isotonic contraction can induce an aftercontraction of equal size to the standard fixed, isometric induction (chapter 2), seems consistent with this theory.

Involuntary movements often feel uncontrollable. We showed that precise voluntary control of the involuntary aftercontraction was, in fact, possible, through voluntary inhibition (chapter 5). Postural control must be subservient to the voluntary system if movements are to be efficient. Previous observers have noted that voluntary movements appear to sum on top of aftercontractions (Hick, 1953). This may be related to the finding of biases in force generation following isometric contractions (Hutton et al., 1984, 1987; Knight et al., 2008; Shea, Guadagnoli, & Dean, 1995; Shea et al., 1991). Our experiments on voluntary inhibition (chapter 5), suggest that the output of the Kohnstamm generator can be voluntarily reduced, to

precisely match the strength of the contraction to stabilise the arm. Since this was done without peripheral involvement of the antagonist, we hypothesised a central “negative motor command”. Though the “negative motor command” may have broad focus initially, it can be quite rapidly targeted to the specific limb, indicating a degree of peripheral feedback is involved in this form of cognitive action control. We also found that aftercontractions are likely controlled via negative-position feedback control in a similar manner to voluntary movements (chapter 4). This may explain how voluntary and involuntary drives within the motor system can be efficiently integrated.

## 6.6. A model of the Kohnstamm phenomenon

### 6.6.1. The Kohnstamm generator

Our experiments show that purely peripheral theories of the Kohnstamm generator are inadequate. Moreover, we found no evidence across multiple experiments for increased firing rates/sensitivity in muscle spindles, relative to voluntary movements. Many accounts see the increase in spindle afferent firing as the initial generator of the Kohnstamm movement. It can therefore be concluded that the Kohnstamm generator is not just a simple reflex loop between afferent input and efferent output (Fig. 6.1. left panel). Instead, the Kohnstamm generator consists of a central adaptation, which includes a low-frequency integrator (Fig. 6.1. right panel). This Kohnstamm generator may be activated by afferent and/or efferent input during the induction (see *outstanding questions* below). Upon the cessation of the induction and the relaxation of the muscle, output from the Kohnstamm generator causes the aftercontraction. The Kohnstamm generator remains active throughout the aftercontraction, but gradually becomes less active as central adaptations decay. While the generator is central, rather than peripheral, it receives some important peripheral inputs (see next section). Thus, the aftercontraction is not controlled by purely central, feed-forward mechanisms.

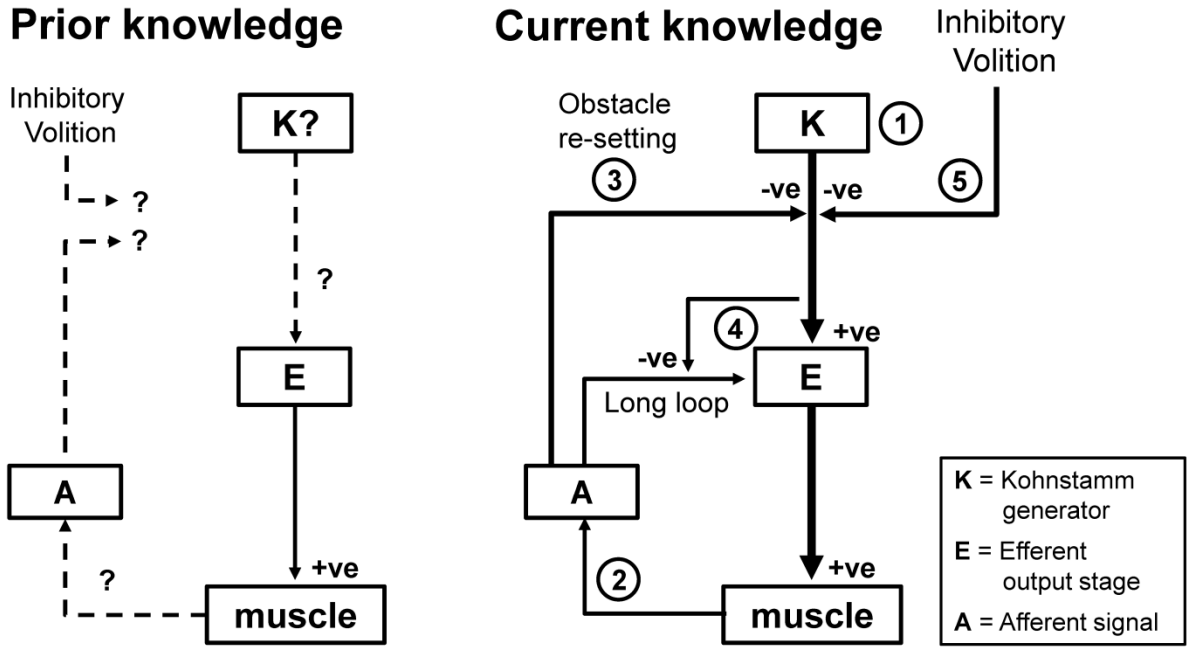



***Figure 6.1. A model of the Kohnstamm phenomenon.*** *The figure shows the prior and current state of knowledge regarding the control principles of the aftercontraction. Previously (left panel), the state of afferent signal [A] was not known. It had been proposed that spindle firing/sensitivity could be strong enough in itself to drive the aftercontraction, which would locate the putative Kohnstamm generator [K] in the periphery. It was not known if changes in the afferent signal affected a central Kohnstamm generator or only a subsequent efferent output stage [E]. It was known that voluntary inhibition of aftercontractions was possible. However, it was not established if this signal affected the Kohnstamm generator directly or just the efferent output stage. It was also not known if this inhibitory volition signal could precisely oppose the aftercontraction. The current state of knowledge, resulting from the experiments in this thesis is shown on the right panel. The key new knowledge added is: 1. Low frequency integration of afferent and/or efferent signal activates a central Kohnstamm generator during the induction. 2. Afferent signalling from muscle spindles does not appear to differ to that during voluntary movements, and the Kohnstamm generator is not purely peripheral. 3. A strong negative afferent signal caused by physical obstruction of the aftercontraction continually re-sets/gates the signal from the Kohnstamm generator, without directly affecting the generator itself. 4. The aftercontraction is caused by a strong central signal, combined with a peripheral negative position feedback control loop. The gain on the efferent arm appears to be large, while the gain on the afferent input appears to be small, relative to voluntary movements. 5. Negative inhibitory volitional commands can precisely oppose the positive output from the Kohnstamm generator, without directly affecting the generator itself.*

### 6.6.2. Negative position feedback control

The aftercontraction involves an interaction between a central generator, and a peripheral negative position feedback control loop. An adaptation of the central generator may specify a muscle equilibrium point, and spindle input may specify the disparity between current arm position and the equilibrium value (Fig.6.1. right panel (4)). This circuit includes two gains, one on the afferent input to the putative Kohnstamm generator, and one located either in the generator itself or on the efferent arm. The reduced response to perturbations for Kohnstamm movements

compared to voluntary movements, suggests the Kohnstamm generator has a limited ability to adjust to external changes once an aftercontraction has begun, implying a low afferent gain. The high background EMG during the Kohnstamm suggests a strong efferent gain. The arm eventually stops moving/begins to fall because the weight of the arm in the gravitational field creates a larger oppositional torque to the torque generated by the aftercontraction, and because the central adaptations gradually decay. Maximum arm angle during aftercontraction therefore depends on a complex interplay between the physical properties of the arm (weight, muscle characteristics, moment arm), the level of activation in the Kohnstamm generator, and the relative weighting of efferent and afferent gains in the negative-feedback loop.

### 6.6.3. Afferent resetting/gating

Afferent input has a more dramatic effect on the aftercontraction in the case of the limb encountering a physical obstacle. The afferent signal gates or continually re-sets the output from the Kohnstamm generator (Fig. 6.1. right panel (3)). This process limits the size of the aftercontraction. The limb maintains a relatively constant force against the obstacle, while EMG shows slow oscillations about a constant value. Afferent re-setting does not affect the Kohnstamm generator itself. When the obstacle is removed the arm immediately begins to rise because the Kohnstamm generator is still active and the activity level is higher than that which is required to maintain the arm against gravity (hence an upward force on the obstacle). Once the obstacle is removed, afferent signal contributes to the aftercontraction again via negative position feedback control (see above). As a result, the final arm angle is the same as if the aftercontraction had not been obstructed (providing obstruction is brief enough for the decay of the central adaptation to be minimal).

### 6.6.4. Voluntary inhibition

The Kohnstamm aftercontraction is under voluntary inhibitory control, so it is not completely ‘automatic’. Nevertheless, these negative motor commands do not appear to affect the Kohnstamm generator directly. Negative motor commands sum with positive motor commands below the level of the Kohnstamm generator (Fig. 6.1. right panel (5)). Negative motor commands can be specified precisely to match the positive motor command and thus produce a stable efferent signal. Our experiments

suggest that these commands need to be sent continuously to maintain stable output. It is not known if negative motor commands are modified by afferent feedback.

### 6.7. Outstanding questions

A key outstanding question is whether afferent input during the voluntary induction is necessary for the aftercontraction to occur. Our experiments show that the aftercontraction is probably not driven by a high afferent discharge. However, we were not able to show what aspects of the afferent signal, if any, contribute to 'activating' the Kohnstamm generator during the induction. The finding that variable and near-isotonic inductions produce aftercontractions of equal size to standard isometric inductions is potentially informative, since the afferent signal during induction clearly differed across the conditions. However, due to the complexity of muscle afferent signalling and the inter-related nature of efferent and afferent commands, firm conclusions are not possible. One account posits that the Kohnstamm phenomenon is caused by discharge from muscle spindles during the induction (Duclos et al., 2004, 2007; Gilhodes et al., 1992; Gurfinkel et al., 1989). Discharge from muscle spindle primary endings strongly increases during an isometric contraction or a vibratory stimulation (Edin & Vallbo, 1990). For isometric contractions during the Kohnstamm induction this may be due to co-activation of α and γ motoneurons (Edin & Vallbo, 1990; Vallbo, 1974). This proprioceptive signal could cause a central adaptation within brain areas signalling muscle length, essentially setting up a new-equilibrium point for the muscle, which would in turn trigger the aftercontraction (Duclos et al., 2004). We did not find any evidence to contradict the theory that spindle signalling initially sets up the generator during the induction phase. However, we showed that continued spindle discharge is not itself the likely generator of the aftercontraction.

The location of the central component of the Kohnstamm generator remains unknown. Spinal (Matthaei, 1924b; Pinkhof, 1922; Schwartz, 1924; Schwartz & Meyer, 1921; Zigler, 1944), sub-cortical (Foix & Thevenard, 1923; Rothmann, 1915) and cortical (Salmon, 1915, 1916, 1925; Sapirstein et al., 1936, 1937, 1938) loci have all been proposed. Our experiments, and others (Ghosh et al., 2014), suggest

that M1 is an output region for the generator, rather than housing the Kohnstamm generator itself. It seems unlikely that higher regions such as the SMA could be the locus of the generator, given they are not active during the aftercontraction (Duclos et al., 2007). It remains unknown what regions of the cortex are necessary for the aftercontraction to occur.

The basal ganglia appears to play an important modulatory role in Kohnstamm phenomenon. Several studies have noted abnormally prolonged aftercontractions in patients with Parkinson's when a single muscle is tested (Laignel-Lavastine et al., 1927; Salmon, 1915, 1916, 1925, 1929; Sapirstein et al., 1938). However, when multiple muscles are involved, the Kohnstamm induction fails to produce the usual involuntary air stepping in patients with Parkinson's (Selionov et al., 2013). The role of the cerebellum is less clear. The cerebellar vermis was found to be more active during aftercontractions than during matched voluntary movements (Duclos et al., 2007). However, a single case has been reported of a jerky aftercontraction in a patient with cerebellar damage (Sapirstein et al., 1938). Thus, it seems it may also have a modulatory role, rather than being the location of the Kohnstamm generator. A spinal origin is plausible, given the emerging similarity between certain features of the Kohnstamm phenomenon and spinal central pattern generators studied in animals (Selionov et al., 2009). However, no direct evidence exists.

Another unresolved issue is the overlap between the Kohnstamm and voluntary motor system. The experiments presented in this thesis show that the two systems closely interact and share some of the same control mechanisms. Studies involving fMRI and TMS have not found any dramatic differences between Kohnstamm and voluntary movements (Duclos et al., 2007; Ghosh et al., 2014; Mathis et al., 1996; Parkinson et al., 2009). It could be argued that the two forms of movement are highly similar, only differing in terms of how they are attributed, based on the presence or absence of an efference copy. However, subtle differences, such as the higher EMG activity coupled with low afferent gains observed in our experiments (chapter 4) and the observed differences in the frequency of single motor units (Kozhina et al., 1996), suggest the underlying mechanisms may be dissociable.

A question related to voluntary control is whether putative negative motor commands operate via the same control principles as normal, positive motor commands. The concept of “negative motor command” is a novel one, based largely on results obtained in this thesis. However, it may be related to the negative motor areas identified neurosurgically, where stimulation leads to movement arrest (Filevich et al., 2012a, 2012b). However, the functional relevance of such areas remains controversial. We showed that putative negative commands can precisely oppose the output from the Kohnstamm generator, are apparently continuously updated, and can rapidly adjust from an initially broad ‘motor focus’ (both arms inhibited) to a narrower ‘motor focus’ (only one arm inhibited). These findings imply that the putative negative motor command may be modified by sensory feedback. However, this has not been directly tested.

An intriguing finding reported in the literature is that the aftercontraction can switch muscles (Ghafouri et al., 1998; Gilhodes et al., 1992; Gurfinkel et al., 1989; Meigal et al., 1996). It is apparently induced by large alterations in the light entering the eyes (Gilhodes et al., 1992). However, the mechanisms for how light causes this switch remain unknown. Exploring how this light-induced switching occurs may reveal where the Kohnstamm generator is located and how its anatomical pathways are organised.

Numerous papers have reported that there are reliable individual differences in how strongly the aftercontraction manifests. It has been reported that the aftercontraction is pronounced in emotionally positive people and weaker in emotionally negative people (Kohnstamm, 1915; Laignel-Lavastine et al., 1927; Salmon, 1925, 1929; Sapirstein, 1948, 1960; Sapirstein et al., 1937). These differences are amplified in the psychiatric range (Salmon, 1915; Sapirstein, 1948, 1960). However, there have been no modern studies. It could be that such differences are merely due to levels of task compliance. Alternatively, there may be genuine differences, reflecting variation in sensorimotor organisation. Now that more is known about the control mechanisms of the Kohnstamm phenomenon, studies of individual differences will become informative. Moreover, it could be useful to examine if the aftercontraction is altered by pharmacologically induced state changes. It remains unknown why the aftercontraction manifests such large individual differences, being sustained in some healthy participants, rapid or

oscillatory in others, and completely absent in the remainder (Meigal & Pis'mennyi, 2009).

### 6.8. Future Directions

The most obvious way to establish the role of afferent signals in the Kohnstamm phenomenon is to create a complete afferent block and measure the effect on the aftercontraction. Prior to the completion of the experiments in this thesis, it could have been argued that a lack of aftercontraction under these circumstances occurred because the generator itself was a muscular signal (i.e. the thixotropy account). Now, it is clear that if the aftercontraction fails to develop following an afferent block, then afferent signalling during the induction is necessary for the aftercontraction to occur. Alternatively, if no difference is found in the aftercontraction following an afferent block, then it can be concluded that efferent signals are sufficient to 'activate' the Kohnstamm generator. One previous report in the literature found that anaesthetising the deltoid muscle does not affect the aftercontraction (Matthaei, 1924b). However, the extent of the afferent block was not established. Nor were any statistical comparisons performed on the size of the aftercontraction before and after the injections of novocaine. Establishing a full afferent block for large muscles like the deltoid is difficult. A better approach would be to focus on wrist extensor and flexor muscles. These muscles have been studied in Kohnstamm experiments (Duclos et al., 2007; Forbes et al., 1926; Pinkhof, 1922; Rothmann, 1915) and in the context of the effects of afferent blocks on the TVR (Kaji et al., 1995).

If afferent signals do transpire to be necessary for the aftercontraction to occur, it will be further evidence of an overlap with the TVR. Testing if the findings reported in this thesis hold for movements induced by tendon vibration would be informative. In particular, it would be interesting to establish if obstacles, inhibition and perturbations have the same effect on agonist EMG. TVR may be preferable to Kohnstamm for future experiments requiring high numbers of trials, since fatigue is less of an issue.

If afferent signals are not necessary for the aftercontraction to develop, the use of an afferent block could reveal if putative negative motor commands are modified by sensory feedback. This would be tested by comparing several seconds

of voluntary inhibition of the aftercontraction under baseline conditions and following an afferent block. If sensory feedback modifies negative motor commands, one would expect the EMG signal during inhibition to be significantly more variable in the afferent block condition compared to baseline. Bilateral aftercontractions could also be investigated. If sensory feedback modifies negative motor commands, then a unilateral afferent block should impair participants' ability to selectively inhibit the aftercontraction of one wrist, while allowing the aftercontraction of the other wrist to develop normally.

Determining if cortical regions besides M1 are necessary for aftercontraction to occur will ultimately require studying patients with focal lesions or experiments where TMS is used to temporarily disrupt function during specific phases of the Kohnstamm phenomenon (induction, latent period, aftercontraction). Sensorimotor integration regions in the posterior parietal cortex are active during the aftercontraction (Duclos et al., 2007). On one hypothesis, this region contributes to processing a proprioceptive signal, which sets up a new-equilibrium point for the muscle, which in turn causes the aftercontraction (Duclos et al., 2004). Effects of applying offline repetitive TMS to this region prior to the induction could be compared to a control region elsewhere in the cortex to determine if putative shifts in the equilibrium point develop during the induction. Alternatively, it may be that signals from parietal regions trigger the aftercontraction. This could be tested by selectively applying TMS during the latent period. If the aftercontraction was prevented or systematically delayed it would support posterior parietal involvement in the generation of the Kohnstamm phenomenon. However, the activity found in such regions could just reflect normal sensory feedback relating to the movement that accompanies an aftercontraction.

fMRI experiments could systematically modify the latent period. The Kohnstamm latent period can be extended for several seconds via the use of a physical obstacle. If enough trials could be collected, it would therefore be possible to create a jittering effect in the onset of the aftercontraction. This would negate the poor temporal acuity of fMRI and could reveal the pattern of cortical activity that immediately precedes an aftercontraction, whilst avoiding problems associated with correlated regressors, and confounding afferent signals during the aftercontraction itself. Furthermore, effective connectivity analysis could be employed using the

primary motor cortex as a seed region. Regions that showed connectivity to the primary motor as a function of the imminent occurrence of an aftercontraction would be candidates for causing the aftercontraction. Contrasts with voluntary movements would be informative, as well subtractions against baseline conditions where no induction was completed.

One way to address questions of separation between the Kohnstamm and voluntary system is to test if adaptations acquired through voluntary movement translate to the aftercontraction. Force field adaptation has shown that voluntary movement control is highly adaptive and that these adaptations can be long lasting (Huang & Shadmehr, 2009; Hussain & Morton, 2014; Shadmehr & Mussa-Ivaldi, 1994). Similar techniques could be applied to the Kohnstamm phenomenon. Participants could repeatedly encounter obstacles or perturbations during a voluntary training session involving slow movements matched to their average aftercontraction velocity. Next, aftercontractions would be elicited in the same environment. If adaptations where transferred, one would expect to see anticipatory changes in aftercontraction velocity and EMG as participants' arm approached the location of the former perturbation. Effects could be compared to purely voluntary conditions to determine the degree of transference between the systems. A related question concerns whether the Kohnstamm system learns over larger timescales. It would be interesting to compare aftercontractions before and after several days of wearing arm weights. Larger aftercontractions after this period would imply that the Kohnstamm phenomenon reflects activation of a postural support system which provides support to the limb based on its weight. If no change in aftercontraction was observed it would suggest that the Kohnstamm system cannot learn. Smaller aftercontractions might suggest feedback between an internal model of limb weight and afferent signals during the aftercontraction.

Light-induced muscle switching could provide a means to study cortical involvement in the aftercontraction. Previous experiments have found that the size of MEPs is proportional to the background level of EMG in the contracting muscle (Mathis et al., 1996). This could reflect the excitatory state of the motor cortex, but it could also be due to the general level of excitation in the descending motor pathways. One way to dissociate these possibilities would be to set up an experiment where an aftercontraction was established in one muscle and then

switched to another. First, the time taken from the onset of the light to the onset of the muscle switch would be established. TMS could be time-locked to occur at a specific interval (e.g. 150-200 ms) after the onset of the light, but before the onset of the muscle switch (experimental condition). The coil would be located such that an MEP would be elicited in the as-yet-inactive muscle. The size of the MEP would be compared to conditions where the TMS pulse occurred immediately before the onset of the light (baseline). If MEP amplitude was larger in the experimental condition than baseline it would suggest cortical activity causes the aftercontraction. It may also be informative to apply TMS to other selective regions of sensorimotor cortex, again time-locked to the onset of the light. If switching could be prevented then it would indicate that visual information was not only operating at a subcortical level, as has previously been suggested (Gilhodes et al., 1992).

Individual differences in the Kohnstamm phenomenon have not been explored with modern techniques. It would be interesting to determine if aftercontraction size depended on the level of fine motor control in a specific muscle. This could be assessed by testing groups of individuals with expertise in different disciplines. For example, one could assess leg and arm aftercontractions in professional footballers and archers. If aftercontractions were systematically larger in the legs of footballers and the arms of archers, one might conclude that fine motor control contributed to the excitability of the Kohnstamm generator.

The association between emotional traits and states and the aftercontraction may seem only superficially interesting. However, one can speculate that there may be a more fundamental linking mechanism. The finding that the Kohnstamm phenomenon involves a strong central adaptation invites the question of what constitutes this adaptation. One possible cause is plateau potentials. Plateau potentials, caused by persistent inward currents, are a type of electrical behaviour found in spinal cord cells. They are thought particularly important to spinal motor systems (Hultborn, Zhang, & Meehan, 2013; Svirskis, Gutman, & Hounsgaard, 2001). These sustained, positive inward currents produce lasting depolarisation, causing the cells to fire independently of synaptic input. Persistent inward currents are established by descending serotonergic inputs (Abbinanti, Zhong, & Harris-Warrick, 2012; Perrier, Rasmussen, Christensen, & Petersen, 2013). The association between low serotonin, low mood, and clinical depression is well

established (Casacchia, Pollice, Matteucci, & Roncone, 1998). Perhaps the reduced aftercontraction found in these groups reflects a common underlying serotonergic mechanism. Low mood causes a slumped posture (Oosterwijk, Rotteveel, Fischer, & Hess, 2009). There is also a circular relationship, with upright posture causing positive emotions and self-evaluations (Briñol, Petty, & Wagner, 2009; Peper & Lin, 2012) and reducing negative memory bias in clinically depressed patients (Michalak, Mischnat, & Teismann, 2014). Participants describe the aftercontraction as pleasant and interesting. It may be that large aftercontractions are caused by a postural system that normally provides a high level of tonic muscle activity, which in turn enhances serotonergic function. One way to begin tackling this hypothesis would be to measure aftercontractions in a large sample of participants who also completed extensive mood trait/state questionnaires. Another approach would be to use acute tryptophan depletion (ATD) in healthy participants. ATD produces a marked reduction in plasma tryptophan and consequently brain serotonin synthesis and release (Bell, Abrams, & Nutt, 2001). ATD has been found to increases movement latency in healthy controls (Mace, Porter, Dalrymple-Alford, Wesnes, & Anderson, 2010), indicating that motor effects are detectable. If the serotonin hypothesis is correct, ATD should also reduce the size and duration of the aftercontraction. Other approaches, such as interventions involving good posture training, could also be employed to explore whether the aftercontraction can be modified. Positive correlations with mood ratings would be expected.

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