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# Brain activation in an involuntary human action

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ABSTRACT

This work investigated human brain activity in healthy subjects during an involuntary movement. The involuntary movement was driven by an involuntary postural aftercontraction of the deltoid muscle of the shoulder which follows the cessation of a prolonged isometric voluntary contraction. Previous authors have suggested that this aftercontraction phenomenon does not involve the cerebral cortex. To test this idea we examined brain activation using functional magnetic resonance imaging (fMRI) during the involuntary movement and during a matched voluntary movement. In contrast to the conjectures of earlier authors, during the involuntary movement there was widespread activation of the cerebral cortex. There were also clear activation differences between conditions. The voluntary movement showed activation of the putamen whereas the involuntary movement showed much greater activation of the anterior cingulate cortex (BA 24/32). There were also some similarities in the brain areas activated under both movement conditions namely in the left hemisphere precentral gyrus (BA 4), the left hemisphere superior parietal lobe (BA 7), and the bilateral superior temporal gyrus (BA 22). Activity was also present in the caudate nucleus, the thalamus, and the cerebellum. The results are discussed in relation to theories of aftercontraction generation and error processing by the anterior cingulate.



## 1. Introduction

Normally our limbs move only when instructed to do so; however, involuntary movement can occur following brain damage as in the case of 'alien limb' syndromes (Scepkowski and Cronin-Colomb, 2003). Imaging of the brain in patients with these syndromes may throw light on the brain regions which can generate movement in the absence of intention on the part of the subject. However, clinical cases have the major defect that the cortical damage is rarely discrete and widespread areas of the cerebral cortex may be affected. The brain areas responsible for involuntary movement would be easier to identify if such a response could be generated in the undamaged neural networks of a healthy person. We have recently investigated (Parkinson and McDonagh, 2006; Adamson and McDonagh, 2004) an involuntary movement in healthy subjects in which there is no conscious selection of a behavioural response and no voluntary release of a prepared action. There is no sense of agency (de Vignemont and Fourneret, 2004) but the sense of ownership of the limb is maintained. This involuntary movement can be evoked by voluntarily contracting the muscle as strongly as possible against a fixed resistance for one minute (maximal voluntary contraction—MVC). After this contraction ceases, the resistance is removed, and following a latency of 2 to 7 s (Fessard and Tournay, 1950), a movement of the limb caused by an involuntary contraction of the muscle takes place which is referred to as an aftercontraction or Kohnstammsche Phano-

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men (Kohnstamm, 1915). The aftercontraction is most easily evoked in the deltoid muscle of the shoulder and some other proximal and axial muscles. The strength and duration of the aftercontraction are in proportion to the strength and duration of the preceding voluntary contraction (Sapirstein et al., 1936; Allen and O'Donoghue, 1927; Brice and McDonagh, 2001) and the involuntary movement comes as a surprise to a subject experiencing it for the first time (Sapirstein et al., 1938; Fessard and Tournay, 1950).

It is not known which areas of the CNS are responsible for the involuntary aftercontraction. Electrical stimulation of the muscle does not evoke the phenomenon (Kohnstamm, 1915; Matthaei, 1924) which suggests that it is not a type of intramuscular contracture as suggested by Pereira (1925) but instead is driven from the nervous system. Different regions of the nervous system have each had their advocates: the peripheral nerves (Forbes et al., 1926), the spinal cord (Matthaei, 1924), the brainstem (Foix and Thevenard, 1923), the basal ganglia (Gurfinkel et al., 2006), or the cerebral cortex (Sapirstein et al., 1938). All these theories have been based on conjecture and circumstantial evidence.

In the current experiments, we used functional magnetic resonance imaging (fMRI) to directly examine which cortical and subcortical areas are involved during the involuntary flexion of the shoulder joint caused by an aftercontraction. Brain activation during a matched voluntary flexion was also recorded as was the activation during an isometric maximal voluntary contraction (MVC), which preceded and evoked the involuntary movement.

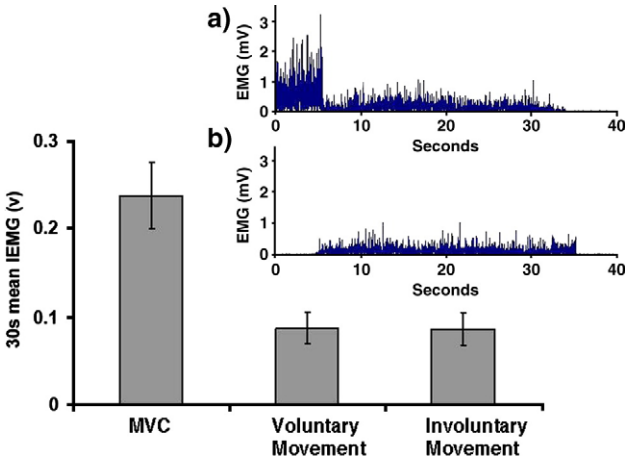


**Fig. 1 – EMG activity of the right deltoid muscle during the experimental conditions. Group data (*n*=6) are shown as an average integrated EMG (IEMG) for the final 30-s activation period for the isometric maximal voluntary contraction (MVC), the voluntary, and the involuntary (AC) movement conditions. Error bars are SEM. Inset: Raw-rectified EMG from a single subject. Traces show (a) the last 5 s of the isometric MVC followed by the 30-s involuntary aftercontraction and 5-s rest and (b) a single repetition of the voluntary movement with 5-s rest either side of the movement.**

## 2. Results

### 2.1. *Arm motion and muscle activity*

In the involuntary condition (Fig. 3, lower panel), aftercontraction of the deltoid muscle and movement of the arm occurred after the cessation of a 60-s *isometric* maximal voluntary contraction (MVC) of this muscle and lasted for 30 s before the arm was voluntarily brought down to the initial position. The arm movement involved flexion at the shoulder joint.

In the voluntary condition (Fig. 3, upper panel), the subject performed a voluntary flexion of the shoulder joint which matched the involuntary flexion. Three-dimensional motion tracking was used during scanning to record arm movement trajectories. Final arm position was measured as the final displacement of the hand from the standard rest position. It was similar in the involuntary (115.4±13.6 mm) and voluntary (105.4±17.7 mm) conditions (mean ± SEM, $N=5$).

The electromyograms (EMGs) during the voluntary and involuntary movement were of similar magnitude in the sample of 6 subjects (Fig. 1) tested which was 10%–30% of that recorded during the subjects 60-s maximal isometric contraction. The EMG recordings during the whole protocol were acquired in a prior session in the laboratory.

### 2.2. *Functional imaging data*

Group results ($N=11$) from the quantitative analysis of the most activated volumes will be considered first (Tables 1 to 5). All the regions listed are separate significant clusters with the maximum z-score for each cluster being reported. For those activations which are central in the mid sagittal plane, only the coordinate for the strongest Z score of activation is reported. To complement this statistical analysis, a visual display is given of the activations in each condition at identical coordinates (Fig. 2).

#### 2.2.1. *Analysis*

The fMRI data were modelled in blocks (see Experimental procedures). The 30 s of the final voluntary movement, the 30 s of involuntary movement and the 60 s MVC were all modelled as separate blocks and compared with the preceding/rest period for that condition (Fig. 3). We deal first with the main conditions vs rest and then with the contrasts.

*2.2.1.1. Main conditions vs rest (Table 1 and Fig. 2).* The coordinates shown for all experimental conditions shown in Fig. 2 are $z=54$, $x=0$, $y=16$. This is done to allow direct visual comparison between activations for all conditions at identical coordinates. The coordinates chosen are those which give the clearest visual display of activations during the involuntary movement. Thus, Fig. 2 represents a snapshot at one set of coordinates so not all of the areas mentioned in Table 1 appear activated.

In general, there appears to be much more widespread activity at the chosen coordinates in the involuntary condition (Fig. 2a) as compared to voluntary condition (Fig. 2b). During the voluntary movement of the right arm (Table 1 and Fig. 2), activation occurred in the left precentral gyrus: primary motor cortex. In Brodmann area 4 (BA 4), activation was also present bilaterally in the superior temporal gyrus (BA 22) and area V5/MT: middle temporal visual area (BA 19). There was activation

**Table 1 – Significant functional brain activations present during voluntary movement, involuntary movement (aftercontraction), and during the isometric 60 s MVC.**

| Condition | Cluster area (Gyral location) | Side L/R | Brodmann area | Z score (max) | MNI co-ordinates of max voxel |
|---|---|---|---|---|---|
| Right arm voluntary movement | V5/MT | R | 19 | 6.10 | 46, −76, −6 |
| | | L | 19 | 6.01 | −46, −84, −6 |
| | Caudate (head) | R | | 5.73 | 4, 18, 2 |
| | | L | | 2.52 | −4, 18, 4 |
| | Precentral gyrus | L | 4 | 4.76 | −24, −26, 58 |
| | Superior temporal gyrus | R | 41/42 | 4.55 | 40, 6, 2 |
| | | L | 41/42 | 4.37 | −46, 6, −2 |
| | Superior parietal lobule | L | 7 | 4.43 | −38, −52, 62 |
| | Cerebellum | R | | 4.39 | 10, −62, −18 |
| | Thalamus | L | | 3.85 | −12, −16, 22 |
| | Putamen | L | | 3.59 | −24, −14, 20 |
| Right arm involuntary movement | Superior temporal gyrus | R | 41/42 | 6.85 | 60, 8, 14 |
| | | L | 41/42 | 3.82 | −50, 4, 16 |
| | Cerebellum | R | | 5.93 | 2, −66, −20 |
| | Precentral gyrus | L | 4 | 5.85 | −32, −26, 48 |
| | Superior parietal lobule | L | 7 | 5.35 | −38, −44, 68 |
| | Anterior cingulate | R | 24/32 | 5.31 | 8, 14, 38 |
| | Thalamus | L | | 4.11 | −18, −28, 16 |
| | Medial frontal gyrus (SMA) | L | 6 | 3.56 | −4, −18, 54 |
| | Caudate (head) | R | | 3.00 | 2, 14, 16 |
| MVC of deltoid (isometric contraction) | Cerebellum | R | | 10.13 | 2, −68, −18 |
| | | L | | 6.80 | −2, −70, −16 |
| | Precentral gyrus | L | 4 | 6.36 | −26, −26, 56 |
| | Thalamus | L | | 5.44 | −4, −38, 2 |
| | Caudate (tail) | R | | 5.29 | 24, −38, 16 |
| | Superior temporal gyrus | R | 41/42 | 4.27 | 60, 2, −2 |
| | Superior parietal lobule | L | 7 | 3.67 | −38, −42, 66 |

All movements were performed with the right arm. Areas are ranked according to z scores.

in the left hemisphere superior parietal lobule (BA 7), caudate nucleus, thalamus and putamen, and right hemisphere of the cerebellum.

The brain activation during the involuntary movement (Table 1 and Fig. 2a) had similarities to that of the same matched voluntary movement. There was clear activation present in the left hemisphere precentral gyrus (BA 4), the superior temporal gyrus (BA 22), and the left hemisphere superior parietal lobe (BA 7). Activity was also present in the caudate nucleus, the thalamus, and the cerebellum.

The involuntary movement also showed activations which were unique to this condition. There was strong activity in the right anterior cingulate cortex (BA 24/32) which is also clearly illustrated in Fig. 2 where the most striking result of a comparison of panel (a) with panel (b) is the prominent activation of the anterior cingulate in the involuntary movement a feature not present in the voluntary movement. Also unique to the involuntary condition was activation of the left medial frontal gyrus: supplementary motor area SMA (BA 6). The activation of area V5/MT seen in the voluntary movement was not present in the involuntary movement.

The lowest section of Table 1 and Fig. 2c shows activations during the isometric MVC which precedes the involuntary movement. Data from the MVC are shown because it could be suggested that the involuntary movement activation is purely a weak copy of that produced during the powerful isometric voluntary muscle action which precedes it. A comparison of Fig. 2a with Fig. 2c and of the two lower sections of Table 1 suggests that this could be true with respect to the right superior temporal gyrus, the left superior parietal lobe ,the left precentral gyrus, the right cerebellum and the left thalamus, i.e., continuing activity of these areas during the involuntary movement. However, the high activation of the right anterior cingulate (BA 24/32) and left medial frontal gyrus SMA (BA 6) and right head of caudate appears unique to the subsequent involuntary movement. Again examining the two lower sections of Table 1, significant activations present in the MVC and not in the involuntary movement are the left cerebellum and the right tail of caudate.

*2.2.1.2. Voluntary vs involuntary movement (Tables 2 and 3).* Due to the physical constraints of creating more than one involuntary movement in any single run, the data analysis compared the involuntary movement to the last single voluntary movement of the run. This allowed a fair comparison across the two conditions.

Activation during the voluntary movement minus that during the involuntary movement (Table 2) detected activation in the right middle temporal gyrus (BA 21), the caudate, the putamen, and the thalamus. The reverse contrast, involuntary movement data minus that during the voluntary movement condition (Table 3), showed activation in the left anterior cingulate (BA 32), the left dorsal premotor area (BA 6), the right inferior frontal gyrus (BA 44), and the left superior temporal gyrus (BA 22).

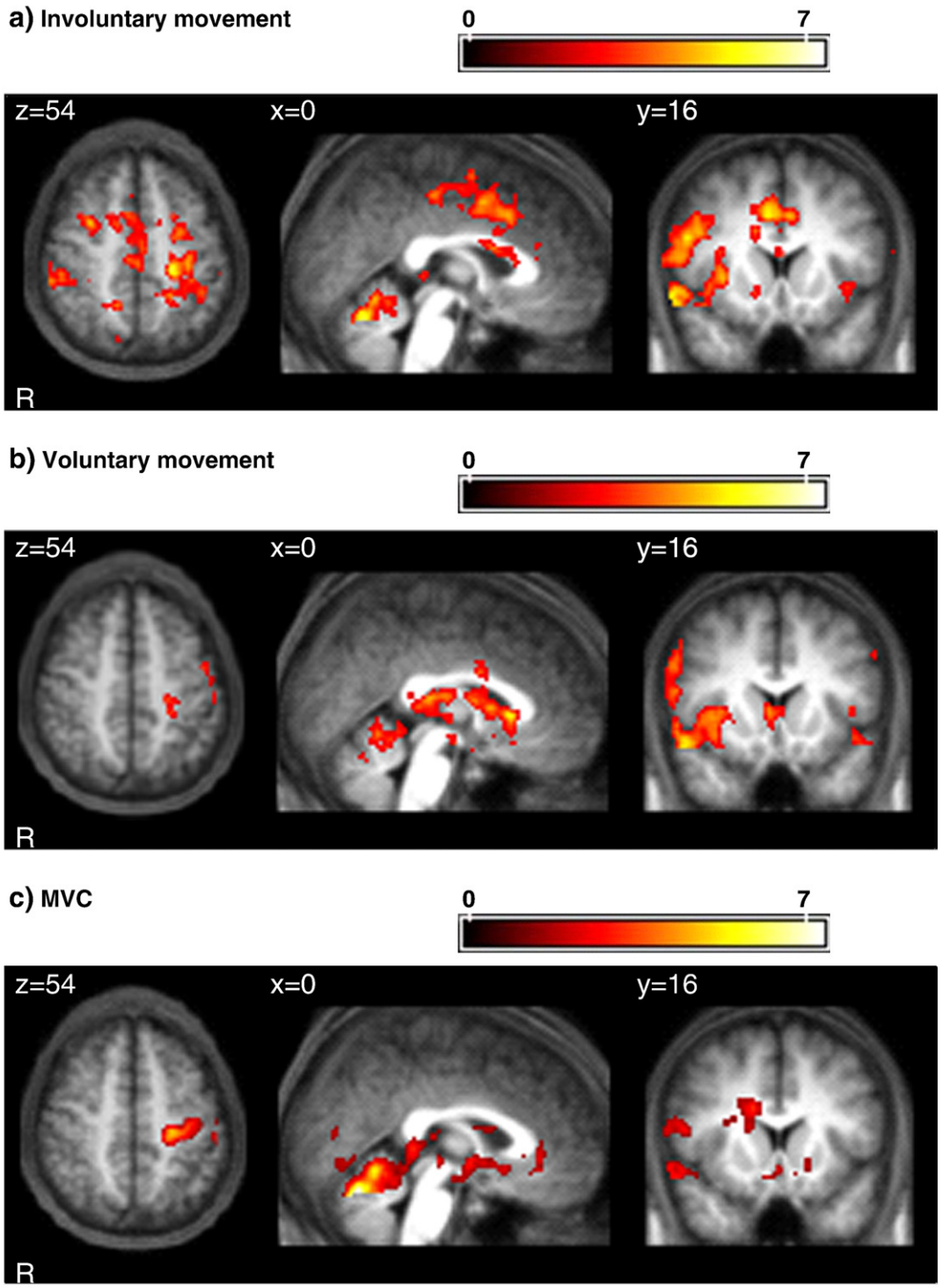


**Fig. 2 – Brain regions showing a significant increase in BOLD signal ($n=11$) during the (a) involuntary movement, (b) voluntary movement, and (c) 60-s isometric maximal voluntary contraction (MVC) which preceded the involuntary movement and initiated it. 'R': right hemisphere.**

2.2.1.3. *Maximal voluntary contraction (MVC) vs subsequent involuntary movement (Tables 4 and 5).* The isometric MVC initiated the subsequent involuntary movement. Activation during the involuntary movement minus that during the 60 s MVC (Table 4) showed significant activation in the right medial frontal gyrus (SMA, BA 6), the left precuneus (BA 7), the left posterior cingulate (BA 31), and the right anterior cingulate (BA 24). When the activation during involuntary movement was subtracted from that during the MVC (Table 5) significant activation was detected in the right cerebellum, putamen, and caudate.

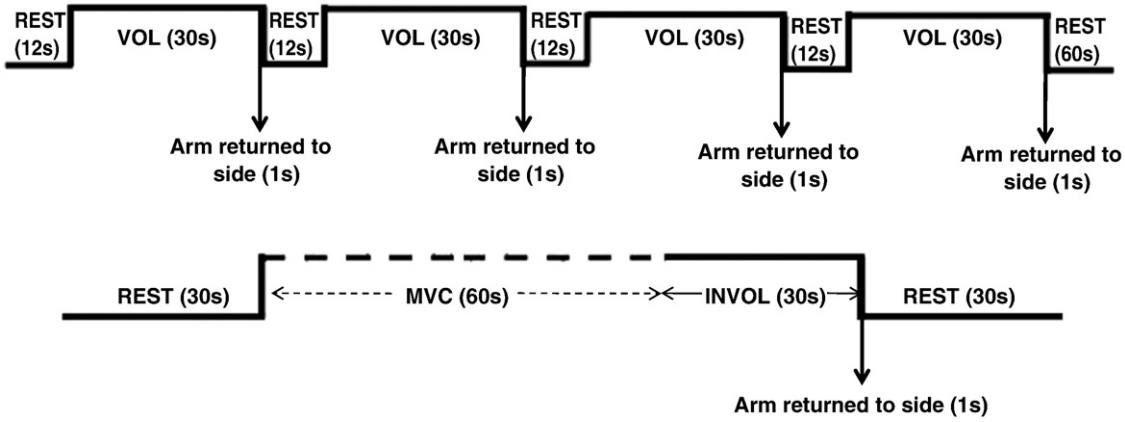


**Fig. 3 – Experimental design. Voluntary movement protocol (upper panel): There were four trials of the 'voluntary movement condition' in one run. These were performed before and separate to the involuntary aftercontraction protocol. The arm was raised for 30 s and was lowered for 1 s. There was a rest period of 12 s before and after each movement. After the last rest period, a further 60 s elapsed before the next run for the involuntary protocol took place. Involuntary movement protocol (lower panel): Following a 30-s rest period, the subject performed a maximal voluntary isometric contraction of m.deltoid for 60 s against a fixed resistance. Following this minute, the hand released the fixed resistance and an involuntary movement then ensued which produced flexion of the shoulder joint. After 30 s of involuntary movement, the arm was then lowered voluntarily for 1 s and returned to its supine position for a rest period of 30 s. The second run was then terminated.**

## 3. Discussion

One of the most surprising findings in this work is the widespread activation of cerebral cortex motor areas during a simple involuntary movement produced by an aftercontraction. This finding helps to settle some very old arguments about the anatomical origin of the involuntary aftercontraction which have continued for nearly a century. In particular, some authors suggested that the aftercontraction is purely a spinal phenomenon. This is clearly not the case. The discussion will focus on the statistically significant results recorded in the tables rather than the qualitative data of Fig. 2. However, many of the points are also well illustrated in Fig. 2.

### 3.1. *Similarities between brain activations during involuntary and voluntary movements*

The voluntary and involuntary movements showed similar activations in the precentral gyrus (motor area BA 4), superior parietal area (BA 7), and the thalamus. These are all areas involved in movement (Wise and Shadmehr 2002). The superior parietal area is believed to contain a model of the current limb orientations (Pellijeff et al., 2006). The involvement of these motor areas in the involuntary aftercontraction suggests that the phenomenon is driven from the cerebral cortex, a possibility not allowed for in the hypotheses of some earlier authors (Matthaei 1924; Forbes et al 1926).

The activations were strongest on the left side of the brain in both conditions as might be expected from a movement of the right arm. The superior temporal area, caudate nucleus, and the cerebellum also showed similar activations under both conditions but here they were strongest on the right side of the brain. This would be particularly expected for the cerebellum where it is known that ipsilateral processing is dominant. Cerebellar activation indicates the importance of sensory feedback under both conditions (Thickbroom et al., 2003). The role of the caudate and cerebellum in movement production and control has been extensively investigated. The role of the superior temporal area has been less studied in relation to movement. However, recently, it has been implicated in biological motion perception (Saygin, 2007). In all the above areas except the caudate, the activations were, surprisingly, strongest in the involuntary movement.

### 3.2. *Differences between brain activations during involuntary and voluntary movements*

There were also clear differences in activations between the involuntary and voluntary movements (Table 1). The anterior

**Table 2 – Significant differences: voluntary minus involuntary movement.**

| Cluster area (gyral location) | Side L/R | Brodmann area | Z score (max) | MNI co-ordinates of max voxel | Cluster size (voxel) |
|---|---|---|---|---|---|
| Middle temporal gyrus | R | 21 | 6.17 | 46, −78, −4 | 3579 |
| Caudate (head) | R | | 4.69 | 8, −2, 16 | 1007 |
| Putamen | L | | 4.50 | −22, −14, 18 | 607 |
| Thalamus | R | | 4.26 | 4, −26, 12 | 998 |

**Table 3 – Significant differences: involuntary minus voluntary movement.**

| Cluster area (gyral location) | Side L/R | Brodmann area | Z score (max) | MNI co-ordinates of max voxel | Cluster size (voxel) |
|---|---|---|---|---|---|
| Superior temporal gyrus | L | 22 | 5.81 | −36, −16, 2 | 711 |
| Inferior frontal gyrus | R | 44 | 4.00 | 50, 4, 18 | 1268 |
| Anterior cingulate | L | 32/24 | 3.95 | −14, −6, 64 | 1070 |
| Dorsal premotor | L | 6 | 3.52 | −28, −6, 44 | 1070 |

cingulate cortex (ACC) was strongly activated in the involuntary movement but not in the voluntary movement. The medial frontal gyrus (SMA BA 6) was also more active in the involuntary movement. In the contrast analysis, between conditions activation of the more dorsal lateral parts of area 6 was greater in the involuntary movement than in the voluntary (Table 3). In the contrast (Table 4) of involuntary movement minus prior isometric contraction (MVC), area 6 was again prominent. The preferential activation of the SMA in the involuntary movement is surprising as this area is believed to be concerned with motor planning. The planning of postural arm positioning may be carried out elsewhere, perhaps in the brain stem (Takakusaki et al., 2004). It is also possible of course that this activation of the SMA in the involuntary movement could be related not to motor causation of movement but to the detection of an involuntary action as discussed below for the anterior cingulate.

In the voluntary movement area, V5/MT and the left putamen were strongly activated but in the involuntary movements activations in these areas were insignificant (Table 1). Area V5/MT is a region of the visual cortex which is involved with motion perception and the integration of local motion into more global percepts (Born and Bradley, 2005). Interestingly, activation of this area was not present during the isometric 60 s MVC when there was no limb movement.

The activation of the putamen taken with the results from the caudate above suggests a higher general level of activation in the basal ganglia during the voluntary efforts. This would fit with evidence that the basal ganglia are associated with the production of internally rather than externally triggered movements (Cunnington et al., 2002). The basal ganglia have also been implicated in the preparation of actions (Elsinger et al., 2006) and in the selection of motor programmes which are essential for survival (Grillner et al., 2005).

### 3.3. *Activation of the anterior cingulate in the involuntary movement*

The activation of the anterior cingulate cortex (ACC), during the involuntary movement requires further discussion. This region was also found to be highly activated in a study which investigated aftercontractions in the wrist extensor muscles (Duclos et al., 2007). The ACC is a region where motor control, homeostatic drive, emotion and cognition come together. It has reciprocal connections with limbic structures ,brainstem nuclei, thalamus, prefrontal cortex, spinal cord and motor cortex (Paus, 2001). One possibility is that the ACC has a motor role in driving the postural centres in the brain stem (Takakusaki et al., 2004) and or the primary motor cortex to produce the involuntary movement. However, cingulate activation was not strongly present in the 60 s of MVC which preceded the involuntary movement, but it was present during the involuntary movement. This indicates that the pattern of brain activations in the involuntary movement is not just a weak version of the activation in the prior isometric MVC. Furthermore, it also suggests that the ACC might be performing processing functions other than those relating to motor activity.

The processing functions usually associated with strong activation in the ACC are the production of response errors and the monitoring of conflicting response alternatives (Taylor et al., 2007; Carter et al., 1998). This conclusion from human studies is supported by unit activity in the ACC of the monkey associated with behavioural errors (Niki and Watanabe, 1979). Subsequently, event-related fMRI studies in humans have shown extensive ACC activation during response errors in a go/no-go task (Kiehl et al., 2000) and when subjects actively reject a trial to avoid errors (Magno et al., 2006).

ACC-related errors could arise during the involuntary movement in the following manner. There is evidence that the brain predicts the sensory consequences of a motor command using a forward model (Wolpert and Kawato, 1998). This sensory feedback is compared to the predicted model, and a subsequent error signal is generated (Frith et al., 2000). Errors could arise at two stages in the generation of the involuntary movement. First error: we assume that the 60-s maximal voluntary contraction has the effect of increasing the magnitude of the stored value for the predicted load signal (for the sake of argument from 1 to 3 units). On cessation of the

**Table 4 – Significant differences: involuntary movement minus the 60-s isometric MVC.**

| Cluster area (gyral location) | Side L/R | Brodmann area | Z score (max) | MNI co-ordinates of max voxel | Cluster size (voxel) |
|---|---|---|---|---|---|
| Medial frontal gyrus | R | 6 | 4.84 | 14, 2, 56 | 690 |
| Precuneus | L | 7 | 4.42 | −22, −50, 54 | 3072 |
| Posterior cingulate | L | 31 | 4.13 | −12, −36, 34 | 3072 |
| Anterior cingulate | R | 24/32 | 3.23 | 20, 0, 46 | 690 |

**Table 5 – Significant differences: 60-s isometric MVC minus the involuntary movement.**

| Cluster area (gyral location) | Side L/R | Brodmann area | Z score (max) | MNI co-ordinates of max voxel | Cluster size (voxel) |
|---|---|---|---|---|---|
| Cerebellum | R | | 5.32 | 2, −68, −20 | 504 |
| Putamen | R | | 3.87 | 28, −6, 10 | 462 |
| Caudate | R | | 3.71 | 18, 2, 22 | 462 |

voluntary maximal isometric contraction, the actual load on the muscle reduces to 1 unit but the predicted value of 3 units remains in store. The difference between the predicted and the actual load then creates an error signal of 2 units prior to the initiation of arm movement. This error probably drives the subconscious movement control system to issue a signal to the motor nerves which is too strong. This causes the involuntary movement of the arm as the force produced is greater than that required to balance the weight of the arm (e.g., 1 unit).

Second error: as this movement is clearly not voluntary there will subsequently be a further large error between the predicted and real sensory states with respect to *dynamic* movement. The predicted sensory state will be that the arm is stationary whereas the actual physiological state is movement. It is certainly true that subjects who are naive to the aftercontraction phenomenon experience surprise when their arm moves. The activity in the cingulate cortex could be related to one or both of these sources of error. We believe that this involuntary movement paradigm has much future potential for studies investigating error processing during motor acts.

### 3.4. *Summary*

In summary, we have shown that the involuntary aftercontraction is not a purely segmental phenomenon as hypothesised by some early authors; on the contrary, it involves widespread activation of the cerebral cortex. Further, in the general context of the current debate about unconscious and conscious processing (Koch and Crick 2001; Sumner and Husain 2008), this paradigm provides further evidence of the breadth and intensity of cortical processing in unconscious actions.

## 4. Experimental procedures

### 4.1. *Participants*

Eleven subjects, three of whom were female, completed the scanning experiments (age 28±10 yr, height 1.76±0.06 m, weight 76.9±9 kg). We were able to obtain 3D movement data from five of these and EMG data from six. All subjects were right-handed, and the paradigms required the use of the right arm. All tasks were practiced by subjects outside the scanner and laboratory prior to acquiring data in order to familiarise them with the protocol. All the participants who were scanned completed safety screening forms and passed the screening conditions. Participants signed informed consent to all procedures in accordance with the Declaration of Helsinki.

### 4.2. *Procedures*

Each subject was required to attend two sessions. The first session was in the laboratory where the full protocol was administered to the subject the day prior to the scanning. In addition, the subjects practiced matching with a voluntary movement the trajectory and final position of the hand attained during the involuntary movement. EMG and arm movement recordings were taken with the subject lying supine. In the second session, the same protocol was administered to the subject in the fMRI scanner.

In all experimental conditions, subjects were lying supine with a cushion beneath their knees to keep their legs slightly flexed in both laboratory and scanner sessions. The subject's arm with elbow extended lay parallel to their trunk and was supported by a foam wedge so that the angle between the limb and the horizontal was approximately 40°. The head was strapped firmly to the head plate of the coil, and subjects were told to prevent head movement. Instructions relating to the different aspects of the task were delivered over a high-resolution audio system (Philips head phones using an air-driven (pneumatic) system which blocked out 25 dB of noise).

Subjects performed two different conditions; right arm voluntary movement (Fig. 3, upper panel) and then right arm involuntary movement caused by an aftercontraction (Fig. 3, lower panel). The less-fatiguing voluntary movement protocol preceded the involuntary movement protocol so as to minimize any effects of fatigue.

#### 4.2.1. *Voluntary arm movement condition*

The sequence for the voluntary movement condition was a 12-s rest period followed by a 30-s period in which the movements were performed. This sequence was performed four times. At the command "rest," the subject lowered the arm over 1 s with gravitational assistance. Subjects were instructed via an audio system to "lift" when they were required to make the arm movement and to "rest" and return their arm to their side for the rest phase of the protocol (Fig. 3, upper panel). The data reported and analyzed for the voluntary movement are that of the last repetition so as to allow a fair comparison with the involuntary condition in which only one movement was recorded. The last repetition was chosen as this was the one nearest in time to the involuntary protocol. Following the voluntary protocol, there was a rest period of 60 s.

#### 4.2.2. *Involuntary arm movement condition*

The supine subject grasped a rubber ball which was secured by a strong cord to their waist. This provided a fixed resistance. The sequence for the involuntary condition began with a 30-s rest period then on hearing the command 'MVC' the subjects were instructed to make a maximal effort for 60 s to flex the

shoulder joint by exerting an upwards force on the ball with a straight arm. This force was produced by a maximal isometric contraction of the deltoid muscle of the shoulder. On the command "release," the subject released the fixed resistance and ceased the maximal effort. There was a delay of 1–2 s and the involuntary aftercontraction then developed for approximately 30 s during which an involuntary movement ensued. At the command 'rest,' the subject lowered the arm over 1 s with gravitational assistance. Muscle lengthening switches off the aftercontraction (Adamson and McDonagh, 2004). This was then followed by 29 s of complete rest (Fig. 3, lower panel).

### 4.3. *MRI parameters*

All structural and fMRI data were acquired on a 3-T Philips Achieva Scanner (Best, Netherlands) using an 8-channel SENSE head coil. T1 sagittal structurals were acquired with a resolution of 1 mm isotropic. T2-weighted BOLD images were acquired using the following parameters; FOV 240 mm, 34 slices, slice thickness of 3 mm and a matrix size of 96×96, a TE of 35 ms and a TR of 2 s. Slices were planned on the high-resolution sagittal structural so that the entire brain was covered, and tilted slightly so that much of the frontal sinus was avoided to prevent inhomogeneity distortion.

### 4.4. *fMRI data analysis*

Functional data were analyzed using a block design. The voluntary movement protocol was modelled (Fig. 3, upper panel) as one parameter. As mentioned above, the data reported and analyzed for the voluntary movement are that of the last repetition and its preceding rest period. Scan protocols were separated into two different runs, the first being the voluntary protocol which was then followed 60 s later by the involuntary protocol which began with a 30-s rest period (Fig. 3, lower panel). The involuntary movement protocol functional data were modelled such that the MVC for 30 s was modelled as one parameter, and the involuntary arm movement was modelled as a second, separate parameter. The gravity assisted lowering of the arm over 1 s was included in the subsequent rest period for both protocols. In the involuntary protocol following the MVC, there is a 1–2-s delay before the involuntary movement occurs. This was included in the involuntary movement period. In each session during which the voluntary and involuntary movements were both scanned the subject remained supine throughout.

fMRI data were analyzed using FSL software (http://www.fmri.ox.ac.uk/fsl/) Version 5.64. Prior to fMRI analysis, all images were viewed fully in FSLview to ensure there were no significant artifacts or areas of signal loss. The FEAT tool was used to analyze BOLD fMRI responses relating to the different tasks. All images were motion corrected using MCFLIRT, and motion correction reports were studied for any outstanding head movements. Images were smoothed using a FWHM 5 mm Gaussian kernel, and time series were analyzed using the FILM algorithm with local autocorrelation correction. Z statistic images were thresholded using clusters determined by $Z>3.5$ and a corrected cluster significance threshold of $P=0.05$.

The fMRI data was modelled in blocks. Experimental conditions were first compared with rest (Table 1); then in order to further elucidate the cortical areas implicated in the involuntary contractions, a higher-level analysis was carried out to mask out the cortical areas that were consistent in the voluntary contractions condition from the involuntary conditions. This was done using the higher-level analysis algorithm in the FEAT toolbox of FSL and by combining the responses for each subject with a fixed effects treatment of the variance (FMRIB's Local Analysis of Fixed effects) by forcing the random effects variance to zero in FLAME (FMRIB's Local Analysis of Mixed Effects) (Beckmann et al., 2003; Woolrich et al., 2004). In summary, specific contrast analyses were as follows: (i) involuntary condition (IC) only, (ii) voluntary condition (VC) only, (iii) MVC only, (iv) VC–IC, (v) IC–VC, (vi) IC–MVC, and (vii) MVC–IC.

### 4.5. *Motion capture*

A MRI-compatible motion tracking system became available for our use towards the end of our data collection, and we were able to obtain motion data for 5 of the 11 subjects during scanning. Motion data were recorded for both experimental conditions in the scanner. Infrared reflective markers (2 cm diameter) were attached to each wrist using double-sided tape. Actions were measured using a 4-camera Qualysis pro-reflex system (MCU240) in order to compare movement trajectories in the voluntary control and involuntary movement conditions on each subject. Offline analysis was performed using the Qualysis Track Manager software.

### 4.6. *EMG recording apparatus*

Electromyographic activity in the anterior deltoid muscle was measured in the laboratory practice sessions which employed an identical protocol to that used in the scanner. This was recorded by means of bipolar, silver, surface electrodes (10 mm diameter, 17 mm centre to centre) coated with a thin layer of conductive gel. The electrodes were part of a skin-mounted preamplifier (×1000) encapsulated in epoxy resin. They were developed from an original design by Johnson et al. (1977). These electrodes have a common mode rejection ratio at 50 Hz and 500 Hz of 100 dB and 80 dB, respectively, with a filter pass band from 10 Hz to 1000 Hz. The noise-to-signal ratio of the signals was <5%.The EMG signals were also passed via a RMS integrator with a time constant of 500 ms (NL7005; Digitimer Ltd, UK). The electrodes were placed over the midpoint of the anterior deltoid in a direction parallel to the orientation of the muscle fibres. Significance of EMG differences was computed using a double-tailed Student's *t* test for paired values.

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