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Abnormalities in the awareness and control of action

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Much of the functioning of the motor system occurs without awareness. Nevertheless, we are aware of some aspects of the current state of the system and we can prepare and make movements in the imagination. These mental representations of the actual and possible states of the system are based on two sources: sensory signals from skin and muscles, and the stream of motor commands that have been issued to the system. Damage to the neural substrates of the motor system can lead to abnormalities in the awareness of action as well as defects in the control of action. We provide a framework for understanding how these various abnormalities of awareness can arise. Patients with phantom limbs or with anosognosia experience the illusion that they can move their limbs. We suggest that these representations of movement are based on streams of motor commands rather than sensory signals. Patients with utilization behaviour or with delusions of control can no longer properly link their intentions to their actions. In these cases the impairment lies in the representation of intended movements. The location of the neural damage associated with these disorders suggests that representations of the current and predicted state of the motor system are in parietal cortex, while representations of intended actions are found in prefrontal and premotor cortex.

Keywords: motor control; awareness; prediction; abnormalities

1. INTRODUCTION

In this review we will present a framework designed to provide a coherent account of a number of disparate observations concerning abnormalities in the awareness and control of action. Our framework is based on established models of normal motor learning and control (for a review, see Wolpert 1997). However, we are particularly concerned to explain abnormal experiences of motor control such as phantom limbs and the passivity phenomena associated with schizophrenia. In § 2 we will summarize the components of our model of motor control and learning. In § 3 we will outline the application of this model to a number of specific signs and symptoms of motor disorders.

2. AN OUTLINE OF THE MOTOR CONTROL SYSTEM

A well-functioning motor system is an essential requirement if we are to move through our environment safely, reach and grasp objects and learn new skills. Making movements involves the production of an appropriate sequence of muscle contractions. At the same time sensory information is critical for deciding what movements to make and for observing the consequences of those movements. Motor control and motor learning can best be understood in terms of an engineering system (Craik 1948). In this system the motor commands emanate from controllers within the central nervous system (CNS). The brain also has access to the various kinds of sensory feedback that result from the movements generated by the motor commands. The basic task of the motor control system is to manage the relationships between motor commands and sensory feedback. This management is necessary for two reasons. First, it ensures that our movements achieve their goals. Second, it enables us to learn by experience to make more accurate and effective movements. Motor commands are transformed into sensory feedback every time our musculoskeletal system interacts with the environment, since every movement we make has immediate sensory consequences. Activity in the musculoskeletal system transforms efferent motor actions into reafferent sensory feedback. Once a sequence of motor commands has been issued it is possible to predict the subsequent behaviour of the motor system and the sensory consequences of that behaviour. However, these predictions cannot be made solely from knowledge of the sequence of motor commands. An additional set of variables, called state variables, also needs to be known. These are the configurations of parts of the body, such as joint angles and angular velocities and include the state of the system prior to the implementation of the motor commands. These state variables provide the basis for internal models of the motor system. On the basis of the motor commands and these state variables it is possible to determine the future behaviour of the system.

(a) *Internal models of the motor system*

There is evidence that the CNS contains transformations, or internal models, which mimic aspects of one's own body and the external world (Wolpert *et al.* 1995;

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Wolpert 1997). Here we shall be concerned with two varieties of internal model, predictors and controllers (also known as forward and inverse models, respectively). Whenever a movement is made, a motor command is generated by the CNS and a predictor estimates the sensory consequences of that motor command. A controller, on the other hand, captures the relationship between the desired state and the motor command required to achieve that state. An important issue to stress in our discussion of such representations is that they do not need to be detailed or accurate models of the external world. Often an internal model need only provide a rough approximation of some external transformation in order to play a useful role. The function of predictors and controllers requires that at least three states of the motor system are represented: the current state of the system, the desired state of the system and the predicted state of the system.

(i) *Predictors (forward models)*

Predictors model aspects of the external world and of the motor system in order to capture the forward or causal relationship between actions and their outcomes (Ito 1970; Jordan 1996; Wolpert *et al.* 1995). Every time a motor command is issued to make a movement, an efference copy of the motor command is produced in parallel. Based on the efference copy, the predictor estimates the sensory consequences of the ensuing movement. This prediction can be used in several ways (Miall & Wolpert 1996; Wolpert 1997) and there is a substantial body of evidence that the CNS makes use of such prediction.

(i) Prediction is needed to anticipate and compensate for the sensory effects of movement. For example, during eye movements an efference copy of the motor command is used to predict the effects of the movement (Von Helmholtz 1886; Sperry 1950; Von Holst & Mittelstaedt 1950). In order to determine the location of an object relative to the head, its retinal location and the gaze direction must be known. As the eye muscles are thought to contain no sensory receptors used to determine the gaze direction, Von Helmholtz (1886) proposed that the gaze direction is determined by predicting the eye location based on the efference copy of the motor command going to the eye muscles. Using this estimate of eye position together with the object's retinal location, its true position in space can be determined. When the eye is moved without using the eye muscles (for example, by gently pressing on the eyelid with the finger), the retinal location of objects changes, but the predicted eye position is not updated, leading to the perception that the world is moving.

(ii) Prediction can also be used to filter sensory information, attenuating the component that is due to self-movement (reafference) from that due to changes in the outside world. The sensory consequences of self-generated movements are predicted from the efference copy produced in parallel with the motor command. Self-produced sensations can be correctly predicted from motor commands. As a result there will be little or no sensory discrepancy resulting from the comparison between the predicted and actual sensory feedback. In contrast, externally generated sensations are not associated with any efference copy and therefore cannot be predicted and will produce a higher level of sensory discrepancy. As the discrepancy between predicted and actual sensation increases, so does the likelihood that the sensation is externally produced. By using such a system it is possible to cancel out or attenuate sensations induced by self-generated movement and thereby distinguish sensory events due to self-produced motion from sensory feedback caused by the environment, such as contact with objects. Such a mechanism underlies the finding that the same tactile stimulus is perceived as much less intense when it is self-applied in comparison with when it is applied by another person (Weiskrantz *et al.* 1971). The perceived intensity of a self-applied tactile stimulus increases with the degree of discrepancy introduced between the predicted and actual sensory feedback (Blakemore *et al.* 1999).

(iii) Prediction can also be used to maintain accurate performance in the presence of feedback delays. In most sensorimotor loops the feedback delays between the issuing of a motor command and the perception of its sensory consequences are large. This is due to both neural transduction and processing delays, which can be as long as 250 ms. These delays can result in inaccuracy if the motor system compares the desired outcome with the perceived outcome to determine the performance error. As the perceived outcome is delayed relative to the actual outcome the motor system will respond to a perceived error which may no longer exist, thereby generating a potentially inappropriate response. A predictor can be used to estimate the actual outcome of the motor command without delay and compare this with the desired outcome. Such internal feedback of the estimated outcome of an action is available before the true sensory feedback (Miall *et al.* 1993).

(iv) Prediction also plays a critical role in a process that integrates sensory and motor information in order to estimate the current state of the system. The state of the motor system is not directly observable by the CNS, which has access only to the outgoing motor commands and the subsequent sensory feedback. Instead, the state has to be estimated by observing these signals. To produce optimal estimates, two processes can be used. The first uses a predictor to estimate the next state of the system. The second process uses sensory feedback to modify this estimate (Wolpert *et al.* 1995; Wolpert 1997). By using both sources of information the uncertainty of the state estimate can be reduced. The recognition that the representation of a limb position depends not only on current sensation but also on predictions based on motor commands can explain a number of the bizarre experiences associated with abnormalities of the motor system (see § 3(b)).

(ii) *Controllers (inverse models)*

Controllers provide the motor commands necessary to achieve some desired outcome. For a simple reaching and grasping movement, the first step would be to plan the

trajectory to be followed by the hand in order to reach the desired final position. The trajectory represents the desired configuration of the body at each point in time. The muscle activations necessary to achieve this trajectory depend on the dynamic parameters of the body such as the inertia and link lengths of the body segments. The controllers must learn to generate the appropriate motor commands such that the muscle activations achieve the desired trajectory. The controllers, therefore, receive a desired configuration of the body and produce motor commands which should achieve this configuration

Recently it has been proposed that our ability to interact with many different objects in a variety of different environments relies on a 'divide-and-conquer' strategy. Complex tasks are decomposed into simpler subtasks, each learned by a separate controller (Ghahramani & Wolpert 1997; Wolpert & Kawato 1998; Blakemore *et al.* 1998*a*). Therefore, rather than having a single controller, multiple controllers develop, each tuned to a particular sensorimotor context. At any given time, one or a subset of these controllers contributes to the final motor command. The contribution each controller makes to the final motor command is determined by two distinct processes. The first uses sensory contextual information (affordances), such as the visual appearance of an object, to select controllers prior to movement initiation. For example, the apparent size and weight of an object would determine whether we try to pick it up with a precision grip or a power grip. The second process uses the errors in the predictions made by a set of predictors each tuned to a different context. As these predictors capture distinct dynamic behaviours of the motor system, their prediction errors can be used during movement to determine in which context the motor system is acting and thereby switch between controllers during a movement. For example, when we pick up a milk bottle which appears full, but is in fact empty, we select the inappropriate controller based on the visual information, but are able to switch controllers when the predicted outcome of our action does not match the actual outcome. This modular learning system, known as the multiple paired predictor-controller model (Wolpert & Kawato 1998), is capable of learning to produce appropriate motor commands under a variety of contexts and can switch rapidly between controllers as the context changes. These features are important for a full model of motor control and motor learning, as the human motor system is capable of very flexible, modular adaptation.

(b) *Motor representations*

Our outline of the motor control system postulates several kinds of motor representation. These are listed below and shown graphically in figure 1.

(i) Actual state of the system. The actual state of the system is not directly available to the CNS. Instead an estimated actual state of the system is inferred on the basis of the stream of motor commands and sensory feedback. For simplicity we will refer to the estimated state as the actual state as it represents the best estimate of the actual state available to the CNS.
(ii) Desired state of the system. This representation holds the instantaneous goal of the system.

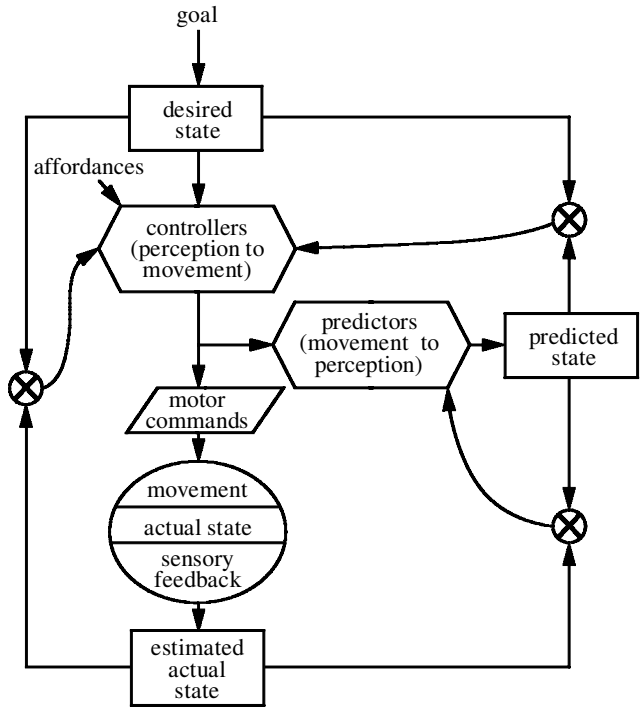

Figure 1. The basic components of a motor control system based upon engineering principles.

(iii) Predicted next state of the system. This representation provides an estimate of the future state of the system derived from the predictors.
(iv) Motor commands. These are derived from the controllers and are fine-tuned by sensory information (affordances) about the current state of the world (e.g. visual information about the position and shape of the object that is to be grasped).
(v) Sensory feedback. This is the consequence of the action performed, plus any environmental events.

Comparisons of these representations provides error signals that can be used to improve the functioning of the predictors and the controllers.

(i) Errors derived from differences between the desired and the actual state of the system can be used to improve the functioning of the controllers.
(ii) Errors derived from differences between the predicted and the actual state of the system can be used to improve the functioning of the predictors.
(iii) Errors derived from differences between the desired and the predicted state of the system can be used to improve the functioning of the controllers during mental practice.

In terms of this model the performance of a simple action involves the following stages. Current wishes and plans are used to formulate the desired state (instantaneous goal) of the system. The controllers generate appropriate motor commands on the basis of the difference between the actual state and the desired state. Computation by the controllers is 'fine-tuned' by the context in which the action is occurring. For example, if the action requires the grasping of an object, knowledge of the shape and position of the object provides 'affordances' which allow a more accurate computation of the appropriate

motor commands (Greeno 1994). Once the motor commands have been computed the predictors calculate the expected state of the system. Subsequently, or in parallel with this process the action is performed. Once the movement has been made the new state of the system can be estimated on the basis of sensory feedback and knowledge of the motor commands that have been executed. If there are discrepancies between the new state and the desired and predicted states then modifications can be made to the predictors and controllers and further actions can be performed to correct the situation.

(c) *Awareness of motor representations*

One major concern in this paper is to consider the extent to which we are aware of the functioning of some aspects of our motor control system (see also Jeannerod 1994). Here we shall review evidence indicating which components of the motor control system outlined in § 2(b) are available to consciousness and which are not.

(i) *Motor imagery and motor preparation*

The awareness of selecting and controlling our actions is a major component of consciousness. We can also readily imagine making movements in the absence of any overt behaviour. Furthermore this mental activity can have detectable consequences. First, mental practice of various tasks can lead to a significant improvement in subsequent performance (for a review, see Feltz & Landers 1983). Mental training affects several outcomes of motor performance such as muscular strength (Yue & Cole 1992), movement speed (Pascual-Leone *et al.* 1995) and temporal consistency (Vogt 1995). Second, prolonged performance of tasks in the imagination can lead to marked physiological changes. Subjects who performed or mentally simulated leg exercise increased heart rate and respiration rate in both conditions (Decety *et al.* 1991). Third, changes in brain activity associated with movements made in the imagination can readily be detected using brain imaging techniques such as positron emission tomography. Decety *et al.* (1994) asked subjects to imagine grasping three-dimensional objects presented to them. Stephan *et al.* (1995) compared execution of a sequence of joystick movements with imagining making such a sequence. These studies showed that the brain regions activated during motor imagery are a subset of those activated during motor execution. Jeannerod (1994) argued that motor imagery is closely related to motor preparation. Preparing a movement in advance and holding it in readiness while waiting for a signal to release the movement engages the same processes as those involved in imagining making that movement. Brain imaging studies of motor preparation and motor imagery highlight activity in the anterior cingulate cortex (ACC), the anterior supplementary motor cortex (SMA), inferior lateral premotor cortex and inferior parietal lobe (Decety *et al.* 1994; Stephan *et al.* 1995; Krams *et al.* 1998). Since these areas are engaged by motor preparation and motor imagery they are presumably involved with representations of intended and predicted movements. It has been argued that covert attention, that is attending to a particular object without actually moving the eyes or the hand towards it, is equivalent to mentally reaching for that object with the eyes (foveation) or the hand (e.g. Rizzolatti *et al.* 1987; Corbetta 1998). During the performance of covert attention tasks activity is observed in areas which overlap with those seen during motor imagery tasks: ACC, SMA, lateral premotor cortex (frontal eye fields) and intraparietal sulcus (IPS) (Corbetta *et al.* 1993; Nobre *et al.* 1997).

(ii) *Limited awareness of affordances and motor commands*

These observations confirm that we can be aware of intended movements and can perform movement sequences in imagination. Furthermore, this motor imagery has specific neural correlates. There are a number of other observations, however, which demonstrate that the motor control system can also function in the absence of awareness. Goodale *et al.* (1986) (see also Bridgeman *et al.* 1981) report a pointing experiment in which the target occasionally jumped several degrees, unnoticed by the subjects. Nevertheless the subjects were able to adjust the trajectory of their moving hand to the target position. In this case the subjects were aware neither of the sensory information that elicited the movement correction nor of the change in the motor programme that was elicited. In another experiment involving reaching and grasping, Castiello *et al.* (1991) found that awareness of an unexpected target jump occurred more that 200 ms after the motor system had initiated an appropriate movement correction. Furthermore, appropriate grasping movements can be made even when conscious perception of the object to be grasped is incorrect. In the Ebbinghaus (Tichener) Circles Illusion two identical circles appear to be of different sizes because of the context in which they occur. The strength of this illusion can be measured by asking subjects to adjust the size of the circles until they appear to be identical. However, the size of this illusion is greatly reduced if it is measured in terms of the distance between the finger and thumb when grasping the central circles (Aglioti *et al.* 1995). The result from studies of this illusion and others (e.g. Gentilucci *et al.* 1996) suggests that there can be a dissociation between our perception of objects and the information which the sight of objects (their affordances) provides to fine-tune our reaching and grasping movements. An extreme example of this lack of awareness is provided by the case of D.F. described by Milner & Goodale (1995). D.F. was unaware of the shapes of objects and was unable to describe them or to discriminate between them, but she could nevertheless produce appropriate grasping actions based on the shapes of which she was unaware. A similar pattern of behaviour has been observed in another patient by Perenin & Rossetti (1996).

In terms of the model for motor control presented in § 2(b) these results suggest that we are not aware of the precise details of the motor commands that generate our actions, nor of the way in which immediate sensory information (affordances) is used to fine-tune these commands. Thus, it would appear that our awareness of our actions and of the sensory information on which these actions are based is derived from other sources. There are likely to be good reasons for this separation. For example, we have suggested (Frith 1995) that representations used for reaching an object need to be coded in egocentric coordinates, while representations for reporting the

position of an object need to be in coordinates that are independent of a personal view. To reach for an object it is necessary to know where that object is in relation to our hand, not in relation to other objects in the environment. There are many different frames of reference that could be used for representing the position of an object (Andersen 1995). Some possibilities include the position of the object on the retina (retinotopic coordinates), the position of the object relative to the head (head-centred coordinates; Vetter *et al.* 1999), and the position of the object relative to the shoulder (shoulder-centred coordinates; Flanders *et al.* 1992). Animal studies suggest that cells exist which code in terms of each of these different coordinate systems. Cells of this type tend to be found in parietal cortex (Colby *et al.* 1995; Andersen *et al.* 1997). This brain region has a major role in the control of movements, including reaching and grasping with limbs and eyes (Rizzolatti *et al.* 1997). Evidence from the behaviour of cells in this region suggests that its role in motor control derives in part from an ability to translate from one coordinate system to another. For example, to use visual cues to make a limb movement necessitates a translation from retinotopic to body-centred coordinates (Jeannerod *et al.* 1995). The appropriate reach depends on where our arm is in relation to the target, and is independent of where we happen to be looking. Thus, in the region of parietal lobe concerned with reaching, objects are represented, not in terms of what they are, but in terms of how they may be reached (equivalent to the dorsal 'how' pathway of Milner & Goodale (1995)).

For such representations to be maintained the coordinates associated with each object must be altered, not only when the objects move, but also every time we move our eyes, limbs or body (Kalaska & Crammond 1992; Galletti *et al.* 1993). Consistent with this is the evidence that the receptive fields of cells in some regions of parietal cortex are 'remapped' prior to eye or limb movements (e.g. Duhamel *et al.* 1992). Awareness of these constant remappings would be confusing. In addition awareness of the remapping is unnecessary. The changes in representation that result from our own movements are entirely predictable on the basis of those movements and therefore do not require our attention. It seems plausible that to be aware of representations which changed every time we moved our bodies, or even our eyes, would be a positive disadvantage. Indeed, the mechanisms that underlie our conscious perception seem designed to maintain stability and to emphasize the unexpected.

(iii) *Limited awareness of the actual state of the motor system*

In the outline of the motor control system presented in § 2(b) a major role is played by representations of the predicted state of the system that will result from intended acts. In most situations, especially those that are routine, the actual state of the motor system will correspond closely to the state predicted before the action was performed. If awareness puts an emphasis on the unexpected, then we would predict that there would be only limited awareness of the actual state of the motor system whenever this has been successfully predicted in advance. We may only be aware of the actual sensory consequences of our movements when they deviate from what we expect.

An extreme example of a lack of awareness of action resulting from predictability comes from overlearned tasks. With sufficient practice many tasks can become 'automatic' and can performed without any need to think about the actions required to perform the task. This automaticity can be proved by showing that a second, attention-demanding task can be carried out at the same time as the overlearned task without impairing performance (e.g. Passingham 1996). While performing such tasks we are not aware of the actual state of our motor system, nor are we aware of our intended actions or their predicted consequences.

A more specific example of a reduced awareness of the actual state of the system, or at least of the sensory feedback that indicates the actual state of the system comes from studies of tickling. It is well known that the intensity of the tactile experience when we tickle ourselves is greatly reduced in comparison with the sensation when someone else tickles us (Weiskrantz *et al.* 1971). Corresponding to this reduction in tactile sensation is a reduction of activity in somatosensory cortex (Blakemore *et al.* 1998*b*). This phenomenon occurs because self-generated tactile sensation can be predicted from the motor commands that generated the movements that created the sensations. This prediction is based on a rather precise specification. Thus, the perceived intensity of a self-generated tactile sensation is markedly affected by small deviations in the timing or trajectory of the tactile stimulus from the movement that generated it (Blakemore *et al.* 1999). For example, if there is a delay of 100 ms between the movement and the tactile stimulation, then the perceived intensity of the tactile stimulation increases even though the subject is unaware of the delay.

In some circumstances we are unaware of even quite large deviations of actual movements from those expected. This seems to happen as long as the desired state is successfully achieved. For example, Fourneret & Jeannerod (1998) gave false feedback about the trajectory of an arm movement so that subjects, who could not see their arm or hand, had to make considerable deviations from a straight movement in order to generate a straight line on a computer screen. The subjects could achieve the desired result of drawing a straight line by making deviant movements. However, verbal reports indicated that they were unaware that they were making deviant movements. It seems then that we are largely unaware of sensory feedback about the actual state of our motor system as long as our intentions have been achieved. In most cases successful achievement implies that sensory feedback has been correctly predicted, but in some circumstances we remain unaware even of unexpected sensory feedback. When we come to consider abnormalities in the control of action (§ 3(a,b)) we shall see that a major insight derived from the engineering model is that estimates of the current state of the system are not only derived from sensory inputs, but also from the preceding stream of motor commands. In many situations information from this latter stream seems to be more important in determining the experience of the patient.

(iv) *The timing of awareness*

In addition to examining which aspects of the motor control system are accessible to awareness attempts have

also been made to investigate the time at which awareness emerges during the generation of an action. Libet *et al.* (1983) and McCloskey *et al.* (1983) asked normal volunteers to estimate the time at which they initiated a finger movement (i.e. the time at which the finger started to move). This reported time of awareness consistently anticipated the actual starting time of the movement by 50–80 ms. If transcranial magnetic stimulation is applied to the motor cortex then there is a substantial delay in the initiation of a movement, but there is a far smaller delay in the perceived time of initiating the movement (Haggard & Magno 1999). These observations imply that our awareness of initiating a movement is not derived from sensory signals arising in the moving limb. This information will not be available until after the limb has started moving. In terms of the model of motor control we are formulating here, the most likely representation relating to awareness of movement initiation is the predicted state of the system (e.g. the predicted position of the limb and associated sensations; see also Haggard *et al.* 1999). This can be formed as soon as the predictors have calculated the expected sensory consequences of making the intended movement.

More controversial are studies in which volunteers try to indicate the time at which they are aware of having the 'urge' to make a movement (Libet *et al.* 1983). This can precede the production of the movement by *ca.* 300 ms and might correspond to the formation of the representation of the intended position of the limb that precedes motor preparation. Haggard & Eimer (1999) asked subjects to indicate the time at which 'they first began to prepare the movement' and related this to various components of the motor readiness potential. In this study subjects moved either their left or their right index finger. Haggard & Eimer (1999) found that the onset of the lateralized readiness potential, rather than earlier components of the readiness potential, covaried with the perceived time at which preparation of the movement began. This observation suggests that the awareness of preparing to move is associated with the exact specification of the movement (i.e. which finger will be moved) rather than some more abstract representation of action. In terms of our framework of the motor system, specification of the goal of the movement seems not to be sufficient for awareness of preparing to move. Awareness of preparing to move requires that the controllers have completed the specification of the sequence of motor commands needed to make the movement. Awareness of initiation of the movement, on the other hand, has to wait further until the predictors have specified the sensory consequences of the movement. It is these predicted consequences that form our awareness of initiating the movement.

In this brief review we have presented evidence that some, but not all aspects of the motor control system are accessible to awareness. In the remainder of this paper we will discuss a variety of human movement abnormalities and attempt to convince the reader that the model of the motor system illustrated in figure 1 provides a useful and unifying framework for understanding these various disorders. We shall also suggest that to understand these disorders it is important to consider the patient's awareness of different aspects of the motor control system. In some cases the problem resides principally in an abnormality of awareness rather than an abnormality of control.

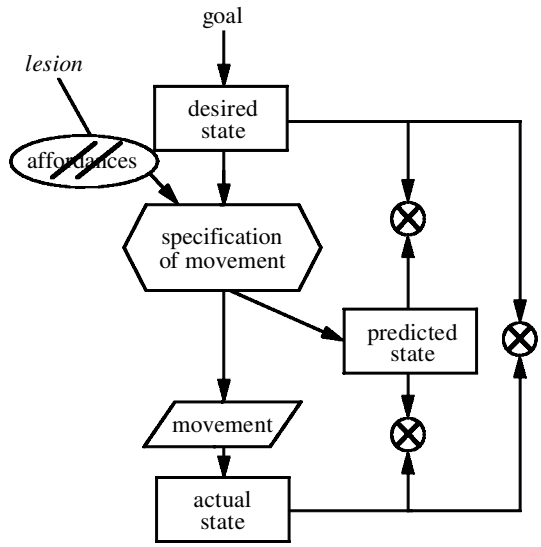

Figure 2. The underlying disorder leading to optic ataxia. The fine tuning of grasping actions afforded by the precise shape and position of objects is no longer available to the patient. The patient is aware that actions are clumsy.

3. ABNORMALITIES OF THE PERCEPTION AND CONTROL OF ACTION

(a) *Abnormalities in the control of action while awareness remains unimpaired*

(i) *Optic ataxia and other forms of apraxia*

Patients with optic ataxia (Bálint's syndrome) (Bálint 1909, translated by Harvey 1995; Perenin & Vighetto 1988) have difficulty grasping objects which they can see quite clearly. Their difficulty has at least three components: the arm fails to extend correctly in space, the wrist fails to rotate to match the orientation of the object to be grasped, and the hand fails to open in anticipation of gripping the object (Jeannerod *et al.* 1994). However, although clumsy, the attempted movement matches the patient's intentions and the patient is aware of having a problem with reaching, although this is often attributed to a problem with vision rather than a problem with movement. In terms of our characterization of the motor system the problem in optic ataxia occurs because the controllers are not properly finely 'tuned' by the immediate context (i.e. the affordances offered by the shape of the object to be grasped). All other aspects of the control of movement and the awareness of that control remain intact (figure 2).

However, the controllers do not rely solely on the immediate affordances provided by the sight of the object that is to be grasped in order to derive an appropriate sequence of motor commands. Relevant information is also available from memory and can be used in the absence of affordances. As a result some patients can grasp a well-known object such as a lipstick more accurately than an unknown object of exactly the same shape (Jeannerod *et al.* 1994). In this example the information used by the controllers comes from long-term knowledge about objects. Relevant information is also available from

short-term memory, although this is not as useful as actual sight of the object. If vision of the object to be grasped is removed for only a few seconds then the reaching and grasping of normal subjects is impaired (Goodale *et al.* 1994; Rossetti 1998). The information available in short-term memory in this situation may be derived from representations about the position and nature of the object rather than representations specifically tailored for grasping the object. Patient D.F., who could grasp objects without being able to recognize them, completely lost her ability to grasp objects after a short delay during which the object was not visible. In contrast, the reaching behaviour of a patient with optic ataxia can improve after a short delay in the dark (Milner *et al.* 1999). Presumably this is because, for this patient, information about the object in short-term memory, although not ideal for grasping, is better than the faulty information provided by the sight of the object.

Optic ataxia is one of many forms of apraxia: difficulties in making voluntary movements in the absence of a primary motor defect. In terms of our model, apraxia occurs when there is insufficient information for the controllers to construct an appropriate sequence of motor commands. This suggestion relates closely to the suggestion of Pause *et al.* (1989, p. 1599) that 'the motor disability ... does not lie in the loss of kinetic memory to perform movements, but in the loss of their evocation by appropriate sensory stimuli'. Because relevant information comes from many different sources there can be many different forms of apraxia. We have already mentioned patients who can grasp a lipstick (information derived from long-term knowledge), but not a neutral cylinder of the same shape (information derived from immediate sight of the object). Other patients are unable to produce an action to command, e.g. they cannot obey the command 'to blow', but, when presented with a lit candle will blow it out. In these cases information can be used from the sight of the object, but not from verbal commands. De Renzi *et al.* (1982) have formally demonstrated other such dissociations, finding patients who can mime the use of an object to verbal instruction, but cannot imitate the same gesture when performed by someone else, and also finding patients with the opposite pattern of disorder.

In § 2(c)(ii) we discussed the need to translate between different coordinate frames in order to use visual information to generate movements (Andersen 1995). Patients with apraxia seem to have lost the ability to translate certain kinds of information into coordinates appropriate for constructing actions. We also mentioned the evidence from animal studies that the parietal cortex may have a major role in translating from one coordinate frame to another (Colby & Duhamel 1996). Apraxia can occur after damage to many brain regions, but is particularly associated with damage to the parietal lobe (De Renzi & Lucchelli 1988). With regard to optic ataxia, lesions in the superior parietal cortex (or more precisely in the IPS between BA7 and 39) impair the ability to make accurate reaching and grasping movements in both man (Perenin & Vighetto 1988) and monkey (Gallese *et al.* 1997; Rushworth *et al.* 1997) (for a discussion of the precise location of the critical area in parietal cortex see Passingham (1998)). Imaging studies of grasping in healthy volunteers also implicated the IPS in the control of visually guided reaching (Clower *et al.* 1996). There is as yet little evidence that other forms of apraxia can be related to specific lesions, largely because there is so little agreement as to how to classify the different forms of apraxia.

(ii) *The 'anarchic hand' sign*

Patients showing the anarchic hand sign (sometimes known as the alien hand sign, see Marchetti & Della Salla (1998)) have a hand that moves 'of its own accord' without the will of the patient. In one case it was noted that the patient had picked up a pencil and 'had been scribbling with the [affected] right hand ... She then indicated that she had not herself initiated the original action of the right arm ... She experienced a feeling of dissociation from the actions of the right arm, stating ... that "it will not do what I want it to do"' (Goldberg *et al.* 1981, p. 685). In another case the patient's 'left hand would tenaciously grope for and grasp any nearby object, pick and pull at her clothes, and even grasp her throat during sleep .... She slept with the arm tied to prevent nocturnal misbehaviour. She never denied that her left arm and hand belonged to her, although she did refer to her limb as though it were an autonomous entity' (Banks *et al.* 1989, p. 456). Typically the anarchic hand grasps objects in its vicinity in an inappropriate manner; it will grasp doorknobs or pick up a pencil and scribble with it. Patients clearly recognize that there is a discrepancy between what the hand is doing and their desired actions. The patients are upset by the actions of the hand and will often try to prevent it from moving by grasping it firmly with the other hand.

In many ways the patient with an anarchic hand shows the converse problem to the patient with optic ataxia. We have just reviewed (§ 3(a)(i)) the evidence that the parietal cortex contains representations of the various objects in our immediate environment in terms of the appropriate movements needed to reach and grasp them. The patient with optic ataxia fails to form these representations and therefore has difficulties with reaching and grasping. In the patient with an anarchic hand these representations are activated inappropriately. The sight of an object is sufficient to elicit the movement even though this does not fit with the patient's current goals. In terms of our characterization of the motor system, the movements of the anarchic hand occur because the effects of the affordances supplied by the immediate visual environment are no longer inhibited by the currently intended action (figure 3). However, the rest of the system is intact. Representations of the intended and actual positions of the hand are available, so that patients know that the behaviour of the hand does not conform with their intentions.

What is the brain mechanism which prevents us from responding to every graspable object in our environment? The anarchic hand sign is often associated with unilateral damage to the SMA contralateral to the anarchic hand (Goldberg *et al.* 1981). The anterior part of the SMA is considered to one of a number of 'higher-order' motor areas in contrast to areas, such as the primary motor cortex, with are directly concerned with execution (Pickard & Strick 1996). In contrast to executive motor regions, the anterior SMA does not show increasing activation with increasing force (Dettmers *et al.* 1995). On

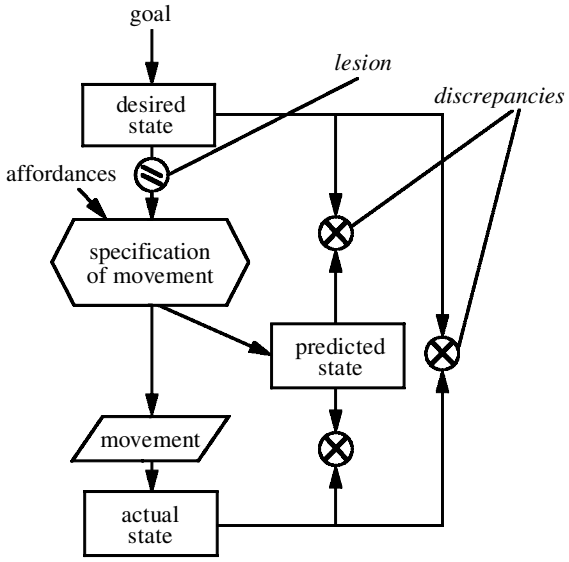

Figure 3. The underlying disorder leading to an anarchic hand. The actions of the hand are no longer controlled by the intentions of the patient. Instead the hand makes stereotyped responses to objects in the environment. The patient is aware of the discrepancies between intentions and the actions of the hand.

the other hand, unlike executive motor regions the anterior SMA is activated specifically in tasks requiring selection between different movements (Deiber *et al.* 1991), especially when these movements have to be made only in the imagination and not actually executed (Stephan *et al.* 1995). When the precise timing of events is investigated there is evidence that some neurons in the anterior SMA are active during movement preparation, but not during movement execution (Rizzolatti *et al.* 1990). Ball *et al.* (1999) using combined electroencephalography and functional magnetic resonance imaging observed a sharp drop in activity in an area referred to as intermediate SMA that coincided with a sharp increase in activity in primary motor cortex just before execution of a movement. They suggest that the function of this region of the SMA may be essentially inhibitory, so that a movement can only be initiated by primary motor cortex when activity in the anterior SMA drops. This would account for the preferential activation of the anterior SMA when movements are imagined because in such cases execution must be inhibited. Such a role for the anterior SMA could explain why an 'anarchic hand' should be released when this region is damaged.

The major projections to motor cortex (area 4) come from lateral and medial premotor areas (area 6, including the SMA) and from parietal cortex (areas 5 and 7b in the monkey, probably equivalent to areas 5 and 40 in man; see Passingham 1993). This pattern of projections is consistent with the idea that signals arising in parietal cortex enable motor cortex to generate appropriate reaching and grasping movements to any object in the immediate environment, while signals arising in the SMA permit selection of the one movement appropriate to current intentions. Unilateral damage to what is probably a rather circumscribed region of the SMA releases inappropriate reaching and grasping in the contralateral hand, creating an anarchic hand.

(There is some evidence that the anarchic hand is often associated with damage to the anterior corpus callosum as well as the SMA (Parkin 1996). In these cases the unwanted actions of the anarchic hand often consist of interference with the actions of the other hand, rather than unintended grasping behaviour. For example, the anarchic left hand might undo buttons that the right hand had just done up. This behaviour would also be explained in terms of a failure of inhibition. However, in these cases the inhibition arises from signals concerning the behaviour of the hand selected for performing the action. These inhibitory signals fail to be transmitted through the corpus callosum.)

We have argued that patients with optic ataxia and the anarchic hand have disorders of motor control, but no associated disorder in their awareness of the motor system. This is because the impairment concerns the mechanisms by which the controller constructs and selects the precise movements required for an action. These processes are not available to consciousness. In § 3(b) we shall consider syndromes in which motor impairments are associated with abnormalities of awareness.

(b) *Abnormalities of motor control and awareness*

(i) *Phantom limbs*

After amputation of all or part of a limb many patients report that they experience a phantom limb. Although they know that there is no limb they still feel the presence of it (Ramachandran & Hirstein 1998). Some patients report being able to move their phantoms voluntarily, while others experience their phantom as paralysed and cannot move it even with intense effort. If the limb was paralysed before amputation the phantom normally remains paralysed. If not, then typically immediately after amputation patients can generate movement in the phantom. However, with time they often lose this ability (Ramachandran 1993). Some finger amputees experience their phantom fingers only when they flex the fingers in the intact hand as when making a fist or grabbing a cup. There is frequently a latency of 2–3 s before the phantom emerges and when the normal fingers are extended again the phantom takes 2–3 s to disappear (Ramachandran 1993). In these cases the position of the phantom is determined by the actions of the contralateral limb and there is a marked delay in the formation of the phantom.

The existence of phantom limbs has long seemed deeply mysterious. How is it possible to experience a limb in a particular position in space when there is no limb and, as a result, the brain is no longer receiving any relevant somatosensory or proprioceptive information? There is now substantial evidence for neural plasticity in the mature human brain. After amputation of a limb there is reorganization of the deafferented region of cortex. As a result stimulation of the skin of distant areas such as the face or the chest can elicit sensation in a phantom arm (Ramachandran *et al.* 1992; Aglioti *et al.* 1994; Kew *et al.* 1997). Thus the experience of the presence of a phantom limb can be supported by somatosensory signals coming from other parts of the body. The presence of proprioceptive signals from other limbs can also explain how a patient can experience a phantom in the positions occupied by the intact contralateral limb. However, these mechanisms cannot explain cases in

which the position of the phantom is not determined by the positions of other limbs or cases in which the patient can voluntarily move the phantom. Our explanation of these phenomena is that the estimated position of a limb is not solely based on sensory information, but also on the stream of motor commands issued to the limb muscles. On the basis of these commands the predictor can estimate the new position of the limb before any sensory feedback has been received. Indeed, as we have already argued, the normal experience of the limb is often based on this predicted state, rather than the actual state. Even in the absence of a limb, streams of motor commands can still be issued. If these commands lead to the prediction of movement then the phantom will be experienced as moving. However, the motor control system is designed to adapt to changing circumstances. Since the limb does not actually move there is a discrepancy between the predicted and the actual consequences of the motor commands. With time the predictors will be modified to reduce these discrepancies. At this point the issuing of a stream of motor commands will not lead to the prediction of a change in limb position. Such adaptation in the predictors could explain why patients eventually lose the ability to move their phantoms.

Such adaptation of the predictors would also explain how Ramachandran & Rogers Ramachandran (1996) were able to reinstate voluntary movement of the phantom by providing false visual feedback of a moving limb corresponding to the phantom. This was achieved by placing a mirror in the midsaggital plain. With the head in the appropriate position it was possible for the patient to see the intact limb at the same time as the mirror reflection of this limb. This reflection so closely resembles the missing limb that the patient has the strong illusion of seeing the missing limb. If the intact limb is moved then the patient receives from the mirror visual feedback of movement in the missing limb. For most patients moving their hand in this mirror box rapidly leads to the perception that they are now able to move the phantom limb again. In some cases this perception continues even when the mirror box is no longer being used.

In a reformulation of the proposals of Ramachandran & Rogers Ramachandran (1996), we suggest that the false visual feedback supplied by the mirror box allowed the predictors to be updated. In consequence the efference copies produced in parallel with the motor commands now generated changes in the predicted position of the missing limb corresponding to what the patient had seen in the mirror.

Ramachandran & Hirstein (1998) proposed that dynamic images of the body are formed in the parietal lobes and provide the basis for the experience of phantom limbs. This formulation resembles our suggestion that parietal cortex is involved in the representation of predicted limb positions. However, as we have seen, the parietal lobe contains representations of limb positions in terms of many different coordinate systems. Which of these particular coordinate systems relates to the experience of phantoms and the precise locations for such representations remains to be determined. Evidence that phantom limbs are represented in the parietal cortex comes from the observation that a phantom limb patient lost his phantom after a right parietal stroke (Sunderland 1978). Unfortunately the precise location of the lesion in this case was not specified.

(ii) *Missing limbs*

After peripheral deafferentation of a limb the patient will often develop a phantom even though the deafferented limb is still present. This phantom may be contained within the real limb, but, in some circumstances, may become separated from the limb and become supernumerary (e.g. Kew *et al.* 1997, subject 2). However, in other cases patients do not develop phantoms, but rather are unaware of the existing limb unless it can be seen. We are not aware of any systematic comparison of deafferented patients who develop phantoms with those in whom the limb fades. However, a study of cases reported in the literature suggests that the critical difference lies in whether or not the deafferented limb is also paralysed. The cases described by Kew *et al.* (1997) who developed phantoms had limbs that were deafferented and paralysed. In contrast the patient described by Cole (1991) was completely deafferented for touch, but was not paralysed and achieved a remarkable degree of motor control which was largely based on visual feedback. This patient never developed a phantom, but for him and his body it was literally 'out of sight, out of mind' (Cole 1991).

Most deafferented patients in whom the motor output system remains intact are unwilling to attempt movements because they are so inaccurate. Rothwell *et al.* (1982) demonstrated that a patient with peripheral deafferentation was unable to make automatic reflex corrections to movements and was unable to sustain constant levels of muscular contraction or maintain long action sequences in the absence of visual feedback. The lack of a sensation of the current position of the limb is not only a problem for checking the success of movement through feedback. It also creates a problem because the computation by the controllers of the appropriate movement requires that the starting position of the limb must be known.

Similar problems can occur after brain damage in somatosensory areas as a result of which the patient can no longer experience the limb contralateral to the lesion. Jeannerod *et al.* (1986) described a patient with hemianaesthesia after damage involving the right inferior parietal lobe. The patient could initiate simple single-component movements, but could not make complex multicomponent movements with his left hand in the absence of visual feedback. Wolpert *et al.* (1998) describe an interesting variant of this phenomenon. Patient P.J. had a large cyst in the left parietal lobe and reported the experience of the position and presence of her right limbs fading away over seconds if she could not see them. Her experience of a constant tactile stimulus or a weight also faded away, but changes in such sensations could be detected. Slow reaching movements to peripheral targets with the right hand were inaccurate, but reaching movements made at a normal pace were unimpaired. In this case there seemed to be a circumscribed problem with the representation of the current limb position in that it could not be maintained in the absence of changing stimulation.

In all these cases of deafferentation without paralysis, visual signals provide the only sensory information for

making accurate movements. They provide information about the position of a limb prior to movement and provide feedback about the accuracy of the movement. As a result the motor control system will learn to ignore somatosensory and proprioceptive signals when predicting the outcome of movements or estimating the current state of the system. It will learn to base such estimates solely on the stream of motor commands and upon visual information. In the absence of visual signals the estimates cannot be made and the experience of the limb fades away.

In patients with deafferentation and paralysis no movements can be made and so the system has no chance to learn to attend to one modality of sensation rather than another. The experience of a phantom can therefore be driven by sensations from other limbs that have been remapped into the deafferented cortical region.

(iii) *Supernumerary limbs*

Patients sometimes report experiencing one or more supernumerary limbs in addition to their real ones (Vuilleumier *et al.* 1997). Of particular interest is the patient described by Hari *et al.* (1998) who reported experiencing an additional left arm. The extra arm occupied the position vacated by the real left arm a minute or so previously. The felt position of the phantom extra arm mirrored the voluntary (but not passive) movements of the right arm. Experience of the extra arm ceased if the patient moved her left arm or looked at it or had it touched.

The estimated position of a limb is based on integrating information from motor commands and sensory feedback. Failure to integrate these two sources of information could lead to the experience of two limbs rather than one. At the time of initiating action the patient of Hari *et al.* (1998) has the normal awareness of movement based on the representation of the predicted position of the arm. However, the representation of the estimated actual position of the arm fails to get updated on the basis of the motor commands. This discrepant representation of the estimated position of the arm emerges into awareness some time after the movement has been completed leading to the experience of an extra arm. Correct updating of this representation occurs on the basis of signals from the somatosensory or visual system. However, false updating can also occur based on motor commands controlling the right limb. This false updating must be based on motor commands rather than sensory feedback since passive movements of the right arm do not affect the phantom. Presumably the effect of signals concerning movements of the right limb are normally suppressed when they are discrepant from the motor commands driving the left limb. We are suggesting that movement of the phantom in this case derives from motor signals relating to the contralateral limb. This is different from the mechanism underlying the phenomenon described by some amputees in which the fingers of the phantom follow the movements of fingers on the contralateral hand. In these cases it is assumed that the experience is driven by somatosensory and proprioceptive signals from the contralateral fingers. If this is so, then movements of the phantom in amputees should be experienced whether the contralateral finger movements are active or passive.

The extra phantom limb experienced by the patient of Hari *et al.* (1998) emerged after a subarachnoid haemorrhage leading to an infarction in the right frontal lobe including damage to the most anterior region of the right SMA. However, brain scans suggested that there was also a congenital abnormality in the corpus callosum.

In our discussion of the anarchic hand sign (§ 3(a)(ii)) we suggested that the SMA, in particular the anterior part, has a major role in initiating movements and interacts with the parietal cortex in order to ensure that the movement initiated corresponds to the desired action. The case described by Hari *et al.* (1998) in which damage to the anterior SMA was associated with an extra phantom left arm suggests that this interaction between SMA and parietal cortex may also ensure integration between representations of predicted and actual limb positions. Damage to the anterior SMA can result in a failure of this integration.

(iv) *Anosognosia*

A patient with anosognosia is unaware of some impairment that has resulted from brain damage (Babinsky 1914). Here we shall be concerned only with those patients in whom the impairment concerns the motor control of a limb. Such patients typically have right-hemisphere damage leading to paralysis (or weakness) on the left side usually associated with hemianaesthesia. In § 3(b)(ii) we argued that this combination provides the appropriate circumstances for the development of a phantom limb. However, these patients, rather than developing a phantom limb, develop the false belief that there is nothing wrong with the paralysed limb. For example, the left side of Mrs F.D.'s body was completely paralysed as the result of a stroke.

Doctor: 'Mrs F.D., can you walk?'
F.D.: 'Yes.'
Doctor: 'Can you move your hands?'
F.D.: 'Yes.'
Doctor: 'Are both hands equally strong?'
F.D.: 'Yes, of course they are.'

(Ramachandran 1996, p. 124)

Sometimes patients will attempt to 'explain away' the lack of movement in the paralysed limb.

Doctor: 'Mrs L.R., why aren't you using your left arm.'
L.R.: 'Doctor, these medical students have been prodding me all day and I'm sick of it. I don't want to use my left arm.'

(Ramachandran 1996, p. 125)

In some cases the patient will claim to have moved a limb to command even though no movement has occurred.

Doctor: 'Can you clap?'
F.D.: 'Of course I can clap.'
Doctor: 'Will you clap for me?'
The patient proceeded to make clapping movements with her right hand as if clapping with an imaginary hand near the midline.
Doctor: 'Are you clapping?'
F.D.: 'Yes, I am clapping.'

(Ramachandran 1996, p. 124)

This disorder is often associated with unilateral neglect for the left side of space. Geschwind (1965) suggested that anosognosia arises from a disconnection such that sensory feedback (both somatosensory and visual), indicating that the limb is not working, is no longer available to a left-hemisphere monitoring system. However, making sure that the paralysed left arm can be seen in the right visual field does not alter the denial of impairment. Heilman *et al.* (1998) have proposed a 'feed-forward' theory of anosognosia. According to this account anosognosic patients receive no signals indicating movement failure because the comparator which contrasts intended and actual movements receives no signal that a movement has been intended. Because patients do not try to move the paralysed limb they never discover that it is paralysed. While this account can explain denial of impairment, it is not clear how it can explain cases, such as the one described above, in which the patient apparently experiences having made a movement when none has actually occurred.

How is it possible to experience a limb movement when none has actually occurred? On the basis of our review of evidence concerning the normal awareness of motor control we suggested that awareness of initiating a movement was based on a representation of the predicted consequences of making that movement, rather than its actual consequences. A representation of the predicted consequences of a movement can be formed as long as the controllers can compute the appropriate motor commands and the predictors can derive from these the expected consequences. Thus, a patient with a paralysed limb would have the normal experience of initiating a movement with that limb as long as the controller and predictor were functioning normally. However, to continue to believe that he or she had initiated that movement would require further abnormalities in the system. First, there would have to be a failure to register the discrepancy between the predicted consequences and the actual consequences of the movement that was initiated. We have already quoted the work of Fourneret & Jeannerod (1998) demonstrating that normal people can have a remarkably limited awareness of the actual form of the movements they have made. Thus, the patient with anosognosia is showing, in exaggerated form, a tendency already present in the normal state. The exaggeration of this tendency could be related to the neglect of the left side of space often shown by such patients. Second, there would have to be a failure to update the operations of the predictor. With experience the predictor should learn that the motor commands issued by the controller result in minimal movements of the paralysed limb. In the patient with anosognosia this updating does not occur.

We suggest, then, that the false experience of movement reported by patients with anosognosia occurs because, while representations of the desired and predicted positions of the limb are appropriate, the patient is not aware of the discrepant representation of the actual position of the limb. The controllers issue the appropriate motor commands, but, due to paralysis, do not generate a limb movement. However, the predictors have estimated, on the basis of these commands and from past experience prior to brain damage, the new position of the limb. The lack of a discrepancy between intended and predicted positions indicates success. Contrary information derived from sensory feedback concerning actual limb positions is not available, since the relevant brain regions have been damaged or else this contrary information is neglected (figure 4). As a result the estimated position of the limb is based on sequences of motor commands and not upon sensory feedback.

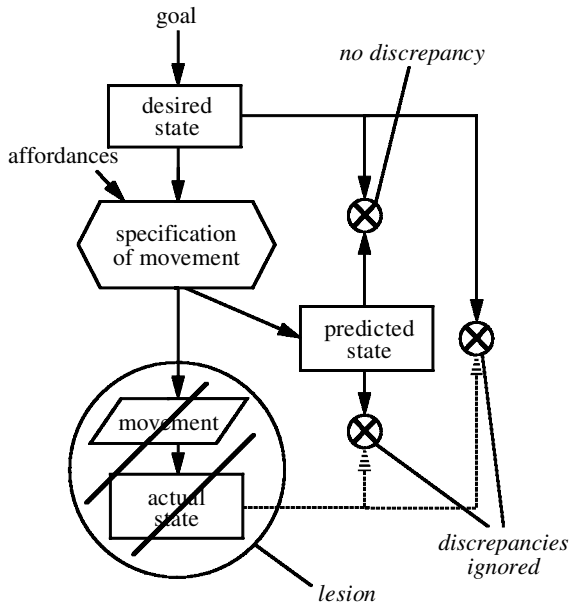

Figure 4. The underlying disorder leading to anosognosia. The patient formulates the action needed to fulfil his intention and is aware that the action initiated is appropriate. No information about the actual position of the limb is available to indicate that no action has actually occurred.

Anosognosia is usually associated with damage to the right hemisphere, especially the parietal lobe. However, there is, as yet, no information about the precise location of the lesions that lead to the illusion that a paralysed limb is being moved normally. Damage to the parietal lobe is most frequently associated with apraxia rather than anosognosia and apraxic patients can sometimes show features of anosognosia. For example, Sirigu *et al.* (1999) studied three patients with apraxia while they performed gestures to command (e.g. extend index and little finger). On some trials the patients saw their own hand performing the gesture, but on other trials they saw the hand of an experimenter performing the same or a different gesture. On nearly 90% of trials in which the patients saw the experimenter making accurately the gesture that they were trying to make they believed that they were observing their own hand even though they had actually made a very clumsy gesture. In these cases false visual feedback elicited a form of anosognosia. However, the patients were not generally anosognosic. When they saw their own hand they recognized and were distressed by the clumsiness of their gestures. Furthermore, the lesions in these cases were in the left parietal cortex, which is typical for apraxia, rather than the right parietal cortex, which is typical for anosognosia.

In another experiment Sirigu *et al.* (1996) investigated the effects of parietal lobe lesions (both left- and right-sided) on the time needed to make imaginary movements.

How long we take to make movements in the imagination depends upon the functioning of the predictor, not upon the actual state of the system. Sirigu *et al.* (1996) showed that a patient with unilateral damage to the motor cortex showed strong correlations between the time to make actual movements and the time to make imaginary movements with both the intact and the impaired hand. For the impaired hand the times for actual and imagined movements were much slower. In this case the predictor had been updated to take account of the changed abilities of the impaired hand. In contrast patients with parietal lesions did not show this close link between actual and imagined movements in the limb contralateral to the lesion. In these cases the discrepancies between predicted and actual movements have not been used to update the estimates made by the predictors.

Clearly damage to parietal cortex can impair awareness of the actual state of the motor system and also lead to failure to take note of discrepancies between the actual and predicted states of the system. However, though these problems may be necessary for anosognosia they do not seem to be sufficient. Another consequence of parietal lesions is unilateral spatial neglect. This syndrome, especially in its perceptual form, is usually associated with lesions in the right inferior parietal lobe (Vallar & Perani 1986) and is often associated with anosognosia. Patients with neglect fail to notice or respond to objects and events in their left hemifield. Neglect of this kind would allow even visual evidence that a movement had not been made to be ignored.

Ramachandran (1996) considered that the accounts of anosognosia of the kind outlined above are not sufficient to explain the extent to which anosognosic patients can ignore the wealth of evidence indicating that they are paralysed. He proposes that there are additional factors at work which enable patients to ignore sensory anomalies. These factors have parallels with those associated with delusions and confabulations. Confabulations are more usually associated with memory impairments. The patient recollects past events which did not and, indeed, could not have happened. The patient seems to be unaware of the impossibility of what he or she is reporting. Such problems are typically associated with damage to the right frontal cortex (Burgess & Shallice 1996), which is believed to have a role in monitoring the consequences of action at a high level. There is evidence that this role also applies to the motor system. For example, if a normal volunteer is performing a task with two hands, but one hand is hidden behind a mirror, then the illusion is created that both hands are seen, when, in fact, the subject is viewing a single hand and its mirror image. In this case, if the task is to move the hands out of phase, the visual feedback falsely indicates that the hands are moving in phase. Performance of this somewhat disturbing task in which there is a discrepancy between expectations derived from intended movements and what is actually seen, elicits activity in right dorsolateral prefrontal cortex (Fink *et al.* 1999). It is plausible that damage to this region might result in failure to respond to such discrepancies.

(v) *Utilization behaviour*

Some patients with damage to the frontal lobes show 'utilization behaviour' (Lhermitte 1983) in which the patient uses objects inappropriately. The sight of an object elicits a stereotyped action which is inappropriate in the wider context. For example, if there is a glass within reach of the patient, he will grasp it. If a bottle of water is placed on the desk he will grasp this too and then pour water into the glass and drink it. Such behaviour is not shown by normal subjects put in the same situation or by patients with posterior lesions.

> 'If the examiner asks the patient why he grasped the objects and used them, then the answer is always the same, "You held them out to me, I thought I had to use them."
>
> The examiner then ... gives the instruction, "You are mistaken; from now on don't grasp any of the objects I will show you, and in no case must you use them."
>
> After about 20–30 s, during which time the patient's attention has to be distracted ... the behaviour remains unchanged. If the examiner asks if the patient remembers the instruction, the latter replies, most of the time, "It's true, I remember."
>
> "Then why [did you grasp the objects]?"
>
> "Because you held out the objects to me and I thought I had to reach and grasp them."' (Lhermitte 1983, p. 246)

Much more complex actions can also be elicited by the environment in which the patient finds himself. 'Patient 1 ... came to see me at my apartment .... We returned to the bedroom. The bedspread had been taken off and the top sheet turned back in the usual way. When the patient saw this he immediately began to get undressed [including taking off his wig]. He got into bed, pulled the sheet up to his neck and prepared to go to sleep.' (Lhermitte 1986, p. 338).

On the surface this behaviour is very similar to that associated with the anarchic hand. Actions are elicited by objects in the environment even though such actions are not appropriate. However, there is an additional problem which is reflected in the patient's experience of this disorder of control. The patient showing utilization behaviour does not perceive a discrepancy between his actions and his intentions. He is not upset by the actions and he does not develop strategies to prevent the actions occurring. On being asked why he performed the actions the patient will 'rationalize', saying that he performed the action because he thought that is what the examiner wanted him to do. Our formulation of utilization behaviour is that the patient's actions are involuntarily elicited by objects in the environment, but that the patient erroneously experiences these actions as intended.

Problems with the experience of intention are not unique to these patients. Normal three-year-old children do not distinguish between an intentional movement and a knee-jerk reflex. Only at five years do children state that the knee-jerk reflex was unintended (Schultz *et al.* 1980). Three-year-old children, however, do state that their movement was unintended in the interlaced finger game. In this task the child can see that the designated finger remains stationary while the wrong finger moves. The child has a clear goal which has not been achieved. The lack of success is taken to indicate a lack of intention. In the case of the knee-jerk reflex there is no simple prior goal, and thus a judgement cannot be made as to whether or not the movement was successful. Smith (1978) suggested that, without an explicitly stated goal, the

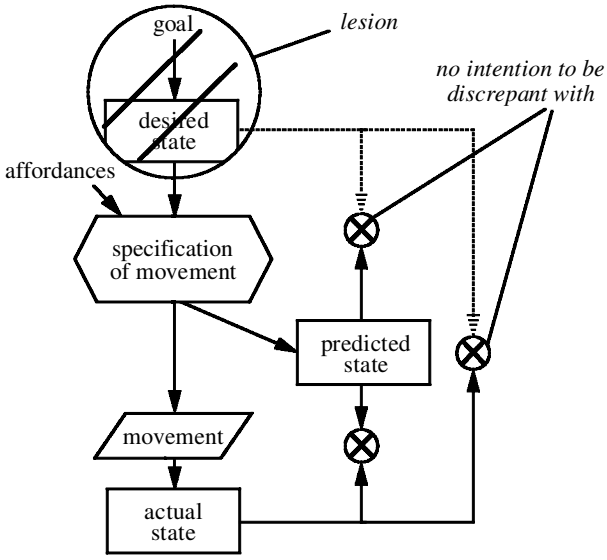

Figure 5. The underlying disorder leading to utilization behaviour. The patient does not form any intentions and so makes stereotyped responses to objects in the environment. The patient is not aware that these responses are inappropriate.

default judgement is that actions are intended. Only by the age of five can the child form the much more abstract goal of 'not moving' in order to interpret the knee-jerk reflex correctly.

A corollary of this argument is that, if an explicit goal is formed just prior to an action which achieves that goal, then the action will be perceived as intended. Wegner & Wheatley (1999) have used just this technique to elicit the erroneous perception of intended action in normal adults. A subject and a confederate simultaneously used a single mouse to control the position of a pointer on a screen. If the attention of the subject was drawn to an object on the screen shortly before the pointer stopped near that object, then the subject frequently believed that he had intentionally moved towards the object even though in reality his arm had been moved passively by the confederate. As long as the action did not conflict with some explicitly formed goal then the action was perceived as intended.

These results suggest that the experience of an action as intended depends on the relationship between the action and a prior goal. If the action does not match the goal then the action is unintended. If, however, there is no prior goal then, by default the action is perceived as intended. In these terms utilization behaviour can be explained as resulting from a failure to represent goals. We suggest that the problem causing utilization behaviour occurs at an earlier stage in the development of an action than that causing the anarchic hand. The problem has two components. First, there is no awareness of goals and intended actions (figure 5). The patient is not aware of what he is going to do until after he has done it. Second, inappropriate responses elicited by objects in the environment are not inhibited. These components can be related if we assume that a lack of awareness of intentions reflects a failure to develop such intentions. Responses to objects in the environment are normally inhibited until an intention has been developed. The system that develops intentions also inhibits inappropriate responses.

The high-level control system we are describing here is based on the supervisory attentional system developed by Shallice (1988, pp. 328–352) to explain the behaviour of patients with frontal lobe lesions. These patients have no problems in routine situations, but have difficulty coping with novel tasks. With such tasks they may make inappropriate routine responses (a form of utilization behaviour) or they may fail to respond. This response failure occurs because, in novel situations it is not only necessary to inhibit inappropriate responses elicited by objects in the environment, but also to initiate responses when there is no external stimulus to elicit them.

While there is good evidence that this high-level control system is instantiated in prefrontal cortex (Shallice 1988), it has proved more difficult to relate particular components of this system to specific regions within prefrontal cortex. Imaging studies suggest that dorsolateral prefrontal cortex (BA46 and 9) is particularly involved in selection between alternative actions when there are no external cues to indicate which action is the most appropriate (Jahanshahi & Frith 1998). However, utilization behaviour seems to more concerned with failure to inhibit inappropriate movements rather than a failure select appropriate ones. There is some evidence that the lesions that produce utilization behaviour are more likely to involve the ACC (Degos *et al.* 1993). Such lesions are also associated with difficulties in inhibiting routine responses, for example, inhibiting saccades to peripheral stimuli (Paus *et al.* 1991).

There is also, as yet, little evidence concerning brain areas concerned with awareness of intended actions. In one of the few relevant imaging studies Jueptner *et al.* (1997) trained volunteers until they could perform a paced sequence of button presses routinely and without thought. The volunteers were then scanned, either while performing this task in routine mode, or when deliberately thinking of which button had to be pressed next in the sequence. The requirement to be aware of their intended action increased activity in dorsolateral prefrontal cortex and in the ACC (BA32). There are no direct connections between these regions and motor cortex, so that their influence on movement is mediated via their connections with premotor regions including the SMA (Lu *et al.* 1994). On the basis of their study of patients with medial frontal lesions, Paus *et al.* (1991) concluded that the ability to inhibit routine responses depends upon input from the ACC to the SMA. Thus, the same system has been implicated both in the awareness of intended actions and in the inhibition of routine actions. These proposals are also in accord with our suggestion that utilization behaviour is caused by damage to an earlier stage in the system that generates actions than that associated with the anarchic hand.

(c) *Abnormalities in the perception of action while the control of action is largely intact*

(i) *Delusions of control; passivity experiences associated with schizophrenia*

Many patients with schizophrenia describe 'passivity' experiences in which actions, thoughts or emotions are made for them by some external agent rather than by their own will. 'My fingers pick up the pen, but I don't control them. What they do is nothing to do with me.'

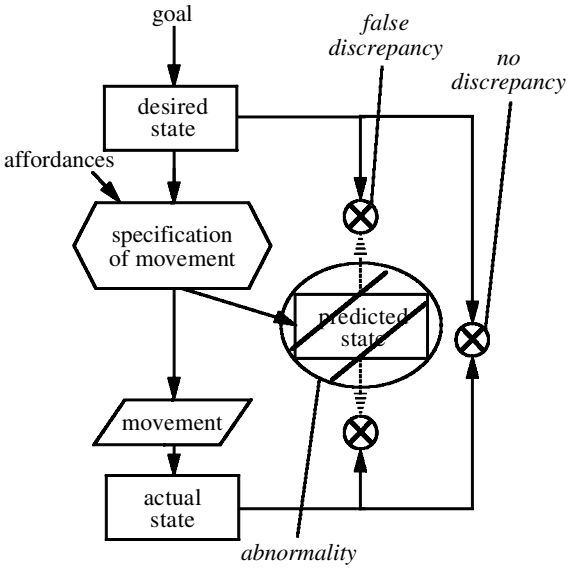

Figure 6. The underlying disorder leading to delusions of control. The patient formulates the action appropriate to his intention and the action is successfully performed. The patient is aware that the action matches the intention, but has no awareness of initiating the action or of its predicted consequences. The patient feels as if his intentions are being monitored and his actions made for him by some external force.

'The force moved my lips. I began to speak. The words were made for me.' (Mellors 1970, p. 18). In most cases the actions made when the patient 'feels' that he is being controlled by alien forces are not discrepant with his intentions. Thus the patient may be correctly performing the task set by the experimenter (e.g. making random movements of a joystick) at the same time as having the experience of passivity (see Spence *et al.* 1997). The patient does not try to correct these 'controlled' actions or prevent them from occurring. Clearly actions are being correctly selected and irrelevant affordances are being suppressed.

We have previously suggested that these abnormal experiences arise through a lack of awareness of intended actions (Frith 1987). However, this formulation is inconsistent with the patients' ability to follow the commands of the experimenter, to avoid showing utilization behaviour, and to correct errors on the basis of sensory feedback about limb positions (which requires comparison of intended actions and their consequences). Instead we suggest that the experience of alien control arises from a lack of awareness of the predicted limb position (figure 6). As a result the patient is not aware of the exact specification of the movement. He is aware of his goal, of the intention to move and of the movement having occurred, but he is not aware of having initiated the movement. It is as if the movement, although intended, has been initiated by some external force. In a variation on this theme Spence (1996) suggested that the problem is to do with the timing of awareness. Normally we are aware of initiating a movement *ca.* 80 ms before the movement actually begins and, therefore well before any sensory feedback resulting from the movement (Libet *et al.* 1983). Spence suggested that, in the presence of delusions of control, the awareness of the sensory consequences of the movement precedes the awareness of initiating the movement, which is in the opposite order to the normal experience of our own agency. We suggest that, in the presence of delusions of control, the patient is not aware of the predicted consequences of a movement and is therefore not aware of initiating a movement.

There is nothing obviously abnormal in the motor control of these patients. This suggests that accurate representations of predicted states are available and used by the motor system. However, these representations are not available to awareness. A number of experiments confirm that there are subtle problems consistent with a lack of awareness of predicted actions. These patients fail to make rapid error corrections based on awareness of discrepancies between intended and predicted limb positions, although they have no difficulty correcting errors based on visual feedback about actual limb positions (Malenka *et al.* 1982; Frith & Done 1989). These patients have difficulty remembering the precise details of actions made in the absence of visual feedback (Mlakar *et al.* 1994; Stirling *et al.* 1998). They also have difficulty distinguishing between correct visual feedback about the position of their hand and false feedback when the image of the hand they see is in fact that of another person attempting to make the same movements as the patient (Daprati *et al.* 1997).

Jeannerod (1999) suggested that conscious judgement about a movement requires a different form of representation from that needed for comparisons of predictions and outcomes within the motor system. Following Barresi & Moore (1996) (see also Frith 1995) he suggests that conscious judgements about movements require 'third-person' information while control of movement depends upon private 'first-person' information. In terms of this formulation he suggests that schizophrenic patients fail to monitor the third-person signals that enable them to make judgements about their own actions. We would suggest, rather, that, in schizophrenia, something goes wrong with the mechanism that translates the first-person representations that are involved in motor control into the third-person representations that are needed for conscious monitoring of the motor control system. This is part of more general problem that these patients have in escaping from a first-person, egocentric view of the world.

Spence *et al.* (1997) used brain imaging to identify brain regions associated with the experience of delusions of control. They scanned schizophrenic patients with and without such delusions while they performed a response selection task. The presence of delusions of control was associated with overactivity in right inferior parietal cortex. We suggest that this overactivity reflected a heightened response to the sensory consequences of the movements the patients were making during scanning. Normally activity associated with sensory stimulation is much reduced if this stimulation is the direct consequence of our own movements (Blakemore *et al.* 1998*b*). This is because the sensory consequences of our movements can be predicted. In the presence of delusions of control, modulation of sensory areas based on such predictions fails, and the regions are overactive. Although the patient is making an active movement, the brain activity and the associated experience resembles

that seen with passive arm movements (Weiller *et al.* 1996).

We have already discussed (§ 2(c)(ii)) the evidence that the parietal cortex has a major role in the control of action which depends upon forming representations in many different coordinate systems (e.g. retinotopic, head centred, body centred, etc.). As yet, however, we have not considered in any detail the nature and location of the subset of these representations that are available to consciousness. Such consideration is crucial for understanding abnormalities in the awareness of the motor system as observed in anosognosia and delusions of control. Frith (1995) and Jeannerod (1999) theorized that representations suitable for awareness need to be in viewer-independent or 'third-person' coordinates, and not in the private, egocentric coordinates that a more suited for the direct control of movement. Is there any evidence for segregation of these kinds of representation in parietal cortex? We have already presented evidence that there is a general lack of awareness of the details of motor commands and their fine-tuning by affordances as in reaching and grasping. The IPS seems to have a major role in this activity as revealed by single-cell neurophysiology, imaging studies and the effects of lesions. Of particular interest is the observation that optic ataxia, which is caused by lesions to the IPS, is defined by problems with reaching and grasping, but is not associated with any disorder of awareness. In contrast, imaging studies of motor preparation and motor imagery, which emphasize awareness of motor representations, tend to activate the inferior parietal lobe (supramarginal gyrus, BA40; Stephan *et al.* 1995; Krams *et al.* 1998). Lesions in this region, particularly in the right hemisphere, are associated with disorders of awareness such as neglect and anosognosia (Vallar & Perani 1986). This is also the region that is overactive when patients with schizophrenia are experiencing delusions of control.

Given that schizophrenic patients do not have fundamental problems with the control of action it seems unlikely that the brain abnormality associated with delusions of control is located in parietal cortex where the overactivity is observed. It is more likely that the damage involves the system that normally modulates activity at this site. Fletcher *et al.* (1999), for example, provide evidence of abnormal modulation of long-range cortico-cortical connections in patients with schizophrenia and suggest that the anterior cingulate has a key role in this modulatory system. We have already mentioned evidence from imaging that the ACC is involved in attention to future actions (Jueptner *et al.* 1997) and may have a role in suppressing inappropriate actions via its connections with the SMA (Paus *et al.* 1991). We speculate that it may also have a role in modulating representations in the inferior parietal cortex which underpin awareness of the current and future states of the motor system.

4. CONCLUSIONS

In this paper we have attempted to develop a framework based upon well-established principles of motor control in such a way that the components of the system can be related first to the subjective experience of motor control, and second to the underlying physiology upon which motor control depends. We have devoted much attention to the abnormalities of control associated with various neurological and psychiatric disorders. Careful consideration of these abnormalities provides important evidence linking awareness of control to the underlying components of the system. Indeed, we consider that these abnormalities cannot be properly understood without taking into account the subjective experience of the patients. As yet the physiological underpinnings of the motor control system are understood only in the broadest terms. However, there is a rapidly increasing body of evidence from studies of patients with circumscribed lesions and from functional brain imaging studies to aid in generating a more detailed account. On the basis of this evidence it is now possible to explore the brain systems concerned specifically with awareness of the different aspects of the motor control system.

In this paper we have only considered relatively simple motor functions such as reaching and grasping or learning sequences of movements. However, the control system we have described, involving representations of desired and predicted states and models for generating these states, could apply equally well to much more difficult problems. It is simple, in principle, to extend the concept of internal models of the motor system to internal models of the external world, of other people's mental processes, or of states of one's own mind. For example, rather than changing the position of an arm, one might wish to change someone else's belief about the world. Of course, we have no direct knowledge of their belief, we have to estimate this just as we have to estimate the current position of our own limbs. Given an estimate of a person's current belief, a controller could used to compute the behaviour (or speech) we need to adopt in order to produce the required change. A predictor could be run to check whether this behaviour would indeed produce the desired change in the belief of the other person. Similar analysis could be applied to the control of many aspects of the external world.

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REFERENCES

Aglioti, S., Bonazzi, A. & Cortese, F. 1994 Phantom lower limb as a perceptual marker of neural plasticity in the human brain. *Proc. R. Soc. Lond.* B **255**, 273–278.

Aglioti, S., DeSouza, J.-F.-X. & Goodale, M.-A. 1995 Size-contrast illusions deceive the eye, but not the hand. *Curr. Biol.* **5**, 679–685.

Andersen, R. A. 1995 Encoding of intention and spatial location in the posterior parietal cortex. *Cerebr. Cortex* **5**, 111–122.

Andersen, R. A., Snyder, L. H., Bradley, D. C. & Xing, J. 1997 Multimodal representation of space in the posterior parietal cortex and its use in planning movements. *A. Rev. Neurosci.* **20**, 303–330.

Babinsky, M. J. 1914 Contribution a l'etude des troubles mentaux dans l'hemiplegie organique cerebrale (agnosognosie). *Revue Neurologique* **1**, 845–847.

Bálint, R. 1909 Seelenlämung des 'Schauens', optisches Ataxie, räumliche Störung der Aufmerksamkeit. *Monatsschift für Psychiatrie und Neuologie* **25**, 51–81.

Ball, T., Schreiber, A., Feige, B., Wagner, M., Lücking, C. H. & Kristeva-Feige, R. 1999 The role of higher-order motor areas in voluntary movements as revealed by high-resolution EEG and fMRI. *NeuroImage* **10**, 682–694.

Banks, G., Short, P., Martinez, A. J., Latchaw, R., Ratcliff, G. & Boller, F. 1989 The alien hand syndrome; clinical and post-mortem findings. *Arch. Neurol.* **46**, 456–459.

Barresi, J. & Moore, C. 1996 Intentional relations and social understanding. *Behav. Brain Sci.* **19**, 107–154.

Blakemore, S.-J., Goodbody, S. J. & Wolpert, D. M. 1998*a* Predicting the consequences of our own actions: the role of sensorimotor context estimation. *J. Neurosci.* **18**, 7511–7518.

Blakemore, S.-J., Wolpert, D. M. & Frith, C. D. 1998*b* Central cancellation of self-produced tickle sensation. *Nature Neurosci.* **1**, 635–640.

Blakemore, S.-J., Frith, C. D. & Wolpert, D. W. 1999 Spatiotemporal prediction modulates the perception of self-produced stimuli. *J. Cogn. Neurosci.* **11**, 551–559.

Bridgeman, B., Kirch, M. & Sperling, A. 1981 Segregation of cognitive and motor aspects of visual function using induced motion. *Percept. Psychophysiol.* **29**, 336–342.

Burgess, P. W. & Shallice, T. 1996 Confabulation and the control of recollection. *Memory* **4**, 1–53.

Castiello, U., Paulignan, Y. & Jeannerod, M. 1991 Temporal dissociation of motor responses and subjective awareness. A study in normal subjects. *Brain* **114**, 2639–2655.

Clower, D. M., Hoffman, J. M., Votaw, J. R., Faber, T. L., Woods, R. P. & Alexander, G. E. 1996 Role of posterior parietal cortex in the recalibration of visually guided reaching. *Nature* **383**, 618–621.

Colby, C. L. & Duhamel, J.-R. 1996 Spatial representations in parietal cortex. *Cerebr. Cortex* **5**, 105–115.

Colby, C. L., Duhamel, J. R. & Goldberg, M. E. 1995 Oculocentric spatial representation in parietal cortex. *Cerebr. Cortex* **5**, 470–481.

Cole, J. D. 1991 *Pride and a daily marathon*. London: Duckworth.

Corbetta, M. 1998 Frontoparietal cortical networks for directing attention and the eye to visual locations; identical, independent, or overlapping neural systems? *Proc. Natl Acad. Sci. USA* **95**, 831–838.

Corbetta, M., Miezin, F., Shulman, G. & Petersen, S. E. 1993 A PET study of visuospatial attention. *J. Neurosci.* **13**, 1202–1226.

Craik, K. J. W. 1948 Theory of the human operator in control systems. II. Man as an element in a control system. *Br. J. Psychol.* **38**, 142–148.

Daprati, E., Franck, N., Georgieff, N., Proust, J., Pacherie, E., Dalery, J. & Jeannerod, M. 1997 Looking for the agent: an investigation into consciousness of action and self-consciousness in schizophrenic patients. *Cognition* **65**, 71–86.

Decety, J., Jeannerod, M., Germain, M. & Pastene, J. 1991 Vegetative response during imagined movement is proportional to mental effort. *Behav. Brain Res.* **42**, 1–5.

Decety, J., Perani, D., Jeannerod, M., Bettinardi, V., Tadary, B., Woods, R., Mazziotta, J. C. & Fazio, F. 1994 Mapping motor representations with PET. *Nature* **371**, 600–602.

Deiber, M.-P., Passingham, R. E., Colebatch, J. G., Friston, K. J., Nixon, P. D. & Frackowiak, R. S. J. 1991 Cortical areas and the selection of movement. *Exp. Brain Res.* **84**, 393–402.

Degos, J. D., Dafonseca, N., Grey, F. & Cesaro, P. 1983 Sever frontal syndrome associated with infarcts of the left anterior cingulate gyrus and the head of the right caudate nucleus—a clinicopathological case. *Brain* **116**, 1541–1548.

De Renzi, E. & Lucchelli, F. 1988 Ideational apraxia. *Brain* **113**, 1173–1188.

De Renzi, E., Faglioni, P. & Sergato, P. 1982 Modality-specific and supramodal mechanisms of apraxia. *Brain* **105**, 301–312.

Dettmers, C., Fink, G. R., Lemon, R. N., Stephan, K. M., Passingham, R. E., Silbersweig, D. A., Holmes, A., Ridding, M. C., Brookes, D. J. & Frackowiak, R. S. J. 1995 Relation between cerebral activity and force in the motor areas of the human brain. *J. Neurophysiol.* **74**, 802–815.

Duhamel, J. R., Colby, C. L. & Goldberg, M. E. 1992 The updating of the representation of visual space in parietal cortex by intended eye movements. *Science* **255**, 90–92.

Feltz, D. L. & Landers, D. M. 1983 The effects of mental practice on motor skill learning and performance. A meta-analysis. *J. Sport Psychol.* **5**, 27–57.

Fink, G. R., Marshall, J. C., Halligan, P. W., Frith, C. D., Driver, J., Frackowiak, R. S. J. & Dolan, R. J. 1999 The neural consequences of conflict between intention and the senses. *Brain* **122**, 497–512.

Flanders, M., Helms-Tillery, S. I. H. & Soechting, A. J. F. 1992 Early stages in a sensorimotor transform. *Behav. Brain Sci.* **15**, 309–362.

Fletcher, P., McKenna, P. J., Friston, K. J., Frith, C. D. & Dolan, R. J. 1999 Abnormal cingulate modulation of fronto-temporal connectivity in schizophrenia. *NeuroImage* **9**, 337–342.

Fourneret, P. & Jeannerod, M. 1998 Limited conscious monitoring of motor performance in normal subjects. *Neuropsychologia* **36**, 1133–1140.

Frith, C. D. 1987 The positive and negative symptoms of schizophrenia reflect impairments in the perception and initiation of action. *Psychol. Med.* **17**, 631–648.

Frith, C. D. 1995 Consciousness is for other people. *Behav. Brain Sci.* **18**, 682–683.

Frith, C. D. & Done, D. J. 1989 Experiences of alien control in schizophrenia reflect a disorder in the central monitoring of action. *Psychol. Med.* **19**, 359–363.

Gallese, V., Fadiga, L., Fogassi, L., Luppino, G. & Murata, A. 1997 A parieto-frontal circuit for hand grasping movements in the monkey: evidence from reversible inactivation experiments. In *Parietal lobe contributions to orientation in 3-D space* (ed. P. Thier & H. O. Karnath), pp. 255–269. Heidelberg, Germany: Springer.

Galletti, C., Battaglini, P. P. & Fattori, P. 1993 Parietal neurons encoding spatial locations in craniotopic coordinates. *Exp. Brain Res.* **96**, 221–229.

Gentilucci, M., Chieffi, S., Daprati, E., Saetti, M. C. & Toni, I. 1996 Visual illusion and action. *Neuropsychologia* **34**, 369–376.

Geschwind, N. 1965 Disconnexion syndromes in animals and man. *Brain* **88**, 237–294.

Ghahramani, Z. & Wolpert, D. M. 1997 Modular decomposition in visuomotor learning. *Nature* **386**, 392–395.

Goldberg, G., Mayer, N. H. & Toglia, J. U. 1981 Medial frontal cortex and the alien hand sign. *Arch. Neurol.* **38**, 683–686.

Goodale, M. A., Pélisson, D. & Prablanc, C. 1986 Large adjustments in visually guided reaching do not depend on vision of the hand or detection of target displacement. *Nature* **320**, 748–750.

Goodale, M. A., Jacobson, L. S., Milner, A. D., Perrett, D. I., Benson, P. J. & Hietanen, J. K. 1994 The nature and limits of orientation and pattern processing visuomotor control in a visual form agnosic. *J. Cogn. Neurosci.* **6**, 46–56.

Greeno, J. G. 1994 Gibson's affordances. *Psychol. Rev.* **101**, 336–342.

Haggard, P. & Eimer, M. 1999 On the relation between brain potentials and awareness of voluntary movements. *Exp. Brain Res.* **126**, 128–133.

Haggard, P. & Magno, E. 1999 Localising awareness of action with transcranial magnetic stimulation. *Exp. Brain Res.* **127**, 102–107.

Haggard, P., Newman, C. & Magno, E. 1999 On the perceived time of voluntary actions. *Br. J. Psychol.* **90**, 291–303.

Hari, R., Hanninen, R., Makinen, T., Jousmaki, V., Forss, N., Seppa, M. & Salonen, O. 1998 Three hands: fragmentation of bodily awareness. *Neurosci. Lett.* **240**, 131–134.

Harvey, M. 1995 Psychic paralysis of gaze, optic ataxia, spatial disorder of attention. Translated from Bálint 1909. *Cogn. Neuropsychol.* **12**, 266–282.

Heilman, K. M., Barrett, A. M. & Adair, J. C. 1998 Possible mechanisms of anosognosia: a defect in self-awareness. *Phil. Trans. R. Soc. Lond.* B **353**, 1903–1909.

Ito, M. 1970 Neurophysiological aspects of the cerebellar motor control system. *Int. J. Neurol.* **7**, 162–176.

Jahanshahi, M. & Frith, C. D. 1998 Willed action and its impairments. *Cogn. Neuropsychol.* **15**, 483–533.

Jeannerod, M. 1994 The representing brain—neural correlates of motor intention and imagery. *Behav. Brain Sci.* **17**, 187–202.

Jeannerod, M. 1999 To act or not to act: perspectives on the representation of actions. *Q. J. Exp. Psychol.* A **52**, 981–1020.

Jeannerod, M., Michel, F. & Prablanc, C. 1986 The control of hand movements in case of hemianaesthesia following a parietal lesion. *Brain* **107**, 899–920.

Jeannerod, M., Decety, J. & Michel, F. 1994 Impairment of grasping movements following bilateral posterior parietal lesions. *Neuropsychologia* **32**, 369–380.

Jeannerod, M., Arbib, M. A., Rizzolatti, G. & Sakata, H. 1995 Grasping objects: the cortical mechanisms of visuomotor transformation. *Trends Neurosci.* **18**, 314–320.

Jordan, M. I. 1996 Computational aspects of motor control and motor learning. In *Handbook of perception and action: motor skills* (ed. E. H. Heuer & E. S. Keele), pp. 71–118. New York: Academic Press.

Jueptner, M., Stephan, K. M., Frith, C. D., Brooks, D. J., Frackowiak, R. S. & Passingham, R. E. 1997 Anatomy of motor learning. I. Frontal cortex and attention to action. *J. Neurophysiol.* **77**, 1313–1324.

Kalaska, J. F. & Crammond, D. J. 1992 Cerebral cortical mechanisms of reaching movements. *Science* **255**, 1517–1525.

Kew, J. J. M., Halligan, P. W., Marshall, J. C., Passingham, R. E., Rothwell, J. C., Ridding, M. C., Marsden, C. D. & Brooks, D. J. 1997 Abnormal access of axial vibrotactile input to deafferented somatosensory cortex in human upper limb amputees. *J. Neurophysiol.* **77**, 2753–2764.

Krams, M., Rushworth, M. F. S., Deiber, M.-P., Frackowiak, R. S. J. & Passingham, R. E. 1998 The preparation, execution and suppression of copied movements in the human brain. *Exp. Brain Res.* **120**, 386–398.

Lhermitte, F. 1983 'Utilisation behaviour' and its relation to lesions of the frontal lobes. *Brain* **106**, 237–255.

Lhermitte, F. 1986 Human autonomy and the frontal lobes. II. Patient behavior in complex and social situations: the 'environmental dependency syndrome'. *Annls Neurol.* **19**, 335–343.

Libet, B., Gleason, C. A., Wright, E. W. & Pearl, D. K. 1983 Time of conscious intention to act in relation to onset of cerebral activity (readiness potential): the unconscious initiation of a freely voluntary act. *Brain* **106**, 623–642.

Lu, M.-T., Preston, J. R. & Strick, P. L. 1994 Interconnections between prefrontal cortex and the premotor areas in the frontal lobe. *J. Comp. Neurol.* **341**, 375–392.

McCloskey, D. I., Colebatch, J. G., Potter, E. K. & Burke, D. 1983 Judgements about the onset of rapid voluntary movements in man. *J. Neurophysiol.* **49**, 851–863.

Malenka, R. C., Angel, R. W., Hamptom, B. & Berger, P. A. 1982 Impaired central error correcting behaviour in schizophrenia. *Arch. Gen. Psychiat.* **39**, 101–107.

Marchetti, C. & Della Salla, S. 1998 Disentangling the alien and anarchic hand. *Cogn. Neuropsychiat.* **3**, 191–208.

Mellors, C. S. 1970 First-rank symptoms of schizophrenia. *Br. J. Psychiat.* **117**, 15–23.

Miall, R. C. & Wolpert, D. M. 1996 Forward models for physiological motor control. *Neural Net.* **9**, 1265–1279.

Miall, R. C., Weir, D. J., Wolpert, D. M. & Stein, J. F. 1993 Is the cerebellum a Smith predictor? *J. Mot. Behav.* **25**, 203–216.

Milner, A. D. & Goodale, M. A. 1995 *The visual brain in action*. Oxford University Press.

Milner, A. D., Paulignan, Y., Dijkerman, H. C., Michel, F. & Jeannerod, M. 1999 A paradoxical improvement of misreaching in optic ataxia: new evidence for two separate neural systems for visual localization. *Proc. R. Soc. Lond.* B **266**, 2225–2229.

Mlakar, J., Jensterle, J. & Frith, C. D. 1994 Central monitoring deficiency and schizophrenic symptoms. *Psychol. Med.* **24**, 557–564.

Nobre, A. C., Gitelman, D. R., Sebestyen, G. N., Meyer, J., Frackowiak, R. S. J., Frith, C. D. & Mesulam, M.-M. 1997 Functional localisation of the system for visuospatial attention using positron emission tomography. *Brain* **120**, 515–533.

Parkin, A. J. 1996 The alien hand. In *Methods in madness: case studies in cognitive neuropsychiatry* (ed. P. W. Halligan & J. C. Marshall), pp. 173–183. Hove, UK: Psychology Press.

Pascual-Leone, A., Dang, N., Cohen, L. G., Brasil-Neto, J., Cammarota, A. & Hallett, M. 1995 Modulation of motor responses evoked by transcranial magnetic stimulation during the acquisition of new fine motor skills. *J. Neurophysiol.* **74**, 1037–1045.

Passingham, R. E. 1993 *The frontal lobes and voluntary action*. Oxford University Press.

Passingham, R. E. 1996 Attention to action. *Phil. Trans. R. Soc. Lond.* B **351**, 1423–1432.

Passingham, R. E. 1998 The specialisation of the human neocortex. In *Comparative neuropsychology* (ed. A. D. Milner), pp. 271–298. Oxford University Press.

Paus, T., Kalina, M., Patockova, L., Angerova, Y., Cerny, R., Mecir, P., Bauer, J & Krabec, P. 1991 Medial vs lateral frontal lobe lesions and differential impairment of central-gaze fixation maintenance in man. *Brain* **114**, 2051–2067.

Pause, M., Kunesch, E., Binkovski, F. & Freund, H.-J. 1989 Sensorimotor disturbances in patients with lesions of the parietal cortex. *Brain* **112**, 1599–1625.

Perenin, M.-T. & Rossetti, Y. 1996 Grasping without form discrimination in an hemianopic field. *NeuroReport* **7**, 793–797.

Perenin, M.-T. & Vighetto, A. 1988 Optic ataxia: a specific disruption in visuo-motor mechanisms. I. Different aspects of the deficit in reaching for objects. *Brain* **111**, 643–674.

Pickard, N. & Strick, P. L. 1996 Motor areas of the medial wall: a review of their location and functional activation. *Cerebr. Cortex* **6**, 342–353.

Ramachandran, V. S. 1993 Behavioural and magnetoencephalographic correlates of plasticity in the adult human brain. *Proc. Natl Acad. Sci. USA* **90**, 10 413–10 420.

Ramachandran, V. S. 1996 What neurological syndromes can tell us about human nature: some lessons from phantom limbs, Capgras syndrome, and anosognosia. *Cold Spring Harb. Symp. Quant. Biol.* **61**, 115–134.

Ramachandran, V. S. & Hirstein, W. 1998 The perception of phantom limbs. *Brain* **121**, 1603–1630.

Ramachandran, V. S. & Rogers-Ramachandran, D. 1996 Synaesthesia in phantom limbs induced with mirrors. *Proc. R. Soc. Lond.* B **263**, 377–386.

Ramachandran, V. S., Rogers-Ramachandran, D. & Stewart, M. 1992 Perceptual correlates of massive cortical reorganization. *Science* **258**, 1159–1160.

Rizzolatti, G., Riggio, L., Dascola, I. & Umilta, C. 1987 Reorienting attention across the horizontal and vertical meridians—evidence in favour of a premotor theory of attention. *Neuropsychologia* **25**, 31–40.

Rizzolatti, G., Gentilucci, M., Camarda, R. M., Gallese, V., Luppino, G., Matelli, M. & Fogassi, L. 1990 Neurons relating

to reaching–grasping arm movements in the rostral part of area 6 (area 6a beta). *Exp. Brain Res.* **82**, 337–350.

Rizzolatti, G., Fogassi, L. & Gallese, V. 1997 Parietal cortex: from sight to action. *Curr. Opin. Neurobiol.* **7**, 562–567.

Rossetti, Y. 1998 Implicit short-lived motor representations of space in brain damaged and healthy subjects. *Conscious. Cogn.* **7**, 520–558.

Rothwell, J. C., Traub, M. M., Day, B. L., Obeso, J. A., Thomas, P. K. & Marsden, C. D. 1982 Manual motor performance in a deafferented man. *Brain* **105**, 515–542.

Rushworth, M., Nixon, P. D. & Passingham, R. E. 1997 Parietal cortex and movement. I. Movement selection and reaching. *Exp. Brain Res.* **117**, 292–310.

Schultz, T. R., Wells, D. & Sarda, M. 1980 The development of the ability to distinguish intended actions from mistakes, reflexes and passive movements. *Br. J. Soc. Clin. Psychol.* **19**, 301–310.

Shallice, T. 1988 *From neuropsychology to mental structure.* Cambridge University Press.

Sirigu, A., Duhamel, J. R., Cohen, L., Pillon, B., Dubois, B. & Agid, Y. 1996 The mental representation of hand movements after parietal cortex damage. *Science* **273**, 1564–1568.

Sirigu, A., Daprati, E., Pradat-Diehl, P., Franck, N. & Jeannerod, M. 1999 Perception of self-generated movement following left parietal lesion. *Brain* **122**, 1867–1874.

Smith, M. C. 1978 Cognizing the behavior stream: the recognition of intentional action. *Child Dev.* **49**, 736–743.

Spence, S. A. 1996 Free will in the light of neuropsychiatry. *Philosoph. Psychiat. Psychol.* **3**, 75–90.

Spence, S. A., Brooks, D. J., Hirsch, S. R., Liddle, P. F., Meehan, J. & Grasby, P. M. 1997 A PET study of voluntary movement in schizophrenic patients experiencing passivity phenomena (delusions of alien control). *Brain* **120**, 1997–2011.

Sperry, R. W. 1950 Neural basis of the spontaneous optokinetic response produced by visual inversion. *J. Comp. Physiol. Psychol.* **43**, 482–489.

Stephan, K. M., Fink, G. R., Passingham, R. E., Silbersweig, D., Ceballos-Baumann, A. O., Frith, C. D. & Frackowiak, R. S. J. 1995 Functional anatomy of mental representation of upper extremity movements in healthy subjects. *J. Neurophysiol.* **73**, 373–386.

Stirling, J. D., Hellewell, J. S. E. & Quraishi, N. 1998 Self-monitoring dysfunction and the schizophrenic symptoms of alien control. *Psychol. Med.* **28**, 675–683.

Sunderland, S. 1978 *Nerves and nerve injuries*, 2nd edn. Edinburgh, UK: Churchill Livingstone.

Vallar, G. & Perani, D. 1986 The anatomy of unilateral neglect after right hemisphere stroke lesions. A clinical/CT-scan correlation study in man. *Neuropsychologia* **24**, 609–622.

Vetter, P., Goodbody, S. J. & Wolpert, D. M. 1999 Evidence for an eye-centred representation of the visuomotor map. *J. Neurophysiol.* **81**, 935–939.

Vogt, S. 1995 On relations between perceiving, imagining and performing in the learning of cyclical movement sequences. *Br. J. Psychol.* **86**, 191–216.

Von Helmholtz, H. 1886 *Handbuch der Physiologischen Optik.* Leipzig: Voss.

Von Holst, E. & Mittelstaedt, H. 1950 Das Reafferenzprincip (Wechselwirkungen zwischen Zentralnervensystem und Periferie). *Naturwissenschaften* **37**, 464–476.

Vuilleumier, P., Reverdin, A. & Landis, T. 1997 Four legs—illusory reduplication of the lower limbs after bilateral parietal lobe damage. *Arch. Neurol.* **54**, 1543–1547.

Weiller, C., Juptner, M., Fellows, S., Rijntjes, M., Leonhardt, G., Kiebel, S., Muller, S., Diener, H. C. & Thilmann, A. F. 1996 Brain representation of active and passive movement. *NeuroImage* **4**, 105–110.

Weiskrantz, L., Elliot, J. & Darlington, C. 1971 Preliminary observations of tickling oneself. *Nature* **230**, 598–599.

Wegner, D. M. & Wheatley, T. 1999 Apparent mental causation—sources of the experience of will. *Am. Psychol.* **54**, 480–492.

Wolpert, D. M. 1997 Computational approaches to motor control. *Trends Cogn. Sci.* **1**, 209–216.

Wolpert, D. M & Kawato M. 1998 Multiple paired forward and inverse models for motor control. *Neural Net.* **11**, 1317–1329.

Wolpert, D. M., Ghahramani, Z. & Jordan, M. I. 1995 An internal model for sensorimotor integration. *Science* **269**, 1880–1882.

Wolpert, D. M., Goodbody, S. J. & Husain, M. 1998 Maintaining internal representations: the role of the human superior parietal lobe. *Nature Neurosci.* **1**, 529–533.

Yue, G. & Cole, K. J. 1992 Strength increases from the motor program. Comparison of training with maximal voluntary and imagined muscle contractions. *J. Neurophysiol.* **67**, 1114–1123.