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# Possible mechanisms of anosognosia: a defect in self-awareness

Kenneth M. Heilman<sup>1,3\*</sup>, Anna M. Barrett<sup>1,3</sup> and John C. Adair<sup>2,3</sup>

<sup>1</sup>Department of Neurology, University of Florida College of Medicine, PO Box 100236, University of Florida, Gainesville, FL 32610-0236, USA

<sup>2</sup>Department of Neurology, University of New Mexico South, 221 Lomas Boulevard North East, Albuquerque, NM 87131, USA

<sup>3</sup>Neurology Service, Department of Veterans Affairs, Medical Center, Albuquerque, NM, USA

Anosognosia of hemiplegia is of interest for both pragmatic and theoretical reasons. We discuss several neuropsychological theories that have been proposed to explain this deficit. Although for psychological reasons people might deny deficits, the denial hypothesis cannot account for the hemispheric asymmetries associated with this disorder and cannot explain why some patients might deny one deficit and recognize another equally disabling deficit. There is some evidence that faulty feedback from sensory deficits, spatial neglect and asomatognosia might be responsible for anosognosia in some patients. However, these feedback hypotheses cannot account for anosognosia in all patients. Although the hemispheric disconnection hypothesis is appealing, disconnection is probably only a rare cause of this disorder. The feedforward intentional theory of anosognosia suggests that the discovery of weakness is dependent on attempted action and some patients might have anosognosia because they do not attempt to move. We present evidence that supports this theory. The presence of one mechanism of anosognosia, however, does not preclude the possibility that other mechanisms might also be working to produce this disorder. Although a large population study needs to be performed, we suspect that anosognosia might be caused by several of the mechanisms that we have discussed.

On the basis of the studies of impaired corporeal self-awareness that we have reviewed, we can infer that normal self-awareness is dependent on several parallel processes. One must have sensory feedback and the ability to attend to both one's body and the space where parts of the body may be positioned or acting. One must develop a representation of the body, and this representation must be continuously modified by expectations (feedforward) and knowledge of results (feedback).

**Keywords:** anosognosia; self-awareness; unawareness; denial; feedback; neglect

## 1. INTRODUCTION

Anosognosia (*a*, without; *noso*, disease; *gnosia*, knowledge) was a term coined by Babinski (1914) to denote the loss of recognition or awareness of a hemiplegia. Subsequently, unawareness of other forms of illness were described. Patients with blindness, especially from cortical lesions, might be unaware that they are blind (Anton 1896); patients with a hemianopia might be unaware of their limited vision. Patients with aphasia and other cognitive deficits might be unaware of these deficits, and when these patients make speech errors they might not attempt to correct these errors. Patients with memory loss associated with either Korsakoff's syndrome or basal fore-brain lesions might be unaware of their memory loss.

Anosognosia might not be a trivial problem. Failure to detect illness often delays medical care, and some of the new therapies for stroke make early intervention important. In addition, it is difficult to compensate for an illness when one is unaware of it (Bisiach & Geminiani 1991), and rehabilitation in the absence of recognition might not be successful (McGlynn & Schacter 1989).

Unawareness of illness might also preclude patients from recognizing their disabilities and avoiding potentially dangerous activities. For example, we had a patient who had a left hemianopia and left-sided neglect. The patient worked in a factory where a crane would carry steel beams along the ceiling. Although the patient was advised against going back to work, he returned to his job. Because he was unaware of his deficits he sustained a serious injury when a steel beam carried by this crane hit him on the left side of his head. Studying brain-damaged patients with unawareness of their illness might also provide insight into the neuropsychological mechanisms underlying consciousness awareness of self.

Critchley (1953) used the term anosodiaphoria to describe patients who verbally acknowledge that they are ill or have a disability but who seem to be unconcerned about their disability. Anosodiaphoria might be a milder form of anosognosia in which patients have partial awareness. Partial awareness might be related to being repeatedly told that one is impaired, rather than to self-discovery, or might be related to the flattened expression of affect associated with brain injury (see Heilman *et al.* 1993a). Although there might be many forms and degrees of unawareness, this discussion will focus on that which

\*Author for correspondence ([heilman@medicine.ufl.edu](mailto:heilman@medicine.ufl.edu)).

Babinski called anosognosia, an explicit unawareness of hemiplegia.

## 2. PSYCHOLOGICAL DENIAL

Perhaps more than any other investigators, Weinstein & Kahn (1955) in their book *Denial of illness* generated interest in anosognosia. Weinstein & Kahn asserted that the various forms of anosognosia are not discrete entities that can be localized in different areas of the brain. 'Whether a lesion involves the frontal or parietal lobe determines the disability that may be denied, not the mechanism of denial' (Weinstein & Kahn 1955). According to Weinstein & Kahn the motivation to deny illness and disability exists in everyone; they postulated that unawareness of disease, including hemiplegia, was psychologically motivated denial, an unconscious defence mechanism that attenuates the potential distress of a catastrophic event such as hemiplegia. Weinstein & Kahn tested the denial hypothesis by ascertaining each stroke patient's premorbid personality characteristics from relatives and close associates. They found that before their illness, patients who demonstrated anosognosia used denial as a coping strategy more frequently than did patients who were aware of their deficits.

Although there might be patients who use denial as a psychological defence mechanism, the denial postulate is not supported by clinical observations. The coping strategy that one uses in life should not influence which side of the brain becomes damaged by stroke. The finding that anosognosia is more commonly associated with dysfunction in the right hemisphere than in the left might be critical in testing the coping–denial hypothesis.

Babinski's cases had left hemiplegia from right-hemisphere lesions. Although many clinicians attribute unawareness of hemiplegia primarily to right-sided lesions, according to Babinski the diagnosis of anosognosia requires a verbal, explicit denial of illness. Because patients with a right hemiplegia from left-hemisphere lesions frequently have an accompanying aphasia, the frequency of anosognosia from left-hemisphere lesions might have been underestimated. In a recent review of anosognosia related to hemiplegia, Bisiach & Geminiani (1991) stated, 'the issue of right–left differences and the incidence of unawareness of hemiplegia and hemianopsia is a thorny one . . .'.

Hemispheric functions can be studied in patients undergoing intracarotid barbiturate procedures also called the Wada test. During these procedures, in our laboratory, a short-acting barbiturate (Brevital) is injected sequentially into each internal carotid artery. The barbiturate anaesthetizes either the right or the left cerebral hemisphere independently. This technique is used in preoperative evaluation of patients who are being considered for resective surgery for the treatment of epilepsy. This procedure is used to determine the cerebral dominance for language and memory. Terzian (1964) was among the first to report the behavioural changes occurring with Wada testing. However, he reported that at the end of the test the subjects did not remember their neurological deficits regardless of the side of injection. In a pilot project we studied eight consecutive patients who had Wada testing with methohexital (Brevital) as part of their

pre-surgical evaluations for intractable epilepsy (Gilmore *et al.* 1992). All eight patients developed a contralateral hemiparesis within 5 s of injection to either the right or the left carotid artery. This hemiparesis was associated with contralateral moderate-amplitude slowing to delta frequencies in their EEGs. All eight patients became globally aphasic with left carotid injection. This global aphasia persisted for 1.5–3 min with each injection. Although all patients developed a right hemiplegia with left-sided injections, after the subjects recovered from their aphasia and hemiparesis, all subjects were aware that they had a hemiparesis. After right-hemisphere injections, however, none of the eight patients recalled their hemiplegia. No patient experienced loss of consciousness or transitory confusion. Dywan *et al.* (1995) could not replicate our findings. We remain uncertain why our findings differ from those of Terzian (1964) and Dywan *et al.* (1995); perhaps it is because we used a shorter-acting barbiturate. Discrepant results might also reflect different criteria for, or a different definition of, anosognosia. In any case, there have been several other studies, using the amobarbital infusion, that were able to replicate our findings of hemispheric asymmetries (Carpenter *et al.* 1995; Durkin *et al.* 1994). Perhaps most importantly, our findings are consistent with the literature on strokes (see Starkstein *et al.* 1992; Stone *et al.* 1992) and support the hypothesis that unawareness of hemiplegia is primarily associated with right-hemisphere dysfunction.

Although the marked hemisphere asymmetries reported by Gilmore *et al.* (1992) seem to refute the denial hypothesis, there is an alternative explanation of these findings. Whereas after selective left-hemispheric anaesthesia the subjects recalled being weak, after right-hemisphere anaesthesia they did not recall being weak. Therefore a selective amnesia might have been induced by right-hemisphere anaesthesia. To test this selective amnesia hypothesis, Adair *et al.* (1995) assessed the occurrence of anosognosia for hemiplegia during and after right-hemisphere anaesthesia and after left-hemisphere anaesthesia. The proportion of subjects with anosognosia during anaesthesia was the same as the proportion of subjects who had anosognosia of hemiplegia after anaesthesia. These results suggest that the anosognosia we reported after anaesthesia is not related to a selective amnesia. In addition, although we observed subjects who had anosognosia with left-hemisphere anaesthesia, we again demonstrated that anosognosia was more common with right-hemisphere than with left-hemisphere anaesthesia.

In our pilot and follow-up anosognosia studies, each patient served as his or her own control, and there were right–left asymmetries. The premorbid personality–denial hypothesis of Weinstein & Kahn (1955) cannot account for the observation that these patients were primarily anosognosic with right-hemisphere dysfunction. There is a second observation during Wada testing in our laboratory that also does not support the psychological denial hypothesis. Most people who prefer to use their right hand to perform skilled tasks are left-hemisphere dominant for speech and language. Therefore selective anaesthesia of the left hemisphere might induce both a right hemiplegia and impaired speech or aphasia. If Weinstein and Kahn's postulate was correct, we would expect that if

a person denies a hemiplegia he or she should also deny aphasia and vice versa. Breier *et al.* (1995) studied a group of patients who had selective anaesthesia of their left hemisphere. Some patients were unaware of their hemiplegia but were aware of their aphasia. This dissociation of anosognosia concurs with observations made in patients with focal brain lesions (see Prigatano & Schacter 1991). Other patients were aware of their hemiplegia but unaware of their aphasia. If patients were using denial as a defence mechanism, we would expect them to be unaware of both disorders. However, these observations do not preclude the possibility that pre-morbid personality might influence the severity of anosognosia.

Starkstein *et al.* (1992) demonstrated that the presence of anosognosia did not prevent patients with hemispheric lesions from developing depression. Although the depression observed by these investigators might have been a direct result of their brain injury, their observations suggest that if denial is a defence mechanism, it might be inadequate to prevent depression.

### 3. CONFUSION AND EMOTION

A second hypothesis that has been used to explain anosognosia is that these patients are confused (Hecaen & Albert 1978). Confusion has been attributed to right-hemisphere dysfunction (Mesulam *et al.* 1976). Cognitive functions are difficult to test in patients who are confused. It has also been noted that patients with right-hemisphere dysfunction have a flattened affect, whereas those with left-hemisphere dysfunction are often depressed and demonstrate a catastrophic reaction (Gainotti 1972). The right hemisphere seems to be dominant for the expression of emotions (see Heilman *et al.* (1993a) for a review). Therefore patients with right-hemisphere lesions might also be impaired in expressing their emotions. Because patients with right-hemisphere disease have a flattened affect or are even euphoric and might be impaired at expressing emotions, they might seem to be unconcerned about a motor disability, whereas those with left-hemisphere dysfunction, because of their depression, might show greater alarm or concern toward their disabilities. However, we asked our Wada test subjects about their hemiparesis after hemispheric anaesthesia had resolved. Because at the time of testing our patients were not confused and did not have emotional changes, our findings are also not compatible with these explanations of anosognosia.

### 4. IMPAIRED FEEDBACK

#### (a) *Sensory deficits and hemispacial neglect*

The finding that unawareness of hemiplegia and the unawareness of aphasia seemed to be dissociable suggests that awareness of dysfunction is mediated by modular systems and that there might not be a single explanation that can account for the varieties of anosognosia seen in the clinic. One reason that patients with anosognosia for hemiplegia might be unaware of their hemiplegia is that they do not get the sensory feedback that the limb is weak (Levine *et al.* 1991). There are at least two modalities that one uses to get feedback that a limb is not properly

working: somatosensory and visual. We have examined several subjects during right-hemisphere anaesthesia by putting a number on their left hand and bringing this hand into the subject's right body and head hemispace, as well as into the right visual field. To make certain that they visualized their left hand, we asked them to identify the number on the hand. After naming the correct number, we asked the subject if their hand was weak. Some subjects, upon seeing their hand and being asked if that hand was weak, attempted to move their left hand and discovered that their hand was weak. It is possible that, in these subjects, sensory defects might have contributed to their unawareness. However, even in the presence of a hemianopia and a somatosensory deficit, if subjects explored the left side of their body while they were trying to move their hand, they might have discovered that their hand was weak. Therefore sensory loss in the absence of the type of exploratory defect seen with hemispacial or unilateral neglect (neglect) might be insufficient to explain the failure to be aware of a deficit. We did not test our subjects for neglect in this experiment. However, it has been well established that neglect is more commonly associated with right-hemispheric than with left-hemispheric dysfunction (see Heilman *et al.* (1993b) for a review). Although unilateral spatial neglect with or without sensory loss might have accounted for anosognosia observed in some of our patients, most subjects remained unaware of their hand weakness even when they were able to see their left hand on the right side.

#### (b) *Asomatognosia: personal neglect*

There are several possible reasons that these subjects remained anosognosic when their hand was brought into their ipsilesional visual field and hemispace. One possibility is that they failed to recognize that the hand with the number on it was their own hand. To learn whether patients undergoing hemisphere anaesthesia had personal neglect or asomatognosia (unawareness of one's own body parts) of their contralateral forelimb, we performed the following procedure. After the barbiturate injection and the onset of a hemiparesis, the examiner either moved one of the subject's hands into a restricted viewing space, or, while the examiner moved the subject's hands outside the range of vision, the examiner put his or her own hand into this same restricted viewing space. To make the hands look similar we selected an examiner who was the same sex and race as the patient and of approximately the same age. After a hand was presented, the subject was asked, on multiple randomly ordered trials, if the hand that they were viewing was their own hand or that of the examiner. Whereas there were some subjects with anosognosia who did not recognize their own hand (asomatognosia or personal neglect), the majority of subjects with anosognosia accurately recognized their hand (Adair *et al.* 1995). Bisiach *et al.* (1986) studied right-hemisphere-damaged stroke patients and also noted that there were patients without asomatognosia or personal neglect, as determined by their reaching for their paretic left hand on command, who demonstrated anosognosia. These results suggest that, although there are some subjects whose anosognosia might be related to asomatognosia or personal neglect, in most patients anosognosia cannot be accounted for entirely by asomatognosia.

Roth (1949) posited that the brain stores memories or representations of the body and that these representations are stored in the parietal lobe. Therefore parietal lobe injury might induce asomatognosia. Asomatognosia might also be related to an attentional deficit such that patients do not attend a portion of their body (Mark & Heilman 1990). Alternatively, abnormal attention might be responsible for positive symptoms whereby brain dysfunction leads to misidentification of movement planning as movement experienced.

## 5. PHANTOM LIMB MOVEMENT

We saw a patient with a large right-hemispheric stroke who told us that when he attempted to move his arm, he had the feeling that his arm moved. We termed this phenomena 'phantom limb movement'. The patient who told us about the phantom movement did not have anosognosia. However, it is possible that some subjects do not report that their arm is paralysed because they actually feel the limb moving, and unless they inspect their arm they might be unaware that they are weak. We are currently studying the relationships between phantom movement and anosognosia.

## 6. CONFABULATION

Confabulation is the production of responses that are not rooted in reality. Fabricated responses can be either spontaneous or in response to a question. In our discussion, confabulation refers to fabricated responses that are generated from neurological impairments rather than a desire to mislead the examiner. Feinberg *et al.* (1994) studied the relationship between anosognosia and confabulation. Although anosognosia can be considered a form of confabulation, these investigators wanted to learn whether patients with anosognosia have a propensity to confabulate in other domains. These investigators tested hemiparetic subjects, with and without awareness of their motor deficit, on their ability to identify stimuli presented in the contralesional visual field. They found that the subjects with anosognosia were more likely to make a confabulatory identification of an object than those without anosognosia. In our laboratory, we wanted to study the relationship between confabulation and anosognosia by using the Wada test to induce hemispheric dysfunction (Lu *et al.* 1997). We studied 17 patients with intractable epilepsy who were being evaluated for surgery by Wada testing. Test stimuli were three different textured materials (sandpaper, metal and cloth) or no stimulus. After barbiturate injection and a contralateral hemiparesis, different tactile stimuli were randomly applied or not applied to the subject's finger tips. These materials were also placed on response cards. In addition to these three materials, these response cards also contained a question mark to indicate uncertainty of the perception and a blank square to indicate that there was no stimulus. The subjects were never allowed to see the textured stimuli that touched their fingers. After the subjects were either touched or not touched with a tactile stimulus, the response cards were presented on the side ipsilateral to the dysfunctional hemisphere, and the subjects were asked to point to the correct texture or to the blank if they were

not touched. If they were uncertain, they were to point to the portion of the card with the question mark. The order of presentation of the stimulus and no-stimulus trials was randomized. Subjects were fully trained on this task before the Wada study and performed flawlessly. The types of response were classified as confabulation (e.g. pointing to a texture during a no-touch trial), failure to perceive (e.g. pointing to the blank or question mark when touched with a textured material), and correct. We found no significant relationship between anosognosia and confabulation on this test. Therefore there were subjects who were aware of their hemiparesis but confabulated what they had felt, and there were other subjects who were anosognosic but did not confabulate on this tactile task. Although there were some subjects who did have anosognosia and who did confabulate, the brain mechanisms that induce confabulation have not been elucidated. In the next section we discuss the disconnection hypothesis, which is one of the possible explanations of both anosognosia and confabulation.

## 7. DISCONNECTION

Geschwind (1965) posited that anosognosia is related to hemispheric disconnection. According to the disconnection hypothesis, right-hemisphere lesions might not only destroy sensory monitors but, unlike left-hemisphere lesions, right-hemisphere lesions might also disconnect these sensory monitors from the left hemisphere, which mediates speech and language. Without veridical information from the right brain, the undamaged left hemisphere, or what Geschwind termed the 'eloquent brain', fabricates responses to questions regarding the functions subserved by the injured right hemisphere. Therefore, when questioned about a hemiparesis, the disconnected left hemisphere might confabulate a response. Geschwind's disconnection postulate is attractive because not only can it help to explain anosognosia but, because in over 90% of the population the left hemisphere is dominant for language, the disconnection hypothesis might also explain right-left asymmetries. Bisiach *et al.* (1986) argued against the disconnection hypothesis because patients who verbally deny their deficit should retain the capacity to express their deficits non-verbally, and dissociations between verbal and non-verbal expression of deficits have not been reported. Ramachandran (1995) tested patients with anosognosia for hemiplegia to learn whether they had 'tacit, or non-verbal, knowledge of their hemiplegia by giving them a choice of a unimanual or bimanual task. In 17 out of 18 trials the subjects chose the bimanual task. Although results suggest that these patients did not have 'tacit' knowledge and the disconnection theory would predict that they should, both verbal and pointing responses by the right (non-paretic) hand are mediated by the left hemisphere. We therefore wished to perform a more direct test of the disconnection hypothesis. If the disconnection hypothesis is correct, supplying the left hemisphere with information that the left hand is weak should modify the patient's appreciation of the deficit. As we discussed, moving the paretic left hand into the right head/body space and into the right visual field, so that the left hemisphere could be made aware of the left arm, helped only a minority of

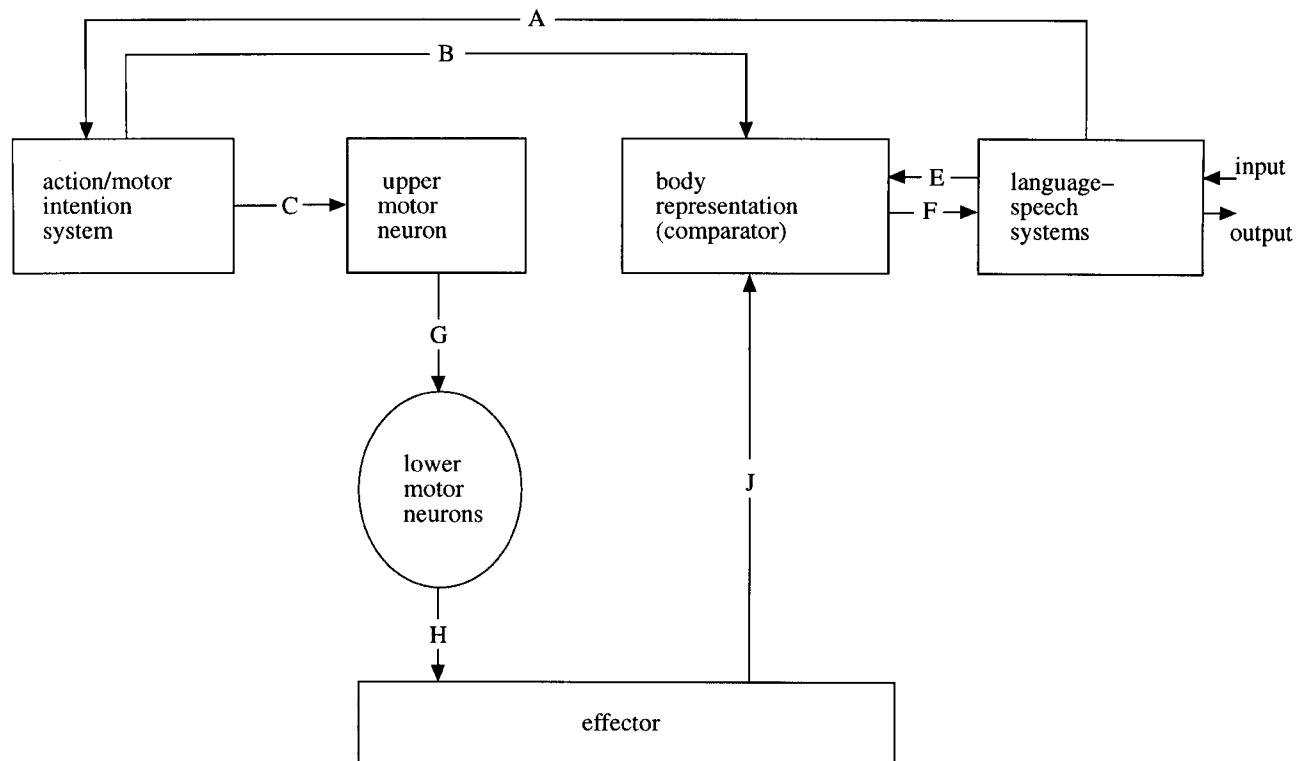


Figure 1. Normally when one is given a command to move an arm, this command is decoded by the speech–language systems that activate the motor action–intentional systems, which include the premotor systems. These premotor systems not only activate the motor system (including the upper motor neurons, the corticospinal tract, the lower motor neurons and the effector), but also activate a representation of how the body (arm) position will change (body representation). When the arm moves, the afferent neurons feed back to the comparator (body representation) that a movement has taken place. However, if there is a defect in the motor system (upper motor neuron, corticospinal tract, lower motor neurons, motor roots, motor nerves, the myoneural junction or the muscle), the feedback does not match the representational set and the person becomes aware of his or her weakness. According to this model, anosognosia might be associated with several deficits: (1) deficits of the action–intentional system induce a failure to develop expectations of movement (i.e. a failure to set the monitor); (2) feedback failures, such as those induced by deafferentation or neglect (inattention) might prevent the comparator from learning that there was a motor deficit; (3) dysfunction of comparator would not only induce anosognosia (personal neglect) but also an inability to detect a mismatch between the expected and implemented movement; (4) a disconnection between the comparator or body representation and the left hemisphere’s language speech areas might produce verbal confabulation.

subjects (5 out of 15) to discover their weakness (Adair *et al.* 1997). Although anosognosia of hemiplegia is more common with right-hemispheric than left-hemispheric dysfunction, as we mentioned above, some subjects with left-hemispheric anaesthesia were also unaware of their hemiplegia and this unawareness could not be accounted for by hemispheric disconnection. However, when the paretic left hand was moved to a position where that hand could be seen by the left hemisphere, some subjects did discover they were weak. Although the discovery of weakness might have been related to bypassing hemispheric disconnection, discovery of weakness when the hand was viewed on the right could have also been related to the neglect-induced faulty feedback that we have discussed. There is a third possible explanation of why some subjects discovered their weakness with this procedure. That patients only discovered their weakness after they were asked to move might suggest that these patient’s anosognosia was related to a motor activation or intentional feedforward deficit. The motor activation-intentional mechanism that could account for anosognosia (the feedforward hypothesis) as well as the supporting evidence will be discussed in the next section.

## 8. FEEDFORWARD

Because confusion, confabulation, disconnection, psychological denial and defective feedback could not entirely explain anosognosia in all subjects, Heilman (1991) proposed a feedforward theory of anosognosia. The feedforward theory deals with expectations. In general, one can recognize failure only if one has expectations. In regard to anosognosia, if one does expect movement but does not detect movement, why should one recognize that they failed to move? For example, if your left arm were totally relaxed at your side, so that you did not attempt to move this arm, and a physician asked you whether your arm was weak you would probably say, ‘no, it is not weak’. However, if, unknown to you, just before being asked whether your arm was weak, your brachial plexus was painlessly injured by an invisible and silent beam of radiation, you would be anosognosic. The primary means of learning that your arm was weak would be by attempting to move this arm. As long as your arm remained totally relaxed at your side, you would presumably never be able to discover the effects of the injury. Therefore recognition of a disability might rely, in part,

on comparator functions. A model of the proposed comparator system can be found in figure 1.

According to this model, weakness is detected when there is a mismatch between the expectancy of movement and the perception of movement. Therefore, if there is a defect in the motor system (e.g. corticospinal system or motor units) and one attempts a movement, the hypothetical monitor-comparator notes the discrepancy between the expected movement and what is observed. A patient who is weak thereby becomes aware that he or she is weak and can explicitly describe the weakness. However, if a patient has an intentional motor activation deficit and therefore does not intend to move or prepare to move, as seen with akinesia, then the monitor-comparator is not set for the expectation of movement. When there is a subsequent failure of movement, with or without motor system dysfunction, there is no mismatch generated and thus no recognition of a disability.

To test this motor-intentional expectancy model, Gold *et al.* (1994) tested motor intention by measuring the activation of proximal muscles (pectoralis majores) while subjects squeezed a dynamometer with each hand. The subjects included normal controls, patients with hemiparesis who were without anosognosia, a patient with neglect, a patient with resolved anosognosia and a patient with anosognosia for a left hemiparesis. When the patient with anosognosia squeezed with his normal ipsilesional hand, both the right and left pectoralis contracted. However, when asked to squeeze with his contralesional paretic hand, he did not contract either pectoralis muscle. The normal controls, the patients with hemiplegia who were not anosognosic, the recovered anosognosic and the patient with neglect all contracted both pectoral muscles when asked to squeeze with either the right or left hands. These results suggest that anosognosia for hemiplegia is associated with a loss of motor intention, results that lend support to the feedforward hypothesis.

Additional support for the intention hypothesis comes from the study of Adair *et al.* (1997), examining the disconnection hypothesis. They examined 17 patients with intractable epilepsy during Wada testing. In this study, awareness of deficit was assessed in three stages after the induction of a hemiparesis by selective hemispheric anaesthesia. First, while the extremity remained on the paretic side, the subjects were asked if they felt weak. Second, if they were anosognosic, the arm was moved to the centre of their body but the hand was placed in a position where special viewing goggles prevented the subjects from seeing their hand. The examiner put his fingers in the subject's paretic hand, asked the subject to squeeze the examiner's fingers, and then asked whether the subject thought they were weak. If the subject still denied being weak, the hand was brought to a position in right body/head hemisphere and right visual field where the subject could view his or her hand. To be certain that the hand was seen by the left hemisphere the subject was asked to read the number in the hand and then to squeeze the examiner's fingers. After seeing his or her own hand while attempting to squeeze the examiner's fingers, the subject was again asked if the hand was weak. Of the total 17 subjects tested, two were without anosognosia. Of the 15 subjects with anosognosia, 11 subjects had anosognosia in

all three stages. Of the four subjects who discovered their weakness, three discovered the weakness after attempting to squeeze the examiner's hand without visual feedback and only one discovered the weakness after both attempting to squeeze the examiner's hand and watching the attempt. The observation that asking patients to move helped them recognize their weakness supports the postulate that these patient's anosognosia might have been related to a motor-intentional deficit.

It is unclear why the other subjects who were asked to move did not become aware of their hemiparesis. It is possible that the examiner's instructions were insufficient to help the anosognosic subjects to overcome the intentional deficit.

The intentional deficit of limb akinesia or motor neglect have been described with both dorsolateral (Brodmann's areas 6 and 8) and medial frontal lesions (supplementary motor area and cingulate gyrus), and with lesions of the inferior parietal lobe, the thalamus (ventrolateral, anterior lateral and medial) and basal ganglia (striatum and substantia nigra). For a full discussion of these motor intentional systems see Heilman *et al.* (1993b). Starkstein *et al.* (1992) studied a large population of patients who had anosognosia associated with a stroke. They found that lesions commonly involved the regions that comprise this intentional network, including the inferior parietal lobe, the basal ganglia, the thalamus, and in the frontal white matter.

The motor intentional hypothesis of anosognosia for hemiplegia might also help to account for the right-hemisphere predominance of anosognosia. Coslett & Heilman (1989) demonstrated that limb akinesia or motor neglect was more often associated with right-hemispheric than left-hemispheric lesions. Warning stimuli might reduce reaction times because the warning stimuli prepare the brain to act. Using a reaction-time paradigm, Heilman & Van Den Abell (1979) demonstrated that warning stimuli directed to the right hemisphere reduced the reaction time for both hands but warning stimuli directed to the left hemisphere primarily reduced the reaction time of the right hand. These results suggest that in normal subjects the right-hemisphere intentional systems can help activate the motor systems for both the right and left hands. However, the left hemisphere's intentional system primarily activates the right hand. Therefore with left-hemisphere injury there might be less intentional deficit because the right hemisphere can compensate for the injured left hemisphere. However, with right-hemisphere injury, the left hemisphere cannot compensate for the right and results in a motor-intentional deficit.

In their book *Denial of illness*, Weinstein & Kahn (1955) described a patient with anosognosia for hemiplegia who was asked why she was not using her left arm if she was not weak. The patient responded that her arm was 'lazy'. Laziness is not a psychological defence mechanism and is not caused by disconnection or a failure of feedback. Laziness is a symptom of a defective intentional system.

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