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Chapter 13

Anosognosia and denial after right hemisphere stroke

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Anosognosia: general presentation

Anosognosia is a puzzling condition. It refers to patients suffering from various neurological impairments who deny or lack awareness of their deficits, although these deficits may have serious consequences in their everyday life. Studying anosognosia is important for two reasons. The first is practical. Indeed, as pointed out by Heilman [1], anosognosia is not a trivial problem. It may impair patients' motivation to engage in a rehabilitation program, and anosognosia has repeatedly been found associated with poor functional recovery from stroke. Second, studying anosognosia is also of great theoretical importance, as it may help in understanding the complex relationships between brain injury and various forms of consciousness. Its pathophysiology is still poorly understood, but recent experimental studies provided new insights into the cognitive mechanisms underlying anosognosia for hemiplegia [2,3].

Two detailed historical reviews of anosognosia [4,5] outline the first observations of patients with impaired awareness of neurological deficits in the late nineteen century, by von Monakow in 1885 and by Anton in 1889 for cortical blindness. It seems that Pick, in 1898, was actually the first to report unawareness of left hemiplegia, but it was Joseph Babinski who first coined the term "anosognosia," and his contribution, at a meeting held by the French Neurological Society on June 11, 1914, remains a landmark in the field [6].

Anosognosia may be associated with many disorders. Perhaps the most dramatic is unawareness of hemiplegia. However, there are many other defects of which patients may be unaware. These include cortical blindness, hemianopia, cortical deafness, aphasia, memory loss, and the dysexecutive syndrome [7]. Whether common neuropsychological mechanisms produce anosognosia for different types of deficit remains unknown. This chapter will focus on anosognosia associated with right hemisphere stroke, that is mainly anosognosia for hemiplegia, and/or hemianopia.

Anosognosia after right hemisphere stroke: clinical features

Babinski [6], in his seminal report, described a patient who, although "her intellectual and affective functions seemed grossly preserved … seemed to ignore the existence of a nearly complete hemiplegia …. She never complained of it, she even never mentioned it. … When asked to move her left arm, she remained immobile, silent, just as if the request concerned someone else." Babinski also introduced the term "anosodiaphoria" to refer to some hemiplegic patients who, without ignoring their hemiplegia, seemed indifferent and did not appear to be affected by their paralysis [6]. Other clinical manifestations have been described since, such as misoplegia [8], where patients express hatred toward the contralesional limb, or somatoparaphrenia [9], corresponding to a variety of bodily delusions where patients fail to acknowledge ownership of their own contralesional limbs, attribute them to someone else or treat the limb(s) as a separate person or object.

Anosognosia is a complex disorder and surprising dissociations and contradictions have been reported in anosognosic patients (see key points box). This leads some authors to distinguish between different types or degrees of anosognosia [2,10,11]. Specificity – the degree to which lack of awareness is restricted to a particular deficit – is an important issue of anosognosia for hemiplegia [10]. For example, an individual

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might not mention his flaccid hemiparesis but complain elaborately about some trivial discomfort of infirmity. One of the first anosognosic patients reported by Babinski [6] complained of an old phlebitis, while being unaware of hemiplegia. Even within the domain of neurological deficits, patients may deny weakness while retaining full awareness of language or visual impairments. In addition, different levels of awareness can be observed within a specific modality regarding different ipsilateral body parts (e.g. some patients acknowledge leg weakness while failing to appreciate their paretic arm). Bisiach *et al.* [12] found that a few patients with severe anosognosia for visual field defect had minimal, if any, anosognosia for motor impairment. In a study of 206 patients with subacute–chronic neglect, Azouvi *et al.* [13] found that anosognosia for hemiplegia, for visual impairments and for neglect behavior in everyday life were weakly correlated, suggesting that they may dissociate one with each other. Marcel *et al.* [10] systematically studied specificity of anosognosia for hemiplegia. They found that unawareness of hemiplegia, somatosensory loss and visual field defect were all doubly dissociated.

Key points Main characteristics of anosognosia

- Denial or lack awareness of a clear neurological deficit
- Concerns deficits in the motor (hemiplegia), visual (hemianopia, cortical blindness), auditory (cortical deafness) or cognitive (aphasia, dysexecutive syndrome) domains
- May be dissociated (anosognosia concerns only one of the multiple deficits of the patient)
- May be associated with misoplegia (patients express hatred toward the contralesional limb) or somatoparaphrenia (bodily delusions)

In addition, anosognosia is not an all-or-none phenomenon. There are various degrees of severity, which may range from uncritical underestimation to explicit, intractable denial of neurological deficits. A distinction has been made between awareness of a deficit, awareness of its consequences, and concurrent awareness of instances of failures entailed by the deficit [10]. Several terms have been used to distinguish between these different degrees of anosognosia, such as complete versus incomplete anosognosia, systematic versus partial denial, or verbal versus behavioral anosognosia [9,14]. Anton (1889; quoted by Bisiach and Geminiani [5]) pointed out that a patient may admit his or her hemiplegia and nonetheless set about walking or knitting. Marcel *et al.* [10] suggested that unawareness of hemiplegia is frequently encountered at the acute phase of stroke, while only unawareness of its consequences commonly persists late after stroke. Moreover, the degree of awareness may wax and wane across the course of a single day. Some patients may temporarily acknowledge their paresis during the demonstration of the motor impairment by the examiner, but again deny their problems just a few minutes later.

In addition, dissociations have been repeatedly reported between explicit verbal awareness and patient's behavior, suggesting that some degree of implicit knowledge may persist in anosognosic patients. Patients may deny any motor impairment but accept to stay in hospital without ever trying to leave their chair on their own. This may be taken as informal evidence of partial knowledge, or as a dissociation of knowledge [5,10,15]. Marcel *et al.* [10] found that some patients who overestimated their current task ability did not overestimate when asked how well the examiner, if he was in their current condition, could do each task. This suggests some form of implicit awareness. Some degree of implicit awareness in anosognosic patients has recently been supported using different experimental paradigms, such as reaction time measures to disease-related words or a sentence completion test with neutral or disease-related sentences [16–18].

Another peculiar characteristic of anosognosia for hemiplegia is the patient's response to confrontation by the examiner. Some anosognosic patients, when asked why they failed to move their paretic limb, claim that they are too tired or lack interest. Even when faced with obvious contradictions between their statements and their actions, they appear indifferent to these discrepancies, and their false belief may persist despite logical arguments and contradictory evidence.

Characteristics of strokes responsible for anosognosia

Incidence of anosognosia after stroke

There is a wide consensus that anosognosia is more frequent after right than after left hemisphere stroke. A selection bias created by the difficulty of testing patients with left brain damage with aphasia cannot

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Table 13.1. Frequency of anosognosia for hemiplegia in a sample of representative studies

	No.	Right brain damage (%)	Left brain damage (%)
Nathanson *et al.* (1952) [21]	100	68	32
Cutting, 1978 [22]	70	58	14
Hier *et al.*, 1983 [(23]	41	36	
Bisiach *et al.*, 1986 [12]	36	33	
Levine *et al.* (1991) [24]	13	46	
Starkstein *et al.*, 1992 [25]	80	51	16
Stone *et al.*, 1993 [26]	171	28	5
Pedersen *et al.*, 1996 [27]	566	36	9
Jehkhonen *et al.*, 2000 [28]	56	7	
Appelros *et al.*, 2002 [29]	279	20	13
Azouvi *et al.*, 2002 [13]	206	17	
Beis *et al.*, 2004 [30]	78		6
Marcel *et al.*, 2004 [10]	64	12–29	0–9
Baier & Karnath, 2005 [31]	128	10	
Cocchini *et al.*, 2009 [32]	33		29
Vocat & Vuilleumier, 2010 [33]	58		
3 days		32	
1 week		18	
6 months		5	

account for this asymmetry, which has also been found in studies using intracarotid barbiturate injection (the Wada procedure) [19,20]. For example, Adair *et al.* [19] questioned subjects both during and 3 minutes after barbiturate injection, and found that 30 of 31 (97%) denied left hemiparesis after right carotid injection, while 15 of 31 (48%) denied right hemiparesis after left carotid injection. The proportion of subjects with anosognosia during or after the procedure was similar.

Studies of anosognosia after stroke yielded similar results. Table 13.1 shows the results from several representative studies. Differences in the reported rate of incidence might be related to different times and/or different methods of assessment. In some studies, anosognosia was assessed on unselected patients at the acute stage of stroke. Stone *et al.* [26] studied 171 consecutive patients at 2–3 days after stroke and found an incidence rate of anosognosia of 28% after right hemisphere stroke and 5% after left hemisphere stroke. Interestingly, they also found an asymmetric pattern for anosodiaphoria (27% versus 2%, respectively). Similar findings were reported by Pedersen *et al.* [27], who used a standardized questionnaire [12] to assess 566 consecutive, unselected patients with acute stroke included in a large community-based study (the Copenhagen Stroke Study). Incidence of anosognosia after right and left hemisphere stroke was 36% and 9%, respectively. A somewhat lower incidence was reported more recently by Appelros *et al.* [29,34], who studied 349 patients with acute stroke (1–4 days post-stroke) in a population-based stroke incidence study. Out of 276 (79%) patients able to complete an anosognosia questionnaire [25], 48 (17%) showed signs of anosognosia: 26 (out of 126; 20.6%) after right hemisphere damage, 19 (out of 146; 13%) after left hemisphere damage, and 3 after unknown or bilateral damage.

Studies on patients with subacute/chronic stroke also found a right hemisphere prevalence for anosognosia. A French multicenter study included patients with subacute stroke in rehabilitation units, after a right (n = 206) or a left (n = 78) hemisphere stroke [13,30]. The questionnaire devised by Bisiach *et al.* [12] was used to assess anosognosia for motor and for visual impairments. Anosognosia for hemiplegia was found in 17% after right hemisphere stroke and 6% after left hemisphere stroke. Anosognosia for visual impairments was more frequent, but also with a marked right–left asymmetry (incidence 46% and 10%, respectively). Marcel *et al.* [10] systematically assessed different forms of anosognosia in patients with subacute stroke (27 left and 42 right). For all questions, unawareness was more common in the group with right hemisphere damage, but the difference was statistically significant only with respect to anosognosia for arm paresis (29% versus 0%), for touch sensation impairments in the leg (66% versus 19%), and for dependence in activities of daily life (52% versus 14%). However, unawareness of somatosensory loss in the arm and of hemianopia was almost as common in the group with left brain damage as in the group with right.

Cocchini *et al.* [32] recently suggested that the frequency of anosognosia in patients with left brain damage may have been underestimated for methodological reasons. In a group of 33 patients with left hemisphere stroke, they used a new procedure, the Visual Analogue Test for Anosognosia for Motor Impairment, which was less dependent on language than traditional structured interviews. They found a

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much higher frequency of anosognosia (29%) than with a more traditional testing (5%).

A few reviews or meta-analyses have been published [3,35,36]. Pia *et al.* [36], in a meta-analysis, selected four studies published between 1938 and 2001 reporting the prevalence of anosognosia for hemiplegia in relation to the lesion side, with a total of 280 patients. Anosognosia was found in 54.1% after right brain damage and 9.2% after left brain damage, a difference that was highly significant. Jehkonen *et al.* [35], in a review covering the period 1995 to 2005, found that the occurrence of anosognosia after right hemisphere damage ranged from 11 to 60%, while it ranged from 6% to 24% for left hemisphere damage. Baier & Karnath [31] suggested that the wide variation in the incidence rate of anosognosia for hemiplegia in the literature could be explained by the use of different diagnosis criteria. In a sample of 128 patients with acute stroke, the incidence of anosognosia, according to the Bisiach *et al.* [12] scale, was 23%. However, these authors argued that patients who did not mention their paresis spontaneously, but did so when questioned directly about it (grade 1, or "mild anosognosia") were actually more similar to non-anosognosic patients than to the other groups of anosognosic patients, and they recommended that denial grade 1 should not be considered as pathological. Using this restricted diagnosis criteria, the incidence of anosognosia for hemiplegia in their study was only 10%.

Anatomy and stroke topography of anosognosia for hemiplegia

It is difficult to find an agreement between different studies relative to the intrahemispheric localization of lesions associated with anosognosia for hemiplegia. As recently outlined by Pia *et al.* [36], it seems that there is no specific brain area involved in causing the denial behavior. The lesion size seems to be a crucial factor, larger lesions being more likely to be associated with anosognosia [3]. Many different regions, either cortical or subcortical, have been found associated with anosognosia. Bisiach *et al.* [12] found that, for lesions involving the cortical convexity, anosognosia was associated with inferior–posterior parietal damage, while for deep lesions, the more likely anatomical correlates of anosognosia were damage to the thalamus and/or the lenticular nucleus. Quite similar results were reported by Starkstein *et al.* [25]. Other authors emphasized the role of the basal ganglia [37,38]. They found that the majority (70–79%) of anosognosic patients had a lesion involving this region, while this was observed in only 30% of non-anosognosic patients. The size of the lesion may be an important factor to consider. Hier *et al.* [23] found that denial of illness occurred consistently only after larger strokes, a finding similar to that of Levine *et al.* [24]. Pedersen *et al.* [27] also failed to find any particular association of anosognosia with localization of the stroke lesion in any of the cerebral lobes, but did find that anosognosia was associated with larger strokes.

Pia *et al.* [36] conducted a meta-analysis on the anatomy of anosognosia for hemiplegia. When considering lesions restricted to a single cortical structure, anosognosia was equally frequent after damage to frontal, parietal, or temporal lobe. If considering lesions involving more than one lobe, the combination of fronto-parietal and fronto-temporo-parietal lesions seemed to be the most frequent cause of anosognosia. The fronto-parietal combination of lesions was significantly more frequent than all the others. A number of patients (41%) presenting with anosognosia had a lesion involving subcortical structures which was purely subcortical in 44% of cases. In these patients, there was a slight tendency for a more frequent involvement of the basal ganglia. Pia *et al.* [36] concluded that anosognosia could be the consequence of a lesion in a fronto-parietal circuit related to space and motor representation.

Berti *et al.* [39] compared the distribution of brain lesions in patients showing neglect and anosognosia for hemiplegia after right hemisphere damage (n = 17) with those of patients with hemiplegia and neglect but without anosognosia (n = 12). Denial was associated with lesions in areas related to the programming of motor acts, particularly the premotor and motor areas within the frontal lobe and the somatosensory cortex. The authors suggested that monitoring systems (which, if impaired, may produce anosognosia) could be implemented within the same cortical network that is responsible for motor function. Karnath and Baier [40,41] reported different findings. Based on a lesion-mapping study in 27 patients with acute stroke, they emphasized the role of the right posterior insular cortex. The same region was also found associated with disturbed sensation of limb ownership for the paretic limb, such as asomatognosia or somatoparaphrenia [40]. These authors concluded that the right posterior insula is critical for self-awareness

of action and sense of limb ownership. Vocat *et al.* [33] used a voxel-based statistical lesion-mapping method to identify lesions associated with more severe anosognosia and found that damage to the insula (mainly its anterior part) and adjacent subcortical structures was crucial for anosognosia in the early post-stroke period (3 days). However, the persistence of anosognosia in the subacute phase (1 week) was associated with additional lesions in the premotor cortex, cingulate gyrus, parieto-temporal junction, and medial temporal structures. These results suggested that anosognosia for hemiplegia is likely to reflect a multicomponent disorder related to a distributed set of brain regions [33].

Anosognosia is mainly observed after ischemia in the territory of the middle cerebral artery, particularly when it involves the posterior–inferior division. Anosognosia following deep infarct in the territory of the middle cerebral artery has been reported [42,43].

Key points **Main types of stroke associated anosognosia for hemiplegia**

Arterial infarct: middle cerebral artery, posterior–inferior and deep branches, anterior choroidal artery

Hemorrhage: fronto-parietal, lenticular and thalamus

Recovery

Typically, anosognosia for hemiplegia develops immediately after brain injury and resolves within 2 weeks, persisting in less than 10% of the patients. Critchley [44] maintained that anosognosia lasting more than "some days" is a disorder associated with "… psychiatric features, ranging from a mere confusional to an actual demential state." (pp. 232, 234). Hier *et al.* [23,45] found anosognosia at the acute stage in 36% of patients, and the median time to recovery was 11 weeks. More recently, Vocat *et al.* [33] evaluated a prospective cohort of 58 patients with right hemisphere stroke. They found that the frequency of anosognosia decreased with time, from 32% at the very acute stage (3 days), to 18% at 1 week and 5% at 6 months. Interestingly, the determinants of anosognosia, as assessed by means of multiple regressions, differed across these different time points. At the hyperacute phase, proprioceptive loss was the best predictor of anosognosia, while at the subacute phase, spatial neglect and disorientation were more determinant.

Although persisting anosognosia late after stroke is uncommon, a few cases have been reported [46–48]. Several authors have suggested that chronic anosognosia is always associated with intellectual disorders and/or prefrontal dysfunction. However, case reports of chronic anosognosia without overt intellectual and reasoning deficits have been reported. Venneri and Shanks [47] reported the case of a 85-year-old right-handed woman who showed persistent anosognosia for hemiplegia following an hemorrhagic stroke. Anosognosia was associated with left visuospatial neglect, but there were no global reasoning, memory, or language problems, except for poor performance on verbal fluency tasks. The patient confabulated extensively on themes of active excursions and bizarre acts of persecution. A SPECT (single photon emission computed tomography) examination showed an area of hypoperfusion in the right frontal lobe extending beyond the precentral cortex into lateral and orbital associative cortex. Persisting anosognosia was tentatively attributed by the authors to an inability to monitor and check the "real" and particularly to assess the truth of mental contents [47].

Anosognosia as a marker of prognosis after right hemisphere stroke

Clinicians often claim that lack of awareness prevents patients from actively engaging in a rehabilitation program and may be a cause of rehabilitation failure. Early non-systematic observations suggested that left hemiplegic patients have less or slower recovery and less social adjustment than right hemiplegics [49,50]. Several authors have studied the influence of neglect on functional recovery after stroke. However, as outlined by Appelros *et al.* [34], many of these studies did not use multivariate analysis, thus leaving open the intervention of confounding factors. Denes *et al.* [51] found that neglect was the strongest prognostic factor for poor functional recovery in hemiplegia when compared with other cognitive disorders, such as aphasia, intellectual deterioration, or disturbed emotional reactions. This finding has largely been reproduced subsequently by other authors, who showed that neglect has an unfavorable influence upon functional outcome, improvement on rehabilitation, length of hospital stay, and discharge to home [28,52,53]. This remains, nevertheless, a matter of debate, and several recent large and well-designed studies, using multivariate analysis to control for confounding variables,

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Table 13.2. Assessment of anosognosia after right hemisphere stroke

Source	Methodology
Cutting (1978) [22]: standardized questionnaire	Starts with general questions, then focuses on specific impairments; includes assessment of "anosognosic phenomena"
Bisiach *et al.* (1986) [12]: standardized questionnaire	Awareness of motor and visual impairments; score uses a 4-level scale (range: 0–3)
Azouvi *et al.* (2003) [58]: Catherine Bergego Scale	Awareness of behavioral neglect in daily life situations; score based on the difference between therapist's and patient's assessment (range: 0–30)
Prigatano *et al.*, 1990 [59]: Patient Competency Rating Scale	Questionnaire asking the patient and a relative (or staff member) to rate the subject's ability on a variety of practical skills
Anderson and Tranel (1989) [60]: awareness interview	Awareness of various cognitive and motor impairments after brain injury; based on a comparison of examiner's and subject's report; three-level score for each question

found contradictory results. Pedersen *et al.* [54] found no independent influence of neglect on recovery, while two other studies reported that neglect had a significant negative impact on functional outcome after controlling for other confounding variables, such as age or stroke severity [29,34,55].

The influence of anosognosia has been less frequently studied. Gialanella and Mattioli [56] studied patients at 1 and 6 months after stroke. They found that extrapersonal neglect alone was not associated with poor recovery, while patients with neglect and anosognosia had the worst motor and functional outcome. However, they only used univariate analyses. Jehkonen *et al.* [28] reported that hemiparesis and unawareness of illness lengthened the duration of hospital stay, while the presence of a relative reduced it. These three factors were the best predictors of the time from right hemisphere stroke to discharge to home. Sundet *et al.* [57] found the presence of pathological emotional reactions, including denial of illness, to be a more important predictor of self-reported functional status than other cognitive variables in a multivariate analysis. In a large community-based study, Pedersen *et al.* [27] found that, although neglect by itself was not a significant independent predictor of poor recovery, anosognosia did have a profound influence on prognosis. Multivariate analyses showed that, after controlling for the influence of other variables, the presence of anosognosia per se predicted 11.5 points less in discharge Barthel Index, increased the likelihood of death during hospital stay by a factor of 4.4, and reduced the likelihood of discharge to independent living by 0.43 [27]. Appelros *et al.* [29,34], however, found contrasting results in another population-based study. At the acute stage, neglect and anosognosia were both found to influence disability in a multiple logistic regression analysis. Moreover, anosognosia was a stronger predictor than neglect [29]. The level of disability was established in survivors after 1 year, and the authors looked for correlations with cognitive impairments at the acute stage. At that time, only neglect was still significantly associated with dependency [34].

Assessment of anosognosia

Only a few well-standardized and/or semiquantitative assessment methods for anosognosia have been reported, most of which are of unknown reliability (Table 13.2). Some of them have been designed specifically to assess anosognosia after right hemisphere stroke. Cutting [22] was probably one of the first authors to design a standardized anosognosia questionnaire. This questionnaire includes three different levels. The first level uses general questions focusing on awareness of plegia, starting with open questions, such as "Why are you here?" "What is the matter with you?" It then uses more precise terms, such as "Is it weak, paralyzed, or numb ?" Then, if denial has been elicited on general questions, two procedures are used: the examiner picks up the patient's arm and requests the patient to lift his or her arms themselves, while asking questions such as "What is this?" "Can you lift it?" "Can't you see that the two arms are not at the same level?" The third level includes questions addressing anosognosic phenomena, such as anosodiaphoria, non-belongings, misoplegia, and kinesthetic hallucinations.

The scale proposed by Bisiach *et al.* [12] is probably the most widely used. It includes two parts, one related to anosognosia for motor impairment, and one for visual field defect. A four-level score is used. A score of 0 is given if the disorder is spontaneously reported

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by the patient following a general question about the complaints. A score of 1 is given if the disorder is reported only following a specific question about left limbs' strength. If the disorder is acknowledged only after its demonstration by the examiner, the score is 2. A score of 3 corresponds to no acknowledgement at all of the disorder.

A standardized questionnaire has also been designed by Starkstein *et al.* [25], with a four-level score ranging from 0 (normal score, the patient is able to spontaneously report the disorder following a general question) to 3 (severe anosognosia). The Anosognosia for Hemiplegia Questionnaire was developed by Feinberg *et al.* [61]. It includes 10 questions, starting with general questions about the deficit, then focusing on more precise questions on sensations, movements, or opinions related to the affected limbs. Other studies also used various forms of structured interviews or questionnaires addressing different aspects of anosognosia: general questions about health status, awareness of motor impairments, of the consequences of motor impairments, and/or anosognosic phenomena [10,62,63].

Marcel *et al.* [10] proposed a different method based on the comparison of the evaluation (by the patient or by the examiner) of the patient's performance on a motor or verbal task before (prediction) or after the completion of the task. Marcel *et al.* [10] found that 70% of patients at the acute stage overestimated their motor performance with the affected limb before completion of the task, and 50% still overestimated their performance after completion of the task.

The Catherine Bergego Scale [58,64,65] permits an assessment of awareness of neglect behavior in daily life. Neglect behavior is assessed by an examiner from a direct observation of the patient in 10 everyday situations such as grooming, dressing, eating, spatial orientation, or finding personal belongings. A parallel self-assessment version is given to the patient. Scores range from 0 (no neglect) to 3 for each item. An anosognosia score can be computed from the difference between the examiner's score and patient's self-assessment. This anosognosia score has been found significantly correlated with neglect severity [58,65].

Other scales have been designed to assess various forms of unawareness. An awareness interview has been designed by Anderson and Tranel [60] to assess awareness of various cognitive and motor impairments in patients with stroke, head trauma or dementia. It includes seven question related to different problems (reason for hospitalization, motor impairments, thinking ability, orientation, memory, language, and visual perceptual problems) and a post-test question addressing awareness of quality of test performance and ability to return to normal activities. For each question, a three-level score is used. Unawareness was operationally defined as a discrepancy between the subject's description of abilities and measurement of those abilities by the examiner. The Patient Competency Rating Scale, designed by Prigatano and his colleagues [59], is a standardized questionnaire focusing on ability on various practical skills. The subject's scoring is compared with staff or family ratings. This questionnaire is, however, more specifically designed to assess anosognosia after head trauma rather than anosognosia for hemiplegia.

Pathogenesis

The pathogenesis of anosognosia remains unsettled. The role of *concomitant cognitive or neurological disorders* has been emphasized by some investigators, who suggested that disorientation or frontal lobe type deficits could be more frequent in anosognosic patients [22,24,25]. A frontal lobe dysfunction could cause a deficit in reality monitoring. However, global mental confusion or major intellectual disturbances seem insufficient to explain anosognosia. Indeed, there are numerous observations of patients with anosognosia and with normal mentation and orientation. Marcel *et al.* [10] failed to find an association between anosognosia and scores in frontal lobe tests. Moreover, the fractionation of anosognosia into function-specific forms argues against the explanation of anosognosia in terms of general confusion. In a similar way, there does not seem to be any significant relationship of anosognosia with sensory and/or motor loss [10,12,25]. Consequently, it is unlikely that anosognosia could simply result from the lack of direct feedback about the affected limbs' state [2].

The relationship of anosognosia with unilateral neglect represents a more complex issue. Patients with neglect, particularly with personal neglect, lack regard for their contralesional limbs and sometimes fail to acknowledge the existence of their left arm or leg (asomatognosia). Logically, an individual without an adequate concept of half the body might not make reliable judgments about the function of these limbs. The frequent association of neglect and anosognosia for motor loss is a common finding in most studies. For

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example, in the Pedersen *et al.* [27] study, anosognosia was present in 73% of patients with hemineglect and in 6% of those without hemineglect, a difference that was highly significant. Azouvi *et al.* [13] also reported statistically significant, albeit moderate, correlations between three measures of anosognosia and unilateral neglect. However, several studies showed that neglect and anosognosia can be doubly dissociated one from another, thus making unlikely the explanatory power of neglect as a cause of anosognosia. Adair *et al.* [19] tested personal neglect and anosognosia for hemiplegia after selective anesthesia of the right hemisphere using the Wada procedure, and found that both disorders could be dissociated. Bisiach *et al.* [12] found clear examples of dissociation between both personal and extrapersonal neglect and anosognosia for hemiplegia in 97 patients with right hemisphere lesions. Similar findings have been reported by Cutting [22], who observed that, although personal neglect was rare in patients without anosognosia for hemiplegia, it occurred in only one-third of the anosognosic patients. Extrapersonal neglect was more common than personal neglect, but was present in only half of patients with anosognosia. Dauriac-Le Masson *et al.* [66] reported a double dissociation between unilateral neglect and anosognosia in two patients with a subacute right hemisphere stroke. Accordingly, Berti *et al.* [39] reported that 56.7% of their patients showed both anosognosia and neglect, while 40% presented pure neglect and a minority (3.3%) pure anosognosia. These different studies permit the unambiguous conclusion that anosognosia for hemiplegia is not simply a manifestation of inattention to the left side of egocentric space, even though it is frequently associated with neglect.

Psychological motivation can induce denial for severe and/or disabling medical illnesses, such as heart disease, cancer, or AIDS. Refusal to acknowledge illness or putting it out of one's mind is a quite common defense or coping mechanism in such conditions. Healthy people may also show a natural tendency to use denial mechanisms to deal with environmental stress. Weinstein and Kahn [67] studied the premorbid personality of patients with anosognosia and found that anosognosic patients used denial mechanisms before their strokes more frequently than did controls. However, this finding has not been reproduced thereafter, and such mechanisms could not account for the observation that anosognosia for hemiplegia is more frequent after right then left hemisphere stroke. Moreover, several studies found that anosognosic patients do not seem to show a tendency to deny illness of just any kind [5]. Nevertheless, emotional changes have been reported in patients with anosognosia, such as apathy, inappropriate cheerfulness, and depression [22,24,25]. It has been suggested that anosognosia might involve a deficient affective drive to respond to uncertainties about current bodily states or current cognitive abilities [11].

Anosognosia has sometimes tentatively be attributed to a *disconnection mechanism* [68,69]. Within this theory, anosognosia for hemiplegia would be related to the isolation of right hemisphere processes from language centers in the left hemisphere. Anosognosic patients could not verbally report transactions within the injured right hemisphere, a structure lacking direct connection to language centers. Without input from the right hemisphere, the "eloquent" brain "confabulates" a response to questions regarding function subserved by the injured brain. However, such a theory cannot explain why patients who verbally deny their deficit are not able to express deficit awareness non-verbally. Moreover, informal observation indicates that bringing the paralyzed extremity into the right visual field does not enhance recognition of deficit. The disconnection hypothesis has been more recently revisited, and it has been suggested that disconnection of a parietal conscious awareness system from particular input modules may result in specific forms of anosognosia [15,70].

The *discovery theory* has been proposed by Levine *et al.* [24]. This theory rests upon a main premise: deficits are not passively experienced in the same way as other environmental stimuli. In the words of Levine *et al.* [24], neurological deficit is not "phenomenally immediate," creating an ambiguous experience for the patient. Hence, patients must learn of dysfunction through a process of self-observation and inference. Levine *et al.* [24] asserted that two broad forms of deficiency preclude that discovery: sensory loss and some cognitive impairment. Subsequent studies [10] did not support this hypothesis.

The *feedforward theory* was proposed by Heilman [1] to explain deficits in theories based on feedback. Many explanations of anosognosia for hemiplegia viewed the primary problems in terms of inadequate information or "feedback" from the paralyzed limb to a hypothetical monitor of bodily function. This theory had roots in the sensory loss theory and the discovery theory. According to Heilman [1], these theories

cannot explain denial of hemiplegia when the arm is brought into a normal visual and attentional field and the patient is explicitly asked to monitor the arm's function. Heilman [1] suggested then a feedforward or intentional theory based on the fact that, under normal circumstances, the formation of an intention to move generates accurate predictions about the impending sensory feedback. This prediction is based on the efference copy of the programmed motor act (i.e. signals that are sent to sensory and motor structures once a movement is programmed). If an intended movement is not performed as planned, a comparator will detect a mismatch between the predicted sensory feedback and the absence of any actual sensory feedback. According to Heilman and colleagues, anosognosia would be related to a defect of the intention system, which would not be able to set motor intentions. As a consequence, the comparator (although properly functioning) does not receive any information about movement planning. Thus, lack of movement does not create a mismatch between intended and actual movement and so patients are not able to discover that they did not move.

The *motor awareness theory* was proposed in opposition to Heilman and colleagues' motor intention model. In this model, anosognosia for hemiplegia could be related to a dysfunction of the comparator system [39,62]. According to this assumption, patients are able to form motor intentions but are not aware of the discrepancy between their prediction and the actual position. Similar findings have been reported by other authors and have led to the assumption that anosognosia for hemiplegia could be related to a more global deficit of motor awareness [71,72].

The *two-factor theory* was proposed by Aimola-Davies and colleagues [73]. Within this framework, anosognosia is seen as a pathology of belief, similar to delusions. The first factor is a basic neuropsychological or neurological deficit creating an anomalous experience that may answer the question of where did the delusion come from. The second factor should explain why the patient does not subsequently reject the false belief. This second factor is presumably related to impairments of working memory or executive function.

The *ABC model* was proposed by Vuilleumier and colleagues [2,33]. This is a multidimensional model in which the anosognosia may result from a combination of defects in three different processes: appreciation of a deficit, beliefs, and check operations. This framework may account for different subtypes of anosognosia.

In summary, there is, to date, no univocal account for anosognosia. What emerges from the empirical studies is that anosognosia might reflect the impairment of a specific monitoring process rather than being produced by a combination of somatosensory and cognitive deficits. Moreover, anosognosia for hemiplegia seems to be associated with a more global impairment in reality monitoring, beyond the domain of motor control. In addition, as pointed out by Marcel *et al.* [10], there may be more than one kind of awareness and awareness may differ according to its object. Accordingly, anosognosia could be a non-unitary and multifactorial phenomenon.

Therapy

We are not aware of any controlled study of therapy of anosognosia for hemiplegia. When patients have a complete syndrome of impaired self-awareness, it is futile to try to argue with them. Rather, patients should be examined to determine the extent and the nature of their complete syndrome of impaired awareness. Inevitably, these patients will experience a number of failures that they do not understand. Resistance to rehabilitation emerges usually when their syndrome recovers from complete to partial. Therapists should try and evaluate the method of coping the patient is using when partially aware of something wrong.

Caloric vestibular stimulation may produce a temporary remission of unilateral neglect and of anosognosia. Rode *et al.* [74] reported a 69-year-old woman who had sustained a right hemisphere stroke and subsequently presented with a complete hemiplegia and delusions concerning her left hemibody when referred for rehabilitation 6 months later. The patient showed severe extrapersonal and personal neglect as well as a complete anosognosia for both motor and visual deficits. After the first vestibular stimulation, the patient was totally aware of her hemiplegia while unilateral neglect also dramatically improved. The delayed post-stimulation state showed that the anosognosia was less severe as the patient acknowledged that she could not walk like everybody else. This finding has been subsequently reproduced in a group study [75]. However, these effects are short lasting and cannot readily be recommended as a therapy method for anosognosia. A transient remission of anosognosia has also been reported after prismatic adaptation [76].

More recently, a rapid and long-lasting recovery of anosognosia for hemiplegia was reported when

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a patient viewed herself in a video replay [77]. This interesting finding should be replicated in other patients.

In conclusion, anosognosia remains an unresolved issue that has major implications for prognosis and functional recovery after right hemisphere stroke. As outlined by Vuilleumier [2] and Marcel *et al.* [10], there is a need for new experimental approaches and new therapeutic tools to better understand the cognitive and neural mechanisms underlying both awareness of normal functioning and anosognosia of neurological impairments.

Key points Management and treatment of anosognosia for hemiplegia

Anosognosia: impairs motivation to engage a rehabilitation program

Rehabilitation of anosognosia

- compensatory approach
- vestibular stimulation: temporary remission
- further trials required

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