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Unawareness after stroke: A review and practical guide to understanding, assessing, and managing anosognosia for hemiplegia.

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Abstract

How should stroke patients with poor motor awareness be managed? This question is important because unawareness (or anosognosia) is related to poor rehabilitation and prognosis. This narrative review provides a guide for clinicians and (applied) academics to understanding, assessing and managing anosognosia. Questions addressed are: What is anosognosia? What causes anosognosia? How can anosognosia be assessed? And how can anosognosia be managed? We suggest that anosognosia is a multifaceted disorder, with diverse neuroanatomical and psychopathological origins. Assessment should measure various aspects of awareness, and management should be multidimensional to address problems with motor function, awareness, and emotional / motivational disturbance.

Unawareness after stroke: A review and practical guide to understanding, assessing, and managing anosognosia for hemiplegia.

1. *Introduction*

Providing care for patients who are unaware of their motor deficit is a difficulty frequently encountered by health professionals working with stroke patients. Unawareness is a considerable problem for rehabilitation, leading to sub-optimal benefits and significantly poorer prognosis (Appelros, Karlsson, Seiger, & Nydevik, 2002). In practice, unawareness is a problem in acute and subacute rehabilitation because patients refuse treatments that improve prognosis (Di Legge, Fang, Saposnik & Hachinski, 2005; Katz & Segal, 2004) and typically do not take appropriate safety measures (Hartman-Maier, Soroker & Katz 2001; Hartman-Maier, Soroker, Ring & Katz, 2002). Although unawareness is often transient, lasting from days to months, the occurrence of unawareness at the crucial acute stages can considerably impede motor rehabilitation (Gialanella, Monguzzi, Santoro & Rocchi, 2005). Unawareness is linked to a longer stay in hospital (Maeshima et al., 1997), reduced likelihood of returning to independent living (Pedersen, Jørgensen, Nakayama, Raaschou, & Olsen, 1996), and lower scores on measures of functional recovery (Gialanella et al., 2005; Maeshima et al., 1997), Activities of Daily Living (Appelros, Karlsson, Seiger & Nydevik, 2002, 2003; Maeshima et al., 1997), and safety behaviour (Hartman-Maier et al., 2001).

Although no studies have directly assessed the financial impact of unawareness, for every patient who experiences a stroke the average direct and indirect costs to the NHS is over £58,000 (Youman, Wilson, Harraf & Kalra, 2003). Unawareness is likely to increase this sum, given that patients with unawareness have a much poorer prognosis than aware patients.

The degree of unawareness can vary considerably, from a simple failure to appreciate the consequences of motor impairment, to a lack of concern regarding one's problems (termed 'anosodiaphoria'), or outright denial and/or a complete inability to recognise the existence of a movement problem, despite clear evidence to the contrary (e.g. demonstration that a limb is paralysed). This latter condition, termed anosognosia for hemiplegia (AHP), has been a topic of clinical and theoretical speculation for decades. However, it is only recently that empirical research into AHP has advanced our understanding (Jenkinson & Fotopoulou, 2010). Improvements have been made in assessing and characterising anosognosia, and understanding the neuroanatomical and functional bases of the disorder (see Orfei et al., 2007). But despite this progress, the management and rehabilitation of patients with unawareness remains poorly addressed. In particular, there is a frequent failure to translate empirical research into clinical practice.

This article reviews recent research in AHP, in order to provide a practical guide to assessing / managing unawareness following stroke, and generate suggestions for applied rehabilitation and future research. Our primary goal is for this paper to be a useful resource for people working with unaware stroke patients. As such, we attempt to address several practical questions, which have arisen during our frequent interactions with staff providing care for stroke patients. These questions are: (i) What is anosognosia? (ii) What causes anosognosia? (iii) How can anosognosia be assessed? (iv) How can anosognosia be managed? With this in mind, literature was identified searching the databases *PubMed* and *Web of Knowledge*, using combinations of the search terms "anosognosia", "unawareness", "stroke", and "rehabilitation". The reference list of all identified articles were further searched and selected by the authors for inclusion if considered relevant to explaining, assessing or

managing anosognosia. The literature reviewed represents a holistic interpretation of empirical evidence and theoretical models guided by the expertise of the authors.

2. *What is anosognosia?*

2.1. Definition.

The term anosognosia originally referred to a specific unawareness of paralysis following stroke (Babinski, 1914). Since then, use of the term has been broadened and applied to unawareness of a variety of other conditions, such as blindness (David, Owen, & Forstl, 1993), memory problems (Ansell & Bucks, 2006), speech difficulties (Rubens & Garrett, 1991), and movement problems in other conditions (e.g. Jenkinson, Edelstyn, Stephens, & Ellis, 2009). This has resulted in the term anosognosia being used to denote any form of unawareness or lack of insight. In order to develop a coherent explanation for anosognosia, individual studies must describe the phenomenon being considered adequately, since unawareness in these various conditions presents differently and likely has distinct underlying causes. Here, the term unawareness and anosognosia refer specifically to a problem with insight into motor impairment after stroke.

2.2. Presentation.

Motor unawareness can take many forms. Some patients verbally deny their problems but show behaviours consistent with paralysis (e.g. executing a bi-manual tasks using a uni-manual strategy), while others verbally accept their paralysis but behave in a manner inconsistent with this acceptance (e.g. attempting to walk). This suggests that explicit (verbal) and implicit (behavioural) awareness are dissociable (Jehkonen, Laihosalo, & Kettunen, 2006). Unawareness can also vary from a complete failure to recognise paralysis even after its demonstration (i.e. severe anosognosia), to a mild or partial unawareness, in which the patient fails to recognise,

appreciate the severity, or acknowledge the consequences of paralysis (Orfei et al., 2007). Anosognosia can also be specific to a given function or ability, such that patients may accept one deficit (e.g. paralysis of the leg, or arthritis) but not another (e.g. paralysis of the arm). Both complete and partial unawareness have been referred to as anosognosia. This is an obstacle to generating a cohesive theory of anosognosia, but is beyond the scope of this review.

2.3. Incidence.

The reported frequency of anosognosia varies considerably. Pia, Neppi-Modona, Ricci and Berti (2004) report the frequency as 32.3% in a meta-analysis of studies selecting patients independent of lesion location. Jehkonen et al. (2006) performed a systematic review of anosognosia studies between 1995 and 2005, finding the reported frequency to vary from 8-27% when patients were similarly selected. Orfei et al. (2007) reviewed studies from 1990-2007, and identified rates of between 7 and 77%. This variation in reported frequency has been attributed to several methodological factors, such as differences in patient recruitment and selection criteria, diagnostic criteria, and time of assessment (for a review of these issues see Orfei, Caltagirone, & Spalletta, 2009).

The incidence of anosognosia at different stages after stroke is worth specific mention. A recent longitudinal study, in which 58 right hemisphere patients were assessed for AHP during the hyperacute (3 days), subacute (1 week), and chronic (6 months) stages post stroke, found that the reported frequency dropped from 32% during the hyperacute stage, to 18% in the subacute stage, and only 5% past 6 months (Vocat, Staub, Stroppini & Vuilleumier, 2010). However, Cocchini and Della Sala (2010) suggest that the low incidence of anosognosia reported during the subacute or chronic phase may be a result of the diagnostic tools used, rather than a genuine

recovery of awareness. Patients may be overexposed to questions used to assess anosognosia, such that they can provide the 'correct' response based on what they have 'learned' rather than on their actual awareness of deficit.

Finally, anosognosia is more frequent following right-hemisphere brain damage, suggesting that the condition is a right-hemisphere syndrome (Jehkonen et al., 2006; Pia et al., 2004). However, instances of anosognosia following left brain damage may be obscured by language impairment (Cocchini, Beschin, Cameron, Fotopoulou, & Della Sala, 2009), and the true incidence of anosognosia in this group (and overall) may be much higher. This limitation in assessing anosognosia following left-hemisphere lesions has resulted in the recent development of assessments designed specifically for patients with language impairment (see Della Sala, Cocchini, Beschin, & Cameron, 2009 and section 4.5).

2.4. Duration.

Anosognosia typically occurs in the acute or post-acute phase after a stroke, with awareness spontaneously recovering within days or weeks from onset. However, unawareness within a patient can fluctuate over time, and there are several reported instances of anosognosia present after several months or years (e.g. Cocchini, Beschin, & Della Sala, 2002; Preston, Jenkinson, & Newport, 2010). Additionally, explicit and implicit awareness may recover at different times in the same patient. That is, many patients who appear to have recovered explicit awareness based on a verbal assessment, remain unaware of the implicit consequences of paralysis (Cocchini, Beschin, Fotopoulou, & Della Sala, 2010; Marcel, Tegnér, & Nimmo-Smith, 2004; Nimmo-Smith, Marcel, & Tegnér, 2005). Research by Vocat and Vuilleumier (2010) further suggests that different combinations of neurological and neuropsychological deficits may be responsible for the persistence of anosognosia

beyond the acute stage. Specifically, they observed that a resolution of anosognosia between the hyperacute (3 days) and post-acute (1 week) stages coincided with rapid decrease in deficits of proprioception, visuospatial neglect, and temporospatial disorientation, whereas chronic anosognosia (present at 6 months) was linked to ongoing visuospatial neglect, memory impairment, and temporospatial disorientation. These findings suggest that the emergence of anosognosia is a result of both neurological and neuropsychological impairments, while its persistence is a result of ongoing neuropsychological factors. Vocat and Vuilleumier (2010) support these conclusions with a lesion analysis relating to the evolution of anosognosia over time. Persistent anosognosia was found to involve additional damage to brain areas that were not involved in cases of anosognosia limited to the acute period, suggesting that a complex network of cerebral regions is responsible for the occurrence and persistence of anosognosia (see section 3.1 for details).

2.5. Concomitant deficits.

Several concomitant deficits are common in patients with anosognosia. A loss of rudimentary sensation or perception (e.g. proprioceptive loss, hemianopia) is sometimes observed in anosognosia (Jehkonen, Ahonen, Dastidar, Laippala, & Vilkki, 2000; Small & Ellis, 1996), although the link between impaired sensation and unawareness is not ubiquitous, and the extent of sensory impairment does not appear to relate directly to severity of anosognosia (Small & Ellis, 1996). Similarly, global mental confusion or intellectual decline, poor memory, and executive dysfunction are psychological sequelae of stroke that are not uncommon in patients with unawareness; however, these are not necessary for anosognosia to occur (Starkstein, Fedoroff, Price, Leiguarda, & Robinson, 1992, 1993).

The relationship between anosognosia and unilateral neglect is worth specific mention, since the distinction between these two disorders is easily overlooked. In clinical practice a failure to directly attend to contralesional space (neglect), and a failure to acknowledge or recognise contralesional paralysis (anosognosia) can, *prima facia*, appear very similar. However, neglect without anosognosia is common, and reports of anosognosia without neglect (e.g. Bisiach, Vallar, Perani, Papagno, & Berti, 1986; Small & Ellis, 1996) demonstrate that the two processes are independent. As such, the frequent co-occurrence of anosognosia and neglect has been attributed to overlap in the cortical areas responsible for the two conditions, rather than a functional relationship (Berti et al., 2005).

Finally, an often overlooked comorbidity of patients with anosognosia is their disturbed mood, affect or motivation. Unsurprisingly, this may involve an increase in depression (Starkstein, Berthier, Fedoroff, Price, & Robinson, 1990; Starkstein et al., 1992), or apathy (Cutting, 1978; Levine, Calvanio, & Rinn, 1991), which may be linked to damage to subcortical circuits responsible for arousal, motivation and affective drive (Vuilleumier, 2004). Striking emotional disturbances and delusional symptoms can be found in patients showing inappropriate jocularity, fewer catastrophic reactions, an apparent disregard or indifference about paralysis (anosodiaphoria), hatred of the paralysed limb (misoplegia), or feeling of non-belonging or disownership (asomatognosia) (Turnbull, Evans, & Owen, 2005; Turnbull, Jones, & Reed-Screen, 2002). Turnbull and colleagues (2002, 2005) note that the presence of these pathological emotions suggests an intact range of emotions in anosognosia, although the focus of emotions may be abnormal (see section 3.2).

In summary, it is clear from the above description that anosognosia is not a uniform disorder. The heterogeneous presentation suggests that anosognosia is a

multicomponent (Vocat & Vuilleumier, 2010) or multifaceted disorder (Orfei et al., 2007), and that several sub-types may exist (Jehkonen et al., 2006; Marcel et al., 2004).

3. What causes anosognosia?

The aetiology of anosognosia is difficult to establish. A combination of multiple factors, rather than a single deficit, is likely to explain the heterogeneity of the disorder (Vocat & Vuilleumier, 2010). In this section we will briefly review the neuroanatomy and neuropsychology of anosognosia, in order to provide a basis for later discussion of possible treatments.

3.1. Neuroanatomy of anosognosia.

Several recent studies have attempted to identify brain areas that correlate with the presence of anosognosia; however, the diverse presentation and concurrent deficits found in unaware patients mean that multiple lesion sites have been found. To some extent, divergent findings across studies may be the result of disparate methods used to conduct lesion analyses. Scan quality (i.e. spatial resolution of CT vs. MRI) and lesion mapping method (i.e. by hand or computerised) affect the results of analyses, and have varied within and between studies.

A meta-analysis by Pia et al. (2004) included 85 cases of anosognosia reported between 1938 and 2001, with lesions identified using post-mortem examination, or CT and MRI scans. Analyses identified damage in frontal, parietal, temporal and occipital cortical regions, as well as subcortical structures comprising the thalamus, basal ganglia, corpus callosum, internal capsual, corona radiate, insula, lateral ventriculus, and amygdala. Further analyses of these data identified combined fronto-parietal damage as the most frequent site of cortical lesions in anosognosia, whilst unawareness following damage confined to a single subcortical structure was greatest

following basal ganglia and thalamus lesions. In addition, Pia et al. identified three cases of anosognosia in which the combination of cortical and subcortical damage did not involve the parietal cortex. The authors conclude that the observed association with parietal lobe damage may, therefore, be a consequence of the frequent co-occurrence with neglect, and that a combination of frontal and subcortical structure damage (in particular the basal ganglia and thalamus) is crucial in causing anosognosia.

The role of the insula in anosognosia has been a source of recent interest and debate. Berti et al. (2005) performed an MRI lesion analysis comparing three groups of right-brain-damage patients: those with anosognosia and spatial neglect (n=17), those without anosognosia but with spatial neglect (i.e. pure neglect, n=12), and a single patient with anosognosia but without spatial neglect (i.e. pure anosognosia). Their findings indicated that anosognosia was characterised by damage to premotor cortex (Brodmann areas (BA) 6 and 44), somatosensory cortex (BA 3), primary motor cortex (BA 4), and less frequently the dorsolateral prefrontal cortex (BA 46) and the insula. The inferior parietal lobule, which is traditionally associated with spatial neglect, was also frequently involved. These findings were corroborated by the single case of pure anosognosia, in which damage was similarly identified in areas 6, 4, 44, 3 and the insula. Berti et al. (2005) conclude that damage to areas related to motor planning are important in the pathogenesis of anosognosia. They speculate that spared activity in these premotor areas allows a distorted representation of the intended movement to be generated, while damage to the same premotor circuit impairs the monitoring of these intended movements (see section 3.2 for further consideration of this explanation).

Karnath, Baier and Nägele (2005) present an opposing view based on their own analysis of CT and MRI scans in 14 patients with- and 13 patients without-anosognosia. After matching the two groups on demographic and clinical factors (i.e. age, acuity of lesion, size of lesion, degree of hemiparesis, and frequency of sensory loss, neglect and visual field deficits), the posterior insula was the only structure identified as specifically more damaged in patients with anosognosia. These findings are consistent with the earlier finding of insula damage in anosognosia (Berti et al., 2005) and the involvement of the insula in normal monitoring of self-generated actions (Farrer et al., 2003), but suggest that the right posterior insula, as opposed to areas involved in motor planning, is the critical locus of damage in anosognosia. These findings are also contrary to the meta-analysis of Pia et al. (2004), which identified insula damage in only 19 patients out of 85 with anosognosia. However, several methodological differences have been highlighted, which may account for the discrepant findings (see Karnath & Baier, 2010; Orfei et al., 2007; Vallar & Ronchi, 2006).

The idea that the insula plays a crucial role in anosognosia is supported by Craig (2009, 2010), which identifies the anterior insular cortex (AIC) as the anatomical substrate for all subjective feelings and self-awareness. Craig (2009, 2010) proposes a model of self-awareness in which the insula houses a hierarchical set of representations. At the most fundamental level, in the posterior insular cortex, is a neural representation of the physiological condition of the entire body, which forms a foundation for the encoding of all feelings. Moving from posterior to anterior parts of the insular cortex, increasingly complex information concerning the environment, motivation and social cognition are integrated from other parts of the brain and re-represented in the insula. These re-representations culminate in the AIC, where they

are experienced as a subjective, unified meta-representation of the sentient self at one moment in time. The continual stream of these meta-representations produces our ongoing sense of self-consciousness. Accordingly, damage to the insula would be expected to result in altered awareness, like that occurring in anosognosia.

Fotopoulou, Pernigo, Maeda, Rudd and Kopelman (2010) performed a lesion analysis of patients' CT or MRI scans producing results consistent with Craig's (2009, 2010) model. Patients with implicit or explicit anosognosia (see section 2. 2) were compared with hemiplegic control patients without anosognosia, in order to identify the brain areas involved in these different types of awareness. Their findings revealed damaged insular cortex in patients with impaired explicit awareness and intact implicit awareness, whilst patients without implicit awareness tended to have damage in frontal and occipital cortex. These results provide empirical evidence of a neural dissociation between implicit and explicit awareness, and suggest that variability in the presentation of anosognosia can be attributed to the differential involvement of multiple lesion sites.

Finally, Vocat and Vuilleumier (2010) stress that previous lesion overlap studies fail to consider the multiple variations and fluctuations of anosognosia, thereby limiting interpretation; therefore, Vocat et al. (2010) performed a lesion overlap analysis of patients' CT or MRI scans, taking into account these factors. Results indicated that anosognosia in the hyperacute phase (3 days post stroke) was related to damage in the anterior insula and anterior subcortical structures, while persistent anosognosia (still present one-week later) was associated with additional lesions in parietal, frontal, and/or temporal structures. Importantly, these data are able to reconcile the divergent findings of previous research (Berti et al., 2005; Karnath et al., 2005), and coincide with recent findings of Fotopoulou et al. (2010). Vocat et al.

(2010) identify damage to the insula as accounting for the initial occurrence of anosognosia, with additional involvement of motor planning and other cortical areas linked to sustained unawareness.

3.2. Neuropsychology of anosognosia.

Several early accounts of anosognosia were created on an ad-hoc basis to explain clinical phenomenon. However, subsequent empirical investigation of these early proposals failed to provide corroborating evidence. Small and Ellis (1996) failed to identify any difference in personality traits that would be predicted to arise from a psychodynamic explanation (see Weinstein & Kahn, 1950, 1955), and Berti, Làdavas and Della Corte (1996) failed to find a predicted evolution of defence over time, indicating that a defensive explanation of anosognosia is improbable. The fact that providing sensory feedback about one's impairment to the left hemisphere (e.g. by bringing the paralysed hand into the right visual field) does not necessarily improve awareness (Adair et al., 1997) refutes the suggestion that anosognosia arises from disconnection of the verbal left- and sensory right-hemisphere (Geschwind, 1965) Finally, anosognosic patients without concomitant sensory or intellectual impairments (Berti et al., 1996; Small & Ellis, 1996) negate the suggestion that combined sensory impairment (e.g. especially proprioception and spatial neglect) and intellectual deficits impair the discovery of a motor problem or play a major causal role in the pathogenesis of anosognosia (Levine, 1990; Levine et al., 1991). However, all of the aforementioned factors may predispose patients to anosognosia, or lead to greater severity of the disorder (Marcel et al., 2004; Vuilleumier, 2004).

More recent approaches to anosognosia have drawn on the idea that the disorder is caused by a failure to self-monitor. According to an established computational model of the motor system, motor awareness in healthy subjects depends on the comparison

of predicted and actual sensory information (Miall & Wolpert, 1996). Moreover, motor awareness typically relies on predicted sensory information arising from ones intended movements, and relies less on actual sensory feedback (Fourneret & Jeannerod, 1998). Therefore, whenever an intended movement is planned, awareness that this movement has been performed may automatically be constructed (Berti & Pia, 2006). Consequently, movement errors are only detected when a large discrepancy arises during the comparison of the predicted and actual sensory information (Blakemore & Frith, 2003).

Several explanations of anosognosia map on to this model of motor control and awareness. Heilman and colleagues (Heilman, 1991; Heilman, Barrett, & Adair, 1998) suggest that anosognosia is due to an absence of the intention to move (or lack of 'feed-forward'). If the patient does not form an intention to move, a subsequent lack of movement does not create a mismatch between predicted and actual sensory information, and the motor impairment is not detected. However, there is considerable evidence to suggest that patients with anosognosia are able to generate motor intentions, and that such a deficit cannot account for the disorder (see Jenkinson & Fotopoulou, 2010 for a review).

Frith, Blakemore and Wolpert (2000) and Berti and colleagues (Berti & Pia, 2006; Berti et al., 2007) take an opposing position with regards to motor intention in anosognosia, suggesting that patients with illusory limb movements (i.e. the strong sense of having moved) have intact motor intentions and are still be able to form sensory predictions. Thus, patients have the normal experience of having initiated a movement and false sense of having moved because of a failure to detect the discrepancy between predicted and actual sensory information. Several recent experiments in patients with anosognosia support this account (Fotopoulou et al.,

2008; Jenkinson, Edelstyn, & Ellis, 2009), and suggest that anosognosia is the result of an inability to detect large sensorimotor discrepancies. However, the source of this deficit remains unresolved, with Frith et al. (2000) proposing a lack of contrary sensory information, whereas Berti and colleagues (2006, 2007) suggest damage to a comparator mechanism itself. Preston et al. (2010) use evidence from the non-paralysed upper limb to examine this question. They identified an anosognosic patient who was unable to detect large errors made with his unimpaired arm, despite correcting for them during movement. The dissociation between the conscious awareness of discrepancies and online motor corrections suggests that the comparator is not actually broken and that the deficit arises only in the conscious detection of discrepancies. However, these results are not incompatible with the suggestion of faulty comparator mechanisms, as the motor corrections recorded were often unsuccessful, indicating suboptimal functioning of the comparator.

Finally, despite their popularity, it is important to consider that purely motor accounts of anosognosia cannot account for all of the disorder's features (e.g. resilience to counterargument, unusual belief, and abnormal affect). Recent studies have sought to better clarify these non-motor features and account for their occurrence.

Turnbull and colleagues (Turnbull et al., 2005; Turnbull et al., 2002) demonstrated that patients with anosognosia show a normal range of positive and negative emotions, but that the subject or their emotions tended to be abnormal. Patients with anosognosia tend to focus their emotions on matters unrelated to their neurological deficit, whereas non-anosognosic patients with hemiplegia focus their emotions on their neurological impairment. Nardone, Ward, Fotopoulou and Turnbull (2007) further identified a tendency for disability related words to create interference

and increase response latencies in patients with anosognosia, but not non-anosognosic patients with hemiplegia. These findings are supported by Fotopoulou et al. (2010), who found that deficit-related words caused unconscious interference (i.e. slowing) only in patients with anosognosia. Together these results suggest that anosognosia may involve avoidance of deficit related information. This idea is consistent with recent experiments in healthy subjects, which have identified executive control processes that prevent unwanted memories from entering into conscious awareness (Anderson & Green, 2001). Such mechanisms may be recruited to a greater extent in some patients with anosognosia, in order to prevent anxiety and distress. Vuilleumier (2004) stresses how people often use denial as a coping mechanism for severe medical illnesses (Lewis, 1991; Moyer & Levine, 1998), and such tendencies may be exaggerated following brain injury. Therefore, motivational and affective factors may have been too quickly disregarded in the aetiology of anosognosia.

4. How can anosognosia be assessed?

It order to develop a coherent explanation of anosognosia, or therapeutic intervention, unawareness must be assessed accurately and reliably. Unfortunately, the heterogeneous and fluctuating nature of anosognosia has prevented a single 'gold standard' assessment from being developed. Traditionally, an assessment of anosognosia is made on the basis of a clinical observation that the patient does not appear to be aware of deficits that are clearly apparent to the clinician. Unfortunately, this subjective assessment does not produce a robust diagnosis. Thus, several standardised approaches have been developed to formally assess anosognosia. Here we focus on a selection of the most widely used and recently developed assessments (more extensive reviews can be found elsewhere in Orfei et al., 2009; Orfei, Caltagirone, & Spalletta, 2010; Orfei et al., 2007). The psychometric properties of

these assessments are not known; however, these measures are commonly used and general accepted for the identification of anosognosia in research.

Table 1 around here

4.1. Anosognosia Questionnaire (Cutting, 1978).

Cutting (1978) developed a clinician-rated scale that includes general questions, questions directed towards awareness of motor deficit, and items designed to assess concurrent anosognosic phenomena (Table 1). Based on the patient's responses, a simple classification is made of unawareness and associated phenomena being present or absent. This questionnaire has the advantage of assessing both anosognosia and associated phenomena; however, it is limited by an inability to differentiate different extents of unawareness.

4.2. Bisiach Scale (Bisiach et al., 1986).

The prevailing assessment of anosognosia is one developed by Bisiach et al. (1986), in which responses given during a clinical interview are rated on a four-point scale. The questions are not specified by Bisiach et al. (1986) but are typically similar to those specified by the Cutting Questionnaire (Cutting, 1978; Table 1), and classified according to the following criteria: 0= the disorder is spontaneously reported or mentioned by the patient following a general question about his/her complaints; 1= the disorder is reported only following a specific question about the strength of the patient's left limbs; 2= the disorder is acknowledged only after its demonstration through routine techniques of neurological examination; 3= no

acknowledgement of the disorder can be obtained. The scale is popular because of its simplicity and general effectiveness; however, it is not without its limitations. For example, the dissociation between non-verbal or implicit unawareness and verbal or explicit awareness, is not addressed by the Bisiach scale. Moreover, it has been argued that a score of 1 should not be interpreted as anosognosia, since this may occur as a result of patients being aware of hemiplegia, but not mentioning it spontaneously because concomitant deficits are considered more important (Baier & Karnath, 2005).

4.3. Anosognosia for Hemiplegia Questionnaire (Feinberg, Roane, & Ali, 2000).

The Anosognosia for Hemiplegia Questionnaire (Feinberg et al., 2000) comprises a series of 10 questions concerning the paralysed arm (Table 2), which are each scored on a 3-point scale (0= full awareness, 0.5= partial awareness, 1= full awareness). This questionnaire permits better gradation of the extent of unawareness, including an assessment of awareness after clear demonstration of paralysis to the patient. However, the limitation of only assessing explicit, verbal awareness remains present in this measure.

Table 2 around here

4.4. Berti et al., (Berti et al., 1996)

The Berti et al. (1996) assessment provides a more comprehensive test of unawareness, measuring both explicit awareness via a structured interview, and implicit awareness by asking patients to estimate their ability to perform mono- and bi-manual tasks (see Table 3). The test includes separate items for the upper and

lower limbs. Scoring of the structured interview is similar to the Bisiach scale (1986, section 4.2 above). For the upper limb: 0= the patient answered correctly to the first group of questions (normal); 1= the patient acknowledged being in the hospital and/or being affected by a stroke, but denied his or her upper limb impairment. However, the patient acknowledged that the left arm did not reach the examiner's hand (mild anosognosia); 2= the patient claimed that he or she had reached the examiner's hand (severe anosognosia). For the lower limb: 0= the patient either spontaneously reported the motor impairment of the lower limb when first asked about the reasons for his or her being in hospital, or acknowledged the paralysis when specifically questioned about the left leg (normal); 1= the patient answered 'well' to the first question, but acknowledged the impossibility of walking (mild anosognosia); 2= the patient claimed that he/she was able to walk (severe anosognosia).

To measure implicit awareness, patients are asked to estimate their current ability to perform several activities (see Table 3), which are scored on a scale from 0 (very badly) to 10 (very well). An average score of between 0 and 5 is considered to be normal (not anosognosic on this test), while a score between 6 and 10 is considered anosognosic (Berti et al., 1996). Unfortunately, this test, like the others described above, still relies on a verbal response, which might preclude the detection of anosognosia in individuals with language problems or implicit knowledge of motor deficits, which cannot be expressed verbally.

Table 3 around here

4.5. The Visual-Analogue Test for Anosognosia for motor deficit (VATAm; Della Sala et al., 2009).

A recently devised tool for assessing anosognosia in patients with left-hemisphere brain damage, of which 40% are thought to show some evidence of anosognosia (Cocchini et al., 2009), is the VATAm (Della Sala et al., 2009). The VATAm requires patients to estimate their current ability to perform several bimanual (e.g. open a jam jar) and bipedal tasks (e.g. climbing the stairs). To account for verbal communication difficulties, each question is illustrated by a drawing, and patients give their responses using a 4-point visual-analogue scale (see Figure 1). The patient's ratings are then compared with those of his or her caregiver and interpreted according to normative data, such that scores of 3.8-8.0 are taken to indicate mild anosognosia, 8.1-16.0 moderate anosongosia, and 16.1-24.0 severe anosognosia (full details of the VATAm can be downloaded from http://homepages.gold.ac.uk/gcocchini).

Figure 1 around here

4.6. Experimental Bimanual Task (BMT; Cocchini, Beschin, Fotopoulou & Della Sala, 2010).

The BMT assesses unawareness via actual behaviour on everyday tasks involving real objects (e.g. hold a two-handle tray). All of the tasks are better performed using both hands (e.g. holding a two-handled tray by placing one hand at each extremity), but can also be performed using only one hand (e.g. holding the two-handled tray by placing one hand underneath the centre of the tray). The difference in adopting either

the one- or two-handed strategy lies in ones awareness. Patients unaware of their motor difficulties would adopt the two-hand strategy, whereas patients with implicit anosognosia would behave as if they could use both hands (e.g. by placing the unimpaired hand at one extremity) and fail the task (i.e. the tray falls or is inclined on one side).

Cocchini et al. (2010) provide a description of the one-hand (aware strategy) and two-hand (anosognosia strategy) approach to each task, and criteria for failure due to anosognosia. Each task is attempted three times, and scored for performance on the following scale: 0 = the patient promptly carries out the task with one hand using the aware strategy; 1 = the patient carries out the task using one hand but with some hesitation; 2 = the patient started the task as if they could use two hands but then they corrected themselves using the aware strategy; 3 = the patient behaved as if s/he could use two hands resulting in a failure due to anosognosia. Thus, error scores ranged from 0-24 for each of the three (1^{st}, 2^{nd}, 3^{rd}) attempts. On the basis of normative data, the authors specify that an error score equal to or over 9 is evidence of implicit anosognosia.

4.7. Assessing anosognosia using discrepancy scores.

An alternative approach to assessing unawareness is to compare patient estimates of their ability with those of an independent source (e.g. caregiver, clinician or neuropsychological test). This approach is common in evaluating self-awareness following traumatic brain injury (Noé et al., 2005), and has been applied to anosognosia following stroke. A popular method is the Patient Competency Rating Scale (PCRS; Prigatano et al., 1986), a self-report questionnaire covering four domains: (i) activities of daily living (e.g. problems dressing), (ii) emotion (e.g. problems controlling crying), (iii) interpersonal relationships (e.g. problems

conversing), and (iv) cognition (e.g. problems remembering things). The patient and a clinician/caregiver independently complete the 30-item questionnaire, which judges the patient according to his/her current competency with each activity (1 = the patient cannot perform the activity; 5 = the patient can perform the activity with ease). Awareness is defined by the degree of discrepancy between the patient and clinician/caregiver score (i.e. greater discrepancy indicates more unawareness).

The Awareness Interview (Anderson & Tranel, 1989) is a similar discrepancy technique, which compares patients' ratings with independent neuropsychological (e.g. intelligence, memory, language, etc.) and neurological (e.g. motor strength) evaluations. For each ability the discrepancy between these two sources is scored from 0 (no discrepancy) to 2 (maximum discrepancy), such that greater scores indicate increasing levels of unawareness.

Several other measures follow this same discrepancy technique (see Orfei et al., 2009; 2010 for details), and may provide a more objective and valid measure of unawareness; however, they are constrained by several limiting factors. First, the time taken to complete assessments involving a discrepancy score is often greater than that of other methods. For example, the Awareness Interview (Anderson & Tranel, 1989) requires extensive neuropsychological assessment, a neurological examination, and interview with the patient.

A second consideration is the ability to identify a well-informed independent person to provide accurate information about the patient. Typically this role falls on clinical staff or the primary carer. However, brief clinical interactions do not always allow staff to appreciate the full extent of a patient's abilities and/or limitations, making them an unreliable source of information regarding the patient's awareness. Likewise, the patient's carer may not possess sufficient information or expertise to

accurately evaluate the patient's current abilities and/or limitations. A desire for the patient to return home may also lead carers to underestimate the true extent of the patient's limitations. In contrast, carers might overestimate the patient's limitations in order to ensure ongoing hospital treatment. Moreover, patients themselves may not be correctly informed of their condition. This greatly limits the ability to provide a realistic evaluation of their abilities and limitations, leading to a possible overestimation of functional ability, and erroneous diagnosis of anosognosia (Orfei, Caltagirone, & Spalletta, 2010). Thus, discrepancy ratings are constrained by the knowledge and motivations of the individual providing independent information.

5. *How can anosognosia be managed?*

For those working with stroke patients the most important issue is how to manage patients with anosognosia. Anosognosia has been identified as an inhibitory factor in prognosis (Appelros et al., 2002) and rehabilitation outcomes (Prigatano, 2008). Unfortunately, there is no accepted treatment for the disorder. Although the *National Clinical Guidelines for Stroke* (2008) include broad guidance on the assessment and treatment of patients with impaired spatial awareness (i.e. visuo-spatial neglect), a clear distinction is not made between this condition and anosognosia. As such, existing stroke guidelines fail to provide an adequate manual for the management of anosognosia.

Fortunately, early research identified anosognosia as being amenable to physiological intervention (Cappa, Sterzi, Vallar, & Bisiach, 1987; Rode & Perenin, 1994), and guidelines for the management and care of patients with anosognosia are beginning to appear (Prigatano & Morrone-Strupinsky, 2010). These guidelines are outlined below, along with their limitations, and a discussion of recent research with the potential for translation into a rehabilitation setting.

5.1. Existing guidelines.

Prigatano and Morrone-Strupinsky (2010) outline eight clinical guidelines in the management and rehabilitation of patients with anosognosia (see Table 4). These guidelines provide a broad outline of the steps to consider when managing patients with unawareness. Briefly, these consist of recommendations to assess the unawareness and its associated (potentially underlying) deficits, establish a good working relationship with the patient and family, develop an interesting and engaging rehabilitation plan that is tailored to the individual, and continue this program for as long as there is potential for improvement. A more specific plan for the treatment of anosognosia is also outlined (see Table 5), which suggests a hierarchical intervention strategy. This begins with basic sensory discrimination, before introducing tasks to facilitate recognition of body parts in space. Because unilateral neglect may increase the severity of unawareness, intervention then focuses on the resolution of this concurrent deficit. Traditional occupational and physical therapies are then introduced, along with development of a long-term awareness of one's limitations and their impact on everyday life. Finally, the patient's emotional and motivational state is assessed.

These guidelines provide general advice on the management of patients with unawareness after stroke; however, they fail to suggest methods that may be implemented to address the recovery of specific mental and physical disturbances in patients with anosognosia.

Tables 4 & 5 around here

5.2. Improving awareness

It has been suggested that neglect may exacerbate the severity of unawareness (Marcel et al., 2004; Vuilleumier, 2004). As such, the resolution of neglect may correlate with improvement in awareness. Several methods are available to minimise neglect and may be employed in patients with anosognosia, including both cognitive (e.g. training patient to attend to information in neglected hemispace) and noncognitive (i.e. physiological) methods (e.g. wearing prism glasses to shift the visual field to include neglected space, see Rossetti, Rode, Karnath, Milner, & Vallar, 2002 for details). Luauté, Halligan, Rode, Rossetti & Boisson (2006) provide a particularly useful systematic review of left visuo-spatial neglect interventions, identifying six techniques with greatest efficacy (i.e. visual scanning training, trunk rotation, neck muscle vibration, mental imagery training, video feedback training, and prism adaptation; see Luauté et al., for details). These techniques may also be especially useful in patients with anosognosia.

Interestingly, Robinson and colleagues (Robinson, 2006; Robinson, Beitman, & Nair, 2004) reported that antidepressants prevented the worsening of anosognosia. This finding raises the possibility of pharmacological intervention for patients with, or at risk of developing, anosognosia. However, these results were derived from a secondary analysis of data, which did not specifically aim to examine the effects of antidepressants on awareness in anosognosia. As such, they must be approached with caution, until further clinical trials confirm the beneficial effects of antidepressants on unawareness.

A recent case-report by Fotopoulou, Rudd, Holmes and Kopelman (2009) describes an intervention that directly improved awareness in a 67-year-old woman

with AHP. The researchers provided video feedback of the patient answering standard questions about her current state/abilities, and attempting to perform movements involving her paralysed arm. Remarkably, upon viewing the video playback the patient instantly and permanently recovered awareness of her motor problems. This reinstatement of awareness may be due to the fact that video-playback provides the patient with a unique perspective of their body, which is both 'offline' and from the 'outside' (i.e. a 3rd-person perspective of their body). Visual feedback of one's deficits may be important in facilitating motor awareness; however, further research is needed to validate this finding and establish the generalisability of using visual feedback to reinstate motor awareness.

Other recent experiments have found that patients with anosognosia are impaired at reality monitoring (i.e. distinguishing between information of an internal vs. external origin), both motor- and non-motor information (e.g. Jenkinson, Edelstyn, Drakeford and Ellis, 2009). Similar results have been found in patients with other delusional beliefs, such as delusions of control in schizophrenia (Anselmetti et al., 2007; Brébion, Gorman, Amador, Malaspina, & Sharif, 2002; Johnson, 1991; Keefe, 1998), suggesting that such beliefs involve a breakdown of reality monitoring or checking. Therefore, interventions to improve reality monitoring might enhance awareness in anosognosia. Similar techniques have been used to treat delusions in patients with schizophrenia (Landa, Silverstein, Schwartz, & Savitz, 2006) and confabulation (Dayus & van den Broek, 2000), and might be used in anosognosia.

5.3. *Improving motor function*

Unawareness also inhibits recovery of motor function. It is difficult to initiate active physiotherapy while the patient refuses to acknowledge that there is a problem. However, it may be possible to engage unaware patients in some form of motor

rehabilitation even in the acute stage post-stoke, by means of techniques that rely on visual feedback and/or mental rehearsal of movements, without execution of any physical activity. A growing body of research suggests that mirror therapy (in which a mirror placed long the body midline provides false visual feedback that their paralysed limb is moving) and mental rehearsal (practicing movements involving the paralysed limb using imagination) are effective when supplementing traditional motor rehabilitation (see Moseley, Gallace, & Spence, 2008; Ramachandran & Altschuler, 2009 for reviews). Johnson-Frey (2004) suggests that mental rehearsal in hemiplegic stroke patients might induce functional reorganisation of brain areas responsible for representing the impaired limb, providing neurophysiological support for observed behavioural effects. Moreover, these techniques might help improve motor function in patients with anosognosia, particularly given the recent findings by Jenkinson, Edelstyn and Ellis (2009) that suggest anosognosic patients retain some ability to mentally represent movements involving the paralysed limb. This finding indicates that the underlying processes needed for effective mental rehearsal may be preserved (though not at normal levels).

Moreover, patients considered unsuitable for mental rehearsal (due to poor motivation, etc.) may benefit from visual feedback therapy that does not require mental simulation, such as mirror therapy (see Altschuler et al., 1999; Stevens & Stoykov, 2003; Sutbeyaz, Yavuzer, Sezer, & Koseoglu, 2007; Yavuzer et al., 2008), or action observation therapy (Ertelt et al., 2007). Action observation therapy is a pioneering neurorehabilitative program, designed to engage motor imagery processes via observation (typically via a video-playback) of daily-life hand, arm or leg actions. A single, ongoing study provides evidence that this form of therapy may have a positive effect on rehabilitation of motor deficits after stroke (Ertelt et al., 2007).

Eight stroke patients with a chronic (at least 6 months) upper-limb hemiparesis received a 4-week course of action observation therapy, while a matched control group watched geometric figures for an equivalent period. Remarkably, despite receiving no other therapeutic intervention for the duration of the study, patients in the action observation condition exhibited improvement in motor functions. Functional neuroimaging identified reorganisation of the motor system as a result of the training, suggesting that action observation reactivated motor areas to aid recovery. Furthermore, studies in healthy subjects show that active intention to imitate does not appear to be crucial for movement observation to activate motor areas (Maeda, Kleiner-Fisman, & Pascual-Leone, 2002). Therefore, passive movement observation may benefit those patients who lack the motivation or physical ability to engage in more physically active treatment, although the efficacy of this technique requires further investigation.

Importantly, visual feedback or mental rehearsal should not be used as a total substitute for traditional physical therapy. Rather, such interventions may serve as an adjunct or first step in the recovery process in patients with poor awareness and/or motivation. The above research suggests that motor imagery on its own may not provide benefits beyond that of traditional physical therapy, whereas motor imagery techniques combined with traditional physical therapy may lead to better outcomes. Thus, techniques that rely on visual feedback or mental simulation of movement appear to work by priming the motor system to become active, and reorganising motor areas in the brain (e.g. Stinear, Barber, Coxon, Fleming & Byblow, 2008). Patients with anosognosia may benefit from this priming effect regardless of their current state of awareness or motivation. Thus, when patients eventually do engage in

traditional physical therapy, their motor system is already primed to achieve optimal results. These speculations require empirical examination.

5.4. Managing problems with emotion and motivation.

A feature of anosognosia that has received relatively little attention is the frequently abnormal affect (see section 2.5). Neuropsychoanalysis, an emerging field that combines neuroscientific and psychodynamic principles, provides explanations for anosognosia that incorporate these affective elements (see Solms & Turnbull, 2002). Turnbull et al. (2005) suggest that damage to the right hemisphere causes a reawakening of an original hatred for the body as part of the external world, leading to defence mechanisms which serve to protect the patient from the full realisation of their condition, via denial that the previously loved object is lost (anosognosia) and/or a hatred of the previously loved object (misoplegia) (see also Kaplan-Solms & Solms, 2002 for further details of neuropsychoanalysis, psychoanalytic observations of patients with anosognosia, and details of this explanation).

Fotopoulou (2010) suggests that the false statements made by patients with AHP about their body state (e.g. ‘there is nothing wrong with my arm, I can move it’) or general ability (e.g. “I went walking yesterday”) are confabulations generated by an autobiographical memory system that uses fundamental drives existing in psychoanalysis to modulate the accessibility (or inaccessibility) and content of memories. This self-memory system (SMS; Conway & Pleydell-Pearce, 2000) maintains a sense of self-coherence by balancing the competing demands of accurately representing reality whilst constructing a self that is coherent with premorbid goals and self-enhancing (i.e. biased towards promoting a positive image of oneself).

Based on the above, psychotherapy may be useful as part anosognosia treatment.

Prigatano and Morrone-Strupinsky (2010) advocate the use of psychotherapy in cases where denial is used as a defensive mechanism for coping. According to Prigatano and Morrone-Strupinsky, patients with unawareness who use denial as a defensive mechanism require more than systematic cognitive rehabilitation to achieve a realistic view of their disabilities. Instead, the patient's *resistance* to their limitations must be addressed, and this may be done via psychotherapeutic management. A psychotherapeutic approach takes into consideration the fact that the patient may not be aware of feelings and motivations guiding his/her interpretation of a situation, while the context of the psychotherapeutic relationship may allow the individual to discuss the reasons for their resistance, leading to eventual self-discovery, greater compliance and insight.

Fotopoulou (2004) outlines several strategies that may be useful in the rehabilitation of patients with confabulations, and these might be adapted to treat the false statements produced by patients with anosognosia. The key to these techniques is an appreciation of the motivated content of confabulation, and the functions of self-coherence and self-enhancement it serves. Specific recommendations by Fotopoulou (2004) include: (i) liaising with significant others to understand and explain how confabulations in instances of unawareness are cognitively and motivationally constructed and influenced by social context, (ii) discouraging rehabilitation staff from confirming or contradicting patients' confabulations, (iii) using individual sessions to progressively explore the subjective meaning of confabulations and provide non-threatening feedback, and (iv) building rapport with the patient by initially discussing negative experiences, vulnerability and disability using the third- rather than first-person perspective (i.e. talking about "people who suffer a stroke/brain injury" rather than the patient's problems directly).

Again, it must be stressed that the efficacy of these techniques in treating anosognosia has not been established. However, Shelder (2010) reviewed empirical evidence concerning the effectiveness of psychodynamic psychotherapy in a variety of conditions (e.g. mood, personality and somatic disorders), finding effects as large as those observed in other established therapies (e.g. cognitive-behavioural therapy or pharmacological intervention). Moreover, evidence indicates that the benefits of psychodynamic treatments are lasting, with improvement continuing after treatment ends (Shelder, 2010).

Finally, Vuilleumier (2004) suggests that anosognosia may involve a defective affective drive to detect errors, which is linked to damage of subcortical circuits (e.g. basal ganglia) involved in both motivation and error detection. This suggestion is supported by neuroanotomical evidence in patients with anosognosia, who show frequent damage to these areas (Ellis & Small, 1997; Pia et al., 2004). Vuilleumier (2004) proposes that this defective motivational drive may lead to anosognosia because patients fail to engage in verification processes that would normally be used to check knowledge, particularly when uncertain. This proposal is consistent with behavioural evidence of reality monitoring failures in patients with anosongosia (Jenkinson, Edelstyn, Drakeford et al., 2009). Thus, in addition to using reality-checking as a means of improving awareness (see section 5.2, above), it is possible that pharmacological interventions targeting the basal ganglia might improve motivation and awareness. This suggestion is speculative, but receives theoretical support from studies of anosognosia in Parkinson's disease, which suggest damage to mesocorticolimbic dopamine pathways involving the basal ganglia as a cause of impaired self-awareness (see Amanzio et al., 2010; Jenkinson, Edelstyn, Stephens et al., 2009; Vitale et al., 2001).

6. *Conclusion*

In this paper we have reviewed the presentation and possible causes of unawareness following stroke, and provided a practical guide to different methods of assessing and managing patients with unawareness. In doing so, we have highlighted ongoing controversies and recent empirical research into the disorder. It is clear from this review that a definitive explanation for anosognosia remains elusive, as does an accepted form of assessing and treating unawareness; however, several suggestions are made with regards to each of these issues. For the clinician working with an unaware patient, an approach based on the framework of Prigatano and Morrone-Strupinsky (2010) is recommended, in which a hierarchical treatment strategy is adopted. Careful evaluation of the patient's unawareness (using techniques described in section 4) and concomitant deficits (see section 2.5) should be used to inform a program of rehabilitation that is guided by a knowledge of the possible underlying mechanisms of unawareness (see section 3.2), and tailored to improve awareness (section 5,2) and motor function (5.3), whilst appreciating the impact of possible emotional and motivation factors (section 5.4).

Two limitations to our review should be kept in mind. First, we acknowledge that our review was limited to just two databases (*PubMED* and *Web of Science*), with further relevant literature being selected from this initial search. This method may have overlooked some potentially fruitful sources of information (e.g. text books and expert opinion). Whilst there is a potential bias in this approach, we contend that this has the advantage of allowing an understanding of the important issues, based on our own experiences as clinical researchers and practitioners in the field.

A second limitation lies in the speculative nature of the techniques suggested for rehabilitation. These techniques are proposed on the basis of recent empirical research

and a combination of existing rehabilitation techniques. We have extrapolated from these several possible methods for treating anosognosia; however, the efficacy of these techniques is not proven (none have been subject to a randomised controlled trial) and should be the focus of future research before their adoption into clinical practice.

Despite the possible limitations of our work, we hope that this review serves as a useful resource for those working with stroke patients who exhibit a lack of awareness. We further hope that our attempts to identify the most effective means of assessing unawareness, and to translate the results of empirical research into clinical practice, provokes future research and better care for patients with impaired awareness. This research should focus on uncovering the mechanisms that underpin awareness and anosognosia, and exploring the factors that might cause a patient to develop anosognosia or be prone to chronic anosognosia, so that 'at risk' individuals might be identified and treated effectively.

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FIGURE CAPTION

Figure 1. An example item from the VATAm (Della Sala et al., 2009).

TABLE CAPTIONS

Table 1. Anosognosia Questionnaire (Cutting, 1978).

Table 2. Anosognosia for Hemiplegia Questionnaire (Feinberg, Roane & Ali, 2000). Each item is scored as either 0 (shows awareness), 0.5 (partial awareness) or 1 (complete unawareness).

Table 3. Berti et al. (1996) assessment of anosognosia.

Table 4. Clinical guidelines for the management of patients with anosognosia.

Table 5. A hierarchical intervention for anosognosia for motor deficit.