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On the contribution of unconscious processes to implicit anosognosia

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Implicit awareness in anosognosia: Clinical observations, experimental evidence, and theoretical implications

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Unawareness of deficits caused by brain damage or neurodegeneration, termed anosognosia, has been demonstrated in a number of different neurological conditions, including in patients with hemiplegia, hemianopia, aphasia, and memory disorder. Its effects can be very debilitating, with unawareness predicting worse prognosis (Orfei et al., 2007), less compliance with treatment (Patel & Prince, 2001), and greater exposure to dangerous behaviors (Starkstein, Jorge, Mizrahi, Adrian, & Robinson, 2007). In addition, unawareness is associated with greater distress in relatives or caregivers (Seltzer, Vasterling, Yoder, & Thompson, 1997). Apart from these major clinical implications, unawareness also attracts considerable attention from researchers due to its potential to explore topics such as consciousness, self processes, and embodiment, amongst others.

Clinical observation suggests that unawareness paradoxically can be accompanied by signs of understanding or representation of deficit, but not explicitly expressed. Such “implicit awareness,” an apparent oxymoron, is implied by or inferred from actions or statements of the person with neurological disorder. In the current paper, we review clinical observations and experimental evidence which suggest the occurrence of implicit awareness in dementia and hemiplegia, and explore the clinical and theoretical implications of this phenomenon. We present a theoretical framework to understand implicit awareness in these two conditions.

Keywords: Anosognosia; Awareness; Implicit memory; Dementia; Alzheimer’s disease; Hemiplegia.

Unawareness of deficits caused by brain damage or neurodegeneration, termed anosognosia, has been demonstrated in a number of different neurological conditions, including in patients with hemiplegia, hemianopia, aphasia, and memory disorder. Its effects can be very debilitating, with unawareness predicting worse prognosis (Orfei et al., 2007), less compliance with treatment (Patel & Prince, 2001), and greater exposure to dangerous behaviors (Starkstein, Jorge, Mizrahi, Adrian, & Robinson, 2007). In addition, unawareness is associated with greater distress in relatives or caregivers (Seltzer, Vasterling, Yoder, & Thompson, 1997). Apart from these major clinical implications, unawareness also attracts considerable attention from researchers due to its potential to explore topics such as consciousness, self processes, and embodiment, amongst others.

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subject to reflection and overt personal appraisal (Ownsworth, Clare, & Morris, 2006), implicit awareness can be inferred from a combination of indirect verbal utterances and non-verbal behavior.

The theoretical implications of implicit awareness have tended to be considered in a piecemeal fashion in relation to specific neurological disorders. Here a comparison is made across disorders, focusing on two main disorders in which it has been observed, hemiplegia and dementia, including Alzheimer’s disease (AD), firstly to demonstrate the existence of this phenomenon from observation and experimental investigations and secondly to derive theoretical implications. A contrast between conditions and experimental approaches also highlights the potential involvement of multiple neurocognitive mechanisms. Accordingly, a synthesis is attempted, firstly reviewing clinical and experimental studies, then discussing the theoretical issues that surround this topic, and finally proposing our own theoretical conclusions that might account for the described phenomena.

**CLINICAL OBSERVATIONS**

There are plenty of observational accounts of implicit awareness and these encompass a range of phenomena (see Table 1). Often noted is the disparity between verbal acknowledgement and other forms of action, for instance, not acknowledging hemiplegia but nevertheless disengaging from activities that rely on use of both hands (Bisiach & Geminiani, 1991). Furthermore, anosognosic patients may steadfastly refuse to acknowledge problems but are then willing to stay in hospital and receive care (Prigatano & Weinstein, 1996), including staying in bed or using a wheelchair (Bisiach & Berti, 1995).

Unaware patients also make symbolic references to their impairments (Gainotti, 2005; Prigatano & Weinstein, 1996; Ramachandran & Blakeslee, 1999; Weinstein, Friedland, & Wagner, 1994), including joking about them and using metaphors (Prigatano & Weinstein, 1996). When justifying their failure on motor tasks, some patients will deny their deficit but give answers that suggest implicit knowledge, such as “I should use a robot” or “my arm was cold” (Marcel, Tegner, & Nimmo-Smith, 2004). In addition, patients have been shown to deny left paresis, but talk about weakness or lack of sensation on their intact side (Gilliat & Pratt, 1952; Tei, 2000). People with hemiplegia have also been shown to refer to themselves when considering other people with disabilities. For example, House and Hodges (1988) showed a patient with anosognosia for hemiplegia photographs of a range of people with different disabilities from walking unaided to using a wheelchair, and she selected the wheelchair picture as the one “most like her.” More generally, people with anosognosia may be ready to describe deficits in other people (e.g., Clare et al., 2012; House & Hodges, 1988; Ramachandran & Rogers-Ramachandran, 1996) and, in doing so, use language which describes accurately their own condition (Clare et al., 2012; Weinstein & Kahn, 1955). Manifestations of emotion related to the deficit in unaware patients may also suggest implicit awareness. For example, Kaplan-Solms and Solms (2000) present a case of an anosognosic patient who was indifferent to her hemiplegia, but was prone to sudden tearfulness when hearing about disability and related themes. The rare phenomenon of misoplegia (Critchley, 1955) could probably be considered a related manifestation of implicit awareness, in which hemiplegic patients react with apparent hatred to the affected limb, often verbally and physically abusing it; misoplegia may develop before, during, and after unawareness of paralysis (Loetscher, Regard, & Brugger, 2006; Pearce, 2007).

In relation to neurodegenerative disorders such as AD, clinicians have often noted indications of implicit awareness, mainly through the manner in which patients tend to adjust to abilities despite showing little overt awareness. This is illustrated, for example, in research on driving. People with AD are generally not fully aware of their driving limitations (Wild & Cotrell, 2003) and may be more prone to engaging in dangerous behaviors (Starkstein et al., 2007). However, it has been shown that there can be considerable spontaneous adaptations, including, for example, driving fewer miles, restricting driving to familiar environments (Drachman & Swearer, 1993), and relying more frequently on relatives as “co-pilots” (Shua-Haim, Shua-Haim, & Ross, 1999), in spite of poor acknowledgement of driving problems. Support for this notion is provided by a longitudinal study, which suggested that people with AD tend to show voluntary and self-initiated driving restrictions, but not associated with awareness (Cotrell & Wild, 1999). It can be argued that some of these adaptations happen through residual awareness, being also observed in normal ageing, where reduced driving can be a strategic response in relation to other disabilities and factors, such as fatigue. Nevertheless, it is suspected from the way that patients often rationalize their change in behavior that implicit awareness can be a prominent feature.

An additional sign of implicit awareness is emotional responses to material associated with the condition. Turnbull, Jones, and Reed-Screen (2002) have carried out an in-depth analysis of transcripts of
psychotherapy sessions with four brain injured patients, of which two were substantially unaware of their disabilities, exploring affective content, object of emotional expression, and the occurrence of emotional breakdowns (e.g., crying, sobbing). Evidence of possible implicit awareness was the finding of a greater degree of emotional breakdowns in people with anosognosia, occurring often when the theme of the sessions was about loss. There is other evidence for emotional responsiveness despite unawareness, for example, the recent study by Hainselin et al. (2012), who found that unawareness of memory impairment in the acute phase of transient global amnesia was nevertheless accompanied by greater anxiety and depressed mood.

**EXPERIMENTAL EVIDENCE**

The range of clinical observations has led researchers to explore the phenomenon using experimental procedures. Here we review some main studies focusing on hemiplegia and dementia, where most of these studies have been conducted. The juxtaposition of these patient groups allows for the comparison of conditions in which anosognosia can be very specific, as in motor responding, or more generalized, as related to the range of neuropsychological deficits found in AD.

**Implicit awareness in anosognosia for hemiplegia**

There are a number of studies that have demonstrated what could be considered implicit awareness by observing manual responses involving objects. This provides a scenario where implicit awareness could be potentially explored, for example, investigating systematically the patients’ attitude or appraisal of responding, or changes in the pattern of responding that would suggest adjustment or “tacit knowledge.”

A large study by Marcel et al. (2004) involved asking patients with hemiplegia to evaluate their performance on a range of motor tasks requiring unimanual or bimanual responses. Some of the patients identified using this approach as having anosognosia showed indications of implicit awareness. One of their procedures was to ask about the deficit of the person in the third person, by requiring them to indicate how well the examiner, if they had the condition of the patient, would do on the tasks. This was
compared to them rating their performance directly, i.e., in the first person. It was found that frequently patients would overestimate performance in the first-person perspective, but would not do so when asked in the third person, the latter taken to indicate indirect or tacit knowledge of the deficit. Another approach was to ask tentative questions about their plegic limb, expressed in a playful but respectful fashion, such as “Is it ever naughty? Does it ever not do what you want?” This elicited positive responses in five patients not showing awareness on direct questions, such as “Oh yes! In fact, if doesn’t do what I want, I’m going to hit it.”

In relation to adjustment of manual responses, an initial study by Ramachandran (1995) tested patients with anosognosia for hemiplegia by getting them to select from a choice of unimanual or bimanual tasks. His patients selected bimanual tasks on almost all trials, which would tend to suggest no tacit knowledge. However, a different conclusion can be reached when considering bimanual task performance. Specifically, Cocchini, Beschin, Fotopoulou, and Della Sala (2010) asked 30 hemiplegic patients to perform a series of bimanual tasks using real objects (e.g., opening a bottle, holding a tray). While usually these tasks would be performed better with two hands, they could also be carried out with one hand. Unaware patients made more errors than healthy and neurological controls in the first attempt with the bimanual tasks. However, indicative of implicit awareness of disability, they then changed their strategy to a unimanual method, taking into account their paralyzed limbs, and made significantly fewer errors in subsequent trials. Explicit awareness was also measured, using a visual analogue scale in which patients’ ratings of ability to perform a series of everyday motor tasks is compared against informant report (Della Sala, Cocchini, Beschin, & Cameron, 2009). Interestingly, some patients (n = 8) overestimated their abilities on explicit measures but not in the bimanual tasks, while others (n = 2) showed the opposite pattern, providing preliminary evidence for a potential double dissociation between implicit and explicit awareness. This suggests that these two forms of awareness may potentially be mediated by assumed different neurocognitive processes. It is also possible that patients who changed their strategy did so in response to task-specific feedback.

Similar adjustments in patients with anosognosic hemiplegia have been observed by Moro, Pernigo, Zapparoli, Cordioli, and Aglioti (2011) using a task in which the patients had to grasp a heavy object. Normally, the grasping is bimanual to balance the object, but with hemiparesis adjustment is to use one hand and grasp it at the midline. This study also explored dissociations between verbal report of ability and actual motor behavior on the same task, facilitating a direct comparison. Out of 12 patients who verbally denied motor deficits, five showed indications of implicit awareness, suggested by a shift towards the midpoint of the object while grasping. They also studied hemiplegic patients who acknowledged their hemiplegia, and one patient in particular nevertheless did not show any motor adjustment, suggesting a converse preserved explicit but impaired implicit awareness, indicative of a double dissociation. CT lesion mapping found that reduced implicit awareness was associated with damage to the middle temporal cortex and white subcortical frontal matter anterior and around the basal ganglia.

Adjustment of motor responses despite subjective unawareness of movement performance has also been suggested by Preston, Jenkinson, and Newport (2010) in a case study which included an experimental manipulation. Awareness of movement was explored in relation to the non-paralyzed limb, in a computer paradigm which generated distorted visual feedback to reaching movements. In comparison with hemiplegic and healthy controls, the patient could not detect computer-generated distortions and showed abnormal sense of agency, claiming that observed movements accurately represented his actual movements. In addition, he also failed to notice corrective movements made when compensating for the visually distorted feedback.

These studies provide strong support for the presence of implicit awareness in the form motor adjustment in anosognosic hemiplegia, but it should be noted that not all such patients show this phenomena and demonstration may depend on the precise method used. For example, Garbarini, Piedimonte, Dotta, Pia and Berti (2013) explored patients with hemiplegia and motor neglect (n = 8), and in their sub-sample of two patients with anosognosia for hemiplegia, there was no motor adaptation, it being found only in aware hemiplegic patients. In this case, motor adaptation was indicated by full switching to holding with one hand in the middle of the object.

Another experimental approach has been to measure emotional interference effects in tasks that involve attentional bias. In a study of anosognosia for hemiplegia (Nardone, Ward, Fotopoulou, & Turnbull, 2007), a dot probe task was used (MacLeod, Mathews, & Tata, 1986). The participants have to respond to a neutral probe (for instance, pressing a green button in response to a green probe) which is preceded by words, some of which are threatening while others are neutral. Reaction times to the probe are modified by the emotional impact of threat-related stimuli, with slowed reactions generally
indicating higher interference. In this study, disability theme words (e.g., “crippled,” “deformed,” “immobile”) were used as the affective stimuli. Patients divided into “aware” (n = 2) and “unaware” (n = 5) based on clinician ratings and those with neglect syndromes were not included in the study. Patients with preserved awareness were significantly faster at responding to probes preceded by disability words relative to neutral words, while the unaware group showed the opposite pattern, being significantly slower after disability words. Furthermore, a correlational analysis using the full sample suggested a very strong association between unawareness and increased latency for disability-related (or emotional) words. The study may suggest that aware patients showed facilitation because they were primed in relation to illness-related material, while unaware patients were slowed down due to implicit interference in processing of information. It would be of interest to see the study expanded with a larger sample and also using a control condition with negative words unrelated to illness.

In a further study, Fotopoulou, Pernigo, Maeda, Rudd, and Kopelman (2010) also used an emotional interference approach, modifying the Hayling Test (Burgess & Shallice, 1997). In the standard task response inhibition measurement condition, participants are given an incomplete sentence which they are asked to complete with an unrelated word. This requires the inhibition of all semantically-related (prepotent) responses. Rule breaks and time taken in each trial are registered. In the modified task, 10 sentences had neutral content, 10 were emotionally negative and 10 deficit-related (e.g., “A hoist is often used to lift paralysed patients off the ______.”). Participants were also asked to rate how much each sentence related to themselves. Anosognosic patients were significantly slower with deficit-related sentences relative to neutral sentences, suggesting implicit awareness, and in contrast they rated deficit-related sentences as less self-related than aware patients. The neuroanatomical basis for these phenomena are difficult to establish fully using single case studies or small samples, but it was noted that the unaware patients showing implicit awareness had more subcortical (e.g., basal ganglia, amygdala) and less cortical (mostly frontal areas, but also parietal and occipital lobes) damage (Fotopoulou et al., 2010).

Implicit awareness in anosognosia for dementia

Implicit awareness in dementia has been studied so far using three experiments. The first explored anosognosia relating to memory deficit in a mixed group of people with AD and vascular dementia by Martyr and colleagues (2011) as part of the MIDAS study. This investigated attentional bias to illness-related material in a dementia group and a carer control group. A modified emotional Stroop Test was used, in which the time taken to name the print color of words was measured for neutral words and memory-deficit related words (e.g., forgetful, lapse). Here, attentional bias towards deficit associated words is predicted to slow the production of the color names by the participant. The dementia group showed similar levels of increase in response times to illness-related relative to neutral words as their caregivers. The findings suggest that both the dementia group and their caregivers have a heightened susceptibility to memory deficit-related words. Of note, this interference effect in the patients was not correlated with level of explicit awareness (measured by clinician rating based on extensive interviews with patients and caregivers), which would suggest an independent mechanism and support the notion of implicit awareness. Additional data from the MIDAS study has shown that this effect is weaker in non-carer controls. Further research is needed to expand the use of this technique, also using emotional words unrelated to deficit, and to explore the extent to which the atten- tional bias is self-relevant.

A recent study by Mograbi, Brown, Salas, and Morris (2012) explored experimentally induced emotional responsiveness in relation to task failure. The notion was that if there is implicit awareness of failure, the emotional response should be relatively preserved. In other words, reacting emotionally to failure to the normal extent suggests at some level monitoring of success or failure and detection of error. This was explored in an AD group (n = 23) and matched controls (n = 22) using a series of computer tasks measuring memory and reaction time. These computer tasks employed a success-failure manipulation, such that performance was kept constant for participants through the detection of individual performance thresholds in initial trials and automatic adjustment of difficulty. This produced matching error rates between groups, necessary to explore emotional reaction given the same degree of failure (for a full description, see Mograbi, Brown, Salas, et al., 2012). Half of the tasks were rigged to be above participants’ ability level (success tasks), while the other half was below (failure tasks). Results indicated that, although AD groups had less awareness of failure relative to controls, emotional reactivity was preserved, both in terms of self-report and filmed facial expressions, with increased reactivity to failure compared with success (see Figure 1 for an example). In all tasks, emotional
reactivity to failure was not correlated with awareness of performance. This dissociation between awareness and reactivity seems to suggest implicit awareness in AD (for alternative explanations, see Mograbi, Brown, Salas, et al., 2012).

Using a different approach, we have also explored emotional reactivity to film clips, exposing patients with mild AD to a set of stimuli including neutral, negative, and positive materials (Mograbi, Brown, & Morris, 2012). The negative content compared two illness scenarios: One film depicting difficulties experienced by someone coping with dementia and another about a person dealing with cancer. While emotional reactivity was slightly decreased in the AD group in comparison with healthy controls, higher reactivity to the dementia illness-related film, in terms of frequency of negative facial expressions, was found in more unaware patients. One way of considering these findings is to suggest that people with AD with greater awareness might normally select not to reveal their emotional response to negative dementia-related material as a socially conditioned but conscious coping mechanism to avoid stressful material; in those with less awareness this process might not take place leading to a stronger emotional reaction. An alternative is that this may suggest “leakage” of involuntary expressions in unaware patients, with implicit awareness leading to reactivity which bypasses volitional control (or active suppression, within a psychodynamic perspective).

**Conclusions**

In summary, although there are few experimental investigations about implicit awareness in neurological conditions, studies employing different methodologies seem to provide support for this notion beyond clinical observation. This is not to say that the mechanisms for implicit awareness are the same between neurological conditions, but rather that the existence of the phenomenon appears to be common.

**THEORIES OF IMPLICIT AWARENESS**

The phenomenon of implicit awareness, if accurate, has important repercussions for theories of unawareness and potential consequences for explanations about the cause of unawareness (e.g., the psychological/neurocognitive debate). Here we provide three potential explanations for manifestations of implicit awareness. The first is a mundane explanation that makes the phenomenon less interesting, namely, the effects of partial awareness; the second places the phenomenon within a psychodynamic framework and is denial; a third explanation relates to the preservation of neurocognitive implicit processes.

**Partial awareness**

If unawareness exists within a continuum and is not an “all or none” phenomenon (Marcel et al., 2004), apparent implicit awareness may simply indicate partial explicit awareness, i.e., anosognosic patients may have graded levels of awareness or manifest it only in some contexts. Related to that idea, it is also possible that prompting patients about their deficits or exposing them to feedback about failure (for example, in actual motor behavior or tasks) leads to subsequent adjustment which is mistaken for implicit awareness.
There is evidence for the incompleteness and inconsistency of anosognosia (e.g., Marcel et al., 2004; Moro et al., 2011) but its relationship with implicit awareness is not clear. One way to test the hypothesis that cases of implicit awareness are in fact occurrences of partial awareness, is through correlational analyses between implicit behavior and (task or illness) awareness measures. If implicit awareness is merely partial explicit awareness, it is logical to assume that condition-related interference effects, such as those shown with the emotional Stroop and Hayling tests, would increase as a function of explicit awareness. However, studies of implicit awareness showed a lack of association (Martyr et al., 2011; Mograbi, Brown, Salas, et al., 2012) or dissociation (Cocchini et al., 2010; Moro et al., 2011) between these two measures, failing to support the partial awareness hypothesis. In any case, even if partiality of unawareness can be a contributing factor, it is unlikely that it is the single reason behind implicit awareness phenomena, and it is important to highlight that this perspective is not excluded by the psychodynamic and cognitive explanations described below. In addition, it is also worth noting that implicit awareness itself may exist within a continuum and have different degrees, depending on different “objects of awareness.”

Denial or psychological processes

A second explanation, denial of deficit, put simplistically, is that explicit awareness is masked or suppressed by psychodynamic process, which provides no barrier to implicit expression. This assumption has been made in early studies of unawareness (Weinstein & Kahn, 1955) and more recently by other researchers (Nardone et al., 2007; Turnbull et al., 2002). From this perspective, implicit awareness can be understood as the result of defense mechanisms, for instance, as symbolic manifestations of repressed material or proof of psychodynamic conflict. A development of this notion is that some awareness of deficit (implicit) is necessary for the deployment of defense mechanisms such as denial (Cocchini et al., 2010; Turnbull & Solms, 2007).

It follows that more empirical data is needed to understand the relationship between what can be identified as denial and implicit awareness. Although some findings have been consistent with the notion that implicit awareness may be linked with blocking/suppression of conscious reactions (for example, an explanation for people with dementia with less awareness reacting emotionally to illness-related film material—Mograbi, Brown, & Morris, 2012), double dissociations between explicit and implicit awareness of deficit have been observed (Cocchini et al., 2010; Moro et al., 2011), with not only patients who “deny” their problems showing implicit awareness, but also with patients who have explicit awareness of problems trying to approach tasks as if they were healthy (see also Bisiach & Geminiani, 1991). This suggests that implicit and explicit awareness may be supported by different neurocognitive processes, and is not merely a manifestation of a single overarching psychodynamic concept such as denial. Nevertheless, it must be noted that recent conceptual developments have blurred the borders of the distinction between psychological and neurocognitive explanations.

Cognitive or perceptual implicit processing

The extensive demonstration of mental processes not accessed consciously, such as blindsight (Weiskrantz, 1997) and priming (Schacter, 1990), is sufficiently convincing to support the notion of cognitive non-conscious mechanisms, without necessarily indicating motivational and emotional factors or invoking a psychodynamic unconscious. According to this position, implicit processing of information despite unawareness would be a consequence of the normal architecture of the brain and not the effect of psychological defense (Kihlstrom, 1987). Central to this view is the idea of parallel pathways in the brain for processing similar information. This has been shown across species and it has been suggested these reflect subtle functional specializations (Sherry & Schacter, 1987). This includes, for example, neurocognitive pathways associated with emotion, such as the dual route that explains affective blindsight (Weiskrantz, 1997) and two different pathways for processing fear: A subcortical coarse and fast route, with stimuli traveling from sensory organs to the thalamus and then to the amygdala, activating a fear-response in the body; and a slower road, which includes cortical pathways and is responsible for fine-grained processing and the conscious feeling of fear (LeDoux, 1996). By analogy, or even involving shared mechanisms, implicit awareness might exist due to dual representations, one involving a conscious awareness mechanism and another involving direct emotional and behavioral regulation in response to success or failure. A similar suggestion has been made by Vocat and Vuilleumier (2010), who posited the existence of two forms of monitoring, one explicit and another implicit, with patterns of anosognosic response being explained by
dissociation between them. It is interesting to note, considering the above framework suggesting subcortical routes for processing of implicit information, that even though current evidence about neural correlates of implicit awareness is still incipient, there is some support for the involvement of subcortical regions in this phenomenon (Moro et al., 2011).

**MODELING ANOSOGNOSIA AND IMPLICIT AWARENESS USING THE CAM MODEL**

There is very little theoretical modeling of the types of neurocognitive processes that would explain implicit awareness. In this paper we present the Cognitive Awareness Model (CAM, see Figure 2) (Agnew & Morris, 1998; Morris & Hannesdottir, 2004), which is a more general model used to explain anosognosia, but from the outset accommodated implicit awareness and has been reformulated recently by Morris and Mograbi (2013). The reformulation is explained there in more detail, but for the purposes of relating to the issue of implicit awareness we provide it in outline in this paper. At the center of this model is the notion of a metacognitive awareness mechanism (MAS), which receives input concerning appraisal of performance efficacy or failure from cognitive comparator mechanisms (CCMs), that monitor information derived from task performance and compare it to longer lasting memory representations concerning the self, termed the Personal Data Base (PDB). Mismatches between information derived from immediate experience and longer stored self-knowledge gives rise to awareness in the MAS. The information that filters through to the CCMs is abstracted through various mnemonic mechanisms, starting with raw “episodic” autobiographical memory material and then via the autobiographical conceptual memory system (ACMS). This process of consolidation, described in more detail by Morris and Mograbi (2013), takes place over a long time period and leads to the establishment of self-knowledge, including knowledge concerning personal efficacy, a principle facet of awareness of disability. An additional aspect of the model is the General Memory System, included here to distinguish between specialized cognitive systems representing the self and self-representation that can occur using a more generic system, with the notion of a surrogate self-representation as a possibility (Morris & Mograbi, 2013). The model components are thought to be instantiated in different brain areas. The PDB and ACMS would be linked to memory networks such as the temporo-parietal junction, anterior temporal area, and medial temporal lobe structures, whilst the MAS should be seen as a feature emerging from connectivity of brain areas,

![Figure 2. The Cognitive Awareness Model (Morris and Mograbi, 2012).](attachment:figure2.png)
instead of a module. Nevertheless, in the absence of empirical evidence, these suggestions remain speculative.

A feature of this model, developed in the context of understanding anosognosia in AD, is that anosognosia may not have a unitary cause, but impairment can occur in different parts of the system to produce the phenomena associated with unawareness. In relation to implicit awareness, however, an additional feature was added to the model, creating dual output from the comparator mechanism, including an implicit route that modulates affect and behavior and can give rise to such phenomena as the “emotional awareness” investigated by (Mograb, Brown, Salas, & Morris, 2012). Impairment in the MAS would give rise to anosognosia, but this route could still provide a means by which there would be emotional reaction to failure and behavioral adjustment. Hence, this part of the model was incorporated to take into account implicit awareness, in this case in relation to dementias such as AD. It should be noted that intactness in subparts of the main network, such as the PDB and the CCMs, would still be necessary for this phenomenon. In AD it has been proposed that degradation of the mnemonic mechanism, including the PDB, is the cause of anosognosia (Mograb, Brown, & Morris, 2009; Morris & Mograb, 2013), so the notion would be that, nevertheless, some information was being produced at a comparator stage, sufficient to lead to implicit awareness. In addition, there may be information in the PDB and ACMS concerning illness representation or efficacy that may be accessed implicitly, subject to social or cultural factors, for example, accounting for the emotional Stroop phenomenon found by Martyr et al. (2011). In summary, in dementias such as AD the idea is that the range of cognitive impairments are monitored in faulty fashion, with failure to fully consolidate knowledge of loss of personal efficacy, but sufficient information to produce emotional and behavioral change, including the emotional bias phenomena demonstrated through experimental study.

In this model, the processes producing awareness of deficit invoke relatively higher-order cognitive functions. The MAS leads to a phenomenological sense of awareness, and is considered here as an emergent process rather than a system, involving widespread connectivity between multiple brain systems, including those responsible for the CCM and PDB. In contrast, in anosognosic disorders such as hemiplegia, explanations have been proposed in terms of more local mechanisms, embedded in the brain mechanisms concerned with the sensory motor control of action, again including comparator mechanisms but this time very specific. Computational models of the motor system incorporate notions of corollary discharge, in that when movement is made not only is the primary motor sequence initiated via the pyramidal system, but a parallel signal is created that represents the movement intention. When effector mechanisms are stimulated, such as the movement of a limb through neuromuscular activation of muscle fibers, sensory signals then converge with this signal at a comparator mechanism. Normally, the two pieces of information are congruent, but impairment in the motor system produces a mismatch between the signal of intention and the sensory record. Different theories of anosognosic hemiplegia have been proposed taking into account this framework (Jenkinson & Fotopoulou, 2010). According to the feed-forward hypothesis (Heilman, Barrett, & Adair, 1998) hemiplegic anosognosia would be a failure of the action-intentional system, such that there is no feed-forward signal to use at the comparator stage. An alternative view suggests that damage to the comparator mechanism would lead to an inability to detect the mismatch between expected and implemented movements (Berti, Spinazzola, Pia, & Rabuffetti, 2007).

Finally, there is some recent evidence that patients with anosognosia for hemiplegia may have a general difficulty updating beliefs (Vocat, Saj, & Vuilleumier, 2012) or with reality monitoring (Jenkinson, Edelstyn, Drakeford, & Ellis, 2009), including for non-motor information, which would support the view that anosognosia is caused by a combination of primary motor/sensory deficits with higher-order attributional/monitoring distortions (ABC model; Vuilleumier, 2004).

Accordingly, in the reformulated CAM model it is proposed that there are two main types of awareness comparator mechanisms as follows:

1. Type 1 comparators, also termed sensory motor integration comparators (SMICs), which are local modularized feed-forward mechanisms, operating at a lower sensory or motor level. These occur more immediately and automatically, and include the functioning of thalamic and basal ganglia subcortical monitoring systems and their associated cortical-subcortical loops. It is proposed that neurocognitive mechanisms associated with other modularized functions, such as vision and language, also occur at this level with as yet unspecified comparator mechanisms. As with anosognosic hemiplegia, failure to produce mismatch signals produces modality-specific anosognosic deficits.
such as those associated with hemianopia and aphasia.

2. Type 2 comparators, higher-order secondary mechanisms, labeled CMMs, and their associated mechanisms function at a high level and receive information that filters through mnemonic and semantic encoding mechanisms into higher-order cognition, consolidated to provide appraisal or awareness of deficit. These are typically mechanisms that are damaged in neurodegenerative disorders such as AD, rather than SMICs, which we suggest are relatively preserved in earlier stages of dementia; they are supported by integration of mnemonic, attentional, and executive networks that are associated with temporal, parietal, and frontal lobe function. In a normally functioning mechanism, Type 1 comparator mismatch signals eventually cascade up into higher-order systems to produce awareness through Type 2 awareness mechanisms.

To what extent can this model be used to explain implicit awareness in conditions such as hemiplegic anosognosia? Firstly, because the feed-forward control mechanisms operate below the level of the higher-order mechanisms and are modular, this leaves open the possibility that other sources of information concerning disability can be fed up the system and produce awareness of deficit as processed by the MAS. In other words, this information is able to bypass the specific Type 1 mechanism. There are a number of implicit awareness phenomena that can in principle be explained in this fashion. This includes, in particular, thoughts and cognitive biases that reference disability or the effects of disability, such as symbolically referencing impairment (Gainotti, 2005), use of metaphors (Prigatano & Weinstein, 1996), incorporating disability language (House & Hodges, 1988), and emotional attentional biases to disability words or phrases relating to the condition (Fotopoulou et al., 2010; Nardone et al., 2007). It can be argued here that information concerning the effects of disability, rather than the underlying process, can nevertheless be encoded, for example, through conversations concerning disability as a distressing topic for carers, or the experiences of the effects of disability. This type of material could be processed initially by other intact neurocognitive mechanisms, for example, those to do with language function and other intact features of sensory and motor processing. In other words, impairment in a Type 1 comparator associated with motor control is sufficient to prevent the production of information that would produce concurrent awareness of hemiplegia, but substantial sources of other information are available and may account for some of the phenomena associated with “implicit awareness,” in particular, emotional responses/symbolic references to deficit-related material and general adjustments to disability, such as accepting help. It is also possible that acknowledgement of deficit when observed using a third-person perspective, as in the case of patients who are shown videos of themselves (Fotopoulou, Rudd, Holmes, & Kopelman, 2009), occurs because certain patients can be cued by using these pathways.

Other implicit awareness phenomena may be explained in a different fashion. Specifically, this includes the phenomenon reviewed earlier that patients with hemiplegia will adjust their motor responses either by switching to unimanual unaffected limb responding (Cocchini et al., 2010) or when grasping heavy objects moving their unaffected limb to the midline (Moro et al., 2011). Since these phenomena refer to “online” immediate responding, it suggests that there may have been error detection at the sensory motor integration/comparator level or there would be no motor adjustment. Alternative sources of information could be deemed too indirect to produce the very specific change in motor responding in this instance. This finding suggests that the lack of awareness of deficit is operating at a high level in information processing. In terms of the CAM model, one explanation is that damage to Type 2 mechanisms, such as the CCMs, also produces lack of awareness of deficit in hemiplegia, despite lower level motor adjustment. There is the possibility that faulty input to the CCMs or potential disconnection between Type 1 and Type 2 comparators could result in higher-order secondary mechanism-type impairment. It should also be emphasized that there are considerable variations in patterns of association between explicit awareness of hemiplegia and the implicit adjustment responses (Moro et al., 2011). Some patients show lack of explicit or verbal awareness and also show no adjustment but Moro and colleagues (2011) have identified one patient (PS) who appeared to declare their hemiplegia, but showed no behavioral adjustment on their tasks. This type of patient is of interest because it may suggest some double dissociation between types of awareness comparator mechanisms as indicated by the CAM model. Specifically, it is possible that Moro et al. (2011) have identified in the same experimental study patients who have damage restricted to Type 2 mechanisms, and who show motor adjustment but also those who might only have impairment at the Type 1 level and are able to develop awareness through parallel processing reaching an intact higher level mechanism.
Finally, there are patients who show both lack of explicit awareness and motor adjustment who might have damage to both types of mechanisms. Also, of note, is the finding in the same study that patients who did not show motor adjustment have lesions involving the middle temporal cortex as well as white subcortical matter around and anteriorly to the basal ganglia, implicating these regions in lower level awareness processes.

In summary, included in the reformulated CAM model, two types of comparator mechanisms are postulated, designated Type 1 and 2 comparators with their respective implicit outputs and these provide additional explanatory power related to different phenomena associated with implicit awareness, also summarized in Figure 3. At a sensory/motor level, the Type 1 comparators, SCIMs, give rise to localized implicit sensory or motor output, but these can be bypassed to a higher level. Integrating higher-order cognition, including mnemonic processing, the Type 2 CMMs provide the basis for implicit behavioral and affective regulation that include, for example, what could be termed “implicit emotional awareness.” The Type 2 CMMs gives rise to a range of implicit awareness phenomena, but it should be noted that here we do not specify fully the output mechanisms, drawing in a range of explanatory concepts, such as motivational, emotional, and behavior change mechanisms beyond the scope of this paper. Damage to these neurocognitive systems can occur at single or multiple levels to produce different awareness deficits.

**Figure 3.** CAM model lesion scenarios producing differential implicit awareness phenomena. 
MAS—Metacognitive Awareness Mechanism; CCM—Cognitive Comparator Mechanism; SICM—Sensory Motor Integration Comparators; Dashed arrow represents parallel route for information bypassing specified SICM; Right-hand arrows indicate behavioral/affective regulation versus motor implicit outputs. Texture represents damage to relevant neurocognitive mechanism. Scenario a: Implicit awareness in dementia: The SICM mechanisms are intact. Incomplete and degraded material activates the CCM sufficient to produce behavioral/affective regulation, but without explicit awareness due to damage to the MAS. Scenario b: Implicit awareness in anosognosic hemiplegia producing behavioral affective regulation: Damage to the SICM means that “online” adjustment of motor responses does not take place, but preserved CCM activity enables thoughts and cognitive biases suggestive of implicit awareness to occur, CCM receiving material through parallel intact neurocognitive mechanisms; Scenario c: Implicit awareness in anosognosia hemiplegia with “online” adjustment of motoric response: Damage to CCM but a preserved SICM results in motor adjustment; Scenario d: No implicit awareness: Damage to both the relevant SICM and CCM/MAS complex results in no implicit awareness.
INTERACTIVE PROCESSES AND IMPLICIT AWARENESS

In the above theorizing we present awareness processes as essentially modular, perhaps with very higher-order processes such as those associated with the MAS existing as a more diffuse emergent property of information processing. However, here we present interactive notions of cognition that incorporate more dynamic processes.

Implicit awareness caused by top-down modulation

Whilst awareness mechanisms may have modularity, also supported by double dissociation, it is productive to think about awareness as a phenomenon which can be conceptualized at different processing complexity levels, ranging from a basic material to a higher-order attributional level (Ownsworth et al., 2006). These different levels interact, with awareness being seen as an emergent feature of neurocognitive network which is constrained by higher-order processing. At this higher-order level, personal (e.g., pre-morbid personality traits and beliefs about illness), social (e.g., global awareness about a condition), and cultural factors (e.g., collective values) influence awareness (Clare, 2004; Mograbi, Ferri, et al., 2012; Ownsworth et al., 2006).

It is possible that these factors may modulate the emergence of information into explicit awareness, whilst still allowing the occurrence of implicit effects. Indeed, it has long been known that cognitive functions, such as memory and perception, can be affected by higher-order processes, such as attributions, expectations, and socially constructed values.

Additionally, different authors have emphasized how cognitive systems strive to keep coherence (Friston, 2010). One way of achieving this is to limit progression of dissonant information from lower to higher levels, either through blocking or distortion. Human brains are organized such that extensive re-entrant connections exist between regions processing information at different complexity levels (e.g., cortico-cortical connections between association and primary sensory areas; cortico-subcortical loops). Because of that, higher levels (i.e., areas processing more complex information) can influence the course of ascending information from lower levels. We offer here two accounts of top-down modulation: “micro-modulation” across the system and central active modulation.

The former perspective privileges the idea of modulation of information as an inherent property of cognitive systems instead of the activation of a second- or higher-order system. This notion has been discussed, for example, in the formulation of a free-energy principle of the brain (Friston, Kilner, & Harrison, 2006). According to this view, the brain acts as a Bayesian inference machine (i.e., creating probabilistic predictions which are updated as further information is added to the system) about unknown states (Friston, 2010). A key notion in this model is the need to minimize entropy in the system by constraining the mismatch between prior expectations and incoming information. In order to do that, prior beliefs influence incoming sensory data, which by definition is ambiguous (Edelman, 2003; Edelman, Galy, & Baars, 2011), and action, enabling selective sampling of the world (Friston, Adams, Perrinet, & Breakspear, 2012). Under this perspective, cognitive systems can be described as mechanisms which avoid/suppress surprise by probabilistic predictive models (Friston, 2010).

Although empirical testing of a broad principle of brain functioning is bound to remain limited, numerous examples have been provided about inherent modulation of information in cognitive systems. For example, it has been shown that as perceptual information travels through visual areas, higher levels of the neural hierarchy relay information back to primary areas, which may reflect top-down modulation of perception (e.g., Belke, Humphreys, Watson, Meyer, & Telling, 2008; Cardin, Friston, & Zeki, 2011; Moores, Laiti, & Chelazzi, 2003; Murray, Kersten, Olshausen, Schrater, & Woods, 2002; Rao & Ballard, 1999). It has been hypothesized that, at each level, inference based on prior beliefs and expectations about objects will constrain information emerging from lower levels (Friston, 2010). This resonates with the influential notion of perceptions as hypotheses (Gregory, 1980), and suggests that perception would be a compromise between sensory data and priors (Brown & Friston, 2012). A similar dynamic has been proposed in relation to memory functioning, and although less empirical evidence is available, research has suggested the influence of self-reference or previous knowledge (some of which is possibly implicit in nature) on encoding, consolidation, and retrieval of new memories (Conway, 2005; Ellis & Ralph, 2000; Wilson & Ross, 2003).

These notions have already been considered in relation to unawareness, for example, in the context of anosognosia for hemiplegia. Fotopoulou (2012, 2013) suggested that a potential explanation for anosognosia is the conflict between afferent interoceptive or exteroceptive information about movement with prior beliefs and expectations. The current version of the CAM includes the idea that information may be
filtered as it progresses to higher levels of the system (Morris & Mograbi, 2013). Specifically, the model also proposes the existence of affective gating mechanisms, which would restrict access of new data conflicting with previous knowledge, avoiding cognitive dissonance. In addition, the fact that the model includes different levels of consolidation for information about self-ability allows for the influence of previous knowledge on the formation of new memories regarding self-efficacy.

An alternative view is that central top-down modulation of information carried out by the prefrontal cortex (PFC) may play a role in regulating awareness. The PFC has long been implicated in executive control of information via processes such as attention, maintenance of information in short-term memory, updating of information, and switching from one cognitive process to another (Baddeley, 1986; Luria, 1980; Shimamura, 2000). Inhibition has also classically been linked to frontal lobe function, with lesions to this brain area leading to impulsiveness and inability to consider risks (Cummings, 1993). This prefrontal control would also extend to regulation of emotions, in particular, via orbitofrontal cortex activity (Rule, Shimamura, & Knight, 2002). It is possible that awareness may be modulated in a similar fashion as other cognitive functions. This is a particularly compelling hypothesis when considering the ways through which PFC modulation affects cognition, via processes such as suppression and inhibition of information and selective attention. Top-down modulation would provide visibility for certain features of experience, while at the same time filtering and blocking parts of it. This would be done, for example, based on beliefs, emotional states, and conceptual information, keeping coherence in the system by adjusting new experiences to previous knowledge. The main difference in relation to “passive” micro-modulation of information, described above, is that PFC regulation would be a higher-order “central” form of mediation instead of a property of the system. Considering this framework, it is also possible to think about PFC regulation as an on-off control of information, with passive modulation being more graded.

**Implicit awareness and a failure of bottom-up integration**

We highlighted that awareness should be considered an emergent feature of the brain instead of being thought of as caused by activations in a single area. The concept of emergence is based on connectionist models and suggests that, even though there are hierarchical relations between networks, neural systems self-organize without the need of a commanding center (Varela, Thompson, & Rosch, 1991). According to this view, awareness could be seen as the result of widespread connections between brain areas (Edelman, 1992; Tononi, 2004, 2007). This theoretical approach helps to understand a main feature of awareness, namely its integrative power (i.e., the so-called binding problem of consciousness; Revonsuo, 1999). The connectivity between diverse brain areas allows the provision of a unifying context for ongoing experience. Here, awareness can be considered as the binding of different elements in a scene, with re-entrant connections between perceptual and association areas tying them up with context, and language and abstract concepts ultimately contributing to this process.

Connectivity between brain areas would endow ongoing experience with a second-order reflection (Edelman, 1992, 2003). The notion that awareness is characterized by a second-order operation has been echoed in a series of theoretical positions (for a review, see Dienes & Seth, 2010). For example, Weiskrantz (1997) considers that awareness of an event is endowed by a second-order “commentary system,” i.e., a reflection upon experience. This “commentary system” would be the result of cortical-limbic loops, and would provide access to different types of information and wide contextual linking (Weiskrantz, 1997).

At a phenomenological level, this commentary allows individuals to manipulate information in relation to other knowledge (c.f. the concept of access consciousness; Block, 1995). It is this type of commentary that is missing in cases of implicit residual abilities, such as in priming: the individual may show faster reaction times when responding to a primed item or may form conceptual associations, but there is no awareness of the relationship between stimuli (Weiskrantz, 1997, p. 76). When processing something implicitly, the individual lacks the ability to manipulate the information in relation to other contextual knowledge. For example, implicit awareness may lead to increased emotional response to illness-related stimuli, but without explicit awareness patients possibly would not be able to trace this down to a cause or elaborate on the reasons of their responses. The relationship between illness-related material and emotional reactions is present, but anosognosic patients are unaware of this relationship. This is in agreement with the assumption that one of the most prominent functions of awareness is to increase access between separate sources of information (Baars, 1997; Block, 1995).
THEORETICAL AND CLINICAL IMPLICATIONS

The notion of implicit awareness has important clinical and theoretical implications. From a theoretical point of view, it suggests that implicit processing of information extends to more complex stimuli, also encompassing an attributional level. This follows a historical trend. Initially, cognitive views about implicit phenomena considered them to be basic processes, limited in scope and complexity. While it is indeed true that many basic cognitive processes are implicit, evidence gathered in the past decades has suggested that not all implicit cognitive processes are necessarily basic, mirroring the development of priming research, which moved from understanding perceptual forms to conceptual and affective priming, with recent evidence suggesting that complex implicit stimuli can influence social judgements and decision making (Mograbi & Mograbi, 2012). This is not to say that implicit phenomena are not qualitatively different from explicit declarative cognition. As we pointed out above, awareness serves a series of purposes, including extending access to content and allowing manipulation of information. Nevertheless, if results from the implicit awareness literature have been interpreted correctly, they provide further evidence for the idea that implicit processing can be complex in nature. In this paper we have sought to explore the complexity of this topic by drawing out the distinction between the phenomena as observed in dementia and hemiplegia, but as has been indicated in cognitive modeling, the distinction between the two conditions are not quite as definitive as we have portrayed. For example, in anosognosia for hemiplegia, integrity of higher-order cognitive processes has been considered in addition to the feed-forward and sensory-motor elements, and we do not discount the involvement of the latter in dementia, particular in vascular dementia or later stage disorder. In summary, the mechanisms underlying anosognosia are multifaceted and interacting mechanisms need to be considered to understand the range of phenomena associated with different anosognosic conditions and implicit processes.

Clinically, the notion of implicit awareness has important value for assessment and rehabilitation procedures. For example, in relation to neurodegenerative disorders, such as Alzheimer’s disease, it suggests that patients may respond negatively to the experience of failure during assessment despite not explicitly acknowledging impairment, highlighting the need to tailor evaluation procedures to the person’s level of performance (Mograbi, Brown, Salas, et al., 2012).

Regarding rehabilitation procedures, an approach based on preserved implicit abilities has been proposed in the context of patients with cognitive disorders (Harrison, Son, Kim, & Whall, 2007). According to this perspective, constant practice of preserved implicit abilities would compensate for loss of explicit processing, leading to better preservation of functional abilities. Interventions focusing on adaptation to the patient environment, such as problem adaptation therapy, have been shown to successfully reduce depression and disability in older adults with depression and cognitive impairment (Kiosses, Arean, Teri, & Alexopoulos, 2010). Problem adaptation therapy circumvents behavioral and functional limitations by including adaptation tools, such as calendars, checklists, pictures, and diaries, and tailoring the intervention to the patient’s own living environment and this can occur without explicit memory for the learning experience (e.g., Camp, Foss, O’Hanlon, & Stevens, 1996). This type of adaptation relies on implicit memory, but also implicit awareness may be involved in providing a motivational basis for adaptation. These concepts can be extended to the treatment of patients with other neurological conditions.

For clinicians and researchers, the notion that awareness can manifest itself not only explicitly but also implicitly suggests that actual patient behavior needs to be considered equally to gain a full understanding of awareness. From a clinical point of view, it is less relevant if patients are aware of their condition or if they attribute it to the right cause, provided that they comply with treatment, participate in rehabilitation procedures, and avoid risk-taking behavior. Similarly, it is possible that the association between caregiver burden and unawareness in dementia is caused by the practical effects of unawareness (e.g., refusing care when needed), in which case implicit awareness would help to mitigate carer strain. Given that there is a strong association between explicit awareness and depression in various clinical populations (David, 2004), implicit processing may offer the possibility of preserved adaptation to the patient’s condition, while avoiding increased rates of depressive symptoms.

FINAL REMARKS

In conclusion, despite clinical evidence supporting the existence of implicit awareness, a label that has been considered an oxymoron, empirical work is needed to determine its causes and correlates. Given the numerous theoretical and clinical implications, research
efforts into this phenomenon may pay valuable dividends. Perhaps oxymora, as writers would suggest, are indicative of a complex reality. In which case, we should embrace this complexity and can agree with Shakespeare, when Macbeth says: “My dull brain was wrought with many things forgotten.”

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On the contribution of unconscious processes to implicit anosognosia

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Abstract: Mograbi and Morris present a review of the literature on anosognosia of hemiplegia and dementia, including Alzheimer’s disease. Their review focuses on aspects of implicit anosognosia. The authors’ viewpoint is supported by the presentation of a general model on implicit anosognosia in Alzheimer’s disease. The notions have important implications for clinical management, in particular, failure of treatment in Alzheimer’s patients.

Mograbi and Morris are to be applauded for raising a serious discussion about implicit anosognosia in hemiplegia and dementia, including Alzheimer’s disease. Their article includes a thorough review of the literature on phenomena suggesting implicit anosognosia in patients. Their argument and conclusions raise very interesting and important points, particularly regarding the clinical implications of such phenomena. The Cognitive Awareness Model formulated by the authors (Morris & Mograbi, 2013) is presented, in more detail, to explain the symptoms of implicit anosognosia. The model is based on the intersection of three concepts: The metacognitive awareness mechanism (MAS), the cognitive comparator mechanisms (CCMs), and the Personal Data Base (PDB). The authors show how a disturbance of either one of the three concepts can cause implicit anosognosia phenomena. This model is cogently built on both clinical observations and experimental studies.

We would like to add two further arguments in support of such a model. The first concerns the well-established evidence for dissociations between unconscious and conscious processing in various domains of neuropsychology. For example, in neglect patients, stimuli presented within the left hemifield (which go unreported) can still affect patient’s behavior (Marshall & Halligan, 1988; Vuilleumier, Schwartz, Clarke, Husain, & Driver, 2002) and activate selective brain areas (Mogib, 2001, NPsya, 2002). There may also be implicit memory processing in amnesics, without the capacity for explicit memory (Gisky, Schacter, & Tulving, 1986), or implicit recognition in some prosopagosis (see Schacter, 1992). Thus, as suggested by Mograbi and Morris, it is plausible that although neurological and neuropsychological assessments indicate a lack of awareness of motor (or any other) deficit at a conscious level, the brain may still unconsciously detect incongruence between expectations and actual efficiency.

Another argument concerns evidence for some automaticity in mechanisms of error processing which have been widely studied in the field of cognitive neurophysiology. A very specific neural signature of error detection can be measured with event-related potentials (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000), which consists of two main components: The ERN (for Error-Related Negativity) and the Pe (for Positive amplitude and later latency), whose generators might involve partly distinct brain circuits (Vocat, Pourtois, & Vuilleumier, 2011). Crucially, several studies reported that error detection processes (particularly the ERN) may be triggered without people being aware of their errors. Furthermore, brain regions typically associated with ERN generators are similar to those potentially implicated in anosognosia, including insula but also anterior cingulate and striatum (Ullsperger, von Cramon, & Muller, 2002; Vocat, Staub, Stroppini, & Vuilleumier, 2010). These regions also overlap with a network coding for the saliency of behaviorally relevant events, such as pain and emotions (Menon, 2011).

Thus, the current review of the neuropsychological and anatomical correlates of illness denial after brain lesion converges to suggest a multifaceted phenomenon with a multifactorial origin, presumably reflecting the combined effect of different elementary deficits (see also Vuilleumier, 2004). Based on this critical and scholarly review, the model of Mograbi and Morris dovetails nicely with and integrates the general framework of anosognosia that we recently proposed (Vocat & Vuilleumier, 2010; Vuilleumier, Vocat, & Saj, 2013) to account for impaired awareness of self-efficiency in
patients with brain damage (e.g., stroke) or without (e.g., hysterical conversion). In this framework, the assessment of one’s own (e.g., motor) performance depends on access to a plurality of information sources (e.g., motor, proprioceptive, visual attention), which is performed in parallel at both the conscious (explicit) and unconscious (implicit) levels. The existence of distinct pathways (explicit and implicit) for motor control and self-monitoring makes it possible to envisage different forms of anosognosia, involving different degrees of dysfunction in one or the other of these levels, or in both.

Mograbi and Morris make now one further valuable step by showing that Alzheimer’s patients may have preserved emotional reactivity to their failures, even when denying them. This last point is very important in the evaluation, treatment, and rehabilitation of anosognostic patients or dementia, and the management of their deficits, because patients may exhibit unconscious affective responses to these and this may in turn influence their behavior and impairments in everyday life.

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The interaction between implicit and explicit awareness in anosognosia: Emergent awareness

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Abstract: The dissociation between implicit and explicit forms of awareness has been described in various neurological diseases. The way in which these forms of awareness integrate in order to permit the necessary unitary experience of self remains unclear. Here, the hypothesis that a form of emergent awareness (i.e., the emergence of a verbal acknowledgment of deficits as a consequence of attempting to act) may represent a link between implicit and explicit components is proposed for discussion.

The existence of various different subtypes of anosognosia is the topic of an intense debate in neuroscience and neuropsychology due to its potentially twofold contribution, both in comprehension of awareness mechanisms and in clinical approaches to neurological diseases (Marcel et al., 2004; Ramachandran & Ramachandran, 1996). By means of a comparative analysis of the literature concerning implicit awareness for motor deficits in hemiplegia...
and cognitive deficits in dementia, the authors suggest an updated version of the Cognitive Awareness Model (Agnew & Morris, 1998). In this model, they propose that two types of comparators (Type 1 and 2) are damaged in the different expressions of anosognosia and in dissociations between implicit and explicit awareness.

Thus, the question that remains in large part unanswered concerns how implicit and explicit forms of awareness can integrate to give the habitual, global, individual experience of unitary self-awareness.

The authors suggest considering awareness as an emergent feature of brain functioning, endowed with strong integrative power. This view finds confirmation in studies of neuroimaging in anosognosia after stroke that show the involvement of very large cortical and subcortical networks (Fotopoulou et al., 2010; Moro et al., 2011).

From a phenomenological perspective a specific subtype of awareness, called precisely “emergent awareness” has been described (Crosson et al., 1989). In this condition, subjects who are unaware of the consequences of their deficits deny their paralysis when verbally asked, but they become in some way aware of their deficits when faced with a request to perform an action. This indicates that an intention to act (and/or actually acting) may modify explicit, verbal knowledge of the deficits. Moreover, it induces the use of strategies aimed at correcting their behavior, which are not employed in subjects who do not show any signs of emergent awareness (but have, for example, spared implicit awareness; Moro et al., 2011). Finally, emergent awareness might guide decision-making processes in complex contexts, for example, during procedures requesting a subject’s consensus to medical treatment (Clinical Competence; Gambina et al., 2013).

A related question also concerns the possible role of emergent awareness in Personal Data Base updating. Recent lesion studies (Moro et al., 2011; Vocat et al., 2010) in anosognosia for hemiplegia have shown the involvement of memory networks in the persistence of symptoms over time. In line with this, the CAM model emphasizes the role of autobiographical conceptual memory, self-knowledge, and sense of personal efficacy in building the Personal Data Base (PDB). This sends long-lasting memory information to the comparator systems to compare them to information concerning the results of actual performance. In self-knowledge processes, implicit elements, such as memories, meaning and beliefs concerning social roles in specific social context, cognitive styles, and competences are necessarily included. In updating PDB with respect to new conditions, implicit and explicit memory need to be integrated in the formation of long-lasting memory representations.

The mechanisms underlying emergent awareness have until now not been clear, but the involvement of cognitive and metacognitive processes (necessary to intention, monitoring, and evaluation of performance) and executive processes (for action, memory, speech, etc.) makes this form of awareness a possible candidate as one of the components of integration between implicit and explicit awareness (and memories).

This hypothesis is also supported by lesion results showing that, while implicit awareness deficits are linked to lesions involving the middle temporal cortex and the white subcortical frontal matter (anterior and around the basal ganglia), the lack of emergent awareness is linked to lesions of pathways connecting the parieto-temporal and frontal cortices (Moro et al., 2011; Vocat et al., 2010).

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Denial, anosodiaphoria, and emotional reactivity in anosognosia

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Abstract: A central flaw in models of anosognosia is to consider metacognitive awareness and affective regulation and responsiveness as separate functional modular subsystems. This line of reasoning leads to an “either or” conceptualization of the probable causes of implicit awareness in anosognosic patients. Neuroscience research and clinical observations of patients suggest that anosognosia is often associated with a change in the affective status of the individual as well as a change in their explicit verbal descriptions of themselves. Studying anosognosic patients over time and including measures of psychological denial and anosodiaphoria are necessary when interpreting markers of implicit awareness.

Previous knowledge and present beliefs about the “self” are not purely cognitive or perceptual in nature. They automatically evoke feelings. If I consider myself intelligent and successful, that knowledge or belief is associated in an automatic way with feelings of joy and pride. If I consider myself inept and impaired, the “thoughts”, beliefs, or personal perceptions automatically are associated with feelings of loss, sorrow, anger, and at times anxiety and depression. I can also become more cautious in what tasks I choose to engage. How I manage those feelings is clearly related to my personal developmental (i.e., psychodynamic) history.

A central logical flaw in models which separate metacognitive awareness from affective regulation and responsiveness is that they are considered separate functional modular systems when in fact they are most likely not. Davidson, Scherer, and Goldsmith (2003) have noted: “The notion that emotions are somehow limbic and subcortical and cognitions cortical is giving way to a much more refined and complex view..... It is simply not possible to identify regions of the brain devoted exclusively to affect or exclusively to cognition” (p. 5). These observations are important when conducting studies on anosognosia in its implicit and explicit forms.

It is now accepted that anosognosia can exist for a single lost function (like motor movement) and be preserved for another lost function (like vision) in the same patient. It is also now recognized that “anosognosia may not have a unitary cause” (from Mograbi & Morris, this issue). Thus, when studying emotional reactivity as a marker of implicit awareness in anosognosic patients several factors must be considered. In some patients implicit awareness may reveal a partial awareness, for others a denial or psychological process of coping, and for others represent the intactness of a cognitive non-conscious information processing system. In some patients, a combination of these factors seems likely.

Accounting for the potential moderating effect of denial and anosodiaphoria in emotional reactivity studies is crucial. Seldom are measures of denial used in studies of anosognosia (Prigatano, 2012). Likewise, documenting the presence and degree of anosodiaphoria in patients who have implicit awareness of anosognosia has not been done.

It is one thing to infer implicit awareness in a patient who makes derogatory comments about a limb after a stroke (Prigatano & Weinstein, 1996) versus one who shows apparent indifference. If these two groups show different patterns of responding during emotional reactivity studies it could well help separate patients who present with denial (perhaps the former group) from those who have impaired awareness without denial. In short, the co-morbid features of patients who show reported implicit awareness in anosognosia must be included to have a better understanding of probable etiologies and to construct useful models of the phenomena.

A final, but related issue, is to specify when in the recovery course from a brain lesion (or conversely when in the course of brain deterioration) does the patient demonstrate anosognosia in either its explicit or implicit forms. A key question is how does it change with time? Vocat and Vuilleumier (2010) have provided evidence that different patterns of brain lesions are observed when anosognosia for hemiplegia (AHP) exists within three days after a stroke and then resolves, versus if the AHP exists past one week following a stroke. It is likely that complete AHP is common when lesions are “large” and come on abruptly. Bilateral cerebral dysfunction may also be common when a complete syndrome of anosognosia is observed (Prigatano, Matthes, Hill, Wolf, & Heiserman, 2011). Denial phenomena are more likely to occur during the phase of resolving anosognosia or partial awareness. Thus the mechanisms accounting for implicit awareness in anosognosic patients must consider the time frame and co-morbid neurological, neuropsychological, and psychiatric features of the patients studied.
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Connections between mechanisms for anosognosia and implicit memory

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Abstract: Mograbi and Morris review work highlighting an interesting phenomenon whereby individuals are explicitly anosognosic for their deficits despite intact expression of implicit awareness. Parallels exist between this phenomenon and recent cognitive neuroscience findings demonstrating intact memory test performance despite unawareness of performance. We discuss these parallels with regard to the proposed CAM model. Given that it is possible to investigate the neurological underpinnings of explicit and implicit processing in memory tasks, methods from cognitive neuroscience may offer substantial insight into implicit awareness in anosognosia in various forms of dementia as well as in addition to advancing theoretical understanding of anosognosia broadly.

Mograbi and Morris review mechanisms for anosognosia that bear interesting similarities to recent cognitive neuroscience findings regarding implicit memory. Anosognosia is the lack of awareness of a clinical impairment, such as in hemispatial neglect and potentially various dementias. Anosognosia epitomizes several themes in contemporary implicit memory research, including the distinction of performance, with versus without awareness, the possibility that performance awareness is a graded phenomenon, and the many amazing capabilities that do not require conscious awareness (Ryals & Voss, forthcoming). We wish to highlight these connections and suggest how cognitive neuroscience implicit memory methods and findings could enrich investigations of anosognosia.

Although most anosognosia research involves motor and perceptual impairments, the phenomenon is similar to instances whereby individuals can lack awareness for aspects of performance in memory tests. Indeed, many findings indicate that implicit and explicit performance reflects distinct neural signals, and these signals can be used to better understand the interactive nature of implicit and explicit processing (Dew & Cabeza, 2011; Voss, Lucas, & Paller, 2012). For instance, distinct measures of behavioral performance and electrophysiological signals occur when healthy individuals perform with high accuracy yet lack any awareness that performance is accurate (Ryals, Yadon, Nomi, & Cleary, 2011; Voss & Paller, 2009).

The authors mention the possibility that anosognosia occurs for cognitive impairment in dementia. Indeed, even mild cognitive impairment (MCI, a prodromal cluster of Alzheimer’s symptoms) has been associated with variable awareness of memory performance (Cosentino et al., 2007). Recent findings have highlighted the use of implicit memory tests as assays for low levels of preserved function even in cases of extreme dementia (Kessels, Remmerswaal, & Wilson, 2011); thus, methods used to identify neural correlates of implicit memory could likewise be used in anosognosia more broadly. Mograbi and Morris note that hemiplegic anosognosics unknowingly adjust performance over time despite their lack of awareness. If this finding generalizes across anosognosias (i.e., in dementia), it would provide an excellent means of identifying signals of implicit performance (e.g., those that vary over time with unaware performance improvements).

Identifying neural signals of the awareness decrements in anosognosia would ultimately allow better tests of putative mechanisms. For instance, a crucial component of the CAM model proposed by Mograbi and Morris is the distinction between a cognitive comparator mechanism (CCM) that maps incoming information to existing representations and a Metacognitive Awareness System (MAS) that supports reflective monitoring. MAS and CCM interactivity produces accurate portrayals of task successes and failures in healthy individuals. This model is consistent with the
dynamic hierarchical interplay in the frontotemporoparietal system proposed to support awareness in memory. Lack of awareness could simply reflect a breakdown of this interactivity. Alternatively, implicit function could be supported by a distinct set of functions (e.g., Reber, 2013), whereby normal performance (with and without awareness) is dictated by complex interactions between implicit and explicit functions. As Mograbi and Morris propose, it is unlikely that anosognosia is due to a simple neural impairment. The search for mechanisms could thus be enhanced by considering the interactive nature of implicit and explicit processing that is important for memory. In our opinion, many experimental methods from cognitive neuroscience, such as those separating neural signals of memory performance with versus without awareness (e.g., Voss & Paller, 2008) could be used to enrich the understanding of anosognosia.

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**Eliciting the implicit:**

**Metacognition in Alzheimer’s disease**

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**Abstract:** The literature on metacognition in Alzheimer’s disease points to there being implicit and explicit routes to the control and monitoring of memory. For instance, despite not being able to make predictions of performance which reflect future behavior, people with Alzheimer’s disease can regulate effectively the amount of time they spend studying an item. Thus, empirical tasks from the metacognition literature shed some light on the idea of implicit awareness. But the complex pattern of preservation and impairment in metacognitive knowledge also points to other dimensions on which we need to consider patient awareness.

Metacognition is our ability to reflect on our cognitive processes; the very core of Mograbi and Morris’ CAM model is a metacognitive awareness system. The anosognosia and metacognition literatures have grown somewhat independently (for a synthesis see Souchay, 2007), but the empirical paradigms used in metacognition intersect with the idea of implicit awareness. There is the idea that subjective reports of memory function, for instance, derive from implicit information, but become explicit once made declarative or used strategically to change behavior (Koriat et al., 2008).

Morgabi and Morris’ model receives support from the idea that there are two levels of metacognition (Arango-Muñoz, 2011; Koriat, Nussinson, Bless, & Shaked, 2008; Moulin & Souchay, in press). Experienced-based metacognition lends itself to behavioral measurement, and is based on low-level automatic “feelings.” Knowledge-based metacognition relies on higher order mental representations and deliberative processes. We present examples from metacognition in Alzheimer’s disease (AD) to examine the idea of implicit awareness and the two levels of metacognition.

Moulin, Perfect, and Jones (2000) found that whereas people with AD were unable to accurately predict performance with explicit verbal judgments, they nonetheless allocated their study time appropriately. That is, behavior (a reduction of study time for repeatedly presented materials) was dissociated from their metacognitive report (the prediction of performance was not sensitive to repetition). It is difficult to reconcile this apparent simultaneous
“awareness” and lack of it, without recourse to a dual route system such as Mograbi and Morris propose.

But the pattern is not so clear-cut. Some explicit judgments are preserved in AD. Souchay (2007) describes a complex pattern of preserved and impaired metacognition. People with AD have adequate knowledge of what is easy or difficult to remember (e.g., characteristics of words and list lengths) but not the explicit knowledge that their memory is impaired. Thus, explicit awareness depends on what type of knowledge is being assessed. Whilst people with AD can accurately gauge the state of their memory for semantic (general knowledge) tasks, in the same paradigm, they are unable to accurately predict performance on episodic materials.

Mograbi and Morris provide a structure by which we may come to understand the behaviors and beliefs of people with anosognosia, and we argue that the core element of their model can be captured empirically in metacognitive paradigms. However, there are a few caveats. As above, some explicit judgments are preserved in AD: There is not a complete failure to reflect rationally on one’s cognitive activities. For instance, the type of information used to gauge the difficulties of words to be remembered, is not useful for the assessment of one’s limitations in general. For that, we need a more expansive view of metacognition. Moreover, metacognition is nearly only researched with memory tasks, as Mograbi and Morris note.

Perhaps the most critical issue which comes from a metacognitive viewpoint, is that we should be careful about inferring “awareness” from non-aware behaviors. For instance, patients with AD elect to study words for about four times longer than controls. This might be a tacit acknowledgment of their dysfunction (i.e., implicit awareness); but it may also be a failure to respond to low-level feelings which signal the need to terminate study. It may also represent a strategic failure to give up studying when it would be wise to do so. The only way to negotiate these different interpretations may be to avoid the neo-behaviorist interpretation of behaviors as “aware,” and consider instead subjective reports of experience, motivation, and the relation between awareness and function using the framework proposed by Mograbi and Morris.

Finally, Mograbi and Morris suggest that implicit awareness may guide behaviors and therefore have a therapeutic value. The current AD literature shows that if patients are implicitly aware in their metacognition, then it has very little benefit for their function. The exploration of how we might bring to awareness information streams that will help patients is a priority. In MCI we showed that patients were aware of the benefits of errorless learning—a technique based on implicit memory processes—in the explicit predictions of performance that they made (Akhtar, Moulin, & Bowie, 2006).

REFERENCES


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Implicit emotional awareness in frontotemporal dementia

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The preserved “implicit awareness” in patients with Alzheimer disease (AD) presenting anosognosia has opened a new branch of research regarding explicit-implicit integration. The behavioral variant of frontotemporal dementia (bvFTD), contrary to AD, would present impaired anosognosia-related implicit awareness due to a dysfunctional implicit integration of contextual information caused by an abnormal fronto-insular-temporal network. Loss of insight and anosognosia are pervasive in bvFTD, but no reports have assessed the implicit emotional awareness in this condition. We emphasize the need to investigate and extend our knowledge of implicit contextual integration impairments and their relation with anosognosia in bvFTD vs AD.

Mograbí and Morris have highlighted a curious and not well-known phenomenon of “implicit awareness” in patients with Alzheimer disease (AD). Despite unawareness of deficit, these patients preserve the emotional reaction to disability-related material, suggesting some kind of intrinsic knowledge about the illness. Moreover, they have discussed the anosognosia in terms of a general process including explicit-implicit integration through emergent networks influenced by top-down modulation, expectation, and anticipation. Furthermore, their approach to anosognosia is not restricted to basic processes such as perception, but also includes emotional and social cognition processes.

Within this framework, we discuss the hypothesis that the behavioral variant of frontotemporal dementia (bvFTD), in contrast to AD, would present impaired anosognosia-related implicit awareness. This deficit would be triggered by the impaired implicit contextual integration of social knowledge.

Patients diagnosed with bvFTD demonstrate early decline in social interpersonal behavior, early impairment in regulation of personal conduct, as well as early loss of insight and progressive deterioration in their social functioning (Piguet, Hornberger, Mioshi, & Hodged, 2011). Insidious behavioral changes and social cognition deficits are related to disintegration of neural circuits engaged in social behavior (Piguet et al., 2011). Ibáñez and Manes (2012) have proposed that the deficits of bvFTD patients in social cognition (e.g., emotions, empathy, decision making, theory of mind, etc.) can be understood as a general impairment of implicit integration of social context information due to an abnormal fronto-insular-temporal network. The damage to this network is linked to a dysfunctional implicit integration of emotional-social cognition information that prevents patients from updating and anticipating implicit contextual information (Melloni, Lopez, & Ibáñez, 2013). Similarly, Seeley (2010) argues that in bvFTD there takes place a progressive fronto-insular “Salience Network” breakdown that leaves patients unable to model the implicit emotional impact of their own actions or inactions. Contrarily, when explicit information is provided, bvFTD spares multiple cognitive and social domains (Burgess, Alderman, Vollet, Benoit, & Gilbert, 2009; Mesulam, 1986). For this reason, the use of explicit traditional tests is not good for assessment of bvFTD, given that these tasks lack the implicit context of everyday situations (Burgess et al., 2009; Ibáñez & Manes, 2012). Summarizing, integration of implicit social information within awareness seems to be impaired in bvFTD. Thus, bvFTD deficient integration of implicit information would prevent the access to implicit awareness of the own condition.

Complex, multi-factorial concepts such as “loss of insight” require not only inference, but determination of kind and quality of insight failure. A patient may state that he/she has bvFTD, but fail to appreciate the behavioral, functional, or cognitive consequences of his or her illness. In some cases, loss of insight into illness may be indistinguishable from lack of concern.

Loss of insight and anosognosia are a prominent clinical manifestation in bvFTD, presenting more severe affectation than AD patients (e.g., Méndez & Shapira, 2011). In spite of these features, there are no reports about affection of implicit awareness in this condition.

This scenario opens new research challenges: (1) to determine the possible implicit contextual integration impairments and their relation with different levels of anosognosia in bvFTD vs AD; (2) to assess the possible influence of fronto-insulo-temporal networks in the emergence of anosognosia in bvFTD; (3) to design rehabilitation strategies based on implicit awareness, taking into account the importance of teaching implicit rules for interpreting unpredictable social knowledge (Baez et al., 2012, 2013); (4) to develop neuroscience studies to support neuroanatomical models of implicit-explicit integration within anosognosia research. Thus, a fruitful extension of implicit awareness to bvFTD research would shed light on these questions.

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More work on lack of awareness and insight in healthy people and psychiatric patients will assist model building

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Abstract: We comment on the work of Mograbi and Morris and their newly developed Cognitive Awareness Model in terms of metacognition and awareness of disability in health aging, dementia, and psychosis. It is suggested that further research comparing implicit awareness of disability and objective metacognitive processes would be useful for theory development, as well as further understanding cognitive models of insight in the clinical domain.

Mograbi and Morris provide a fascinating overview of the concept of implicit awareness in clinical anosognosia. We would make the point that further study of implicit awareness of performance and behavior, initially in healthy aging, and later in other clinical groups, will enhance understanding and aid theoretical development. The authors state that implicit awareness is “also observed in normal ageing, where reduced driving can be a strategic response in relation to other disabilities and factors such as fatigue”, however, this may not always be the case. A recent study by Ross, Dodson, Edwards, Ackerman, and Ball (2012) investigated age-related decline in awareness of driving abilities in adults between 65 and 87 years. They found the number of self-reported accidents and driving citations significantly increased with age, while time spent driving did not. Interestingly, regardless of previous accident history, 85% of the drivers rated their abilities as “good” or “excellent.” This indicates that driving abilities decline with age, while awareness of this does not, and is not always implicitly addressed by a change in driving behaviors. These results support the CAMs model “gating mechanisms,” suggesting that new information about driving ability is not used to update Personal Data Base, therefore metacognitive judgments are made using out-of-date information. Inappropriate behavior is therefore likely to ensue, such as driving despite increased crashes. Further research into implicit awareness of problems with daily activities would be an interesting addition to healthy aging literature.

The cognitive neuroscience of metacognition is rapidly developing, with obvious clinical implications (David, Bedford, Wiffen, & Gillean, 2012). New methods have been developed to account for varying levels of performance, exposing differences in appraisal of performance (Fleming, Weil, Nagy, Dolan, & Rees, 2010). Hence, an interesting line of enquiry would be whether implicit awareness as defined by Mograbi and Morris was related to more objective measures of metacognition, as patients’ metamemory abilities have already been linked to clinical awareness of illness (Cosentino, Metcalfe, Butterfield, & Stern, 2007). A recent study by Weil et al. (2013) investigated perceptual metacognitive abilities of healthy participants aged 12–41 years, using a two-forced-choice perceptual judgment confidence rating (Fleming et al., 2010). Results indicated that overall there was a negative relationship between perceptual metacognition and age. However, when analyzing data from participants between 12 and 17 years, there was a significant positive relationship between perceptual metacognition and age, and a decrease in metacognitive ability as participants entered middle age (18–41 years). Recently presented data at the 2013 Association for the Scientific Study of...
Consciousness conference by Palmer, Fleming and David (in preparation) furthered this investigation, demonstrating the decline in awareness of one’s own perceptual ability continues into older age (up to 89 years). Hultsch, MacDonald, Hunter, Levy-Bencheton, and Strauss (2000) demonstrated that metacognitive ability declines as we age, in part because of incorrect beliefs about cognitive ability and control over cognition. Lack of awareness of cognitive, physical, and perceptual abilities in “healthy” older adults can be problematic, as Mograbı and Morris note, with over-confidence potentially leading to risky behaviors.

Methods of studying implicit awareness have facilitated scientific progress in this field. Using the Stroop paradigm—along the lines of the authors’ (Martyr et al., 2011)—we examined implicit awareness in people with psychosis. Unlike Alzheimer’s disease, we demonstrated that psychosis patients did not show increased interference with psychosis-related words (e.g., crazy, schizophrenic) as compared with physical-disease-related words (e.g., cancer; Wiffen, O’Connor, Russo, et al., 2013). Furthermore, a measure of interference was positively associated with explicit awareness—unlike the lack of association or paradoxical dissociation between implicit and explicit awareness, which has driven much of the authors’ model. So when patients with psychosis say they are not ill, it seems this is really what they “know.” Using the updated CAM model it therefore suggests that a problem in the MAS or the comparator mechanism will have wide implications for behavior in patients. Another methodology, namely encouraging patients to adopt a third-person perspective and subsequently reveal hitherto hidden awareness of their own predicament, has also provided contrasting, but inconsistent, data from psychiatric patients (David, Ster, & Zavarei, 2012; Wiffen, O’Connor, Gayer-Anderson, et al., 2013). While the term “anosognosia” is often used to convey the “neurological” basis for poor insight in psychosis, it fails to do justice to the differing patterns seen in some hemiplegia and Alzheimer’s patients.

Finally, comparing patterns of “awareness” within and between groups, would also be productive. For example, patients with Alzheimer’s may be more aware of behavioral problems than memory failures while patients with schizophrenia show the opposite pattern (Gilleen, Greenwood, & David, 2010).

**REFERENCES**


* * *
Health professionals are unaware of anosognosia

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Abstract: “Implicit awareness” may be inferred from compliance with medical treatment, even when the patient explicitly denies the need for treatment. Such compliance may cause medics and other health professionals to underestimate the frequency of anosognosia and its effects on the lives of patients and carers. We report survey data showing that health professionals do indeed consider anosognosia following stroke to be relatively uncommon and unimportant, in contrast with evidence on its true frequency and impact. Mograbi and Morris’ emphasis on the distinction between implicit and explicit awareness may promote increased recognition of anosognosia amongst health professionals.

Saul Bellow, in Mr Sammler’s Planet writes: “Both knowing and not knowing” is “one of the most frequent human arrangements”. Mograbi and Morris show that patients with anosognosia for hemiplegia (AHP) frequently demonstrate some level of awareness of their condition through their willingness to stay in hospital and receive medical treatment. This happens even if the reason for the hospital stay is not acknowledged (e.g., Berti, Ladvavs, Stracciari, Giannarelli & Ossola, 1998) or is attributed to a different cause (Cocchini, Beschin, & Della Sala, 2002). Such indirect compliance may cause the frequency or severity of AHP to be underestimated (see also Jenkinson, Preston, & Ellis, 2011).

We asked health professionals to fill in a brief questionnaire marking, on a scale of 1–5, the frequency of different symptoms following stroke and their likely impact on the lives of patients and carers. This method, while not providing exact frequency estimates, enables assessment of the perception of anosognosia relative to other symptoms. A total of 151 delegates of the 2013 European Stroke Conference completed the questionnaire—103 medics, 33 health professionals and 15 others—87% of whom worked directly with stroke patients. The results are shown in Table 1.

Anosognosia was considered to be the least prevalent symptom and the second least impactful, after facial paralysis. If we take the product of rated frequency and impact to be a rough indicator of the total burden associated with each symptom, then anosognosia was rated as the least important symptom on our list. This contrasts with frequencies reported in controlled studies (Orfei et al., 2007) and with experimental evidence associating AHP with poor functional outcome (Appelros, Karlsson, Seiger, & Nydevik, 2002; Di Legge, Fang, Saposnik, & Hachinski, 2005; Gialanella & Mattioli, 1992; Jehkonen, Laihosalo, & Kettunen, 2006). Such a discrepancy may arise from different methods of assessment (Cocchini, Beschin, & Della Sala, 2012) but also partly from lack of knowledge of the phenomenon of implicit awareness. If explicitly anosognosic patients continue to adhere to treatment, clinicians may presume that their unawareness will have little impact on their daily life and rehabilitation.

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Frequency, 1–5</th>
<th>Impact, 1–5</th>
<th>Frequency x Impact, 1–25</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper Limb Paralysis</td>
<td>4.20 (.86)</td>
<td>4.36 (.77)</td>
<td>18.55 (5.31)</td>
</tr>
<tr>
<td>Facial Paralysis</td>
<td>4.09 (.93)</td>
<td>2.69 (1.12)</td>
<td>11.05 (5.39)</td>
</tr>
<tr>
<td>Aphasia</td>
<td>3.77 (.85)</td>
<td>4.68 (.62)</td>
<td>17.64 (4.78)</td>
</tr>
<tr>
<td>Personality Change</td>
<td>3.16 (.98)</td>
<td>4.05 (.88)</td>
<td>13.03 (5.40)</td>
</tr>
<tr>
<td>Visuospatial Neglect</td>
<td>3.11 (.90)</td>
<td>4.12 (.92)</td>
<td>13.03 (5.10)</td>
</tr>
<tr>
<td>Hemianopia</td>
<td>3.10 (.93)</td>
<td>3.88 (.94)</td>
<td>12.15 (4.78)</td>
</tr>
<tr>
<td>Memory Loss</td>
<td>3.04 (.98)</td>
<td>4.18 (.84)</td>
<td>12.80 (4.94)</td>
</tr>
<tr>
<td>Limb Apraxia</td>
<td>2.93 (.88)</td>
<td>3.94 (.87)</td>
<td>11.53 (4.71)</td>
</tr>
<tr>
<td>Anosognosia</td>
<td>2.45 (.86)</td>
<td>3.55 (1.02)</td>
<td>8.70 (4.10)</td>
</tr>
</tbody>
</table>
Mograbi and Morris’ model, which allows for different levels of awareness, therefore has potential for increasing the recognition of anosognosia amongst health professionals, as well as advancing theoretical understanding of the condition. Perhaps in future, tests of implicit knowledge will be incorporated into clinical settings (e.g., Cocchini, Beschin, Fotopoulou, & Della Sala, 2010) to guide rehabilitation strategies and identify those patients that would most benefit from them.

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