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ANOSOGNOSIA, DENIAL, AND OTHER DISORDERS OF PHENOMENOLOGICAL EXPERIENCE

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SUMMARY

Clinical neuropsychologists frequently evaluate patients who present with poor self-awareness of their neurological and/or neuropsychological status. Understanding the nature of various disturbances of phenomenological experience may be important for differential diagnosis, psychotherapy, and brain injury rehabilitation. In this paper, an attempt is made to briefly review anosognosia and associated disturbances in phenomenological states. This is followed by a brief discussion of impaired self-awareness (ISA) seen in individuals who have a history of severe traumatic brain injury (TBI). It is suggested that in this latter group, ISA represents a partial syndrome of anosognosia. Extensive rehabilitative work with these individuals further suggests that persons with severe TBI may show both ISA and denial as a method of coping. This paper then discusses briefly the phenomenon of denial and how it may be manifested. Suggestions for measuring ISA, denial of disability (DD) and denial of ability (DA) are then made.

Key words: Patient Competency Rating Scale (PCRS), Clinical Rating Scale (CRS), self-awareness, traumatic brain injury (TBI)

INTRODUCTION

Our personal, subjective experience allows us to develop beliefs regarding our capabilities and how we function in the “real world.” Neurological (i.e., brain) and psychiatric disorders can negatively influence the person’s phenomenological state, which impedes their ability to engage in rehabilitation (Prigatano et al., 1986) and/or to adhere to a necessary course of treatment (Amador & David, 2004). These disturbances of subjective experience have not been well understood, although some progress has been made (Prigatano & Schacter, 1991; Prigatano 2010).

This paper briefly describes three disturbances in self-awareness that have been recognized in the neurological literature: anosognosia, anosodiaphoria, and somatoparaphrenia. Next, the phenomenon of impaired self-awareness (ISA) as observed after severe traumatic brain injury (TBI) is discussed in light of the literature on anosognosia. These phenomena are then compared to two other types of altered phenomenological states: denial of disability (DD) and denial of ability (DA). These latter phenomena appear to be present in patients who have experienced significant psychological trauma and/or ongoing, overwhelming stress as they attempt to cope with life’s problems.

ANOSOGNOSIA IN THE NEUROLOGICAL LITERATURE

Gabriel Anton (1898) described patients who had focal lesions of the brain, were not demented, yet did not perceive their losses in vision, hearing, and movement (see Förstl, Owen & David, 1993). Several years later, Joseph Francois Felix Babinski (1914) provided the name anosognosia (in French, anosognosie) to describe what has been the most common, classic form of this disorder (Prigatano & Schacter, 1991; Prigatano, 2010). Following a large right hemisphere stroke typically involving the middle cerebral artery (MCA), the patient may become hemiplegic and not report experiencing any disturbance in his or her motor functioning (Bisiach & Geminiani, 1991a; Bottini et al., 2010). This striking phenomenon has come to be called anosognosia for hemiplegia (AHP) and is observed in about 20 to 30% of patients with large right hemisphere strokes involving the MCA (Bisiach et al., 1986; Pederson et al., 1996; Baier & Karnath, 2005).

Research on AHP continues to raise many interesting issues concerning the awareness and control of motor functions (Bisiach & Geminiani, 1991a; Heilman, 1991; Frith et al., 2000; Berti et al., 2005), the mechanisms underlying these phenomena, and later recovery (Karnath & Baier, 2010; Vocat & Vuilleumier, 2010; Prigatano et al., 2011). Lesions of the frontal and parietal cortex are commonly associated with AHP, but other structures of the brain can also be involved, including the thalamus, the insula, and the basal ganglia. Presently there is a tentative conclusion that AHP may be caused by a different combination of lesion locations impacting the emergence of self-awareness of one’s motor functional capacity in the “here and now.”

What makes AHP so startling is that the person appears to have no subjective or phenomenological experience of severe motor loss (of the hand and leg) despite various efforts to demonstrate to the patient their hemiplegia (e.g., Sandifer, 1946). As the patient obtains feedback by initiating a motor task (such as finger tapping), they then may become aware or subjectively experience for the first time some restriction in their motor functioning (Prigatano et al., 2011).

While AHP is the most commonly observed form of anosognosia in neurological patients, anosognosia for complete cortical blindness (i.e., Anton's syndrome) also has been reported, but is clearly less common (Prigatano & Wolf, 2010). The underlying brain lesions responsible for this condition are even less understood. Its natural recovery patterns have not been documented. Interestingly, however, pregnant females may experience a complete loss of vision following obstetric hemorrhage of the posterior cerebral and/or communicating arteries. During this time they may be totally unaware of their complete visual loss (Argenta & Morgan, 1998). This phenomenon, however, is reported to be quickly reversible, but again, no systematic studies have appeared on its natural recovery course.

A third form of anosognosia that has been long recognized in the literature is a lack of awareness of paraphasic speech and problems of language comprehension in patients who suffer lesions in Wernicke's area. These patients are often described as having jargon aphasia, in which they seem to have impaired awareness of their problems with speech production and comprehension (Rubens & Garrett, 1991; Lebrun, 1987). It has been suggested that some of these patients may become aware of their language difficulties once they attempt to actively engage in speech and language therapy (personal communication with speech and language therapists of the German Speech and Language Therapy Association, BLK Conference, 7/22/2011). The natural course of this phenomenon is also not understood, nor are the underlying mechanisms responsible for it (see Kertesz, 2010; Cocchini & Della Sala, 2010).

OTHER PHENOMENOLOGICAL DISORDERS ASSOCIATED WITH AHP

Patients with AHP can also present with two other disturbances of phenomenological experience: anosodiaphoria and somatoparaphrenia. In cases of anosodiaphoria, the patient may recognize or subjectively report that they have limitations in motor movement or functioning of the arm and leg, but appear emotionally indifferent to this normally frightening experience. Heilman & Harciarek (2010) suggest that lesions of the right hemisphere may be responsible for this phenomenon. Anosodiaphoria may be produced by a disturbance in emotional communication and/or emotional experience. The exact nature of anosodiaphoria and the lesions responsible for it, again, have not been identified.

The second phenomenon is somatoparaphrenia. Karnath & Baier (2010) review the literature on this phenomenon, in which the patient reports that their af-

affected (hemiplegic) hand and arm belong to someone else. The phenomenon, like AHP, is often associated with large right hemisphere lesions. Using a variety of subtraction techniques involving MRI technology, Karnath & Baier (2010) suggest that damage to the right insula may greatly contribute to this phenomenon. While insular involvement is often found in patients who show somatoparaphrenia, it does not occur in all patients who demonstrate this disturbance (Prigatano et al., 2011).

A recent study suggests that somatoparaphrenia typically is associated with frontal-temporal-parietal lesions with subcortical involvement (Gandola et al., in press). Deep cortical and subcortical gray matter structures of the temporal lobe may reduce the sense of familiarity experienced by somatoparaphrenic patients concerning their affected arm. While the temporal lobe may be important for identifying what is familiar versus not familiar, this study, however, does not explain why the arm is attributed to another individual, often a person they know in their life (for example, their wife or aunt). Thus, the neuroanatomical and psychological explanation of somatoparaphrenia remains poorly understood.

A PROBLEM IN DEFINING AND CONCEPTUALIZING THESE RELATED PHENOMENA

Bisiach & Geminiani (1991b:262) define anosognosia as “apparent unawareness, misinterpretation, or explicit denial of an illness.” Bisiach & Geminiani (1991b:263) define anosodiaphoria as “lack of emotional reaction to a deficit caused by a brain lesion” and somatoparaphrenia as: “denial of ownership and other delusional beliefs related to the limbs contralateral to the side of a brain lesion” (1991b:264).

The use of the word “denial” to explain, or at least describe anosognosia and related phenomena is unfortunately misleading, because of the way in which the word denial is used in the psychiatric literature. Denial is considered a “defense mechanism” that automatically (i.e., non-consciously and reflexively) protects the individual from experiencing anxiety (DSM-IV, 1994:765, Glossary of Technical Terms). There is very little, if any evidence to suggest that AHP is a non-conscious, reflexive, motivated response to protect the individual from experiencing anxiety (see Bottini et al., 2010; Cocchini & Della Sala, 2010). This does not mean, however, that when a person experiences, perhaps for the first time, a significant disturbance in motor functioning, they do not automatically become anxious (Cocchini, Beschin & Della Sala, 2002). Some patients do, but others do not. What is important to consider, however, is that the term “denial” when describing patients who have anosognosia can be misleading, and gives a false impression as to the etiology of this complex phenomenon.

This becomes an important issue when considering how patients talk about or describe limitations they may experience after the onset of a brain disorder. For example, patients who suffer severe TBI often verbally report less neuropsychological disturbances than others around them observe for days, weeks, months, and even years post trauma. The study of this subgroup of patients provides an important transition for attempting to understand disorders of phenom-

enological experience in patients who have an underlying brain disorder, possible comorbid psychiatric difficulties, and who struggle to cope with the impact of cognitive deficits in everyday life. We will now consider the phenomenon of impaired ISA and DD in this patient population.

IMPAIRED SELF-AWARENESS (ISA) AS A PARTIAL OR INCOMPLETE FORM OF ANOSOGNOSIA AFTER SEVERE TBI

The early clinical observations of Adolf Meyer (1904), Paul Schilder (1934), and Kurt Goldstein (1952) emphasize that persons who survive severe TBI not only have persistent memory difficulties, but at times they seem unconcerned about the consequences of their brain injuries or are not fully aware of how they have been affected. Efforts at holistic approaches to neuropsychological rehabilitation also reveal that patients with severe TBI frequently seem to be unaware of, or deny persistent neuropsychological disturbances, even when these disturbances have been brought to their attention via daily cognitive remediation exercises (Prigatano et al., 1984; 1986).

Later studies documented that persons with severe TBI clearly underreport or underestimate their disturbances in memory and social interaction, which includes their ability to control emotional responses (e.g., Prigatano, Altman & O'Brien, 1990; Sherer et al., 1998; Bach & David, 2006; Trahan et al., 2006; Malec et al., 2007). It has been difficult, however, to determine if the unawareness is a clear reflection of brain dysfunction or a method of coping with residual deficits in some individuals. Two studies, however, have documented that ISA is related to the actual number of neuropathological lesions identified on neuroimaging of the brain (Prigatano & Altman, 1990; Sherer et al., 2005). These studies suggest that ISA is related to an underlying neurological disturbance.

The phenomenon may represent a milder or partial form of anosognosia (Prigatano, 1999). This impression is supported by conducting neuropsychological rehabilitation activities on a daily basis over several months with TBI patients who demonstrate ISA (Prigatano et al., 1986; Prigatano, 1999). Also, psychotherapeutic work with some ISA TBI patients over several years suggests that they did not seem to fully understand or experience their neurocognitive and neurobehavioral difficulties. With this partial knowledge of their cognitive and behavioral problems, some TBI patients also show features of defensive denial. This led to the proposition that after severe TBI, one may show both ISA and DD at the same time, and with various combinations in different patients (Prigatano & Weinstein, 1996).

Measuring these two constructs, however, has been difficult. Prigatano & Klonoff (1997) developed a Clinician's Rating Scale for evaluating these two phenomena. Their scales, however, were based on clinical impressions and have not been empirically verified. The scales reflect basic assumptions concerning

how ISA and DD differ. In ISA, the patient seems truly perplexed when given feedback that they are having more difficulties than what they experience (like anosognostic patients). These patients seldom show any affect when given feedback regarding the nature of their difficulties (like anosognostic patients). These patients also frequently show problems of initiation, self-monitoring, and planning (like some anosognostic patients). They may be slow in terms of speed of finger movement, which has shown to be a marker of the overall severity of TBI (Dikmen et al., 1995).

In contrast, patients with DD do notice change in their abilities, but upon specific questioning are quick to suggest that the deficits have no real impact in their day-to-day life (unlike anosognostic patients). They may show a negative reaction when given feedback that they have limitations, even when that feedback is given in a thoughtful and caring manner (unlike anosognostic patients). They seldom show the same degree of perplexity when given feedback, and often are ready to counteract the feedback with a somewhat logical argument (unlike anosognostic patients).

These clinical observations ultimately led to a model which suggests that ISA could be conceptualized as a partial or incomplete form of anosognosia. Individuals who show ISA may use both defensive and nondefensive methods of coping, as reflected in Figure 1. A common form of defensive coping is the use of denial.

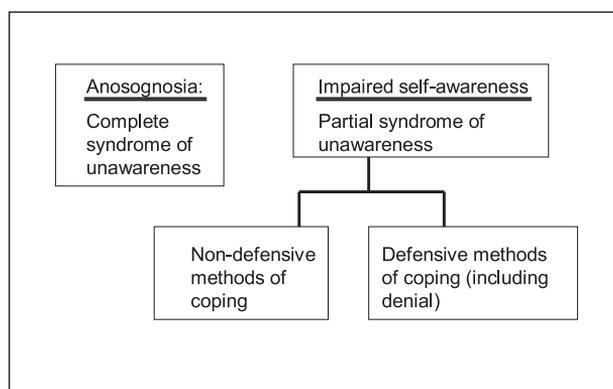


Fig. 1. Adapted from Prigatano, 1999; Prigatano & Morrone-Strupinsky, 2010

DENIAL OF DISABILITY IN NEUROLOGICAL PATIENTS

Weinstein & Kahn (1955) provided an eloquent argument that patients with brain disorders, as well as other medical conditions, can use denial to deal with personal threats caused by serious illness. They note that the level of denial can be “complete” or simply a minimization of the problem. In its most mild forms, denial is represented in patients attributing their difficulties to some benign cause. Weinstein & Kahn (1955) emphasized the following point. “...various forms of

anosognosia are not discrete entities that can be localized in different areas of the brain. Whether a lesion involves the frontal or parietal lobe determines the disability that may be denied, but not the mechanism of denial” (1955:123). Prior to his death, Weinstein (personal communication) wished that he could retract that statement. There has been a growing recognition that, in fact, anosognosia for different neurological impairments can exist in a modular fashion; that is, be related to specific areas of brain dysfunction (for a more thorough discussion of this point see Prigatano, 1999 and Prigatano et al., 2011). It has also become increasingly clear that patients with a variety of neurological conditions can show both DD as well as ISA. The persistent problem has been, however, trying to find a way of measuring these two different but related phenomena in a reliable and predictable fashion.

To help in this regard, it is worthwhile to consider how denial is typically manifested in patients faced with life-threatening issues, as well as patients who report cognitive difficulties, but appear neurologically intact.

DENIAL OF STRESS

Breznitz (1983:ix) has suggested that “stress and denial go hand in hand.” In order to cope with stress it is often necessary for the person to “deny” the reality of the situation. Sustaining the illusion that something is not so may be adaptive in order to cope with the multiple threats that a person experiences daily.

Denial is seen as a form of self-deception. The self-deception may be demonstrated via avoidance behavior. The person may wish to avoid any thoughts or action that brings to conscious awareness a disturbing (threatening) fact. Thus, the cancer patient may not mention their illness, nor do they wish to talk to anyone about their illness, because the mere fact that they discuss it increases their level of stress and anxiety (Breznitz, 1983). Under these conditions, a certain amount of “denial” or “avoidance” would seem adaptive. The problem clinicians often face, however, is that denial can be maladaptive. It can get in the way of the individuals coping with their life situations and becoming more productive, and thereby meaningfully engaging in life. This phenomenon is frequently seen in the situation in which the individual does not deny disability, but actually denies their ability.

DENIAL OF ABILITY

It is common for clinical neuropsychologists to evaluate patients who report memory impairments. Some of these individuals can give detailed accounts of how they have difficulty remembering the names of people that they see often, why they entered a room, and what was said to them the previous day. They are convinced that there is an underlying brain disorder which is accounting for these memory failures. On formal neuropsychological examination, however, they may show no evidence of memory impairment. Speaking to these individuals about this reality can be a challenging task. Having them complete tests such as the

MMPI-2 is at times helpful from both a diagnostic and therapeutic point of view. The individual may show, for example, a 1-3 profile which is commonly observed in somatoform disorders. By reviewing this profile, the clinician can gently point out that the patient seems to be preoccupied with concerns about health and also has a heightened level of anxiety that may not be subjectively experienced. When the individual sees “in black and white” their MMPI-2 profile, it provides the first avenue to discuss the fact that anxiety and preoccupation with health issues may be greater than what the individual consciously recognizes. This may be the basis for them minimizing, or avoiding, or denying their actual strengths and capability of working.

These patients may be overwhelmed by their work and/or home situation, as well as other health problems. They may seek medical disability or early retirement status. Within our culture, having a physical illness justifies ending work and withdrawing from the numerous struggles life can present. Denial of these struggles can reflect a method of coping. These are traditional psychological concepts that are rooted in psychodynamic theory. Measuring these phenomena, however, has remained a challenging task, although the work of Cramer does provide some interesting insights.

MEASURING THE DEFENSE MECHANISM OF DENIAL

Phebe Cramer (2002:104) has suggested that various defense mechanisms including denial can be reliably assessed by analyzing stories on the Thematic Apperception Test (TAT). Cramer (2002) suggests that “...the defense of denial is predominant in preschool years but then declines in importance, while projection increases in use during middle childhood and becomes predominant during the adolescent period of development. Identification, a defense of considerable complexity, develops slowly from early childhood through late adolescence when, at least in college samples, it has been found to replace projection as the predominant defense”.

Within her model, denial is measured by statements of negation, blatant or clear omission of ominous components of reality when telling or relating a story, over-maximizing the positive or minimizing the negative, describing unexpected goodness, optimism, positiveness, or gentleness. The key is a misperception of a situation in which the negative is not attended to and replaced by statements concerning the positive. A key component appears to be a failure to consciously recognize the sad, angry, and threatening aspects of life.

When evaluating patients who present with DA, in the context of a neuropsychological assessment, it is not uncommon for them to report absolutely no anxiety or no depression. That is, the normal amount of anxiety and depression an individual might experience related to some setback in life is not verbally (consciously) reported by the individual. Also, such individuals may overdramatize how wonderful life was in the past or emphasize their excellent abilities prior to

the onset of their medical condition. They may insist that they had a “photographic” memory as opposed to a normal memory.

Thus, when assessing denial it is important to allow the patient to describe their life in their own terms so that one can get a sense of whether they are presenting a balanced view of the “good” versus “bad” in life. Any overemphasis on the positive without adequate recognition of the negative provides a signal that denial may be present.

Applying this to patients who have brain injury, the ability of the person to describe sadness or anger over lost function begins to minimize the likelihood that denial is a major way of coping. The ability of the person to describe themselves in objective terms prior to the onset of the injury as well as after the onset of the injury provides another source of important information. If a reliable informant (e.g., parent) describes an individual as having an overinflated positive view of themselves prior to the injury, this suggests that the mechanism of denial may, in fact, have been a common method of coping prior to the onset of their neurological condition.

Cramer’s (2003) research also suggests that these personality characteristics are relatively stable and can actually predict forms of adjustment later in life. While the use of projective measures has fallen out of favor within the context of neuropsychological assessment, a return to assessing denial through such methods as the TAT may provide some help in measuring this construct in patients with known or suspected brain disorders.

SUGGESTIONS FOR MEASURING ISA, DD, AND DA

While the primary purpose of this paper was to discuss the phenomenon of anosognosia and related disorders of phenomenological experience, it is important to consider methods for assessing impaired ISA, DD, and DA. Orfei et al. (2011) have discussed different approaches to the assessment of frank anosognosia, especially AHP. Bisiach’s et al. (1986) scale is perhaps the prototype for how a behavioral assessment of AHP occurs. Basically, the patient is asked some very straightforward questions regarding their level of motor functioning and that is compared with obvious clinical reality.

In measuring ISA, the process is more difficult. Here the individual is asked to make relative judgments about their strengths and limitations, and those judgments are compared to some reliable others’ perspective. While there are different measures of measuring ISA, one method has been to have patients answer a series of questions reflecting their level of skill or ability and comparing their ratings with a significant other. The Patient Competency Rating Scale (PCRS) was developed with this concept in mind (Prigatano et al., 1986), and has been successful in reliably assessing ISA in patients with TBI (for a recent review as well as a review of its psychometric properties, see Kolakowsky-Hayner et al., 2012)

It has been frequently observed that TBI patients who show ISA report a higher level of competency than what their significant others report. Extreme examples are those in which the individual reports absolutely no difficulty in any area (obtaining a raw score of 150 out of 150 points on the PCRS), while relatives may rate them as having more difficulty than what they report (i.e., relatives' ratings may result in a score in the 80s on the PCRS). Less severe forms of ISA occur in which the disparity is not as great, but the direction of the overestimation of their abilities is reliably seen.

Since the PCRS does not separate denial of disability from impaired self-awareness, Prigatano & Klonoff (1997) introduced the Clinician's Rating Scale as an aid to further separate these two dimensions.

Given the work of Cramer (2003) it may be helpful to obtain from persons who have a history of severe TBI their responses to selected cards on the TAT. Patients who present with primarily ISA and little evidence of DD would be expected to show less independent signs of denial on TAT cards 2, 8GF, 8BM, 10, 15, and 18GF. In contrast, patients who are clinically identified as having stronger indications of DD in addition to their ISA would be expected to show greater independent signs of denial on these cards.

This methodology could also be expanded to assessing DA. Using the PCRS, one would predict that patients who show DA underreport their competency compared to those who know them well. In this regard, it has been my clinical impression that relatives may rate the patient at 120+ points (out of a possible 150 points), indicating they can do most things reasonably well. In contrast, the patient may rate themselves less competent by at least 15 to 20 points on average. It also would be useful to have these patients respond to the TAT cards mentioned above to determine if, in fact, they show greater signs of denial than an appropriate control. Table 1 summarizes predicted psychometric relationships that might help separate out patients with ISA, DD, and DA.

Table 1. Summary of predictive relationships when studying ISA, DD, and DA

On PCRS	Persons with a history of severe TBI who present with ISA will show a total score greater than what relatives report (e.g., in severe cases the patient may have a total PCRS score of 145+ while relatives' total score would be in the 80s).
On PCRS	Persons who report memory difficulties for which no underlying neurological findings can be observed, and therefore appear to show DA, will show the opposite pattern. That is, the PCRS total score will be less than what relatives report (e.g., patients with DA total score may be 110 or less; relatives' score will be 125+).
On CRS	Patients who have a history of severe TBI and DD will have higher scores on the Denial of Disability Scale of the Clinical Rating Scale (Prigatano & Klonoff, 1997) compared to patients not clinically judged to have DD.
On TAT	Patients with DD and DA will show greater indications of denial by their verbal responses obtained from TAT cards 2, 8GF, 8BM, 10, 15, and 18GF [using Cramer's (2003) methodology] compared to persons with anosognosia or ISA.

In closing this section, it should be noted that Lewis (1991) several years ago presented a classic case in which a patient was showing denial as a major method of coping. The patient had extremely angry and hostile feelings toward her mother, which were kept out of awareness for a long period of time during her psychotherapy. As the working alliance developed and negative transference emerged, the patient clearly projected onto the therapist characteristics of her mother. At one point she accused the therapist of being “no better than my mother” and once those words were uttered, she was shocked that they had come from her mouth. It should be emphasized that this revealing of a denied emotion/conflict only occurred after a relationship had been established and the patient was in a position to freely express her feelings in a manner that questionnaires cannot always provide. Thus, the process of assessing denial cannot be relegated to simple questionnaires, but questionnaires can be one useful method to utilize when attempting to conduct scientific studies in this area.

TOWARD A NEUROPSYCHOLOGICAL UNDERSTANDING OF THESE COMPLEX DISORDERS OF PHENOMENOLOGICAL EXPERIENCE

Present research on anosognosia consistently points to the fact that lesions of the cortex may be crucial in producing true anosognostic disturbances. While insular lesions may affect a sense of familiarity or ownership, cortical lesions seem to literally deprive the individual of a conscious representation or experience of an impairment in functioning.

Experimental research on repression provides an interesting model for understanding how denial and anosognosia may be similar or different. Anderson et al. (2004) performed an experimental study in which students were asked to learn a series of paired associate words. Later in the experimental design, he instructed the individual not to think of a certain association when one of the paired words was presented. He evaluated the fMRI correlates of carrying out this task. What he observed was that there were multiple neural systems involved in the suppression of unwanted memories. They seem to involve a complex network. Among the changes he observed was reduced activation in the hippocampus (bilaterally) and the posterior insula. There was increased activation in the dorsal-lateral and ventral-lateral prefrontal cortex bilaterally, as well as the anterior cingulate. The implication was that when there is a conscious effort to suppress information, dorsal and ventral frontal activation may be important. There are also activations in the cingulate gyrus during this time.

It is well known that these brain regions play a key role in problem-solving, self-monitoring, and focusing attention. If the individual wishes not to solve a problem or to monitor their reactions, then frontal lobe activity may play an important role. In classic anosognosia for hemiplegia, damage to the frontal and

parietal lobes may result in the individual not experiencing impaired motor functioning for neurological reasons rather than psychodynamic reasons.

It is important to note, however, that awareness is not “in the frontal lobes.” It has been suggested by many that awareness of various neurological functions/dysfunctions is organized along modular development (Prigatano, 1991; Prigatano, 2010). Thus different structures may be involved in awareness of visual impairments versus auditory or language impairments versus behavioral or emotional impairments. What is important, however, is that the same underlying brain structures may play a key role in anosognosia, ISA, and denial. At first glance these phenomena may look identical, but with further probing, they are clearly different forms of disturbed phenomenological experience.

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