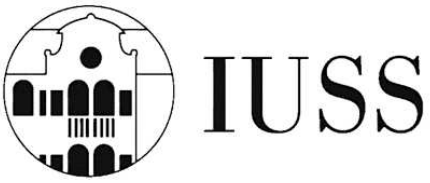
UNIVERSITY OF PAVIA – IUSS SCHOOL FOR ADVANCED STUDIES

PAVIA

Department of Brain and Behavioral Sciences (DBBS)

MSc in Psychology, Neuroscience and Human Sciences



COMPREHENSIVE EVALUATION OF ANOSOGNOSIA FOR HEMIPLEGIA AND THE ROLE OF THE LEFT HEMISPHERE

Supervisors:

Prof. Martina Gandola

Prof. Gabriella Bottini

Thesis written by

Melissa Kennepohl

513178

Academic year 2023/2024

Index

1. Abstract

Anosognosia for hemiplegia (AHP) poses a significant challenge in neurological research and rehabilitation due to its heterogeneous and complex nature. Although it predominantly manifests in patients with right hemispheric lesions, cases of patients with AHP and left hemispheric lesions have been reported in the past. However, less is known about the underlying processes or differences in manifestation. This review aims to investigate the occurrence of AHP in patients with left hemispheric lesions, exploring whether these cases are truly rare or if current diagnostic tools often miss them, particularly by excluding aphasic patients. A total of eleven studies, including 44 stroke patients with AHP, were analysed in this systematic review to explore the difference in pathology of AHP caused by unilateral left hemispheric lesions compared to right hemispheric lesioned patients. The results reveal that only a minority of studies with left hemispheric lesions with AHP exist. However, they also highlight that the current measures often fail to include aphasic patients or extend beyond the assessment of explicit anosognosia. As a result, many cases of AHP related to left hemispheric lesions are overlooked. Furthermore, the results hint at a potential role of reversed hemispheric lateralization in causing AHP, as suggested by several studies, providing a possible explanation and highlighting the need for further investigation. The findings underscore the need for updated diagnostic methodologies that incorporate implicit and integrative measures alongside traditional clinical interviews to provide a more nuanced assessment of awareness deficits and to discover further cases of AHP in left hemispheric lesioned patients.

Keywords: Anosognosia for Hemiplegia; Left Hemisphere; Stroke; Systematic Review

Comprehensive Evaluation of Anosognosia for Hemiplegia and the Role of the Left Hemisphere

2. Introduction

In the complex world of neurological conditions, anosognosia for hemiplegia (AHP) is both an intriguing and perplexing phenomenon. A patient, confronted with the reality of their paralyzed limb, frequently denies their functional impairment, dismissing it with a remark like "I'm not Superman!" (Welman, 1969) after being requested to lift his arm or replying “I am not in the theatre.” (Berti et al., 1998) to a request to clap their hands. This curious interplay between perception and reality captures the essence of AHP, a condition where individuals are blissfully unaware of their paralysis, creating a dilemma for both patients and clinicians alike.

While seemingly bizarre, AHP holds profound implications for our understanding of the human mind and the complexity of brain function. Beyond its intriguing expression, studying AHP unveils critical insights into the neural mechanisms underlying self-awareness and body representation. In attempting to comprehend AHP, researchers have to dissect the complexities of brain functions regarding neural correlates and cognitive processing of underlying motor awareness.

Moreover, the clinical implications of AHP are significant, affecting patient care and rehabilitation strategies directly. Understanding why some individuals fail to recognize their own paralysis is not merely an academic pursuit. The importance of studying AHP extends discoveries of underlying neurological functions and entails essential implications for rehabilitation and improving the quality of life for those affected by this neurological disorder. Due to the lack of awareness of their deficits, individuals with AHP face significant challenges during acute and post-rehabilitation

phases, such as decreased motivation or active resistance to engaging in rehabilitation programs (Fleming et al., 1998; Katz et al., 2002). Consequently, rehabilitation outcomes often fall short of expectations (Gialanella et al., 2005; Ownsworth & Clare, 2006; Winkens et al., 2013). Moreover, the compensatory strategies learned may not be consistently applied afterwards (Ownsworth et al., 2000), raising concerns about safety in daily activities in autonomous living, and adding to the burden on caregivers (Mograbi & Morris, 2013; Orfei et al., 2007).

While AHP was long assumed to be a disorder connected to the right hemisphere (Antoniello & Gottesman, 2020), recent insight and literature suggest that AHP seems to go undiagnosed or underestimated in patients with aphasic features, frequently occurring with left hemispheric lesions (Coccini et al., 2012; Della Sala et al., 2009). The role of the left hemisphere is vague and often overshadowed by the predominant findings and focus of AHP with right hemisphere lesions. Despite potential occurrence, these patients are frequently marginalised due to co-occurring aphasia, and difficulties to assess. Therefore the present systematic review focuses on literature to investigate the notable gap of underrepresentation of AHP cases stemming from left hemisphere damage and whether this gap is justified, or needs more attention in clinical practice. This is essential to determine whether the research gap on left hemispheric patients needs more attention to enhance neurological insights, improve rehabilitation strategies, and ensure potential AHP in these patients is not neglected.

To delve deeper into this topic, comprehensive research is necessary to analyse our current understanding of how AHP manifests, how it is assessed, its anatomical correlates, related theories, and findings of distinct expression between

different hemispheric lesions. With this foundation, a systematic review will explore the current findings of patients with AHP and left hemispheric lesions, and determine if left hemisphere lesions correlate with these findings.

This review underscores the importance of recognizing that studying AHP is not just a matter of intellectual curiosity but a crucial necessity that requires immediate attention. By delving into the complexities of AHP and examining its differences between hemispheres, this review aims to uncover possible interesting discoveries between left hemisphere lesions and AHP over the last few years. These findings could offer valuable insights for improving assessment, intervention, neuro-anatomical insight, novel research approaches, and rehabilitation practices in clinical settings.

2.1 Overview of the Review Structure

In this systematic review, a comprehensive exploration of the pathology, assessment, underlying theories of motor awareness and AHP, and neural correlates of AHP are displayed, with a distinct focus on hemispheric pathology differences, particularly emphasising recent findings of AHP in the left hemisphere. The journey through this review is structured to provide a holistic understanding of AHP. Starting with AHP manifestations in individuals, shedding light on its nuanced clinical presentations and the challenges it poses for accurate diagnosis and management due to its heterogeneous expression. Following the diverse assessment methods employed in clinical and research settings, highlighting their respective strengths and limitations. Furthermore, models of motor awareness are introduced, to lay the basis for the differing underlying theories that attempt to explain the perplexing phenomenon of AHP. Moreover, anatomical correlations implicated in AHP, seek to

unveil the neural underpinnings that contribute to its manifestation, followed by findings of distinct expression of AHP regarding differing hemispheric lesion location will be assessed. The systematic review of the last 50 years findings on AHP associated with the left hemisphere will be analysed and evaluated, ending in a discussion about noticeable manifestations of AHP in the left hemisphere, limitations with assessment, underlying theories of those findings, and analyzation of the anatomical correlations observed in these studies.

Through this systematic review, the review aims to uncover the complex neural mechanisms behind AHP while also advocating for a more inclusive approach that considers the diverse clinical presentations of this condition, thereby fostering a deeper understanding.

3. Anosognosia for Hemiplegia (AHP)

Anosognosia, the lack of awareness or denial of one's medical or psychological condition, poses a unique challenge to understanding self-awareness and the complexities of the human mind. The earliest documentations of anosognosia were found around the end of the 19th century (Marková & Berrios, 2014). Joseph Babinski introduced the term by assembling the Greek words "a"–without, "nosos"–illness, "gnosis"–knowledge to describe the neuropsychological phenomenon of patients lacking awareness or insight of their personal disability or disorder (Babinski, 1914). While the concept of AHP has been present in scientific literature for over 100 years, our comprehension of the phenomenon is evolving, and plenty remains unknown. Anosognosia manifests through various symptoms, impacting behavioural, cognitive, motor, and sensory domains (Mograbi & Morris, 2018). Severity can vary and impact the patient's activities of daily living,

interpersonal relations, personality, and quality of life in various ways. Both cognitive mechanisms and emotional processing play an essential role in anosognosia, altering the awareness of the impairment and its consequences (Gainotti, 2018). This occurs through denial or minimization of the patient's behavioural, cognitive, and functional alterations related to their injury or neurodegenerative disease.

The occurrence of AHP is characterised by a lack of awareness or denial of one's own hemiplegia or hemiparesis. Overall, the severity of hemiplegia is higher in patients with anosognosia compared to those with hemiplegia without anosognosia (Appelros et al., 2007). This condition leads to severe consequences, including difficulties with dressing, eating, walking, and recovery progress, evident to caregivers but not the patient (Gialanella et al., 2005; Di Legge et al., 2005; Orfei et al., 2009).

AHP is a heterogeneous, complex, and multifaceted neurological phenomenon. It is characterised as a transient disorder (Antoniello & Gottesman, 2020; Vocat et al., 2010), typically arising after sudden onset of a moderate to severe traumatic brain injury (TBI), stroke, tumour, or in co-occurrence with neurodegenerative diseases such as dementia or Parkinson's Disease (Orfei et al., 2009, 2018; Prigatano, 2012; Steward & Kretzmer, 2022; Tagai et al., 2020). The presence of anosognosia is frequently viewed as a negative prognostic factor for achieving a positive functional outcome following an injury (Gialanella et al., 2006).

Typically, the lesion is located in the right hemisphere (Antoniello & Gottesman, 2020), leading to a lack of awareness regarding the hemiplegia or weakness on the left body side. However, recent studies also investigate the presence of AHP in patients with bilateral and left-hemispheric damage (Cocchini et

al., 2009) to determine whether left-hemispheric lesions cause AHP less frequently or are less frequently discovered during assessment.

In the hyperacute phase after a stroke, AHP appears to be most robust. Spontaneous recovery can follow within a few days to weeks, with AHP usually becoming less frequent after the first month after stroke onset (Antoniello & Gottesman, 2020; Gialanella & Mattioli, 1992). Only a minority of patients develop AHP chronically (Orfei et al., 2009; Prigatano, 1999). There seems to be no association between age, educational level, gender and AHP, as it appears equally in all groups (Pia et al., 2004). Due to difficulties in the assessment and differentiation of AHP phases, prevalence remains uncertain and varies between studies. Estimated prevalence for AHP varies between 20% and 44% of the patient population (Pia et al., 2004). Epidemiological estimates of incidence rates after brain damage range between 7% and 77% (Jehkonen et al., 2006; Orfei et al., 2007), of probable Alzheimer's disease from 20% to 80% (Agnew & Morris, 1998), and 20%–30% after a stroke (Byrd et al., 2020; Pedersen et al., 1996).

In the upcoming subchapters, we will explore the clinical manifestation, assessment, motor awareness, underlying theories, and anatomical correlation of AHP to enhance understanding and insight into its pathology. This overview reflects our current knowledge up to the present day, emphasising the importance of comprehending AHP in further investigating its potential occurrence in left-hemisphere lesions.

3.1 Clinical Manifestation of AHP

Studies by various researchers (Cocchini et al., 2010, 2018; Fotopoulou et al., 2010; Marcel et al., 2004; Moro et al., 2011; Nardone et al., 2007; Preston et al., 2010; Prigatano, 2014) consistently reveal that anosognosia manifests in different

domains and contexts. The severity of deficits can vary in affective, cognitive, behavioural, motor, sensory, and perceptual functioning. The patient's awareness of their motor impairment can be observed explicitly or implicitly (Fotopoulou et al., 2010; Moro et al., 2021). Different levels of awareness may coexist or differ within the same patient and fluctuate over time (Moro et al., 2021). Capturing patients' full level of awareness is challenging due to discrepancies, inconsistencies, and variations in responses regarding their motor ability (Coccini et al., 2012). Awareness levels (Pacella et al., 2019) can range from differences within anticipatory awareness from "are you able to reach the table with your left hand?" (Moro et al., 2015) to general awareness as "why are you in the hospital?" (Besharati et al., 2016), and mentalization "the doctors think there is some paralysis, do you agree?" (Besharati et al., 2016; Feinberg, 2000). Awareness of AHP can also manifest as modality-specific (Bottini et al., 2018; Cocchini et al., 2012; Orfei et al., 2009). Modality specificity is defined by the unawareness of specific aspects of a patient's situation. Dissociation can be displayed in the expression of explicit and implicit acknowledgement of symptoms within the patient (Cocchini et al., 2010; Mograbi & Morris, 2013). For example, a patient can be aware of their cognitive deficit but neglect their sensorimotor deficit (Bach & David, 2006) or, as stated above, acknowledge their impairment but neglect the consequences. Others acknowledge their hemiplegia (HP), but not their aphasia, or vice versa (Breier et al., 1995; Kinsbourne & Warrington, 1963). These dissociations can extend to the point where patients with AHP may acknowledge the motor impairment of one leg but deny the inability to move their arm or vice versa (Berti et al., 1996; Della Sala et al., 2009; Moro et al., 2011).

Explicit unawareness is more easily recognized and directly explored by asking patients to verbally declare whether they can move, while implicit awareness is typically indirectly inferred based on behavioural observations (Fotopoulou et al., 2010; Moro et al., 2011; Moro et al., 2021; Schacter, 1990). The most frequently observed symptom in AHP regarding explicit awareness is the denial of motor impairment (Fotopoulou et al., 2010; Marcel et al., 2004; Moro et al., 2011; Vocat & Vuilleumier, 2010). Approximately one-third of patients with AHP deny their motor impairment in both the acute and chronic phases (Pia et al., 2004). The robust resistance to awareness or motivated denial can be referred to as "organic repression" (Schilder, 1935). In the context of denial, patients tend to underestimate the implications or overestimate their abilities relative to their current situation (Langer & Bogousslavsky, 2020; Marcel et al., 2004). Behavioural denial can be distinguished from anosognosia as an independent reaction, attributed to adaptation or maladaptation to subjective distress (Ghika-Schmid & Bogousslavsky, 1997; Goldbeck, 1997). Anosognosia, on the other hand, involves a fundamental lack of awareness of one's condition.

Disturbances of mentalization can be explicitly expressed (Moro et al., 2021). Various expressions and responses are observed when patients are asked to move their paralyzed limb. Such expressions include denial, lack of insight, or confabulations (Langer & Bogousslavsky, 2020). Patients may respond in various ways when movement is expected: by moving the opposite arm (allochiria), by refusing to move, by confabulating reasons for their inability to move, or by remaining silent (Antoniello & Gottesman, 2020; Feinberg et al., 2000; Marcel et al., 2004). Moreover, patients with AHP tend to verbally overestimate their motor ability when making judgments about them (Besharati et al., 2022), and often engage in

rationalisation by providing explanations unrelated to their motor abilities (Langer & Bogousslavsky, 2020). They often make causal attributions, underestimating the consequences and severity of their incident and attributing the inability to move to unrelated factors such as back pain, fatigue, diabetes, mood or laziness (Levine, 1990; Langer & Bogousslavsky, 2020; Orfei et al., 2009), as excuses to comprehend their situation. Although some patients may express awareness of their stroke or TBI, they often have unrealistic expectations of recovery and are unaware of the functional implications of daily living related to dressing or eating (Orfei et al., 2009). Consequently, these patients are more challenging to rehabilitate due to their lack of insight into the effect of AHP on their life (Fischer et al., 2004). Interestingly, even those patients who admit, and are aware of their HP, still tell the examiner they had moved their paralyzed limb when requested to move (Antoniello & Gottesman, 2020), hinting at patients' complexity of awareness. Some patients can exhibit emergent awareness after recognizing their failure to perform an action despite prior beliefs (Berti et al., 1996; Cocchini et al., 2009; D'Imperio et al., 2017; Marcel et al., 2004; Moro et al., 2011; Moro, 2013). This emergent awareness was identified as being frequently temporarily limited (Marcel et al., 2004). Due to temporarily limited insight, it was suggested that patients with AHP exhibit a deficit in updating their beliefs even when confronted with evidence of impaired daily living activities or receiving medical, and social feedback (Besharati et al., 2016; Kirsch et al., 2021; Vocat et al., 2013). Patients with AHP do not seem to have a general problem in reasoning but require more time and repeated errors to modify their beliefs (Vocat et al., 2013). They often exhibit an unusual pattern of excessive confidence and low error recognition compared to healthy individuals or those with HP. Confronted with new information about their motor impairment, individuals with AHP persist in their

initial incorrect beliefs rather than adapting and modifying their beliefs with incoming information. They tend to repeat and persist in their erroneous responses more frequently before eventually considering a modification (Vocat et al., 2013). This tendency to be overconfident and persistent in current beliefs is not associated with a more general impairment in executive functions, vigilance, spatial attention, or memory (Marcel et al., 2004; Vocat et al., 2013).

Next to challenges in belief-updating, other manifestations of explicit awareness in AHP include delusional features. The delusional aspects of anosognosia are best understood as a failure to recognize the significance or relevance of sensorimotor errors. This failure to acknowledge context-specific errors results in a disconnection with more abstract and context-independent beliefs about the self, which hold broader applicability across various times and spaces (Kirsch et al., 2021). Additional delusions occasionally observed in AHP include patients describing movements that did not occur, categorised as having a confabulatory nature or termed 'illusionary movement' (Feinberg et al., 2000; Fotopoulou et al., 2008).

Other expressions as implicit knowledge can be evident in their behaviour, challenges in taking allocentric perspectives, intention of motor control, spatial cognition, and conducting reality monitoring beyond the motor domain (Besharati et al., 2016; Jenkinson & Fotopoulou, 2010; Pacella et al., 2019; Vocat et al., 2013). In contrast to explicitly stated awareness, implicit awareness is often observed by inconsistencies between a patient's nonverbal neurological signs, such as behaviour or subtle verbal statements. It can emerge through behavioural tasks or implicitly in conversation (Marcel et al., 2004; Moro et al., 2021). For instance, patients may assert full functionality, but further conversation reveals side sentences that hint at

their inability to move or mention their eagerness to resume activities requiring the use of both hands in the future (Coccini et al., 2012). Alternatively, a patient may acknowledge their hemiplegia and express explicit awareness but attempt to act as they did before the motor impairment, indicating behavioural implicit unawareness (Cocchini et al., 2010; Moro et al., 2021). The inability to perform a specific action while believing they can, indicates anticipatory unawareness (Cocchini et al., 2018; D'Imperio et al., 2017; Marcel et al., 2004; Moro et al., 2021). Such situations are observed where patients in a wheelchair claim no weakness in their limbs but refuse to stand up and walk when requested (Orfei et al., 2009). Other evidence for implicit awareness is observed in other studies of anosognosic patients identifying in picture tasks patients in a wheelchair most similar to them (House & Hodges, 1988). Similarly, when patients were asked about their ability to perform bimanual or bipedal tasks, followed by inquiring how someone with the same condition as the patient would perform those tasks, anosognosic patients rated the ability of others significantly lower compared to their own (Berti et al., 1996; Marcel et al., 2004). Therefore, it is suggested that patients with AHP appear to exhibit a deficit in higher-order mentalizing tasks, such as allocentric and third-person-perspective taking (Besharati et al., 2022). These deficits seem to correlate with the degree of clinical anosognosia. The more severe the deficit in third-person-perspective, the greater the clinical anosognosia for first-person-perspective (Besharati et al., 2016). Additionally, AHP patients seem to have improved insight into their condition when asked from a third-person perspective compared to a first-person perspective (Besharati et al., 2022). They are also able to recognize physical impairment in other patients with AHP (House & Hodges, 1988).

Other observed instances suggesting implicit knowledge of their bodily status can be seen in examinations where the patient is asked to touch the examiner's hand with their left paralyzed arm. The patient fulfils the task by pulling their left hemiplegic hand with their right functional hand to touch the examiner's hand (Antoniello & Gottesman, 2020). Despite refusing to accept that their limb is paralyzed, the patient accomplishes the examiner's request in a strategic way, indicating some underlying awareness of the bodily state. These situations suggest a somewhat unconscious awareness of the state of motor control. Further implicit awareness tasks were tested by displaying words related and unrelated to motor tasks, showing that anosognosic patients have slower reaction times related to motor tasks, like "walking," compared to aware patients (Fotopoulou et al., 2010; Nardone et al., 2007). This was also observed in inhibition tasks with deficit-related sentences (Fotopoulou et al., 2010; Nardone et al., 2007).

Along with explicit and implicit awareness, the manifestation of AHP also involves discussions about cognitive, emotional, executive, and proprioceptive functionality. Cognitive impairment is considered a sub-component in studies, and presents an ongoing discussion about whether it worsens the condition or accompanies AHP (Orfei et al., 2009). Previous studies have shown no strong correlation between pre-existing and present intellectual functioning and the extent of self-awareness (Bach & David, 2006). Consistent with these findings, patients with AHP often exhibit relative intellectual preservation (Berti et al., 1995; Langer & Bogousslavsky, 2020; Marcel et al., 2004; Starkstein et al., 1992; Vocat et al., 2013) and cognitive impairment was suggested to be separate from AHP (Berti et al., 1996; Bisiach et al., 1986; Spinazzola et al., 2008). Previous recorded data on mental deterioration indicated that 50% of patients with AHP had general cognitive

conditions within the standard range (Pia et al., 2004). Other studies identified increased anosognosia and chronic AHP with general or increased cognitive impairment (Goldberg & Barr, 1991; Levine, et al., 1991; McGlynn & Schacter, 1989; Orfei et al., 2018; Vocat et al., 2010). According to other studies, mental impairment can vary greatly between patients, ranging from very intelligent and mentally preserved to below cut-off scores (Berti et al., 1995; Marcel et al., 2004). In numerous case studies, individuals are portrayed as functional in terms of conversation, interaction, and intellectual abilities, with distinctive and unusual reactions observed only when discussing their hemiplegia (Marková & Berrios, 2014).

Emotional regulation plays an intriguing role in the manifestation of AHP. Adverse reactions can be observed, with patients displaying resentful attitudes toward their motor impairment and defensive reactions (Moro et al., 2021). Conversely, they can exhibit anosodiaphoria, a lack of interest or concern (Babinski, 1914; Langer & Bogousslavsky, 2020; Gainotti, 2012). Earlier studies suggested that depression is not influenced by AHP (Starkstein et al., 1992). However, recent studies contradict these findings and provide evidence that patients with AHP exhibit a negative correlation with depression (Besharati et al., 2014), and anxiety compared to HP patients, who were found to be significantly more depressed (Besharati et al., 2022; Fotopoulou et al., 2008). Reduced anosognosia was observed to be related to elevated depression and lowered hedonic tone (Orfei et al., 2018). Interestingly, in an experimental study by Besharati et al. (2014), the induction of negative emotions led to a notable enhancement of motor awareness in anosognosic patients compared to controls, whereas the induction of positive emotions did not yield similar effects.

Proprioception impairments were found in both AHP and HP controls, with AHP patients exhibiting significantly more severe deficits (Besharati et al., 2022; Levine et al., 1991; Orfei, 2007; Vocat et al., 2010). In the acute phase following a stroke, there was a significant correlation between the severity of AHP and the extent of proprioceptive loss (Vocat et al., 2010).

Additionally, executive functions were identified to be much more severe in AHP than in HP patients (Moro et al., 2016). Furthermore, patients with AHP tend to experience more frequent falls than other patients after a stroke (Hartman-Maeir et al., 2001).

AHP can also co-occur with other disorders of consciousness or neuropsychological deficits, including allochiria, asomatognosia, neglect, and somatoparaphrenia (Besharati et al., 2022; Bottini et al., 2018; Fotopoulou et al., 2010; Jehkonen et al., 2006; Jenkinson et al., 2018; Marcel et al., 2004; Orfei et al., 2009; Starkstein et al., 1992). Especially neglect, is often observed more prevalently in AHP patients compared to those with hemiplegia (Berti et al., 2005; Monai et al., 2020; Moro et al., 2016; Pia et al., 2004). Neglect is characterised by an individual's inability to respond to stimuli presented in the hemispace contralateral to the lesion (Kortte & Hillis, 2009). Recent research suggests that only personal neglect is likely to be excluded, indicating that the network for personal neglect seems to be distinct and not involved in other body representation disorders, including AHP (Bertagnoli et al., 2022). However, visuospatial neglect can potentially impact the prospective estimation of the patient's motor ability (Kirsch et al., 2021). Extrapersonal neglect is more prevalent in patients with AHP, with reported frequencies ranging from 52% to 87%, compared to 6% to 23% in patients without anosognosia (Bisiach et al., 1986; Cutting et al., 1978; Hier et al., 1983; Starkstein et al., 1992). While neglect and AHP

can occur simultaneously and influence each other's pathology (Cocchini et al., 2002; Caggiano & Jehkonen, 2018), AHP can also be observed in isolation and does not explain all cases of anosognosia (Berti et al., 1996; Berti et al., 2005; Pia et al., 2004; Gandola et al., 2014). These cases indicate that anosognosia is not simply explained by an attentional deficit or unilateral spatial neglect of contralateral events (Bisiach et al., 1986; Berti et al., 1996; Marcel et al., 2004).

Although disorders of the sense of ownership frequently co-occur with AHP patients (Moro et al., 2021), they have been dissociated from AHP (Gandola et al., 2012; Invernizzi et al., 2013; Romano & Maravita, 2019). Furthermore, Moro et al., (2021) found no correlation between disorders in the sense of ownership, such as anarchic hand syndrome or illusory limb movements. Instead, the sense of agency seems to be disturbed in patients with AHP. The sense of agency, representing the feeling of controlling one's actions and their effects (Moore, 2016; Haggard, 2017), becomes distorted in patients with AHP (Cocchini et al., 2022). Interestingly, this distortion is not only limited to the contralesional side, but also manifests on the unimpaired ipsilesional limb side. AHP patients demonstrate compromised abilities to detect visual incongruences (Preston et al., 2010), and recall whether previous movements were executed or imagined (Saj et al., 2014). They encounter difficulties in remembering the source of action, even for the healthy limb. The extent of the sense of agency and the distortion in monitoring movements are strongly influenced by the severity of motor impairment (Cocchini et al., 2022). It is worth mentioning that, while the monitoring of movement appears to be altered, motor planning seems to remain preserved in AHP (Cocchini et al., 2018; Garbarini et al., 2012). AHP patients perceive the complexity and difficulty of tasks in the same way as healthy controls because they do not struggle to estimate the difficulty of a task itself. Instead

their challenges lie in accounting for their motor impairment while performing the task.

It is exceptionally challenging to study AHP due to lack of internal consistency of movement claims in patients, varying throughout tasks and assessment times (Antoniello & Gottesman, 2020). The dynamic expression of sensorimotor experience in AHP makes it a multicomponent syndrome. The variety of possible combinations of deficits, including impaired body perception, learning and memory, multisensory integration, and spatial neglect, varying in severity, may contribute to the diverse manifestations observed within patients.

3.2 Assessment of Anosognosia for Hemiplegia

Diagnosing AHP necessitates a comprehensive assessment across various dimensions to identify the nature and severity of the condition. The variations in awareness have traditionally been conceptualised as varying degrees of severity (Bisiach et al., 1986). A neurological examination of AHP should encompass assessing the main dimensions identified to current knowledge (Table 1). The diagnostic tools predominantly involve meta-cognitive tasks to examine the awareness and self-evaluation of patients' deficits and motor condition, commonly referred to as "explicit anosognosia" (Coccini et al., 2012). Overall, the primary domains investigated in comprehensive assessment of AHP could include causal attribution, cognitive functions, executive functions, functional implications, implicit knowledge, modality specificity, psychopathology, co-occurring manifestations, and recovery expectations. Explicit anosognosia is evaluated verbally, through structured questions posed to the patient regarding their deficits. Collected data is compared to various sources like medical records, neuropsychological performance, or clinician's evaluation, depending on the assessment method (Table 1). Evaluating implicit

knowledge involves tasks like unimanual and bimanual activities (Cocchini et al., 2010), or experimental designs (Fotopoulou et al., 2010; Jenkinson et al., 2009). Modality-specific testing uses distinct items for varied deficits, such as upper and lower limb paresis (Table 1).

In the case of a mild to moderate deficit, a differential diagnosis from psychological denial has to be made to confirm the diagnosis of AHP. Denial should be excluded due to the similar expression of minimization or denial of the impairment (Kortte et al., 2003; Prigatano, 1998). Additionally, the diagnosis of neglect has to be taken into high consideration due to the frequent co-occurrence of AHP and spatial neglect (Berti et al., 2005; Monai et al., 2020; Orfei et al., 2007; Pia et al., 2004).

Furthermore, aspects of aphasia should be considered and collaborated with through body language and gestures to assess the possibility of AHP (Orfei et al., 2009). Specific functions like mnestic and language functions, including semantic and phonemic fluency impairment in anosognosia, can be assessed (Bogod et al., 2003; Marcel et al., 2004; Starkstein et al., 1992). Executive functions related to attentional control, cognitive flexibility, inhibition, and set-shifting should be explored. Psychopathological aspects like depression, anxiety, apathy, anger, hostility, and alexithymia also need consideration (Fowler et al., 2018; Spalletta et al., 2012). Specific scales and measures for each subdomain summarised can be seen in Table 1.

Scales to assess AHP should not be dichotomous due to its multicomponent manifestation. A rather holistic than isolated view should be integrated when measured. A Likert scale is often preferred and applied in several clinical settings to assess AHP (Orfei et al., 2009). The most utilised diagnostic tools to assess AHP are self-evaluation questionnaires and structured interviews (Coccini et al., 2012).

Common examples are Bisiach's Scale (Bisiach et al., 1986), Cutting's questionnaire (Cutting, 1978), the Anosognosia Questionnaire (Starkstein et al., 1992), Feinberg's scale (Feinberg et al., 2000), and the Structured Awareness Interview (Marcel et al., 2004), all focusing on awareness of the sensorimotor deficit (Orfei et al., 2009), and on measurement of explicit awareness. Recent measures such as the Behavioural Motor Task (BMT) by Cocchini et al. (2010a) and the Visual-Analogue Test for Anosognosia for Motor Deficits (VATAm) by Della Sala et al. (2009) try to extend the variety and options for testing for AHP.

Differing medical conditions, such as AHP caused by TBI or dementia, tend to have more flexible tools of assessment, and clinicians can actively monitor the development of AHP under various aspects and circumstances, such as in cases of daily living, progression, or regression. Therefore, the assessment of patients can differ depending on AHP onset with dementia, stroke, or TBI. In the case of the onset through a stroke, patients can be examined in the hyperacute stroke phase, when AHP is most robust. In the acute phase, the patient should be mainly assessed for awareness deficits with evident impairment. In this stage, due to fatigue and diminished concentration, reliable self-evaluation of cognitive abilities might be too early and yet undiscovered by the patient (Orfei et al., 2009).

Typically, those structured interviews and questionnaires engage the patient in a conversation about their present understanding of their condition before and after the demonstration of their impairment, starting with general questions about their current situation, towards more specific questions regarding their limb function and strength. If denial of impairment occurs, the examiner will require a demonstration of the function by asking the patient to perform specific actions with follow-up questions

if the action was performed by the patient to further deepen the understanding of the degree of awareness. Cutting's questionnaire (Cutting, 1978) also differentiates between patients' varying reactions to the request to move the impaired limb. Furthermore, the patient will be asked to perform unilateral and bilateral tasks such as clapping or walking (Berti et al., 1996; Marcel et al., 2004; Della Sala et al., 2009). Whereas in mild to moderate severity of AHP, simply requesting the patient to walk or clap seemed to be not a sufficient predictor, while opening bottles or washing dishes seemed to be better indicators (Della Sala et al., 2009). Lastly, the patient is asked to predict their ability to move. The total discrepancy score is obtained by subtracting the patients' estimation of their bodily state and an objective standard assessed by a caregiver or clinician's judgement. Clinicians' judgement is one of the most essential factors in the measurements of anosognosia in the case of a stroke. Questionnaires of TBI and dementia rely mostly on relatives or staff reports (Orfei et al., 2009). The assigned score should reflect the degree of awareness of the patient's condition.

The majority of the mentioned measures are in line with the common understanding of anosognosia and are vastly similar, laying the focus on closely related themes. Due to the complexity of awareness impairment in AHP, some criticise that current diagnostic tools of AHP fail to capture the whole diversity of domains of AHP, varying within patients (Antoniello & Gottesman, 2020; Coccini et al., 2012; Orfei et al., 2009; Prigatano, 2010). Orfei et al. (2009) examined the assessments for AHP after stroke and TBI of the last 30 years and reflected that despite the availability of reliable, high-quality measurements, the multifaceted phenomenon of AHP is not fully captured due to too narrow a focus, which might overlook the complexity of AHP, and neglect other areas of interest. Criticism by

Antoniello & Gottesman (2020) and Coccini et al. (2012) emphasise further assessment, going beyond verbal responses, and incorporating performance evaluation and behavioural data. Scales such as the Bisiach Scale (Bisiach et al., 1986) would flatten the severity score due to the multidimensionality of AHP, neglecting essential research opportunities to study various aspects of AHP. Due to interviews and questionnaires requiring the patient to provide verbal fluency, interviewing patients with language impairment is quite challenging, resulting in a high exclusion of patients and a lack of understanding of AHP in patients with lesions in the left hemisphere (Coccini et al., 2012). The VATAm, an innovative diagnostic tool developed by Della Sala et al. (2009), tried to cover this gap by examining patients with AHP and language deficits by evaluating bimanual or bipedal actions with the aid of illustrated drawings and illustrated Likert scales to account for possible language impairment, and to assess left brain-damaged patients more frequently. Compared to the traditional methods of Berti et al. (1996), which only identified 10% of all patients with AHP, the VATAm (Della Sala et al., 2009) identified 40%, due to the inclusion of patients with language impairment (Nurmi & Jehkonen, 2014). Similar to the recently developed ECT (Errand Choice Test) by Cocchini et al. (2018), capturing the assessment of perception of one's motor skills of everyday tasks comparing unimanual and bi-manual tasks, capturing several cases of AHP for patients with left hemispheric lesions. These cases provide further evidence towards underestimation and underassessment of patients with left hemispheric lesions.

Other modern assessment methods address the criticism of excessive focusing on single dimensions, such as explicit and implicit awareness (Coccioni et al., 2018), without considering aspects like sense of agency, body ownership distortion, or unusual emotional reactions. Many assessments fall short in

adequately distinguishing occurring disorders next to AHP. In response, Moro et al. (2021) introduced the 40-item Motor Unawareness Assessment (MUNA), designed and tested for patients with right-hemisphere strokes. Pioneering in their approach, they integrated body and motor awareness to more effectively differentiate explicit and implicit disorders of awareness. MUNA specifically evaluates explicit motor awareness, emotional reactions, implicit motor awareness, impaired sense of ownership, sense of agency, and illusory movement. Still, there is a lack of assessment ability for left hemispheric lesioned patients with language difficulties.

Additional criticism is directed at structured interviews for not capturing various aspects of unawareness in rehabilitation. For instance, there are cases where patients become aware of their impairment after a demonstration but remain unaware of the consequences these deficits may cause (Nimmo-Smith, 2005). Some also note an empirical learning process, where patients adjust their verbal evaluations and behaviour during the interview, making it more challenging to assess AHP (Berti et al., 1996; Cocchini et al., 2012; Marcel et al., 2004). The reliability of interviews assessing cognitive function in individuals post-injury is criticised as the gold standard due to potential biases arising from clinicians' and caregivers' cognitive abilities or emotional responses to incidents, with clinicians lacking prior knowledge of pre-injury functioning (Gasquoine, 2016). Limited correlation exists between clinician and family members' judgments (Fordyce & Roueche, 1986; Sherer et al., 1998), and instead, the discrepancy score between clinician and patient appears to be a more reliable predictor (Sherer et al., 2003). An extensive range and increased holistic approach of diagnostic tools in line with the modern research findings about the development and consequences of AHP are demanded to examine the nature of unawareness patients display to a greater extent. This

holistic approach could potentially reveal the diversity of facets within AHP, promoting better insights for research and more effective rehabilitation strategies.

Table 1

Clinical Rating Scales and Self-Report Measures for Investigation of different Domains of AHP through multiple Aetiology.

Domain	Sub-Domain	Test Administered
Awareness	Degree of awareness	Anosognosia Questionnaire (Starkstein et al., 1992), AHP Questionnaire (Feinberg et al., 2000), AQ [a], Awareness Questionnaire (Hibbard et al., 1992), Bisiach scale (Bisiach et al., 1986), Change Assessment Questionnaire (Lam et al., 1988), Cutting's Questionnaire (Cutting, 1978), The Self-Awareness Multilevel Assessment ScaleImpaired (Bivona et al., 2020), PCRS [b], SADI [c], SAI [d]
	Explicit awareness	Anosognosia Questionnaire (Starkstein et al., 1992), AHP Questionnaire (Feinberg et al., 2000), Verbal assessment of patient's subjective experience of symptoms (Vocat et al., 2010), Berti et al. (1996) Structured Interview, Bisiach scale (Bisiach et al., 1986), Cutting's Questionnaire (Cutting, 1978), Implicit and explicit tests for anosognosia (Fotopoulou et al., 2010), MUNA [e], PCRS [d], SAI [c], Semistructured interview to patients and relatives (Hochstenbach et al., 2005), Structured Interview (Anderson & Tranel, 2007), VATAm [f]
	Implicit knowledge	Anosognosia Questionnaire (Starkstein et al., 1992), BMT [g], Circles - lines bimanual motor task (Garbarini et al., 2012), Grip selection task and reality monitoring task (Jenkinson et al., 2009), Dot-probe Test (Nardone et al., 2008), Experimental tasks for implicit and emergent awareness (Moro et al., 2011), Implicit and explicit tests for anosognosia (Fotopoulou et al., 2010), Rubber hand experiment (Fotopoulou et al., 2008), SAI [c], Task choice method (Ramachandran, 1995), VATAm [f]
	Modality specificity	BMT [g], Bisiach scale (Bisiach et al., 1986), PCRS [d], SAI [c], VATAm [f]
	Suitable for aphasic Patients	Errand Choice Test (ECT; Cocchini et al., 2018), VATAm [f]
	Sensorimotor deficits	Anosognosia Questionnaire (Starkstein et al., 1992), AHP Questionnaire (Feinberg et al., 2000), Bisiach scale (Bisiach et al., 1986), PCRS [d], SADI [b], SAI [c]
Behaviour	Alexithymia	TAS-20 [h]
	Functional implications in ADL [i]	Katz Index of Independence in ADL (Katz, 1983), AQ [a], CBS [j], Functional Independence Measure (Davidson, 2023), ECT (Cocchini et al., 2018), Head Injury Behaviour Scale (Godfrey et al., 2003), PCRS [d], SADI [b]
Cognition	Global cognitive level	AQ [a], Awareness Questionnaire (Hibbard et al., 1992), MMSE [k], PCRS [d], SADI [b], Self-rating questionnaire (Berti et al., 1996), Structured Interview (Anderson & Tranel, 2007), Raven's Coloured Progressive Matrices Test (Raven, 2012), WAIS-R [l]

Domain	Sub-Domain	Test Administered
Cognition	Executive functions and attention	Wisconsin Card Sorting Test (Creque & Kolakowsky-Hayner, 2017), Stroop Test (Stroop, 1935)
	Language functions	Structured Interview (Anderson & Tranel, 2007), Phonological and semantic verbal fluency tests, VATA-L [m], FAS (Patterson, 2011)
	Mnestic functions	Digit Span (Lamar et al., 2018), Corsi Block Test (Schellig, 2012), Rey Auditory Verbal Learning Test (Bean, 2011)
Affective Regulation	Awareness of interpersonal deficits	AQ [a], Awareness Questionnaire (Hibbard et al., 1992), Head Injury Behaviour Scale (Godfrey et al., 2003), MUNA[e], PCRS [d], SADI [b], State Trait Anger Expression Inventory (Spielberger et al., 1988)
	Mood, anxiety, and apathy	Apathy Evaluation Scale (Marin et al., 1991), Beck Depression Inventory (Beck, 1961), HAM-A and HAM-D [n], PSDRS [o]
Co-occurrences	Anosognosia vs Denial	Clinician's Rating Scale for Evaluating Impaired Self-Awareness and Denial of Disability (Prigatano & Klonoff, 1998), LDIS [p], PCRS [d]
	Neglect	CBS [j], Self-rating questionnaire (Berti et al., 1996), Behavioural Inattention Test(Polejaeva & Woods, 2017), Line crossing, letter cancellation, figure and shape copying, and line bisection tests, trials to assess non-visual neglect forms (Lindell et al., 2007)
	Consciousness disorders	AQ [a], AHP Questionnaire (Feinberg et al., 2000), MUNA[e], Cutting's Questionnaire (Cutting, 1978), SAI [c]
Rehabilitation	Compliance	LDIS [q]

[a] AQ = Awareness Questionnaire (Sherer et al., 1998). [b] PCRS = Patient Competency Rating Scale (Prigatano et al., 1986). [c] SADI = Self-Awareness of Deficit Interview (Fleming et al., 2020). [d] SAI = Structured Awareness Interview (Marcel et al., 2004). [e] MUNA = The Motor Unawareness Assessment (Moro et al., 2021). [f] VATAm = Visual-Analogue Test for Anosognosia for Motor Impairment (Della Sala et al., 2009). [g] BMT = Behavioural Motor Task (Cocchini et al., 2010). [h] TAS-20 = Toronto Alexithymia Scale (Bagby et al., 1994). [i] ADL = Activities in Daily Living. [j] CBS = Catherine Berg-ego Scale (Azouvi et al., 2003). [k] MMSE = Mini-Mental State Exam (Crum et al., 1993). [m] WAIS-R = Wechsler Adult Intelligence Scale-Revised (Wechsler, 2012). [n] VATA-L = Visual-Analogue Test for Anosognosia for Language Impairment (Cocchini et al., 2010). [o] HAM-A and HAM-D = Hamilton Anxiety Scale (Hamilton, 1959) and Hamilton Depression Scale (Hamilton, 1960). [p] PSDRS = Post Stroke Depression Rating Scale-Subscale of awareness of illness (Gainotti et al., 1997). [q] LDIS = Levine Denial of Illness Scale (Levine et al., 1987).

3.3 Motor Awareness

A lively debate persists regarding whether AHP can be explained by various facets of motor awareness and behaviours, including cognitive, intentional, motivational, monitoring, motor, sensory, or feedback dimensions. To develop an understanding of such a phenomenon as AHP, awareness of our motor system and subjective experience of motor control has to be comprehended to further understand the pathology of AHP. Many concepts remain unknown regarding what we are aware of and which motor control functions reach our consciousness. Disorders of awareness, attention, and neglect indicate that consciousness can be dissociated and coexists separately, within the stream of consciousness. Therefore, specific lesions can affect single streams of consciousness, while sparing others (Bisiach & Berti, 1995). To further investigate the underlying theories of AHP, it is crucial to establish a foundational understanding of motor control and awareness.

The terms "aware" and "unaware" are frequently applied with considerable flexibility. In this review, the definition of motor awareness refers to the acknowledgment of an individual's actual motor state, who is in charge of performing the movement and the ability to execute specific actions (Pacella & Moro, 2020). The aware experience of motor control and execution is the product of various integrated components, such as the intention to move, sense of agency, motor prediction of sensorimotor feedback, monitoring of execution, the actual sensorimotor feedback of an executed action, and the experience of one's own body in interaction with external factors such as environmental and social contexts. The integration of sensory information from the body is vital for motor awareness, allowing individuals to maintain a sense of bodily self and accurately represent their body's position in space during movement (Craig, 2009; Coslett et al., 2008; Romano et al., 2013;

Sirigu et al., 1991). This complex integration of awareness components contributes to an individual's perception and experience of self (Gallagher, 2000).

Our comprehension of the system accountable for regulating our actions remains narrow, as does our awareness of the components indirectly engaged in action control (Frith, 2002). Research suggests that the brain initiates preparations for a motor act approximately 80 ms before subjective awareness of the decision (Haggard & Eimer, 1999). This phenomenon may be addressed by the hierarchical organisation of the motor system, where details of movement are determined at the lowest level possible, with only the highest levels accessible to consciousness (Haggard, 2005). Conscious awareness of preparing to move is specific to the intended movement rather than a global idea of movement. For example, when reaching for a cup, we have a clear goal and desire but are not consciously aware of the exact muscles involved in achieving this goal. This indicates that our awareness is limited to a global perspective rather than insight into specific computations and motor commands. Typically, awareness of selecting one action over another arises after the choice has been made, while awareness of initiating an action occurs before the movement begins. These temporal distinctions converge in consciousness, forming our sense of agency by combining the intention to act with the outcomes of the action.

Therefore, intention tied to outcomes, is the foundation of the integrated sense of agency (Pacella & Moro, 2022). Sense of agency is referring to the feeling of being in control of one's actions and significantly contributes to awareness of motor performance, and is separate from the sense of body ownership (Tsakiris et al., 2010). Integral to all facets of motor awareness, it differs from feelings of urge or anticipation (Pacella & Moro, 2022). This suggests that the sense of agency involves

integrating information about events in our body or the external world with efferent signals.

Motor intentions are characterised by a strong sense of agency, indicating control over external events (Haggard, 2005). The intention to act can be defined as the consciousness to perform or inhibit an action (Haggard, 2019; Pacella & Moro, 2022; Seghezzi & Haggard, 2022). The term "intention" encompasses various stages of information processing that translate desires and goals into actions (Haggard, 2005). Such actions are typically influenced by task context and memory of past associations rather than immediate stimuli. They often involve cognitive processes such as planning and deliberation before their execution, requiring focused attention and subsequent monitoring for potential learning. Libet et al. (1983) conducted an experimental EEG study to investigate the timing of when an intention to move becomes conscious. Their findings revealed that the conscious intention to move occurs approximately 200 ms before the actual movement, and about 1 s after the readiness potential is detected. This suggests that part of the preparation for movement occurs before the conscious awareness of the intention to move emerges and that intention seems to be a consequence of brain activity instead of the cause of it. Therefore, Guggisberg et al. (2011) emphasise the distinction between the moment when the intention to act is generated and when an individual becomes aware of this intention. Intentional action is not simply a binary decision but rather the culmination of multiple decisions. These decisions encompass not only whether to carry out an action or not, but also considerations regarding the nature of the action and the timing of its execution.

In our daily interactions with others and our environment, motor performance plays a crucial role in expressing our beliefs, desires, and intentions while pursuing

our goals. Successful monitoring of behaviour is necessary for becoming aware of one's own movements, requiring continuous updating of motor performance. Motor monitoring and error detection serve as tools for modifying and optimising behaviour, allowing us to adjust and correct possible mistakes (Boksem et al., 2006; Ullsperger & von Cramon, 2001). Mismatches and mistakes arise into consciousness when there is a significant disparity between actual sensory feedback and predicted sensory feedback, reflecting the internal prediction of motor outcome and the actual outcome (Cocchini et al., 2022; Frith et al., 2000; Serrien & Spapé, 2011; Wolpert et al., 2011). To generate and execute actions effectively, we rely on a motor monitoring process that continuously evaluates the efficacy of our motor acts, facilitating awareness of the movements we perform. Often this monitoring process is achieved by a so-called comparator. Malfunctioning components of the comparator can lead to incorrect beliefs about self-generated movements and a distorted sense of motor control agency (Blakemore et al., 2002; Mograbi & Morris, 2013).

For optimal monitoring and motor control, individuals rely on internal or mental representations of actual, desired, or predicted states of their limbs regarding movement performance and the external world (Blakemore et al., 2002; Frith et al., 2000). These internal representations function like previously created internal models that can simulate and predict the body's interactions with the external environment (Frith et al., 2000). However, only certain aspects of these internal representations are conscious to an individual. Research has shown that unimpaired motor awareness is often narrowed down to the predicted consequences of movements, while corrections of movement occur unconsciously, if the desired goal is still achieved (Blakemore et al., 2003; Fourneret & Jeannerod, 1998; Goodale et al., 1994). Additionally, noisy environments and information overload can hinder

awareness of minor corrections and muscle movements (Preston & Newport, 2014). However, sensory consequences increase awareness if a motor act is unsuccessful or unexpected, even if the individual is unaware of the cause of the deviation between expected and actual movements (Antoniello & Gottesman, 2020). Also, the size of adjustments determines the degree of awareness. Although the exact thresholds for awareness discrepancy are unknown, experimental studies have attempted to estimate them. Blakemore and Frith (2003) identified that individuals become aware, on average, of a discrepancy of 15 degrees spatially or 150 ms temporally when specifically instructed to detect a mismatch. However, without instruction to pay attention to discrepancies, individuals can experience delayed tactile consequences of their movements for up to 300 ms without noticing (Blakemore et al., 1999).

Movement predictions partly account for the delayed tactile consequences of our own movements. Predictions of movements serve several purposes, including anticipating the path of limb movement and forecasting tactile and kinesthetic sensations (Frith et al., 2000). Prediction serves a crucial role in maintaining accurate performance despite feedback delays by estimating the outcome of motor commands before true sensory feedback is received (Miall et al., 1993). When a motor command is issued, an efference copy of the command is simultaneously generated to estimate the sensory consequences of the movement (Miall & Wolpert, 1996). These predictions help filter sensory information and attenuate self-generated sensations from changes in the external environment. Moreover, prediction integrates sensory and motor information to estimate the current state of the system, effectively bridging the gap between outgoing motor commands and subsequent sensory feedback (Frith et al., 2000). Studies have shown that self-produced

sensations are easier to predict compared to externally generated sensations (Frith et al., 2000). This phenomenon underlies the finding that a tactile stimulus administered to oneself feels significantly less intense compared to when administered by someone else (Frith et al., 2005; Shergill et al., 2003; Weiskrantz et al., 1971). The reduction in perceived intensity relies on accurate prediction, as the subjective feeling of self-touching intensifies when the relationship between movement and sensory outcomes is deliberately distorted (Blakemore et al., 1999). This dampening of self-generated sensations is also evident at the physiological level, with activity in the somatosensory cortex notably decreasing when tactile stimulation is self-administered (Blakemore et al., 1998). Interestingly, this effect is not exclusive to self-touching but extends to sensations arising from any type of movement. The forward model implies that awareness of movement initiation is based on predicted sensory consequences of movement, which are available before sensory feedback of the actual movement occurs (Libet et al., 1989). This highlights the proactive nature of the motor control system in anticipating and preparing for sensory outcomes, contributing to our conscious awareness of movement initiation.

The forward model is part of computational and internal models, which include all previously named concepts such as motor- intention, sense of agency, monitoring and prediction to provide a comprehensive view on motor awareness. According to these computational models proposed by Frith et al. (2000) and Blakemore and Frith (2003), movement is closely intertwined with action intention and motor planning. The success of actions is attributed to systems within the central nervous system (CNS) known as predictors and controllers (Frith et al., 2000). Inverse models, also named controllers, are crucial for determining the motor commands necessary to achieve desired outcomes, such as executing simple reaching and grasping

movements (Frith et al., 2000). The process of prediction within the motor system, known as the forward model, is essential for anticipating and compensating for the sensory effects of movement. The operation of predictors and controllers necessitates the representation of at least three states of the motor system: the present state, the desired state, and the predicted state. Movement command and intention play crucial roles in predicting and estimating the consequences of movements. The process of executing movements entails a sequence of muscle contractions while simultaneously relying on sensory information to guide decision-making and observe outcomes (Frith et al., 2000). This model involves generating predictions based on motor commands and sensory feedback to establish a causal relationship between actions and their outcomes (Ito, 1970; Wolpert et al., 1995).

In healthy individuals, the CNS navigates motor control processes with remarkable precision. When initiating a movement, the CNS issues a motor command, while a predictor calculates the expected sensory outcomes of that command. Simultaneously, a controller establishes the connection between the intended state and the necessary motor command to achieve it, essentially bridging the gap between desired and executed actions. The fundamental role of the motor control system is to manage the interplay between motor commands and sensory feedback. Each interaction between the musculoskeletal system and the environment produces immediate sensory consequences, as motor commands translate into sensory feedback. This feedback loop allows for the prediction of more efficient future movements. During intentional movements, coordination between motor and sensory nerves ensures the successful execution of the intended action. When predicting the sensory outcomes of our actions, the response to these

sensations is dampened. This intricate interplay between motor and sensory functions, facilitated by a feedback loop, not only ensures the smooth execution of internal movements but also facilitates learning from past movement experiences. Errors between desired and actual states contribute to improving controller functioning, while errors between predicted and actual states enhance predictor functionality. This learning process enhances the efficiency and effectiveness of future movements (Frith et al., 2000; Fotopoulou et al., 2008). Hemiplegic patients without anosognosia are capable of detecting the mismatch between prediction and the final sensorimotor condition, allowing them to construct normal motor awareness (Blakemore & Frith, 2003). This leads to the underlying theories of unawareness of actual motor performance, the essences of AHP.

3.4 Underlying Theories of AHP

Over the last one hundred years, various theories have been proposed to explain motor awareness models and the pathology of AHP. In earlier studies, anosognosic patients were believed to have a previous general cognitive impairment (Levine et al., 1991; Weinstein & Kahn, 1955; Ullman, 1964). However, the presence of anosognosia in patients with normal mentation and orientation has led to the rejection of many past theories (Babinski, 1914; Berti et al., 1996; Marcel et al., 2004). Contemporary theories on the manifestation of AHP are primarily grounded in neurobiological processes, particularly dysfunctions between the motor control system and the accompanying sensory feedback loop (Bottini et al., 2009; Byrd et al., 2020). Other theories of AHP revolve around the failure of the feedback loop to update the motor cortex regarding the success of the intended movement. Many variations in these theories lie in the mechanisms that trigger the update of the feedback loop (Fotopoulou et al., 2008; Jenkinson et al., 2009; Preston & Newport,

2014; Vocat et al., 2013; Saj et al., 2014). Researchers also proposed that AHP may be caused by a selective deficit in motor planning (Heilman et al., 1998), or higher-level motor monitoring processes (Berti et al., 2005; Fotopoulou et al. 2008; Frith et al., 2000; Garbarini et al., 2012). For instance, studies demonstrating dissociations between various forms of unawareness within the same individual (Berti et al., 2007; Breier et al., 1995; Jehkonen et al., 2000) suggest that denial could be conceptualised as a specific impairment of monitoring. Within this framework, AHP might be understood as a monitoring deficit that specifically affects motor awareness (Berti et al., 2007).

A monitoring deficit or damage to it and its consequences was viewed from different perspectives and angles in past theories. For example, loss of intention to move (Coslett, 2005; Gold et al., 1994; Heilman et al., 1998; Vallar et al., 2003) was proposed, which would provide an explanation of no mismatch about motor planning and execution, and the inability to interpret the absence of movement is abnormal. Contrary to this hypothesis, participants without hemiplegia can have the feeling of intention to move, in the absence of any performed movement (Fried et al., 1991). Conscious intention can be experienced without the actual execution of movements.

Recent findings provide evidence that in AHP, the representation of intended and predicted positions of limbs remains intact. Fotopoulou (2015) developed the idea of the dominance of motor intention over movement sensation, based on direct evidence showing that altered awareness of action in AHP reflects a dominance of motor intention prior to action. In an experimental study by Fotopoulou et al. (2008), evidence was found that patients with AHP tend to detect more movement incorrectly when it did not occur, particularly when they intended to move, compared to situations with no anticipated movement or externally generated movement. Berti

et al. (2005) suggest that the experience of intention to move does not depend on the functioning of a single cortical region but instead arises from a dynamic interaction between different premotor areas. AHP patients never doubt their will when they are programming a movement, despite the impossibility of performing it (Berti & Pia, 2006). This accessibility may play a role in the development of the delusional belief of being able to perform motor acts, despite experiencing complete left hemiplegia (Berti et al., 2005; Berti et al., 2007; Lau et al., 2004).

Furthermore, Berti et al. (2007) also tested the EMG activity of proximal muscles to assess intention for voluntary action in patients with AHP. They proposed a different view, suggesting that anosognosia results from direct damage to the comparator component of the model, rather than a loss of intentionality. They argue that anosognosic patients should still have intentions for voluntary actions, and the neural bases of motor intention should be preserved. Thus, they view anosognosia as a disturbance of the comparator systems of action generation, emphasising the preservation of motor intention in anosognosic patients. Their findings indicate that motor awareness can persist even without perceptual awareness, allowing motor control to depend solely on an internal model. This internal model provides the individual with the normal belief of executing appropriate voluntary movements.

This internal model and computational models previously explained (Blakemore et al., 2002; Frith et al., 2000), attempt to explain AHP by stating that the actual and predicted states of motor movements rely on the stream of motor commands, and sensory signals from the skin and muscles. Damage to these neural networks or components can lead to a diffuse awareness of actions and impaired action control, contributing to the pathology of neuropsychiatric disorders of awareness (Blakemore et al., 2002; Frith et al., 2000). Normally, patients with AHP

should become aware of discrepancies if their expected and predicted movement, such as lifting their hands, does not match the actual movement of not moving at all (Berti & Pia, 2006). Research has shown that patients with AHP are aware of their goal to perform a movement and predict its outcome, but lack awareness when the intended outcome does not occur. Awareness of initiating a movement is rooted in a representation of the anticipated consequences of that movement, rather than its actual outcomes (Frith et al., 2000). This representation of anticipated consequences can be formulated when the controllers compute the appropriate motor commands and the predictors derive expected outcomes from these commands. Thus, a patient with a paralyzed limb might still experience the typical sensation of initiating a movement with that limb if the controller and predictor functions normally. However, persisting in the belief that the movement was successfully initiated would require additional abnormalities in the system. In alignment with Frith et al. (2000), Berti et al. (2007) provided evidence suggesting that patients with AHP retain the ability to program movements and make predictions. Illusory awareness or false sense of movement in AHP stems from the comparison of the intended or desired and predicted positions of the limbs (Blakemore et al., 2003). According to this hypothesis, individuals with AHP can construct accurate mental representations of where their desired and predicted limb positions are but lack awareness of the discrepancy with the actual outcome (Frith et al., 2000). Therefore, they lack access to contradictory sensory feedback indicating movement failure. This lack of feedback occurs due to brain damage affecting regions responsible for registering the limb's actual state or due to neglect of this contrary information (Berti et al., 2005). In the framework of forward models of motor control, efferent copies predict the sensory consequences of motor commands during movements (Blakemore et al., 2002;

Heilman 1991; Wolpert & Ghahramani, 2000). Awareness of actual sensory consequences arises when deviations from expectations occur. If the predictor and controller malfunction due to brain lesions post-stroke, the ability to correctly identify positions of movements is still predicted and estimated based on previous experience or movement memory before the brain lesions (Langer, 2009). The failure to update through a standard feedback mechanism leads to a lack of recognition of impairment or disability, clinically manifesting as delusional features, or denial of deficits (Blakemore et al., 2002; Frith et al., 2000). This supports the notion that awareness in patients with AHP is primarily influenced by an impaired comparator system dominated by intention and prediction, while neglecting or failing processing of sensory evidence contributing to accurate feedback of movement performance with a failure to update the operations of the predictor (Fotopoulou et al., 2008; Frith et al., 2000). Furthermore, experimental studies on motor imagery or mental visualisation processes suggest that these mechanisms may override sensory feedback in anosognosic patients, indicating a failure of the sensory feedback loop to reach the motor cortex (Jenkinson et al., 2009). Indeed, the process of imagining movement and preparing to move activates a subset of brain areas similar to those activated during actual movement execution (Jeannerod & Frak, 1999; Naito et al., 2002; Nyberg et al., 2001). Interestingly, even in healthy individuals, there can be confusion between actions imagined and those actually performed when asked to recall them weeks later (Thomas & Loftus, 2002). In anosognosic patients, intended self-generated movements overshadow the actual sensory feedback, resulting in a disturbed sense of agency for actions that never occurred, as demonstrated in experimental rubber hand studies (Fotopoulou et al., 2008; Cocchini et al., 2022). This hints at damage in areas responsible for

monitoring both motor outflow and sensory inflow (Frith et al., 2000; Fotopoulou et al., 2008).

Simplified, Blakemore and Frith (2003) stated that patients with AHP fail to monitor the mismatch between the prediction and the actual execution due to a malfunctioning comparator. Consequently, an illusory motor awareness is constructed based on predicted or intended movements. In line with this research, recent studies have suggested an impairment in comparator mechanisms responsible for aligning predicted movements based on intention, with actual movements based on sensory feedback (Bottini et al., 2010; Fotopoulou et al., 2008). This discrepancy between actual and intended movement might be the consequence of explicit unawareness in AHP (Frith et al., 2000; Berti et al., 2005; Berti & Pia, 2006). The deficit can be based on solely representation of intended or predicted movement (Berti et al., 2007; Desmurget & Sirigu, 2009; Fotopoulou et al., 2008). Also supported by anatomical correlation studies of areas involved in preparation and planning of movements (Berti et al., 2005) discussed in the next section.

Especially areas of monitoring discrepancies of action and intention are affected (Fotopoulou et al., 2010). Bisiach et al. (1990) suggest AHP stems from a specific lesion within the systems responsible for monitoring and processing sensory information. The monitoring network, a top-down controlled mechanism, interacts with sensory-driven neurological processes to relay information from sensory transducers to an individual (Bisiach et al., 1990). Unawareness levels are predicted not by damage to a specific domain but by the extent of damage to the monitoring module (Bisiach et al., 1990; Berti et al., 1996), thus providing an explanation for

modality specificity in AHP. According to this model, confabulation and denial may arise from lesions in specific monitoring systems of sensory-driven representations, hindering sensory information processing. If damage extends to the monitoring system itself, denial or the creation of false information may emerge as coping mechanisms or due to impaired information processing.

As demonstrated, many theories focus on damage to the comparator system, resulting in the failure to align actual motor movements with accurate motor feedback (Fotopoulou et al., 2008; Jenkinson et al., 2009; Saj et al., 2014). The inability to detect discrepancies between actual and predicted motor performance can explain deficits in motor monitoring, but it does not fully account for the multifactorial manifestation of AHP (Marcel et al., 2004). Other factors, such as implicit awareness (Cocchini et al., 2010; Moro et al., 2011), belief updating theories, and the inability to receive feedback about their hemiplegia (Bisiach et al., 1986), also contribute to the complexity of the condition.

The failure of the feedback loop to accurately adjust and update the individual's belief seems to contribute to the pathology of AHP (Vocat et al., 2013). Experimental studies by Vocat et al. (2013) demonstrate that patients with AHP are more likely to be unable to update their beliefs and modify their responses even when confronted with new information incongruent with their present assumption. Vocat et al. (2023) suggested an inability to adjust beliefs. An impairment in updating beliefs and specific self-related beliefs likely constitutes the central mechanism underlying anosognosia for motor deficits (Davies et al., 2005; Fotopoulou, 2014; Vuilleumier, 2004). As well, delusional explicit aspects of AHP, could be explained by an inability to attribute motor errors and other failures to oneself, leading to difficulty

in updating one's self-representation accordingly (Fotopoulou et al., 2010; Marcel et al., 2004; Ramachandran, 1995; Vuilleumier, 2004).

Belief updating has been suggested also by Vuilleumier's (2004) ‘ABC’ (Appreciation-Belief-Check) model. Due to the absence of error signals and subsequent lack of checking processes, individuals with anosognosia may exhibit an unusual level of confidence in their beliefs. This may result in a failure to incorporate new information when faced with inconsistencies. Consequently, anosognosic individuals might find it challenging to revise their existing beliefs, such as stating that their left arm is fully functional. This difficulty arises from the absence of supportive evidence at both the sensory-motor and affective-motivational levels.

Vocat et al. (2013) hypothesise that AHP is better understood as a breakdown in the ability to recognize specific motor impairments within the context of monitoring one's beliefs. The disrupted experience of primary deficits as motor or sensory weakness in AHP may also be influenced by other deficits, such as attentional deficits, spatial neglect, impaired motor intention, and amnesia, among others (Davies et al., 2005; Venneri & Shanks, 2004; Vuilleumier, 2004). These factors may manifest in varying combinations among different patients (Vuilleumier & Vocat, 2010). The diverse interplay of these factors could contribute to differences in the specificity and selectivity of AHP across individuals. In favour of impairment of belief updating is also the rehabilitation progress in many, but not all patients. Progressive recovery can be related to reception of more information and feedback of their current state, and increase awareness with time and fading anosognosia (Vocat et al., 2013). This is also seen in research exposing patients daily to their deficit to increase their awareness and enhance rehabilitation (Fotopoulou et al., 2009; Ownsworth et al., 2006).

Motivational levels and theories also played an important role since the discovery of AHP. Especially denial behaviours have been further attempted to be explained by neuro-motivational factors (Feinberg et al., 1994; Fotopoulou, 2010; Frith et al., 2000; Marcel et al., 2004; Ramachandran, 1995; Solms, 1995; Turnbull et al., 2005; Vuilleumier, 2004). Emotional factors may influence AHP and explain some of its denial and delusional elements from a motivational perspective (Marcel et al., 2004; Vuilleumier, 2004; Turnbull, 2005). It was suggested that AHP may result from abnormal affective regulation (Nadrone et al., 2008; Turnbull et al., 2005). The primary purpose of denial is to evade emotional distress. Therefore, the inhibition or avoidance of motor deficits could be linked to perceived threat. Also, transient episodes of awareness often evoke feelings of sadness (Kaplan-Solms & Solms, 2000; Turnbull et al., 2002, 2005). From this perspective, a disrupted right hemisphere emotion-regulation system (Kaplan-Solms & Solms, 2000; Turnbull et al., 2002, 2005) may contribute to the inability of individuals with anosognosia to tolerate strongly aversive ideas. Implicit awareness of deficits, dynamic fluctuations in awareness over time, selectively admitting certain deficits while denying others despite evidence, and manipulations of first versus third person perspectives speak in favour of a psychological defence mechanism (Ramachandran & Blakeslee, 1998; Turnbull et al., 2014). Emotional changes and emotional psychotherapeutic intervention accompany fluctuations of awareness, as demonstrated experimentally (Besharati et al., 2014; Kaplan-Solms & Solms, 2000; Ramachandran & Blakeslee, 1998). For instance, experimental manipulations of perspective-taking, in which taking a third person perspective of one's disability can lead to awareness improvements and an increase in depressive emotions (Fotopoulou et al., 2009; Marcel et al., 2004). In the experimental study of Besharati et al. (2014),

self-referential emotion induced by social feedback showed that negative emotion induction resulted in the improvement of motor awareness in patients with AHP, while positive emotions did not. Implicit awareness is thought to occur because denial as a defensive mechanism can only exclude information from explicit awareness (Mograbi & Morris, 2013). This knowledge might persist outside of conscious awareness. Research indicates that consistent avoidance of a particular concept can result in the suppression of memory for that concept, a phenomenon commonly known as repression (Anderson & Green, 2001). In line with the hypothesis of repression in patients, Ramachandran & Blakeslee (1998) informed a patient with AHP that an injection administered would have a side effect of temporary paralysis of her already paralyzed arm. Surprisingly, after the injection, the patient acknowledged her arm's paralysis. The lack of movement was perceived as a temporary side effect, minimising its emotional impact on the patient. This shift in perception seemingly enabled the patient to tolerate awareness of her paralysis. Due to these experiments, Ramachandran suggests that the failure of anosognosic patients to update their intentions in response to actual events may stem from repression. This extends beyond previous sensorimotor models by incorporating cognitive and motivational domains. Studies such as Nardone et al. (2008) have found evidence showing slower reaction times for anosognosic patients when presented with motor-related words compared to non-anosognosic individuals. This aligns with findings that individuals employing repressive coping mechanisms also exhibit slowed responses to threatening stimuli (Calvo & Eysenck, 2000), and high anxiety coupled with repressive coping can lead to various attentional changes. The defence hypothesis posits that patients with anosognosia for hemiplegia repress

aversive neurological deficits, denying their existence in declarative memory despite implicit registration.

Besharati et al. (2014) suggest that unawareness in AHP cannot be adequately explained by either purely motivational or neurocognitive accounts. The motivational theory has not been fully supported by empirical evidence (Besharati et al., 2014). There is still an ongoing debate regarding whether depressive symptoms are elevated or similar to control groups in AHP (Fotopoulou et al., 2010; Kaplan-Solms & Solms, 2000; Nardone et al., 2008; Orfei et al., 2018; Turnbull et al., 2002). However, the theory's expectation of modality specificity is not met, as self-defence mechanisms typically deny hemiplegia in both limbs, unlike some AHP patients who acknowledge hemiplegia in their upper but not lower limb. Furthermore, experimental studies found that the caloric and vestibular reflex can transiently improve AHP when cold water is introduced into the patient’s left ear (Cappa et al., 1987; Ramachandran, 1995). This contradicts the theory, as psychodynamic reactions are not typically influenced by physiological manipulation (Bisiach & Berti, 1995). Specifically, the psychodynamic account of AHP fails to explain the relative neuroanatomical and behavioural specificity of anosognosic behaviours (Bisiach & Geminiani, 1991; Heilman et al., 2011). Thus, it appears that the relationship between AHP and emotion is more complex than suggested by either the psychodynamic or motivational hypothesis.

Next to motivational theories, cognitive theories offer another view on AHP. The Cognitive Awareness Model, outlined by Agnew and Morris (1998) and further discussed by Mograbi and Morris (2013), offers a comprehensive understanding of the multifaceted nature of motor awareness. This model emphasises the role of memory within the comparator system. According to the model, incoming information

regarding self-performance in tasks is monitored by comparator mechanisms. These mechanisms compare this data with long-term memory representations stored in a personal database specifically dedicated to self-related information. When there's a discrepancy between the current experience and the stored self-knowledge, this comparison triggers an update of the personal database. The updated information is then relayed through the metacognitive awareness system, enabling consciousness of decision-making regarding one's abilities. All these processes involved in updating the personal database rely on multiple preserved cognitive functions, particularly memory. Similar with the focus of updating memory and information about oneself Marcel et al. (2004) theorised that there is a failure to integrate awareness of episodic instances of the deficit in the long-term bodily representation. Attentional dismissal leads to the experience of detachment and unconcern about parts of one's body not being perceived as belonging to oneself. The initial denial of impairment may depend on the semantic knowledge that the body segments can move, and the memory prior to the occurrence of the brain lesion (Marcel et al., 2004). In line with this hypothesis, Ramachandran (1995) conducted experiments with AHP patients. Caloric stimulation temporarily reversed anosognosia. The patient acknowledged her paralysis and even admitted to its duration over several days (Ramachandran & Blakeslee, 1998; Turnbull, 1999). Interestingly, the patient retained detailed memory of the caloric episode but lacked declarative memory of admitting to hemiplegia. This selective memory deficit, akin to anosognosia itself, suggests functional mechanisms at play in episodic recall.

Ramachandran (1995) identified the right hemisphere as an 'anomalies detector' in self-perception, responsible for setting up new schemata for new data contrasting with old self-knowledge. Hence, damage to it leads to disturbances in

self-perception and belief updating. This example also illustrates that despite anosognosia, perceptual facts had been encoded in long-term memory but overridden at a higher cognitive level. More recently, Ramachandran (1996) suggested that in normal conditions, the left hemisphere is concerned with managing small discrepancies in perception and thought, in order to make daily life consistent and predictable. When the discrepancies are so prominent that they cannot be ignored or adjusted, the right hemisphere creates new mental schemata or modifies the existing ones. In this view, anosognosia for motor impairment after brain injury would be a failure in this functional balance between the two hemispheres.

Similar, neuropathological theories in the past and present take disconnection syndromes (Geschwind, 1965; Schacter, 1990) and diaschisis between hemispheres into account. Disconnection syndromes (Geschwind, 1965) suggest that damage to a specific brain region can negatively affect the functioning of connected regions by limiting the flow of information between them. A related concept is diaschisis (Monakow, 1914), which implies that uninjured regions of the brain connecting with damaged areas may undergo reduced neural activity, particularly in the early stages following a sudden onset injury. The specific areas affected are not universally agreed upon. Geschwind (1965) suggested a dissociation from somatic function, referring to the impact on interest, attention, memory, and cognitive understanding related to a specific defect, from the right association cortex to the language zones of the left hemisphere. Consequently, the false belief in retained motor ability is reinforced by a left hemisphere "narrator" recounting knowledge about bodily functions (Geschwind, 1965).

Present studies, such as the probabilistic predictive coding theory of AHP (Fotopoulou, 2012; 2014), suggests also that lesions of AHP could disrupt neuromodulatory circuits or suggest weakened connection of functions (Preston & Newport, 2014). The Disconnection hypothesis integrates several aspects of the historical attempt to identify the cause of AHP. Kirsch et al. (2021) suggest pathology of AHP arises due to disconnection of sensorimotor, metacognitive and mentalisation functions related to self-awareness. In this framework, the brain leverages prior learning to create generative models concerning the embodied self. These models encode predictions not only about the hidden causes of current, noisy sensory inputs, but also about the expected sensory experiences related to the body and its interactions with the environment and prior beliefs.

Modern theories such as one by Pacella and Moro (2022) propose a comprehensive multidimensional model of awareness by integrating three key components derived from previous literature and theories: motor intention, motor monitoring, and error recognition, all contributing to one's awareness of their own motor abilities. They emphasise neuropathological theories by stating the role of white matter disconnection is contributing to symptoms of AHP, suggesting that disconnection of distant areas resulting from damage may account for various manifestations observed in AHP. The components of motor intention and monitoring encompass various stages of action, including intention, aim, and planning, as well as the sense of agency, monitoring, control of execution, and error recognition. These components are further linked to anatomical correlational studies in patients with AHP, detailed in subsequent chapters. Their model underscores that motor awareness extends beyond mere sensorimotor processing and monitoring

mechanisms, or vice versa. Instead, it necessitates the integration of both bottom-up and top-down processes involved in maintaining and updating beliefs about oneself.

Recent theories have trended towards suggesting multiple causative factors (Davies et al., 2005; Pacella & Moro, 2022; Vuilleumier, 2004; Vocat et al., 2013), involving different combinations of two or more deficits. AHP presents clinically in various forms in patients, indicating its complex and heterogeneous nature, where each aspect may require an individualised explanation (Marcel et al., 2004; Vuilleumier, 2004). The pathology of AHP might arise from components of the underlying theories proposed so far, matching the diverse observed expressions of this phenomenon. However, not all aspects are fully understood. While none of these factors such as cognitive, intention, motivational, monitoring, prediction, belief-updating appears sufficient to explain the multifaceted nature of AHP alone, the theoretical approach may underlie a heterogeneous nature (Cocchini et al., 2010; Fotopoulou, 2012; Moro et al., 2011; Vocat et al., 2010; Vuilleumier, 2004), including a potential combination of factors (Feinberg et al., 2000; Fotopoulou et al., 2010; Solms, 1995; Turnbull et al., 2005; Vuilleumier, 2004). However, the precise combinations of deficits capable of causing the syndrome and the dynamic relation between the various critical factors remain unknown (Besharati et al., 2015). Anatomical correlational studies may offer additional evidence supporting the relationship between motor awareness and the pathology of AHP and its underlying theories.

4. Anatomy of AHP

AHP is believed to be related to changes in brain function and structure associated with the integration of emotional responses, perception, the processing of

emotional and sensory information, and self-awareness (Gainotti, 2018). Examining the effects of brain damage of AHP is crucial for understanding the neurobiological mechanisms that underlie motor awareness processes.

Previous anatomical correlational studies have frequently identified lesions associated with AHP in the frontal, parietal lobes, and temporal cortical structures (Antoniello & Gottesman, 2020; Berti et al., 2005; Gainotti, 2018; Pia et al., 2004; Pacella et al., 2019), specifically in areas such as the anterior cingulate gyrus, basal ganglia, insula, lateral prefrontal cortex (LPFC), limbic system, premotor cortex (PFC), temporoparietal junction (TPJ), and the ventral attentional system (VAN) (Antoniello & Gottesman, 2020; Berti et al., 2005; Bisiach et al., 1986; Gainotti, 2018; Karnath, 2005; Pia et al., 2004; Pacella et al., 2019; Vocat et al., 2010).

Lesion mapping studies have retraced the medical history of patients with AHP following a stroke and identified the infarct of the middle cerebral artery (MCA) and its surrounding areas as the most frequently observed cause (Baier et al., 2014; Besharati et al., 2014; Fotopoulou et al., 2010). Furthermore, AHP is correlated to lesion size (Pedersen et al., 1996). AHP is observed to have a higher prevalence when lesions in stroke patients tend to have a mean diameter of 5 cm or more (Hartman-Maeir et al., 2003). The anatomical correlation of AHP can be tested in the acute phase of one to seven days post-injury, in the subacute phase eight to 29 days after a stroke, or chronic phase after one month, and is essential for understanding how the condition evolves over time, providing valuable insights into its temporal dynamics and progression. The mixed brain injury aetiology and the time elapsed from the acute event make it more challenging to establish a precise localization of AHP and generalise the measurements (Orfei et al., 2009). Consequently, the

investigation of the anatomical correlation of AHP over the last century has provided inconsistent results, with differing lesions identified as possible causes for AHP. When considering the anatomy of AHP in groups rather than isolation, it becomes more challenging to narrow down a specific lesion prevalence for the expression of AHP pathology.

The following sub-chapters will analyse the anatomical correlational findings of neuroimaging studies, neurophysiological studies, voxel-based lesion-symptom mapping (VLSM) studies to further attempt to explain and identify the neural bases of AHP.

4.1 Neuroimaging studies

Pia et al.'s (2004) meta-analysis compromised neuroimaging studies from 1938 to 2001, establishing an association between the occurrence of AHP and unilateral right-sided or bilateral lesions in various cortical and subcortical brain areas. AHP can arise from single cortical structure lesions in frontal, parietal, temporal cortical, or subcortical areas with distinct etiologies. Notably, fronto-parietal lesions without subcortical involvement appear more prevalent in the cause of AHP than temporal lobe lesions. In cases of involvement of more than one lobe, fronto-parietal structure lesions with or without further extensions were associated with the highest occurrence (Pia et al., 2004). Intriguingly, those meta-analyses highlighted instances where pure anosognosia occurred without parietal lobe damage or spatial neglect, while six patients diagnosed with AHP and neglect exhibited no frontal lobe damage but only parietal lobe damage. Hypotheses propose that in such cases, parietal and frontal damage may induce diaschisis functionally linked to frontal or parietal lobe areas (Pia et al., 2004), yet these cases

highlight the challenge of generalising one specific lesion location for all AHP patients.

The results of the meta-analysis of Pia et al. (2004) entail crucial implications for the explanation of the pathology of AHP. Several studies on humans and primates have provided evidence supporting the involvement of the parietal region in movement planning, sensorimotor integration, and spatial cognition (Halligan et al., 2003). The engagement of the parietal lobe in anosognosia may be attributed to an intrinsic spatial limitation affecting the function of the motor monitoring system (Orfei et al., 2009). From this perspective, anosognosia may be caused by damage to the fronto-parietal circuit dedicated to spatial and motor representation. Within this neural network, the parietal component plays a crucial role in computing spatial information necessary for the execution of motor actions in space. The frontal lobe is commonly associated with self-awareness and metacognitive functions, particularly self-reflection (Orfei et al., 2007). Given that anosognosia is perceived as a disorder of motor awareness (Frith et al., 2000), frontal involvement may be correlated with damage to a motor monitoring system. Damage to the frontal system is often linked to impairments in the monitoring system responsible for action planning and execution. The proposed locations for actual, intended, and predicted movement states, as well as motor awareness and control, are primarily within the PFC, premotor cortex (PMC), and supplementary motor area (SMA) within a frontal-parietal circuit (Desmurget and Sirigu, 2009; Frith et al., 2000; Haggard, 2005). Therefore, the combination of frontal involvement and simultaneous parietal damage might constitute a central factor in a deficit within a cortical circuit associated with space and motor representation (Berti et al., 2005; Rizzolatti et al., 1998). Consequently, neglect and anosognosia could result from impairments to this

shared circuit responsible for spatial-for-action representations, as suggested by studies like Rizzolatti et al. (1998). Lesions affecting specific components of this circuit may give rise to selective and spatially confined disorders of awareness as AHP.

4.2 Neurophysiological studies

Awareness of motor deficits is often associated with a performance-monitoring network. A systematic review conducted by Pyasik et al. (2022) summarised eighteen studies, specifically focusing on patients with lesions in the performance-monitoring network and comparing them to healthy controls. The key components identified in this network included the basal ganglia, dorsolateral PFC, insula, and thalamus, all of which also play a significant role in AHP lesions, as following studies will demonstrate. The anterior frontal network was identified as a crucial risk factor for disorders related to the awareness of motor deficits.

4.3 VLSM studies

Early VLSM studies identified Brodmann areas (BAs) to further narrow down lesions contributing to the pathology of AHP. Berti et al. (2005) conducted an analysis of patients with AHP lesions in the right hemisphere, who explicitly denied their motor impairment and compared them with patients with hemiplegia (HP) without anosognosia. Denial was primarily linked to the frontal cortex, specifically involving the PMC (BA 6), Broca's area (BA 44), the primary motor cortex (BA 4), and the somatosensory cortex (BA 3). These areas play crucial roles in the programming of motor acts and are related with monitoring systems (Rizzolati et al., 1998). Additional areas associated with lesions in AHP included the frontal gyrus (BA 46), and the insula. In contrast Fotopoulou et al. (2010) found no significant

difference in the prevalence of lesions in the PMC (BA 6) when comparing patients with AHP and HP.

In further research by Karnath et al. (2005), the posterior insula was identified as a key differentiator in anosognosic patients, potentially contributing to a deficit in integrating stimuli associated with self-awareness and body perception. The insular cortex is hypothesised to play a crucial role in monitoring internal bodily functions (Karnath et al., 2005; Gomez-Andres et al., 2022). Consequently, a disconnection of fronto-insular tracts would explain the diffusing awareness of motor perceptions in patients with AHP (Monai et al., 2020). Notably, AHP does not solely result from damage to the insula (Karnath et al., 2005). Similar to Berti et al. (2005), Brodmann's areas 44 and 45 were observed to be involved, specifically, the inferior frontal gyrus (IFG) and pars orbitalis (BA 47). Furthermore, lesions in the basal ganglia, parietal and temporal cortical areas, and white matter were observed. The basal ganglia, a component of the subcortical structures within the frontal network (Decety, 1996), could potentially contribute to impaired motor monitoring systems responsible for both action execution and ideation (Pia et al., 2004).

To further explore and gain deeper comprehension into not just the spatial distribution of lesions associated with AHP, but also to uncover distinctions across various phases of AHP, Moro et al. (2016) performed a VLSM analysis to explore lesional differences between acute and chronic AHP. In the acute stage lesions involved the basal ganglia, caudate, insula, putamen, internal and external capsule and the inferior occipito-frontal fasciculus. Chronic AHP was associated with damage to the fronto-temporal cortex, specifically, gyri temporales transversi, temporal superior cortex, the thalamus, and rolandic operculum (ventral premotor cortex). Additionally, alongside the white matter pathways, lesions of the anterior segment of

fasciculus arcuate, cortico-spinal tract, and corpus callosum were observed (Moro et al., 2016). Lesions that overlapped in acute and chronic AHP cases affected the insula, external and internal capsule, superior corona radiata and white matter lesions related to the superior longitudinal fasciculus (SLF). Furthermore, Moro et al. (2016) identified lesions in the anterior temporal superior gyrus in AHP patients as compared to HP controls. The anterior superior temporal gyrus has been previously associated with deficits in mentalization and perspective-taking in AHP (Besharati et al., 2016), potentially contributing to the explanation of why patients cannot update their anosognosic beliefs based on third-person feedback (Fotopoulou, 2015; Moro et al., 2011).

Three networks were especially noticeable in acute AHP, namely the ventral PMC, the insula, and the superior temporal gyri (STG). This aligns with further research that reported similar results for the insular cortex and lateral PMC (Berti et al., 2005; Fotopoulou et al., 2010; Monai et al., 2020; Pacella et al., 2019). Significantly, in addition to the observed basal ganglia and insula damage in acute AHP, there was extended damage involving the fronto-temporal cortex and long white matter pathways. This extended damage appeared to be predictive of symptom persistence beyond 40 days (Moro et al., 2016). The identification of anatomical areas sheds light on the association of greater cortical damage being likely the cause for chronic AHP (Moro et al., 2016). In agreement with previous lesion studies on Disownership Syndrome (DSO) by Gandola et al. (2011), Invernizzi et al. (2013), and Romano et al. (2014), Moro et al. (2016) propose that subcortical grey areas and related white matter tracts might be essential for the emergence of rudimentary feelings of limb ownership. These sensations are then presumably re-represented in higher cortical areas to integrate them with various aspects of

self-awareness, encompassing self-other distinction, spatial and temporal self-awareness, as well as the sense of action awareness and agency (Blanke, 2012; Tsakiris et al., 2010). Additionally, the subcortical involvement appears to be predominantly associated with concomitant disturbances in body ownership.

Like Moro et al. 's (2016), recent VLSM research is taking further changes of white matter connectivity in lesions of patients with AHP into account (Besharati et al., 2022; Monai et al., 2020; Pacella et al., 2019). These findings reveal several damages in long-range white matter pathways within the association cortex. The currently largest investigated sample with 174 AHP patients by Pacella et al. (2019), explored the neural systems contributing to AHP and proposed a tripartite disconnection syndrome of white matter pathways involving the premotor loop, limbic system, and VAN. The results emphasise the joint contribution of these three systems to motor awareness. Consistent with recent analyses (Berti et al., 2005; Moro et al., 2016), AHP is not solely explained by direct grey matter lesions but involves disruptions in white matter connections within these networks as well.

The study of Pacella et al. (2019) highlights the role of white matter disconnection in the limbic system via the cingulum and the VAN and SLF III connections. More specifically, disruptions are observed in the posterior segments of the limbic network, the cingulum connections between the amygdala, the cingulate gyrus, and the hippocampus, the SLF III connections in the right hemisphere between TPJ and ventral frontal cortex of the VAN, and the frontal aslant and fronto-striatal connections between the striatum, the preSMA, and the IFG of the premotor loop (Pacella et al., 2019). The limbic system is commonly linked to emotional and memory processing and operates as an integral component of the default mode network (DMN) (Greicius et al., 2009). This system is associated with a

distinctive pattern of intrinsic connectivity during introspective states and self-referential processes, including autobiographical retrieval, future imagining, and mentalization (Pacella et al., 2019). This association may provide an explanation for deficits in general awareness, anticipatory awareness, and mentalization observed in AHP patients. Building upon previous findings on emotions and arousal in AHP (Besharati et al., 2014; D'Imperio et al., 2017), the disconnection of the VAN might be preventing the recognition of stimuli related to one's own paralysis. The insula, a critical component in AHP, plays a crucial role in updating self-referred beliefs and contributes to both the limbic system and the VAN. It accomplishes this by integrating external sensory information with internal emotional and bodily state signals (Craig, 2009).

To further investigate damage to white matter pathways in AHP, Monai et al. (2020) decided to compare lesions between patients with AHP and HP. In line with previous research, in both groups, lesions in the frontal, parietal, and temporal lobes, along with the insular cortex were observed. Subcortical regions in the basal ganglia and thalamus were also affected, with the brainstem being impacted only in the HP group.

The disconnection of dorsal white matter pathways descending from the motor, premotor, and parietal cortex to the internal capsule and cerebral peduncle was similar in 75% of patients in both the AHP and HP groups (Monai et al., 2020). Additional involvement of basal ganglia-cortical tracts, frontal and temporal white matter tracts, and thalamo-cortical tracts was identified. In the HP group, central damage was observed in the basal ganglia. For AHP, the damage was more diffuse, with a higher prevalence of lesions in frontal and parietal white matter. Overall, no significant difference in the severity of damage between patient groups was found,

but there was a notable difference in terms of structural connectivity. While similar patterns of disconnectivity of descending motor pathways were found between AHP and HP patients, AHP patients exhibited significantly increased and more widespread white matter pathway disconnection in various regions, including the right insula, fronto-insular tract, right TPJ, right lateral and medial prefrontal cortex (mPFC), specifically the inferior and superior parietal lobule (IPL, SPL), STG, middle temporal gyrus (MTG), PMC, anteriorly in the IFG, SLF, and subcortically in the putamen. These disconnections within the neurocognitive hierarchy can manifest at various levels. For instance, observed damage to the PMC and the ventral part of the SLF may lead to a disconnection between somatosensory areas in the parietal cortex and ventral premotor and prefrontal regions. This disconnection could result in an impaired ability to detect and monitor incongruent sensorimotor feedback (Enriquez-Geppert et al., 2014), aligning with findings from previous studies (Berti et al., 2005; Kortte et al., 2015).

The IFG entails different nodes responsible for various networks, such as the CON, a network belonging to the insula and cingulate cortex, related to task maintenance and shifting (Sestieri et al., 2014). This network could possibly explain the inability to shift to the correction of diffused body schema or motor planning observed in AHP (Monai et al., 2020). The IFG is also associated with the TPJ, forming part of the VAN, via the SLF II, one of the most damaged pathways in AHP (Monai et al., 2020). These areas play a crucial role in the integration of multi-modal body and visuospatial signals and in switching between perspectives of the body, environment, external and internal stimuli, and self or others (Corbetta et al., 2008). The superior parietal and dorsal frontal cortex contribute to top-down control for spatial attention, possibly linked to the spatial neglect observed in AHP (Corbetta et

al., 2008). The cingulate cortex is involved in the evaluation of emotional stimuli, memory, perception, and mediation of memory retrieval, as well as emotion. These pathways connect regions to various cognitive networks, including the cingulo-opercular, default, and VAN (Monai et al., 2020). Similar to the previously presented study by Pacella et al. (2019) and Kirsch et al. (2021) significant correlation between damage and disconnection of white matter pathways of the VAN, linking the insula, TPJ, and ventral PFC with belief updating in AHP is observed. Importantly, no isolated pattern of lesions or disconnections to any single system can fully explain AHP. The study proposes that deficits in motor monitoring, combined with salience and belief updating deficits, contribute to the multifaceted nature of AHP.

To investigate the multifaceted nature of AHP further, Besharati et al. (2022) conducted a comprehensive analysis and comparison of deficits in updating beliefs from both egocentric first-person perspective (1PP) and third-person perspective (3PP). The study unveiled the neural components involved in these deficits, including limbic white matter connections, the IFG, middle frontal gyrus (MFG), supramarginal gyrus(SMG)/ TPJ, and STG, consistent with findings from prior research (Moro et al., 2016; D'Imperio et al., 2017). Specifically examining egocentric perspectives, impairments in 1PP were associated with lesions to the insula, IFG, pre- and postcentral gyri, and the SLF. Similarly, egocentric 3PP impairments involve these areas, with additional significance in the MFG, extending to the SMG/TPJ, STG, and subcortically to the pallidum. Moving to allocentric perspectives, mentalization deficits in allocentric 3PP were linked to the IFG, MFG, and SMG/TPJ, with white matter disconnection in posterior-temporal areas associated with a failure in counterfactual belief updating (Besharati et al., 2016, 2022; Kirsch et al., 2021).

Interestingly, AHP patients were more aware of their motor paralysis when asked from 3PP than from 1PP, in line with previous research (Marcel et al., 2004). These findings align with the theoretical framework by Samsonovich and Nadel (2005), proposing the existence of an 'egocentric map' in the prefrontal cortex and an 'allocentric map' in the hippocampus to attempt to understand the diverse belief updating deficits seen in AHP.

4.4 Section Summary

Previous perspectives on AHP concentrated on specific cortical lesions, particularly in the lateral premotor cortex and anterior insula, emphasising impairment on action and body monitoring. Specifically, difficulties in monitoring motor signals and learning from action failures are thought to be reflected in the premotor network. However, AHP is not solely linked to a deficit in sensorimotor monitoring (Berti et al., 2005) or multisensory body representation (Karnath et al., 2005). Recent studies (Monai et al., 2020; Moro et al., 2016; Pacella et al., 2019) suggest that AHP is a more complex syndrome, extending beyond motor monitoring functions and involving disconnectivity in several cortical and subcortical pathways. These findings support contemporary multifactorial theories of AHP, proposing that the syndrome arises from a combination of diverse and heterogeneous disturbances (Marcel et al., 2004; Monai et al., 2020; Morris & Mograbi, 2013; Pacella et al., 2019) in various networks, including cognitive, motor, premotor, and spatial computation, and representation.

Therefore, AHP is not confined to a specific functional region or system; instead, it encompasses widespread areas, particularly the fronto-temporo-parietal regions and subcortical white matter. The neural basis of AHP suggests that damage and disconnection in complex cortico-subcortical circuits, within and beyond the

motor system, underlie awareness of motor control, contributing to anosognosic pathology. This involves multiple networks associated with motor and body monitoring, attention, belief updating, and self-referential processes. The diverse findings could suggest that various manifestations and expressions of AHP correspond to distinct anatomical pathologies as well as differing underlying theories.

5. Lateralization of Hemispheres in AHP

The most frequent lesions following stroke or TBI that are associated with AHP are typically localised in the right hemisphere (Azouvi & Peskine, 2013; Appelros et al., 2002; Baier & Karnath, 2005; Coslett, 2005; Pedersen et al., 1996; Starkstein et al., 1992; Turnbull et al., 2005; Vallar et al., 2003). Anosognosic patients with lesions in the left hemisphere represent only a minority of cases (Azouvi & Peskine, 2011; Appelros et al., 2002; Coccini et al., 2012; Coslett, 2005; Pedersen et al., 1996; Stone et al., 1993). For instance, Stone et al. (1993) conducted a study involving 171 patients and found an incidence rate of anosognosia of 28% following right hemisphere stroke compared to 5% following left hemisphere stroke. Similar findings were reported by Pedersen et al. (1996) where the incidence of anosognosia after right and left hemisphere stroke was 36% and 9% respectively. In a systematic review by Jehkonen et al. (2006) covering the period from 1995 to 2005, the occurrence of anosognosia after right hemisphere damage ranged from 11% to 60%, while it ranged from 6% to 24% for left hemisphere damage.

Recent research proves that left hemisphere lesion-associated AHP might be more frequent than expected (Cocchini et al., 2022; Hartman-Maeir et al., 2003). AHP in left-brain-damaged (LBD) patients may have been underestimated due to methodological issues favouring right-brain-damaged (RBD) patients (Cocchini et al.,

2009) throughout history. More recent evidence from Cutting (1978) and Cocchini et al. (2009) further confirmed that the occurrence of AHP might have been underestimated in patients with left brain damage due to methodological limitations. This could stem from differing concepts of AHP, leading to investigations of potentially distinct underlying mechanisms (Jenkinson et al., 2011; Morin, 2017) and, consequently, different results. Additionally, aphasia is a frequent outcome of left hemisphere lesions and stands as the most prevalent neuropsychological effect of stroke, affecting approximately one-third of all stroke patients in the acute phase (Sinanović, 2010). Left-sided infarcts often impact the language centre, complicating the detection of AHP and potentially leading to a selection bias in assessments. Up to 40% of individuals with left-sided infarcts may experience AHP, which could be masked by aphasia. For example, when applying the VATAm, Della Sala et al. (2009) identified that 40% of anosognosic patients had damage to the left hemisphere. Nathanson et al. (1952) and Gross and Kaltenback (1955) were in favour of the hypothesis that aphasia might obscure certain cases of AHP. They discovered that within unselected cohorts of patients with right-sided hemiplegia, the assessment of AHP was hindered by the concurrent presence of global aphasia.

Outcomes from investigations using the intracarotid sodium amobarbital procedure (Wada Test) are inconclusive regarding the relationship between left hemisphere damage and awareness in AHP. When barbiturates are injected into either carotid artery, selectively anaesthetizing one hemisphere and causing weakness on the contralateral body side, a higher frequency of unawareness is evident when the barbiturate is injected into the right hemisphere (Bisiach & Geminiani, 1991; Gilmore et al., 1992; Adair et al., 1997; Lu et al., 2001; Pia et al., 2004). Left hemiplegia occurred in 66% (Dywan et al., 1995) to 100% (Gilmore et al.,

1992) of cases, while hemiplegia for the right side ranged from 0% to 86% (Carpenter et al., 1995; Durkin et al., 1994; Dywan et al., 1995; Gilmore et al., 1992; Kaplan et al., 1993; Lu et al., 1997), and the difference between the two hemispheric conditions ranged from 0% (Dywan et al., 1995) to 100% (Gilmore et al., 1992; Cocchini & Della Sala, 2010). These results were not influenced by findings of neglect, as 26% of LBD patients and 52% of RBD patients displayed similar deficits (Cocchini et al., 2018). For example, Azouvi and Peskine (2011) questioned subjects both during and 3 minutes after barbiturate injection and found that 30 out of 31 (97%) denied left hemiparesis after right carotid injection, while 15 out of 31 (48%) denied right hemiparesis after left carotid injection. These outcomes suggest that AHP may be more frequently associated with anaesthesia or lesions of the right hemisphere. However, the results also indicate that AHP occurring after left hemisphere anaesthesia or lesion cannot be fully excluded as a cause of unawareness of motor impairment, indicating that the left hemisphere might play a different role than the right hemisphere in terms of awareness.

Fowler et al. (2018) investigated the difference in expression of AHP in LBD and RBD stroke patients. LBD patients were more likely to underestimate their motor ability, while RBD patients predominantly seemed to overestimate their motor ability (Fowler et al, 2018; Marcel et al., 2004). Both cases imply that patients have a disturbed view of their post-stroke motor ability, regardless of which lesion side is damaged. Therefore, the expression of symptoms between the lesion sides might differ. When comparing both groups, RBD patients presented more spatial attention problems, while memory scores were somewhat similar for both groups.

The previously mentioned Geschwind's disconnection hypothesis (1965) postulates an interhemispheric disconnection. LBD patients can be of high

importance due to neuroimaging studies identifying bilateral networks and diaschisis as possible inclusion criteria for both hemispheres (Coccioni et al., 2022). Especially the sense of agency is supposed to be integrated into a bilateral network (Seghezzi et al., 2019; Di Plinio et al., 2020; Zapparoli et al., 2020). Recent neuroimaging studies revealed that the left hemisphere can also play a role in the pre-supplementary motor area, intentional binding of a planned action, and integrating agency and body-ownership (Seghezzi et al., 2019; Zapparoli et al., 2020). Therefore, it is hypothesised that the right hemisphere is not exclusively involved and responsible for awareness and monitoring (Coccioni, 2022), and both hemispheres may cover different cognitive aspects. Neuroimaging studies on AHP (Berti et al., 2005; Karnath et al., 2005; Baier & Karnath, 2008; Vocat et al., 2013) do not directly address the potential role of the left insula in body and emotional awareness (Craig, 2009). Nevertheless, it is noteworthy that some neuroimaging studies of motor and emotional awareness have found bilateral activation of the insular cortex (Farrer & Frith, 2002). Thus, it has to be explored if the specific roles of the right and left insular cortex in motor and emotional awareness are important in their combined functional role. Some researchers view AHP after brain injury as a failure of functional balance between the two hemispheres (Ramachandran, 1996). Similar to the findings of Cocchioni et al. (2022), found an impact on bilateral balance in the motor network for actions involving both limbs in AHP, whereby lesions not exclusively affect contralateral motor abilities but also the sense of agency on the ipsilateral side.

Jehkonen et al. (2006) observed that research on AHP with exclusively LBD patients did not exist until 2002. During the past decade, only 10% of the reviewed studies have assessed anosognosia in homogeneous groups of left hemisphere

patients. Further investigation is necessary to evaluate the role the left hemisphere plays in AHP to prevent possible under evaluation of these patients due to challenging assessment procedures and to further develop a comprehensive understanding of AHP. The aim is to investigate whether patients with LBD need to be more frequently assessed and studied, given contemporary research evidence suggesting that AHP is more prevalently, but not exclusively, caused by RBD. Additionally, the aim is to discover if patients with lesions to the left hemisphere are less frequently assessed or identified due to different symptomatology (Gainotti, 2018; Pia et al., 2004; Monai et al., 2020). Therefore, the present systematic review investigates further the relation of AHP and the left hemisphere. Like other aspects of hemispheric asymmetry (Gasquoine, 2016), the precise nature of this lateralized effect is little understood. Due to the inconclusive findings of LBD and awareness in AHP, the present systematic review focuses on the findings of research regarding this relationship to further comprehend the current findings and to attempt to close the gap, providing possible explanations for the discrepancies in findings. The identification of the relation between the left hemisphere and AHP is of high importance due to possible consequences for assessment methods and study designs necessary to focus and include more frequently patients with LBD.

6. Materials and Methods

6.1 Selection Criteria

A systematic review was conducted on research articles sourced from the electronic database PubMed, with the aim of analysing and summarising the findings of AHP in left hemiplegia spanning from 1954 to 2024. The research articles referenced in this report were restricted to English translations or originals. The selection criteria included adult patients experiencing anosognosia for hemiparesis

or hemiplegia with unilateral left hemispheric lesions, and without specific aetiology, who had been evaluated using any applicable measurement outlined in Table 1. The analysis encompassed the anatomy of lesions, assessment methods, the prevalence and underlying shared theories and findings regarding AHP in patients with LBD.

6.2 Selection of Articles

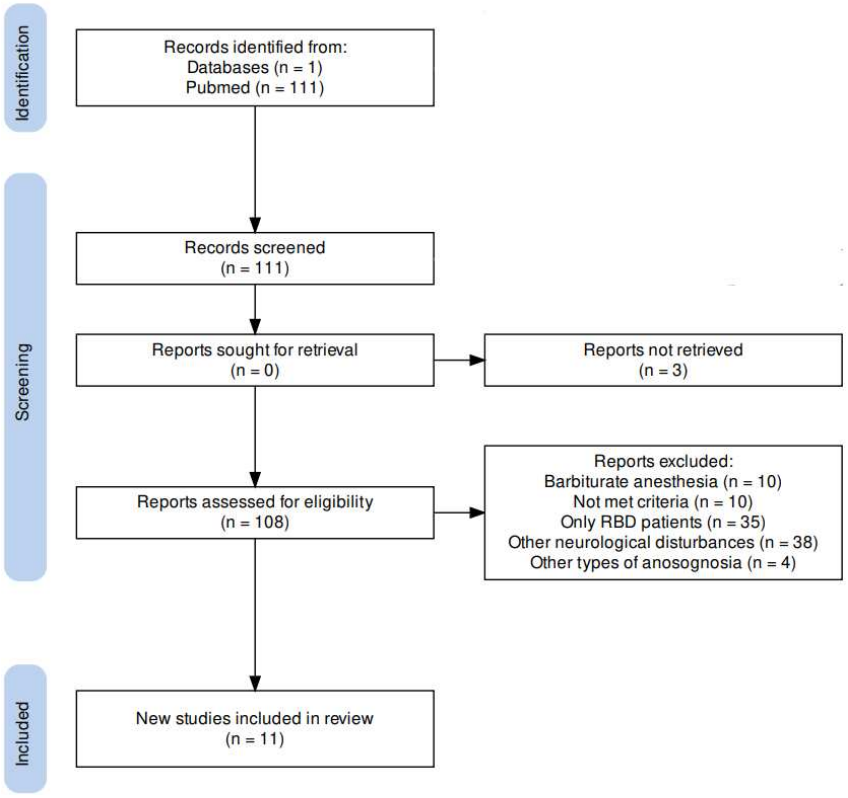
The search was conducted using the following string: "Anosognosia for hemiplegia" OR "AHP" OR "Anosognosia for motor impairment" AND "left hemisphere" OR "left brain damage" OR " left lesion" on April 11, 2024. The process of identification, screening, and inclusion is summarised in Table 2. A total of 111 articles were retrieved from PubMed. After a comprehensive screening of the abstracts 100 articles were excluded. Among these, 35 articles focusing on AHP in left hemiplegia or RBD patients were included, while four papers addressing different types of anosognosia, such as anosognosia for mirror writing, hemianopic anosognosia, anosognosia for PD, and anosognosia for optic ataxia were excluded. Additionally, 38 articles were excluded due to their focus on other neurological disturbances, such as agnosia, agraphia, aphasia, apraxia, gnosis, motor conversion disorder, neglect, object recognition impairment, optic ataxia, prosopagnosia, supernumerary motor phantom limb, somatoparaphrenia, and others. Six papers investigating AHP in non-paralyzed limbs or unrelated reviews not specifically addressing AHP in LBD patients were also excluded.

Furthermore, four studies involving LBD patients did not identify AHP in the sample, either due to the inability to assess all participating patients or the absence of AHP, and thus were not included in the analysis. Additionally, ten Wada test studies, which investigated AHP through barbiturate anaesthesia, were excluded because the patients did not exhibit AHP as a result of their brain lesion but rather

were induced to display AHP for the purpose of intracarotid sodium amytal testing (ISA) prior to epilepsy surgery. After excluding three case studies that met the criteria but were inaccessible and in a different language, eleven articles remained (as shown in Table 2). These eleven articles were thoroughly reviewed and their findings are comprehensively presented in Table 3 of the result section.

Table 2

Overview of Systematic Review Process



7. Results

Among the eleven articles selected, only seven studies focus specifically on AHP in LBD patients (Baier et al., 2014; Cocchini et al., 2009, 2018; Formica et al., 2022; Green & Hamilton, 1976; Hartman-Maeir et al., 2001; Ronchi et al., 2013). The

remaining articles explore various other neurological disturbances, such as neglect (Beis et al., 2004; Dronkers et al., 1989; Stone et al., 1993), and minor hemisphere syndrome (Cohen et al., 1991), wherein AHP co-occurs. Table 3 provides a detailed overview of the studies included. The studies consisted of five case studies and seven group studies (Table 3). Within the group studies, the prevalence of AHP in LBD patients ranged from 2% to 48%.

Table 3

Systematic Review of 11 Articles with the Focus on Extracting Information about AHP Limited to Patients with Unilateral Left Hemispheric Lesions.

Reference	Design	*N*	Patient Characteristic [a]	Aetiology	Time of Assessment (*SD*)	Assessment tools	Aphasia [b]
Green et al. (1976)	Experimental Study	25 (1)	N/A	Stroke	Onset and 9 days post stroke	Clinical Interview	N/A
Dronkers et al. (1989)	Case study	1 (1)	49-year-old woman, left- handed	Stroke	24 hours, 3, and 9 days post stroke	Clinical Interview	No
Cohen et al. (1991)	Case study	1 (1)	68-year-old woman, right-handed	Stroke	First 3 weeks post stroke	Clinical Interview	No
Stone et al. (1993)	Correlational Study	56 (3)	Age: 72.37 (*12.11*) Range 28-100 years	Stroke	2-3 days post-stroke	Cutting's Questionnaire (Cutting, 1978)	45 excluded
Hartman-Maeir et al. (2001)	Correlational Study	17 (4)	56-year-old men, right-handed 58-year-old men, right-handed 64-year-old men, right-handed 64-year-old woman, right-handed	Stroke	1 month post onset, 42 days (SD *13.55*), 1 year follow-up	Task choice method (Ramachandran, 1995), Awareness Interview (Anderson & Tranel, 2007), IADLs, ADLs	Included
Beis et al. (2004)	Correlational Study	78 (5)	46 men, 32 women, Age: 54.6 (15.7), 83.2% right-handed	Stroke	10.8 (*SD* 12.4) weeks post stroke	Bisiach scale (Bisiach et al., 1986)	11 excluded

Reference	Design	*N*	Patient Characteristic [a]	Aetiology	Time of Assessment (*SD*)	Assessment tools	Aphasia [b]
Cocchini et al. (2009)	Comparative Study	20 (2) 30 (12)	16 men, 17 women, Age: 69.7 (11.8) Range 42 to 87	Stroke	73.8 days (*SD* 46.0) Range: 7 to 210 post stroke	Berti et al. (1996) Scale VATAm, Caregiver	22 excluded 9 excluded
Ronchi et al. (2013)	Case study	1(1)	73 year-old man, left- handed	Stroke	1.5 months post stroke	Bisiach scale (Bisiach et al., 1986)	Mild
Baier et al. (2014)	Correlational study	44 (1)	62-year-old woman, right-handed	Stroke	Day 3, 4, and 9 post stroke	Bisiach et al. (1986), Baier and Karnath (2005) criteria	22 excluded No
Cocchini et al. (2018)	Comparative Study	29 (14)	14 men and 16 women Age: 64.8 (12.4) Range 40-85	Stroke	101.6 (SD 49.8) day post stroke, range 10–149	VATAm Errand Choice Test, (Cocchini et al., 2018), ADLs	No
Formica et al. (2022)	Case study	1 (1)	53-year-old woman, right-handed	Stroke	Repeated measures 3, 4, 9 days, and 3 months post stroke	VATAm, Caregiver	Mild

Note. *N* = 11. Number of patients actually assessed in the study with LBD (diagnosed with AHP).

[a] Gender, Mean of age (*SD*) and range, right-handed or left-handed.

[b] Patient additionally excluded due to speech and/or comprehension difficulties, not included in *N*.

7.1 Patient Criteria

The total number of patients with left hemispheric lesions in the eleven studies included in this review is 333, while the number of patients with those lesions identified with AHP was 44 (Table 3). Of those patients one study reports about three patients with AHP in LBD in the acute phase (Stone et al., 1993), six studies identified seven patients in the subacute phase, meaning AHP consisted longer than seven days (Baier et al., 2014; Cocchini et al., 2009; Cohen et al., 1991; Dronkers et al., 1989; Formica et al., 2022; Green & Hamilton, 1976), and five studies identified 34 patients with chronic AHP resulting from left hemispheric lesions (Beis et al., 2004; Cocchini et al., 2009, 2018; Hartman-Maeir et al., 2001; Ronchi et al., 2013). All patients listed in Table 3 had experienced unilateral damage to the left hemisphere. In this review, AHP was observed as a result of onset of a stroke. Most patient samples were recruited from hospital admissions and five from rehabilitation units (Beis et al., 2004; Cocchini et al., 2018; Formica et al., 2022; Hartman-Maeir et al., 2001; Ronchi et al., 2013). Displaying the proportion of men and women is challenging due to insufficient data on the gender of all patients in three studies (Baier et al., 2014; Green & Hamilton, 1976; Stone et al., 1993), or missing information about the gender of those displaying AHP in LBD (Beis et al., 2004; Cocchini et al., 2009, 2018). The overall ages of patients ranged from 28 to 100 years. The mean years of education in the studies providing indication ranged from eight to ten years (*SD* = 3-5) with a range of two to seventeen years (Beis et al., 2004; Cocchini et al., 2009, 2018; Formica et al., 2022; Hartman-Maeir et al., 2001). Among the seven studies only two patients were left-handed, while eight were right-handed. Certain selection criteria in the studies included patients who had experienced their first stroke (Beis et al., 2004; Cocchini et al., 2018; Hartman-Maeir

et al., 2001; Stone et al., 1993), with the requirement that comprehension be intact and no prior history of neurological or psychiatric illness (Baier et al., 2014; Cocchini et al., 2009, 2018; Hartman-Maeir et al., 2001).

7.2 Assessment Methods

Assessment methods and details are presented in Table 3. All studies utilised explicit assessment methods, previously visible in Table 1, primarily employing clinical interviews (Table 3). In the study by Cocchini et al. (2009), the assessment methods of the structural interview and the more inclusive interview of the VATAm were compared. In this study the Berti et al. (1996) Scale Structured Interview identified two anosognosic patients, while the VATAm identified twelve with varying severity. In their subsequent study Cocchini et al. (2018) compared the VATAm and ECT measurement. The VATAm identified six patients, and the ECT identified one additional patient with AHP for LBD, and both applied combined diagnosed another seven patients.

Eight studies included patients with AHP and LBD who demonstrated no severe language disturbances as indicated by test batteries and the applicability of structured clinical interviews. The remaining three studies, conducted by Cocchini et al. (2009, 2018) and Hartman-Maeir et al. (2001), also included aphasic patients due to the requirements of the VATAm, ECT, and Task Choice Method of language comprehension but provided aid in form of pictures and pointing instead of a clear verbal response. Therefore, these studies only required comprehension for assessment and aimed to include aphasic patients. In Table 3, the number of excluded patients is provided, with 20 patients being excluded due to comprehension difficulties (Beis et al., 2004; Cocchini et al., 2009), and a total of 67 patients

excluded in two studies due to aphasia (Baier et al., 2014; Stone et al., 1993). Stone et al. (1993) noted a confidence interval of a 32% difference in proportions of unassessable patients with a 95% confidence interval regarding the accessibility of testing RBD versus LBD patients.

7.3 Neuroimaging, Comorbidities and Functional Outcome

Table 4 presents the lesion details extracted from a systematic review of patients with AHP and unilateral left lesions, along with the lesion size. However, specific data for lesions of patients with AHP from Beis et al. (2004) and Stone et al. (1993) were not available. To summarise the frequency of anatomical areas, Tables 5 and 6 display cortical and subcortical areas found in the ten studies reviewed.

Half of the studies indicated occurrences of other neurological disturbances in relation to LBD rather than in relation to co-occurrence with AHP. The few studies identifying co-occurrences with AHP are also presented in Table 4.

Table 4

Neuro-anatomical Correlation of Patients with AHP after Left Sided Stroke, Stage of AHP, the Neuroimaging Technique Applied, the Lesion Details, Comorbidities and if AHP Decreased over Time.

Reference	*N*		Stage	Technique	Lesion	Size [a]	Comorbidities	Remission [b]
Green et al. (1976)	1	Hemiplegia	Subacute	EMI	Thalamic hematomas	N/A	Right hemianesthesia, hemiasomatognosia	N/A
Dronkers et al. (1989)	1	Hemiparesis	Subacute	CT	Infarct in the dorsolateral left PFC extending superiorly into the inferior portions of the frontal eye fields and posteriorly into the centrum semiovale, subcortical extension into the anterior limb of the internal capsule and the lateral surface of the caudate nucleus with some compression of the frontal horn	N/A	Hemi-spatial neglect, contralateral hypokinesia, visual-spatial constructive difficulties, mild dysarthria and aprosodic, monotonic speech	Day 9
Cohen et al. (1991)	1	Hemiplegia	Subacute	CT	Parieto-temporal infarction, territory of the middle cerebral artery (MCA), both deep and superficial, and the territory of the anterior choroidal artery	N/A	Minor hemisphere syndrome, right hemispatial neglect, right hemiasomatognosia, motor aprosodia, variable right asomatognosia, impairment in identifying and producing musical tunes, hypesthesia and tactile extinction right	N/A

Reference	*N*		Stage	Technique	Lesion	Size [a]	Comorbidities	Remission [b]
Stone et al. (1993)	3	Hemiparesis	Acute	CT	Hemispheric stroke, further N/A for AHP	N/A	N/A for AHP	N/A
Hartman-Maeir et al. (2001)	4	Hemiplegia UE [c]	Chronic	CT	Subcortical lesion capsular putaminal (CP), intrahemispheric white matter (WM)	1-3	No	N/A
					Subcortical lesion: CP	1-3	No	
					Subcortical lesion: CP, intrahemispheric WM	1-3	No	
					Cortical lesion: Frontal and parietal lobe, Subcortical lesion: CP, intra hemispheric WM	3-5	Neglect	
Beis et al. (2004)	5	Hemiplegia	Chronic	CT, MRI	N/A for AHP	N/A	Neglect	
Cocchini et al. (2009)	12 10 2	Hemiparesis	Chronic Subacute	CT	Vascular unilateral lesion: fronto-temporoparietal region, two had subcortical lesions, basal ganglia, internal capsule, and the thalamus	N/A	One patient with neglect	
Ronchi et al. (2013)	1	Hemiplegia	Chronic	MRI	Ischaemic cerebrovascular disease: large cortical and subcortical hypodense lesion in the LH including TPJ	N/A	Hemihypoesthesia, right personal and extrapersonal neglect, hemianopia, somatoparaphrenia, mild aphasia, severe apraxia, hemianesthesia	1,5 months with CVS

Reference	*N*		Stage	Technique	Lesion	Size [a]	Comorbidities	Remission [b]
Baier et al. (2014)	1	Hemiparesis	Subacute	CT, MRI	Large left-sided ischemic lesion of the MCA territory affecting frontal and temporoparietal regions like the superior temporal gyrus, the angular gyrus, the insula and the IFG, the postcentral gyrus, and the operculum	N/A	Right spatial neglect, hemihypaesthesia	Day 9
Cocchini et al. (2018)	14	Hemiparesis UE [c]	Chronic	CT, MRI	Lesion encompassing, or limited to the frontal and parietal lobes	N/A	1 out of 3 had extrapersonal and/or personal neglect	
Formica et al. (2022)	1	Hemiplegia	Subacute	FMRI	Cerebral ischemic lesion in the frontotemporal and left insular areas, no blood flow in left internal carotid artery, and the MCA of the left side	7.15	Babinski's sign right side, right facial hemiparesis, poor pneumo-phonatory coordination, lack of verbal initiative, apathy, lack of emotional expression, and AHP on the right hand, mild aphasia	Day 9

Note. *N* = 44. Number of patients diagnosed with AHP and LBD in the 11 articles.

[a] Size in *cm*.

[b] Reflects spontaneous remission or decrease of anosognosia.

[c] UE = Upper Extremity.

Table 5

Most Frequent Involvement of Cortical Anatomical Areas Identified with Neuroimaging Techniques of Patients with AHP and LBD.

Area of Lesion	Anatomical lesion	*N*
Cortical Lesion	Frontal Lobe	6
	Parietal Lobe	6
	Temporal Lobe	5
	Occipital Lobe	0
	Middle Cerebral Artery	3

Note. N = 9. Number of Studies that observed lesions in these areas.

Table 6

Most Frequent Involvement of Subcortical Anatomical Areas Identified with Neuroimaging Techniques of Patients with AHP and LBD.

Area of Lesion	Anatomical lesion	*N*
Subcortical	Basal Ganglia	1
	Caudate nucleus	1
	Centrum semiovale	1
	Insular	2
	Internal Capsule	2
	Inferior Frontal Gyrus	1
	Intrahemispheric white matter	1
	Thalamus	2

Note. N = 9. Number of Studies that observed lesions in these areas.

8. Discussion

8.1 Manifestation

Although AHP is less commonly encountered in its chronic state (Orfei et al., 2009; Prigatano, 1999), the majority of the eleven studies reviewed included a high number of chronic patients with AHP and LBD, providing evidence for the occurrence of AHP in patients with lesions in the left hemisphere. As mentioned previously (Chapter 2.1), different levels of awareness may coexist or differ within the same patient and fluctuate over time (Moro et al., 2021), also captured in the cases of the present review. Similar manifestations of AHP were observed in the cases of AHP in LBD in the present review. Denial of impairment were observed in several studies (Baier et al., 2014; Cohen et al., 1991; Dronkers et al., 1989; Ronchi et al., 2013), even after neurological demonstrations. Dronkers et al. (1989) reported non-related explanations when a patient was asked to move her hand, as she was unable to move her right hand due to being left-handed. Anticipatory awareness (Moro et al., 2015; Pacella et al., 2019) was also observed in several studies (Cohen et al., 1991; Formica et al., 2022; Hartman-Maeir et al., 2001). In a case study by Formica et al. (2022), the patient showed inconsistencies in responses regarding activities she could or could not perform, such as stating she could not drink with her right hand but could clap or wash her hands without problems. Overall, she overestimated her abilities to move, similar to observations in RBD patients with AHP. Hartman-Maeir et al. (2001), utilising an implicit measure to test AHP, identified four patients with implicit anticipatory awareness, where patients attempted to perform bimanual tasks despite their hemiplegia, failing to predict their current ability to perform the task. Cohen et al. (1991) noted verbal persistence of AHP, with patients still asserting their ability to perform bimanual activities, and sometimes implicit awareness, as

evidenced by requesting support with walking. Dronkers et al. (1989) observed a general impairment of awareness in their case study, where the patient provided another reason for her hospitalisation besides her hemiplegia or stroke. Impaired mentalization (Besharati et al., 2016; Feinberg, 2000) processes were noted by Cohen et al. (1991), where the patient disregarded the diagnosis of hemiplegia because she claimed she could walk, despite being informed otherwise by hospital staff. Modality specificity was observed in the study by Cocchini et al. (2009). Additionally, confabulations and claims of illusory movement were present in two studies (Cohen et al., 1991; Ronchi et al., 2013).

Co-occurring disturbances, sometimes accompanied AHP, but not in all cases (see Table 4). Many studies reported no aphasia or only mild aphasia (Baier et al., 2014; Cohen et al., 1991; Dronkers et al., 1989; Formica et al., 2022). Behavioural changes such as lack of verbal initiative, apathy, and lack of emotional expression were observed in some cases (Dronkers et al., 1989; Formica et al., 2022). Proprioceptive loss was less frequently found, but there is insufficient evidence to conclude that it would be less frequent than in RBD patients with AHP. Similarly, cognitive impairment was only found in the study by Ronchi et al. (2013), thus no definitive conclusion can be drawn here either. Regarding ADLs, Formica et al. (2022) and Hartman-Maeir et al. (2001) found concerning levels of impairment in patients ability to perform ADLs and live independently with AHP after a stroke.

Baier et al. (2014), Dronkers et al. (1989), and Formica et al. (2022) reported incidences of spontaneous rehabilitation occurring after nine days, a phenomenon typically observed in patients with AHP and RBD (Antoniello & Gottesman, 2020; Gialanella & Mattioli, 1992). In the case study by Ronchi et al. (2013), the patient displayed a spontaneous and stable regression of AHP after CVS, resulting in only a

minimization of the severity of his impairments, similar to anosodiaphoria. Despite its higher frequency of occurrence in RBD patients, Hartman-Maeir et al. (2001) investigated the functional outcomes of AHP patients in both hemisphere groups and revealed their inability to retain safety measures at discharge from rehabilitation, as well as their need for assistance in basic and instrumental activities of daily living at their follow-up, with no differing awareness observed between the groups. AHP, regardless of the lesion side causing it, presents a significant risk for negative functional outcomes in stroke rehabilitation.

8.2 Assessment

Compared to Jehkonen et al.'s (2006) systematic review covering the period from 1995 to 2005, the prevalence of AHP in LHB patients ranged between 6% to 24%. The present review was conducted almost twenty years later and includes studies from 1976 to 2024 revealing an increase in prevalence from 2% to 48%. As suggested by Cocchini et al. (2022) and Hartman-Maeir et al. (2003), AHP in LBD might be more frequent than previously expected, and recent studies are attempting to address methodological limitations. These limitations not only include making testing available for mild to moderate aphasic patients, but also aligning and updating the current diagnostic measures of AHP with its evolving understanding.

As seen in Table 3, prevalently explicit measures in the form of structured clinical interviews were applied to investigate AHP. However, solely relying on clinical interviews provides limited insight into the heterogeneous expression of AHP, especially in investigations involving LBD patients. As mentioned in the previous chapter (2.1), only one-third of patients with AHP express anosognosia through explicit denial (Pia et al., 2004), yet the most common measure to diagnose AHP remains an explicit interview based on the patient's denial of motor impairment. Apart

from Hartman-Maeir et al. (2001), no other study attempted to investigate implicit awareness, which would be beneficial for including aphasic patients, and measuring nonverbal anosognosic behaviour alongside clinical interviews. Hartman-Maeir et al. (2001) identified 25% of hemiplegic stroke patients with chronic AHP in their post-acute phase through testing for implicit awareness, without finding explicit expression of AHP at this stage, consistent with previous findings by Jehkonen et al. (2000). They state that conservative methods of verbal assessment are not sensitive enough to assess anosognosia in its chronic phase. Cocchini et al. (2009) argue that subacute or chronic patients eventually become accustomed to their environment, making it challenging for structured interviews to effectively reveal their anosognosia for their hemiplegic state.

Overall, comparison studies of RBD and LBD patients do show a higher prevalence of AHP in RBD patients (Stone et al., 1993; Pedersen et al., 1996). However, upon closer examination of these studies, Stone et al. (1993) calculated a confidence interval of a 32% difference in proportions of unassessable patients with a 95% confidence interval regarding the accessibility of testing RBD versus LBD patients. This is partly due to comprehension difficulties and partly due to the application of clinical structured interviews that are unable to be applied to aphasic patients. Unlike structured interviews, which rely on verbal communication and focus on general questions or movement requirements, alternatives such as the VATAm offer a more comprehensive understanding. For instance, the inclusion of visualised tasks and the evaluation of one's motor abilities in various situations promotes higher sensitivity and reduces the requirement for fluent speech. Cases of limitations regarding aphasia are observed in studies by Baier et al. (2014), who had to exclude 33% of their participants, and Stone et al. (1993), who had to exclude 45% of their

LBD sample. Methodological limitations, especially in comparison studies such as the one by Cocchini et al. (2009), reveal that the application of visual tests for AHP over structured interviews leads to a lower exclusion rate and significantly higher detection of AHP. In their sample, the structured interview had to exclude 52.4% of participants, compared to only 28.6% with the VATAm. More importantly, 23% of patients categorised as unassessable for the structured interview exhibited mild to moderate signs of anosognosia. Mild to moderate anosognosia indicated in their study unawareness regarding their impairment, with differing scores below the cutoff compared to those of their caregivers, despite assessment occurring 50 to 70 days post-stroke. Conversely, none of the patients excluded for the VATAm could be assessed by the structured interview. The studies, which implement integrative measures for aphasic patients, clearly demonstrate evidence of overlooked aphasic patients with AHP.

While Baier et al. (2014) criticised Cocchini et al. (2009) for applying too low thresholds for diagnosing AHP, Cocchini et al. (2018) noted that even if they were to apply a more conservative criterion, as advocated by Baier et al. (2014) excluding mild forms of anosognosia, still 33% of their sample displayed more severe forms of anosognosia. Additionally, Marcel et al. (2004) indicated that AHP was more effectively assessed when patients were asked to perform specific bimanual or bipedal tasks instead of answering global questions about their limbs. For example, in the case described by Formica et al. (2022), the patient showed inconsistencies in responses regarding which activities she could or could not perform, which could lead to different score ratings on structured clinical interviews, particularly if only one or two actions such as lifting a limb are required. Comparing the VATAm and ECT method (Cocchini et al., 2018) resulted in different diagnoses of AHP for different

patients, implying that a single measurement may not be sufficient to identify all patients with AHP due to variations in its expression within individuals. Different methods may be able to address different aspects of awareness, and the ECT proved to be capable of detecting less evident forms of awareness.

The heterogeneity of AHP necessitates testing across various domains to capture its diverse expressions (Cutting, 1978; Cocchini et al., 2009), and several factors may underlie deficits in awareness (Cocchini et al., 2002; Davies et al., 2005; Orfei et al., 2007; Vuilleumier, 2004). Often, patients' behaviour is inconsistent with their explicit acknowledgment or denial of their motor impairment, especially in chronic stages (Cocchini et al., 2010; Moro et al., 2011; Ramachandran & Blakeslee, 1998). Studies such as those by Baier et al. (2014) or Ronchi et al. (2013) continue to employ a clinical interview designed almost 30 years ago, despite the availability of new measures. This outdated approach does not align with the current understanding of the heterogeneous expression of AHP. This classification gap leads to different findings regarding AHP in LBD patients. Conservative application of explicit measures contrasts with the recognition of AHP as a multifaceted syndrome with implicit and explicit expressions of awareness. These expressions may not always be evident in direct interviews but are apparent in behavioural tasks. The divergence in concepts and treatment of patients with AHP may explain the infrequent findings of AHP in LBD. Patients with AHP may acknowledge their hemiplegia but still demonstrate unawareness of its consequences, a phenomenon often revealed only in implicit tasks rather than explicit interviews. The understanding of AHP as a neurological disorder has evolved, and our knowledge of it has expanded. This knowledge has to be updated and implemented into the

methodological classification of AHP to ensure more consistent findings in research studies.

8.3 Underlying Theories

Underlying theories mentioned in the review were the feedforward theory, supported by the fMRI analysis conducted by Formica et al. (2022). According to the feedforward theory, anosognosia arises from a discrepancy between the expectation of movement and the perception of the movement. Their analysis revealed activation of motor areas of the patient with AHP, even when movements were only imagined, suggesting that similar neural patterns are engaged whether a movement is physically executed or imagined.

Some researchers view AHP after brain injury as a failure of functional balance between the two hemispheres (Ramachandran, 1996). In the present review, several studies argue against the role of the left hemisphere in self-awareness, despite the occurrence of AHP in response to unilateral lesions of the left hemisphere. Interestingly, five studies suggest a reversed hemispheric specialisation in their patients, where the left hemisphere appears to be responsible for awareness, while the right hemisphere seems more dominant for language, speech generation, and processing. Concerning language lateralization, the vast majority of right-handed individuals exhibit left hemispheric dominance for language (Corballis, 2009; Dorsaint-Pierre et al., 2006; Dronkers et al., 2004; Knecht et al., 2000; Rasmussen & Milner, 1977). However, a small subset of acute neurological patients presents with a reversal of this typical asymmetry, demonstrating right hemisphere dominance for language instead of the expected left hemisphere dominance (Marien et al., 2004; Padovani et al., 1992). In the case study by Dronkers et al. (1989), the left-handed patient exhibited a constellation of

neurological disturbances including visuo-spatial deficits and aprosodic speech, which are typically associated with right hemisphere lesions (Table 4). Due to preserved language ability, the authors proposed a bilateral or reversed lateralization of hemisphere dominance to account for these syndromes in LBD. Similarly, in Cohen et al. 's (1991) case study involving a right-handed patient, reversed lateralization of language was suggested due to the absence of language impairment following stroke. They suggested that if the left hemisphere had dominance for language, or if language was represented bilaterally, significant damage to the left hemisphere would have resulted in aphasic symptoms, which were not observed. Furthermore, they hypothesised that the patient's left hemisphere was primarily dominant for body schema, manual dexterity, and spatial attention. Moreover, in the case described by Ronchi et al. (2013) involving a left-handed patient, cognitive functions appeared to be predominantly localised in the left hemisphere. Despite suffering a significant stroke, the patient displayed severe praxic and visuo-spatial deficits, while aphasic symptoms remained relatively mild. This observation suggests that linguistic functions may be partially lateralized in the left hemisphere, with possible involvement of the intact right hemisphere in verbal production and comprehension. In the cases reported by Formica et al. (2022) and Baier et al. (2014), fMRI analysis were conducted to investigate cerebral activation patterns (Table 4). Even though Baier et al. 's (2014) patient was right-handed, the authors identified reversed spatial neglect and language functions through asymmetric activity during a language task. They suggested that this observation aligns with the existence of a minority of approximately 3% of right-handers exhibiting right lateralization of language ability (Knecht et al., 2000; Marien et al., 2004; Padovani et al., 1992). The fMRI analysis conducted by Formica et al. (2022) highlighted the

involvement of the left hemisphere in motor planning, motor imagery, and motor representation tasks. Conversely, activation during a language task was predominantly observed in the right hemisphere, suggesting lateralization of language functions in the patient's right hemisphere. In contrast, some studies argue against this theory to provide an explanation for all cases of AHP caused by left lesions. The patients examined by Hartman-Maeir et al. (2001) were all right-handed and possessed adequate language skills. Moreover, studies employing measures such as the VATAm did not exclude patients with mild to moderate aphasia and did not find evidence for reversed lateralization. Additionally, the extent to which non-AHP patients with left brain damage exhibited right hemisphere language representation remains unclear. Overall, the occurrence of AHP in patients with left-sided lesions suggests the possibility of certain cases to be the cause of reversed hemispheric lateralization, implying that the left hemisphere may not inherently play a primary role in our self-awareness of limb movement.

8.4 Anatomical-Correlational Findings

Due to the limited availability of lesion details, only a general comparison between LBD and RBD lesions in relation to AHP can be made. However, the available data on lesions in the patients from the systematic review strongly correlates with anatomical-correlational studies on the neuroanatomical basis of AHP in RBD patients (Chapter 3). Similar to RBD patients with AHP, lesions associated with AHP and LBD were primarily located in frontal, parietal, and temporal cortical structures (Table 5). Even though some studies did not find a significant association between lesions in the frontal or parietal lobes and anosognosia (Coccioni et al., 2009). The previously mentioned Geschwind’s disconnection hypothesis (1965) postulates an interhemispheric disconnection. LBD patients can be of high

importance due to neuroimaging studies identifying bilateral networks and diaschisis as possible inclusion criteria for both hemispheres (Coccioni et al., 2022). Especially, the sense of agency is supposed to be integrated into a bilateral network (Seghezzi et al., 2019; Di Plinio et al., 2020; Zapparoli et al., 2020). Anosognosia may arise from damage to the fronto-parietal circuit responsible for spatial and motor representation, with frontal involvement potentially affecting a motor monitoring system (Desmurget & Sirigu, 2009; Frith et al., 2000; Haggard, 2005), regardless of lesion side. Lesions affecting specific components of this circuit may lead to selective and spatially confined awareness disorders, as observed in AHP and its co-occurring neurological disturbances in patients with both RBD (Chapter 2.1) and LBD (Table 4).

Similar to lesion mapping studies in RBD patients, AHP was frequently associated with strokes in the area of the MCA (Baier et al., 2014; Besharati et al., 2014; Cohen et al., 1991; Formica et al., 2022; Fotopoulou et al., 2010). Despite a correlation with lesion size, the present review found cases of AHP even in smaller lesion sizes (Table 4). Limited detailed information on subcortical lesions (Table 6) revealed that, similar to RBD patients with AHP (Berti et al., 2005; Besharati et al., 2022; Fotopoulou et al., 2010; Karnath et al., 2005; Monai et al., 2020; Pacella et al., 2019; Pyastik et al., 2022), left hemispheric lesions affected various regions, including the basal ganglia, caudate nucleus, centrum semiovale, insular cortex, internal capsule, iFG, intra hemispheric white matter, LPFC, thalamus, and TPJ (Baier et al., 2014; Cocchini et al., 2009, 2018; Dronkers et al., 1989; Formica et al., 2022; Green & Hamilton, 1976; Hartman-Maeir et al., 2001; Ronchi et al., 2013).

In a localization study of the performance-monitoring network the basal ganglia, dorsolateral PFC, insula, and thalamus as main components, all frequently

affected in AHP, were identified in both hemispheres (Pyasik et al., 2022). Furthermore, Moro et al. (2016) associated right lesions and AHP with the rolandic operculum, the insula, and the superior temporal gyri, aligning with partial cases of AHP in left lesions in the present study as well (Table 6). Additionally, Moro et al. (2016) discovered in acute-stage lesions involvement of the basal ganglia, caudate, insula, putamen, internal, and external capsule, also observed in the LBD subacute cases after seven days (Table 4). Their chronic association was with the fronto-temporal cortex, gyri temporales transversi, insular, superior temporal cortex, the thalamus, and the ventral premotor cortex, also observed in the present study, but not all in chronic cases.

Additionally, similar to Moro et al.'s (2016) findings, intra-hemispheric white matter lesions were observed in LBD patients. These white matter pathways involving the fronto-temporal cortex were found to be predictive of persistence of AHP beyond 40 days (Moro et al., 2016). Similarly, patients with damage to the white matter pathways (Table 4) were indeed chronic AHP patients (Hartman-Maeir et al., 2001). The tripartite disconnection syndrome of white matter pathways (Pacella et al., 2019), involving the premotor loop, limbic system, and VAN, impacts the iFG of the premotor loop, the insular, and temporoparietal junction, which are also observed in left lesioned patients with AHP. These results emphasise the joint contribution of these three systems to motor awareness. Even though Karnath et al. (2005) claimed that the insular cortex has a high importance in awareness, patients with lesions only in this area did not display anosognosia. Nevertheless, it is noteworthy that some neuroimaging studies of motor and emotional awareness have found bilateral activation of the insular cortex (Farrer & Frith, 2002). Supported by the present review, the insular cortex can play an additional role in both, right and left

hemispheric lesions causing AHP. Therefore, it is necessary to investigate whether the distinct roles of the right and left insular cortex in motor and emotional awareness are significant for their combined functional role. The limbic system is associated with a distinctive pattern of intrinsic connectivity during introspective states and self-referential processes, including autobiographical retrieval, future imagining, and mentalization (Pacella et al., 2019). As previously mentioned, in differentiation from healthy participants (Monai et al., 2020), AHP patients exhibited significantly increased and more widespread white matter pathway disconnection in various regions, including the right insula, fronto-insular tract, right TPJ, right lateral and mPFC, specifically the IPL and SPL, STG, MTG, PMC, anteriorly in the iFG, SLF, and subcortically in the putamen. The IFG is also associated with the TPJ, forming part of the VAN, via the SLF II, one of the most damaged pathways in AHP (Monai et al., 2020). These areas play a crucial role in the integration of multi-modal body and visuospatial signals and in switching between perspectives of the body, environment, external and internal stimuli, and self or others (Corbetta et al., 2008). Similar to the previously presented (Kirsch et al., 2021; Pacella et al., 2019), there is a significant correlation between damage and disconnection of white matter pathways of VAN linking the insula, TPJ, and ventral PFC with belief updating in AHP. Importantly, no isolated pattern of lesions or disconnections to any single system can fully explain AHP.

9. Limitations and Future Research

The limitations of the present study include the limited availability of studies involving patients with LBD and anosognosia, as well as the incomplete information provided by some studies regarding those patients. Consequently, drawing definitive conclusions and establishing neuroanatomical correlations specific to LBD and

anosognosia is challenging. Furthermore, the onset was homogeneously stroke for all patients in this review. Perhaps different aetiology of onset can display differing expressions of AHP within patients and needs further investigation. Despite these limitations, the occurrence of anosognosia in LBD patients was observed, highlighting the need for further investigation in this area. Studies relying solely on conservative measures such as structural interviews may predominantly identify AHP patients with RBD and reversed hemispheric lateralization. In contrast, more inclusive methods may uncover patients with AHP and LHD who exhibit different types of unawareness.

Future studies should explore whether patients with RBD and anosognosia can also exhibit reversed hemispheric lateralization, or if this phenomenon is unique to patients with LBD and anosognosia. Moreover, research should investigate whether the majority of LBD patients with anosognosia demonstrate this reversed lateralization, or if different types of unawareness can also arise from left unilateral damage without reversed lateralization. Additionally, it is necessary to update the concept of AHP in research, allowing for the investigation of various types of unawareness through different assessment measures beyond explicit structural clinical interviews. This inclusive approach should encompass mild to moderate aphasic patients and further explore the rare occurrence of AHP in LBD patients.

10. Conclusion

The exploration of AHP reveals a multifaceted and heterogeneous syndrome that challenges traditional diagnostic approaches. The complexities of AHP, stemming from its dynamic interplay of diverse expressions of awareness, are not yet fully understood. While historical perspectives focused on single-domain theories and anatomical correlations of specific areas, contemporary theories emphasise a

more holistic view. They highlight the involvement of multiple brain regions, the disconnection of white matter pathways, and affected brain circuits, advocating for an integrated explanation for the cause of AHP. Moreover, no single theory proposed can fully explain the diverse expressions of AHP, therefore integration of multiple theories might provide better insights. This observation is also captured in the current findings regarding reversed lateralization as an underlying theory for AHP in LBD patients. While this potential role of reversed hemispheric lateralization is evident in the majority of case studies, it cannot fully explain all cases presented in this review of AHP in LBD patients. Nonetheless, it represents an intriguing pathway for further investigation. It is to explore whether a majority of patients with AHP and left hemispheric lesions are affected by different connectivity patterns in the cerebral hemispheres, and if reversed lateralization plays a significant role in their manifestation.

Overall, the review revealed that while patients with left hemispheric lesions and AHP exist, they appear in a minority of studies over the last 60 years. A gap created by the limitations of current diagnostic tools likely contributes to the sparse research on AHP in LBD patients. Although many researchers indicate that AHP predominantly occurs in RBD patients, the size of this gap cannot be determined. Current diagnostic tools for AHP are based on measures developed almost 30 years ago, primarily focusing on the expression of explicit awareness in AHP. As demonstrated in this review, these outdated measures lead to overlooking other expressions of AHP and excluding patients with post-brain damage speech difficulties. Although more comprehensive tools exist, they are not applied in the majority of cases.

Further research is needed with a mutually updated perspective on AHP that reflects current knowledge and adapts predominantly homogeneous diagnostic tools to more heterogeneous ones. This would better accommodate the diverse expressions of AHP. With this research base, the consequences of the extent of the existing diagnostic gap can be evaluated. It should be determined whether this gap is due to different expressions of AHP in LBD patients that were previously overlooked due to methodological limitations, or if AHP is indeed a minority condition when comprehensive, holistic, and integrative assessment measures, including those for mild to moderate aphasic patients, are largely applied.

Regardless of the hemisphere in which AHP is expressed, similar manifestations and progression were observed for both cases. The functional outcome and prognosis for chronic AHP were similarly worse and worthy of attention. Acknowledging the diverse expressions of AHP, including implicit manifestations and integrating measures for mild to moderate aphasic patients, will also promote treatment efficacy and improve functional outcomes for patients.

To conclude, this review advocates for a paradigm shift in the conceptualization and assessment of AHP, moving towards a holistic understanding that reflects its complex and heterogeneous nature. By embracing this comprehensive perspective, researchers and clinicians can advance our understanding of AHP and investigate its diverse expressions, including those with post-stroke expressions in the left hemisphere.

11. Bibliography

Adair, J. C., Schwartz, R. L., Na, D. L., Fennell, E., Gilmore, R. L., & Heilman, K. M. (1997). Anosognosia: Examining the disconnection hypothesis. *Journal of Neurology, Neurosurgery & Psychiatry*, *63*(6), 798–800. https://doi.org/10.1136/jnnp.63.6.798

Agnew, S. K., & Morris, R. G. (1998). The heterogeneity of anosognosia for memory impairment in Alzheimer's disease: A review of the literature and a proposed model. *Aging & Mental Health*, *2*(1), 7–19. https://doi.org/10.1080/13607869856876

Anderson, M. C., & Green, C. (2001). Suppressing unwanted memories by executive control. *Nature*, *410*(6826), 366–369. https://doi.org/10.1038/35066572

Anderson, S. W., & Tranel, D. (1989). Awareness of disease states following cerebral infarction, dementia, and head trauma: Standardised assessment. *Clinical Neuropsychologist*, *3*(4), 327–339. https://doi.org/10.1080/13854048908401482

Antoniello, D., & Gottesman, R. (2020). The Phenomenology of Acute Anosognosia for Hemiplegia. *The Journal of Neuropsychiatry and Clinical Neurosciences*, *32*(3), 259–265. https://doi.org/10.1176/appi.neuropsych.19010008

Appelros, P., Karlsson, G. M., & Hennerdal, S. (2007). Anosognosia versus unilateral neglect. Coexistence and their relations to age, stroke severity, lesion site and cognition. *European Journal of Neurology*, *14*(1), 54–59. https://doi.org/10.1111/j.1468-1331.2006.01544.x

Appelros, P., Karlsson, G. M., Seiger, Åke, & Nydevik, I. (2002). Neglect and Anosognosia After First-Ever Stroke: Incidence and Relationship to Disability. *Journal of Rehabilitation Medicine*, *34*(5), 215–220. https://doi.org/10.1080/165019702760279206

Azouvi, P., Olivier, S., De Montety, G., Samuel, C., Louis-Dreyfus, A., & Tesio, L. (2003). Behavioural assessment of unilateral neglect: Study of the psychometric properties of the Catherine Bergego Scale. *Archives of Physical Medicine and Rehabilitation*, *84*(1), 51–57. https://doi.org/10.1053/apmr.2003.50062

Azouvi, P., & Peskine, A. (2011). Anosognosia and denial after right hemisphere stroke. *The Behavioral and Cognitive Neurology of Stroke*, 158–169. https://doi.org/10.1017/CBO9781139058988.014

Azouvi, P., & Peskine, A. (2013). Anosognosia and denial after right hemisphere stroke. In O. Godefroy (Hrsg.), *The Behavioral and Cognitive Neurology of Stroke* (2. Aufl., S. 158–169). Cambridge University Press. https://doi.org/10.1017/CBO9781139058988.014

Bach, L. J., & David, A. S. (2006). Self-awareness after acquired and traumatic brain injury. *Neuropsychological Rehabilitation*, *16*(4), 397–414. https://doi.org/10.1080/09602010500412830

Bagby, R. M., Taylor, G. J., & Parker, J. D. A. (1994). The twenty-item Toronto Alexithymia scale—II. Convergent, discriminant, and concurrent validity. *Journal of Psychosomatic Research*, *38*(1), 33–40. https://doi.org/10.1016/0022-3999(94)90006-X

Baier, B. (2005). Incidence and diagnosis of anosognosia for hemiparesis revisited. *Journal of Neurology, Neurosurgery & Psychiatry*, *76*(3), 358–361. https://doi.org/10.1136/jnnp.2004.036731

Baier, B., & Karnath, H.-O. (2008). Tight Link Between Our Sense of Limb Ownership and Self-Awareness of Actions. *Stroke*, *39*(2), 486–488. https://doi.org/10.1161/STROKEAHA.107.495606

Baier, B., Vucurevic, G., Müller-Forell, W., Glassl, O., Geber, C., Dieterich, M., & Karnath, H.-O. (2014). Anosognosia for hemiparesis after left-sided stroke. *Cortex*, *61*, 120–126. https://doi.org/10.1016/j.cortex.2014.07.017

Bean, J. (2011). Rey Auditory Verbal Learning Test, Rey AVLT. In J. S. Kreutzer, J. DeLuca, & B. Caplan (Hrsg.), *Encyclopedia of Clinical Neuropsychology* (S. 2174–2175). Springer New York. https://doi.org/10.1007/978-0-387-79948-3_1153

Beck, A. T. (1961). An Inventory for Measuring Depression. *Archives of General Psychiatry*, *4*(6), 561. https://doi.org/10.1001/archpsyc.1961.01710120031004

Beis, J.-M., Keller, C., Morin, N., Bartolomeo, P., Bernati, T., Chokron, S., Leclercq, M., Louis-Dreyfus, A., Marchal, F., Martin, Y., Perennou, D., Pradat-Diehl, P., Prairial, C., Rode, G., Rousseaux, M., Samuel, C., Sieroff, E., Wiart, L., & Azouvi, P. (2004). Right spatial neglect after left hemisphere stroke: Qualitative and quantitative study. *Neurology*, *63*(9), 1600–1605. https://doi.org/10.1212/01.WNL.0000142967.60579.32

Bertagnoli, S., Pacella, V., Rossato, E., Jenkinson, P. M., Fotopoulou, A., Scandola, M., & Moro, V. (2022). Disconnections in personal neglect. *Brain Structure and Function*, *227*(9), 3161–3171. https://doi.org/10.1007/s00429-022-02511-z

Berti, A. (2004). Cognition in Dyschiria: Edoardo Bisiach's Theory of Spatial Disorders and Consciousness. *Cortex*, *40*(2), 275–280. https://doi.org/10.1016/S0010-9452(08)70122-9

Berti, A., Bottini, G., Gandola, M., Pia, L., Smania, N., Stracciari, A., Castiglioni, I., Vallar, G., & Paulesu, E. (2005). Shared Cortical Anatomy for Motor Awareness and Motor Control. *Science*, *309*(5733), 488–491. https://doi.org/10.1126/science.1110625

Berti, A., Làdavas, E., & Corte, M. D. (1996). Anosognosia for hemiplegia, neglect dyslexia, and drawing neglect: Clinical findings and theoretical considerations. *Journal of the International Neuropsychological Society*, *2*(5), 426–440. https://doi.org/10.1017/S135561770000151X

Berti, A., Ladavas, E., Stracciari, A., C, G., & A, O. (1998). Anosognosia for Motor Impairment and Dissociations with Patients' Evaluation of the Disorder: Theoretical Considerations. *Cognitive Neuropsychiatry*, *3*.

Berti, A., & Pia, L. (2006). Understanding Motor Awareness Through Normal and Pathological Behaviour. *Current Directions in Psychological Science*, *15*(5), 245–250. https://doi.org/10.1111/j.1467-8721.2006.00445.x

Berti, A., Spinazzola, L., & M, P. (2007). Motor awareness and motor intention in anosognosia for hemiplegia. In *Sensorimotor Foundations of Higher Cognition* (S. 163–181). https://doi.org/10.1093/acprof:oso/9780199231447.003.0008

Besharati, S., Forkel, S. J., Kopelman, M., Solms, M., Jenkinson, P. M., & Fotopoulou, A. (2014). The affective modulation of motor awareness in anosognosia for hemiplegia: Behavioural and lesion evidence. *Cortex*, *61*, 127–140. https://doi.org/10.1016/j.cortex.2014.08.016

Besharati, S., Forkel, S. J., Kopelman, M., Solms, M., Jenkinson, P. M., & Fotopoulou, A. (2016). Mentalizing the body: Spatial and social cognition in anosognosia for hemiplegia. *Brain*, *139*(3), 971–985. https://doi.org/10.1093/brain/awv390

Besharati, S., Jenkinson, P. M., Kopelman, M., Solms, M., Moro, V., & Fotopoulou, A. (2022). Awareness is in the eye of the observer: Preserved third-person awareness of deficit in anosognosia for hemiplegia. *Neuropsychologia*, *170*, 108227. https://doi.org/10.1016/j.neuropsychologia.2022.108227

Besharati, S., Kopelman, M., Avesani, R., Moro, V., & Fotopoulou, A. (2015). Another perspective on anosognosia: Self-observation in video replay improves motor awareness. *Neuropsychological Rehabilitation*, *25*(3), 319–352. https://doi.org/10.1080/09602011.2014.923319

Bisiach, E., & Berti, A. (1995). Consciousness in dyschiria. In M. S. Gazzaniga (Hrsg.), *The Cognitive Neurosciences*. MIT Press.

Bisiach, E., Geminiani, G., Berti, A., & Rusconi, M. L. (1990). Perceptual and premotor factors of unilateral neglect. *Neurology*, *40*(8), 1278–1278. https://doi.org/10.1212/WNL.40.8.1278

Bisiach, E., & Geminiani, G. (1991). Anosognosia related to hemiplegia and hemianopia. In G. P. Prigatano & D. L. Schacter (Eds.), *Awareness of deficit after brain injury: Clinical and theoretical issues* (pp. 17–39). Oxford University Press.

Bisiach, E., Vallar, G., Perani, D., Papagno, C., & Berti, A. (1986). Unawareness of disease following lesions of the right hemisphere: Anosognosia for hemiplegia and anosognosia for hemianopia. *Neuropsychologia*, *24*(4), 471–482. https://doi.org/10.1016/0028-3932(86)90092-8

Bivona, U., Ciurli, P., Ferri, G., Fontanelli, T., Lucatello, S., Donvito, T., Villalobos, D., Cellupica, L., Mungiello, F., Sterzo, P., Ferraro, A., Giandotti, E., Lombardi, G., Azicnuda, E., Caltagirone, C., Formisano, R., & Costa, A. (2020). The Self-Awareness Multilevel Assessment Scale, a New Tool for the Assessment of Self-Awareness After Severe Acquired Brain Injury: Preliminary Findings. *Frontiers in Psychology*, *11*, 1732. https://doi.org/10.3389/fpsyg.2020.01732

Blakemore, S.-J. (2003). Deluding the motor system. *Consciousness and Cognition*, *12*(4), 647–655. https://doi.org/10.1016/j.concog.2003.07.001

Blakemore, S.-J., & Frith, C. (2003). Self-awareness and action. *Current Opinion in Neurobiology*, *13*(2), 219–224. https://doi.org/10.1016/S0959-4388(03)00043-6

Blakemore, S.-J., Frith, C. D., & Wolpert, D. M. (1999). Spatio-Temporal Prediction Modulates the Perception of Self-Produced Stimuli. *Journal of Cognitive Neuroscience*, *11*(5), 551–559. https://doi.org/10.1162/089892999563607

Blakemore, S.-J., Wolpert, D. M., & Frith, C. D. (1998). Central cancellation of self-produced tickle sensation. *Nature Neuroscience*, *1*(7), 635–640. https://doi.org/10.1038/2870

Blakemore, S.-J., Wolpert, D. M., & Frith, C. D. (2002). Abnormalities in the awareness of action. *Trends in Cognitive Sciences*, *6*(6), 237–242. https://doi.org/10.1016/S1364-6613(02)01907-1

Blanke, O. (2012). Multisensory brain mechanisms of bodily self-consciousness. *Nature Reviews Neuroscience*, *13*(8), 556–571. https://doi.org/10.1038/nrn3292

Bogod, N. M., Mateer, C. A., & Macdonald, S. W. S. (2003). Self-awareness after traumatic brain injury: A comparison of measures and their relationship to executive functions. *Journal of the International Neuropsychological Society*, *9*(3), 450–458. https://doi.org/10.1017/S1355617703930104

Boksem, M. A. S., Meijman, T. F., & Lorist, M. M. (2006). Mental fatigue, motivation and action monitoring. *Biological Psychology*, *72*(2), 123–132. https://doi.org/10.1016/j.biopsycho.2005.08.007

Bottini, G., Magnani, F. G., Salvato, G., & Gandola, M. (2018). Multiple Dissociations in Patients With Disorders of Body Awareness: Implications for the Study of

Consciousness. *Frontiers in Psychology*, *9*, 2068. https://doi.org/10.3389/fpsyg.2018.02068

Bottini, G., Paulesu, E., Gandola, M., Pia, P., Invernizzi, P., & Berti, A. (2010). Anosognosia for hemiplegia and models of motor control: insights from lesional data. *The Study of Anosognosia (2010)*, pp. 363-379.

Bottini, G., Sedda, A., Ferrè, E. R., Invernizzi, P., Gandola, M., & Paulesu, E. (2009). Productive symptoms in right brain damage. *Current Opinion in Neurology*, *22*(6), 589–593. https://doi.org/10.1097/WCO.0b013e328332c71d

Breier, J. I., Adair, J. C., Gold, M., Fennell, E. B., Gilmore, R. L., & Heilman, K. M. (1995). Dissociation of anosognosia for hemiplegia and aphasia during left-hemisphere anaesthesia. *Neurology*, *45*(1), 65–67. https://doi.org/10.1212/WNL.45.1.65

Byrd, E. M., Jablonski, R. J., & Vance, D. E. (2020). Understanding Anosognosia for Hemiplegia After Stroke. *Rehabilitation Nursing*, *45*(1), 3–15. https://doi.org/10.1097/rnj.0000000000000185

Caggiano, P., & Jehkonen, M. (2018). The 'Neglected' Personal Neglect. *Neuropsychology Review*, *28*(4), 417–435. https://doi.org/10.1007/s11065-018-9394-4

Calvo, M. G., & Eysenck, M. W. (2000). Early vigilance and late avoidance of threat processing: Repressive coping versus low/high anxiety. *Cognition and Emotion*, *14*(6), 763–787. https://doi.org/10.1080/02699930050156627

Canzano, L., Piccardi, L., Bureca, I., & Guariglia, C. (2011). Mirror writing resulting from an egocentric representation disorder: A case report. *Neurocase*, *17*(5), 447–460. https://doi.org/10.1080/13554794.2010.532143

Cappa, S., Sterzi, R., Vallar, G., & Bisiach, E. (1987). Remission of hemineglect and anosognosia during vestibular stimulation. *Neuropsychologia*, *25*(5), 775–782. https://doi.org/10.1016/0028-3932(87)90115-1

Carpenter, K., Berti, A., Oxbury, S., Molyneux, A. J., Bisiach, E., & Oxbury, J. M. (1995). Awareness of and memory for arm weakness during intracarotid sodium amytal testing. *Brain*, *118*(1), 243–251. https://doi.org/10.1093/brain/118.1.243

Cocchini, G., Beschin, N., Cameron, A., Fotopoulou, A., & Della Sala, S. (2009). Anosognosia for motor impairment following left brain damage. *Neuropsychology*, *23*(2), 223–230. https://doi.org/10.1037/a0014266

Cocchini, G., Beschin, N., & Della Sala, S. (2012). Assessing anosognosia: A critical review. *The Official Journal of the Polish Neuropsychological Society*, *10*(3), 419–443. https://doi.org/10.5604/17307503.1023693

Cocchini, G., Beschin, N., & Della Sala, S. (2018). Unawareness for Motor Impairment and Distorted Perception of Task Difficulty. *Journal of the International Neuropsychological Society*, *24*(1), 45–56. https://doi.org/10.1017/S1355617717000662

Cocchini, G., Beschin, N., Fotopoulou, A., & Della Sala, S. (2010). Explicit and implicit anosognosia or upper limb motor impairment. *Neuropsychologia*, *48*(5), 1489–1494. https://doi.org/10.1016/j.neuropsychologia.2010.01.019

Cocchini, G., Gregg, N., Beschin, N., Dean, M., & Sala, S. D. (2010). Vata-L: Visual-Analogue Test Assessing Anosognosia for Language Impairment. *The Clinical Neuropsychologist*, *24*(8), 1379–1399. https://doi.org/10.1080/13854046.2010.524167

Cocchini, G., & S, D. (2010). *Assessing anosognosia for motor and language impairments* (S. 123–144).

Cocchini, G., Scandola, M., Gobbetto, V., Cioffi, M. C., Bartolo, A., Moore, J., & Moro, V. (2022). The 'healthy side' of anosognosia for hemiplegia: Increased sense of agency for the unimpaired limb or motor compensation? *Neuropsychologia*, *177*, 108421. https://doi.org/10.1016/j.neuropsychologia.2022.108421

Cogliano, R., Crisci, C., Conson, M., Grossi, D., & Trojano, L. (2012). Chronic somatoparaphrenia: A follow-up study on two clinical cases. *Cortex*, *48*(6), 758–767. https://doi.org/10.1016/j.cortex.2011.08.008

Cohen, L., Remy, P., Leroy, A., Geny, C., & Degos, J. D. (1991). Minor hemisphere syndrome following left hemispheric lesion in a right handed patient. *Journal of Neurology, Neurosurgery & Psychiatry*, *54*(9), 842–843. https://doi.org/10.1136/jnnp.54.9.842

Corballis, M. C. (2009). The Evolution of Language. *Annals of the New York Academy of Sciences*, *1156*(1), 19–43. https://doi.org/10.1111/j.1749-6632.2009.04423.x

Corbetta, M., Patel, G., & Shulman, G. L. (2008). The Reorienting System of the Human Brain: From Environment to Theory of Mind. *Neuron*, *58*(3), 306–324. https://doi.org/10.1016/j.neuron.2008.04.017

Coslett, H. (2005). Anosognosia and Body Representations Forty Years Later. *Cortex*, *41*(2), 263–270. https://doi.org/10.1016/S0010-9452(08)70912-2

Coslett, H. B., Buxbaum, L. J., & Schwoebel, J. (2008). Accurate Reaching after Active But Not Passive Movements of the Hand: Evidence for Forward Modeling. *Behavioural Neurology*, *19*(3), 117–125. https://doi.org/10.1155/2008/972542

Craig, A. D. (2009). How do you feel — now? The anterior insula and human awareness. *Nature Reviews Neuroscience*, *10*(1), 59–70. https://doi.org/10.1038/nrn2555

Creque, C., & Kolakowsky-Hayner, S. A. (2017). Wisconsin Card Sorting Test. In J. Kreutzer, J. DeLuca, & B. Caplan (Hrsg.), *Encyclopedia of Clinical Neuropsychology* (S. 1–3). Springer International Publishing. https://doi.org/10.1007/978-3-319-56782-2_1913-2

Crum, R. M., Anthony, J. C., Bassett, S. S., & Folstein, M. F. (1993). Population-based norms for the Mini-Mental State Examination by age and educational level. *JAMA*, *269*(18), 2386–2391.

Cutting, J. (1978). Study of anosognosia. *Journal of Neurology, Neurosurgery & Psychiatry*, *41*(6), 548–555. https://doi.org/10.1136/jnnp.41.6.548

Davidson, M. (2023). Functional Independence Measure. In F. Maggino (Hrsg.), *Encyclopedia of Quality of Life and Well-Being Research* (S. 2612–2615). Springer International Publishing. https://doi.org/10.1007/978-3-031-17299-1_1099

Davies, M., Davies, A. A., & Coltheart, M. (2005). Anosognosia and the Two-factor Theory of Delusions. *Mind and Language*, *20*(2), 209–236. https://doi.org/10.1111/j.0268-1064.2005.00283.x

Decety, J. (1996). The neurophysiological basis of motor imagery. *Behavioural Brain Research*, *77*(1–2), 45–52. https://doi.org/10.1016/0166-4328(95)00225-1

Della Sala, S., Cocchini, G., Beschin, N., & Cameron, A. (2009). Vata-m: Visual-Analogue test assessing Anosognosia for motor impairment. *The Clinical Neuropsychologist*, *23*(3), 406–427. https://doi.org/10.1080/13854040802251393

Desmurget, M., & Sirigu, A. (2009). A parietal-premotor network for movement intention and motor awareness. *Trends in Cognitive Sciences*, *13*(10), 411–419. https://doi.org/10.1016/j.tics.2009.08.001

Di Legge, S., Fang, J., Saposnik, G., & Hachinski, V. (2005). The impact of lesion side on acute stroke treatment. *Neurology*, *65*(1), 81–86. https://doi.org/10.1212/01.wnl.0000167608.94237.aa

Di Plinio, S., Perrucci, M. G., Aleman, A., & Ebisch, S. J. H. (2020). I am Me: Brain systems integrate and segregate to establish a multidimensional sense of self. *NeuroImage*, *205*, 116284. https://doi.org/10.1016/j.neuroimage.2019.116284

D'Imperio, D., Bulgarelli, C., Bertagnoli, S., Avesani, R., & Moro, V. (2017). Modulating anosognosia for hemiplegia: The role of dangerous actions in emergent awareness. *Cortex*, *92*, 187–203. https://doi.org/10.1016/j.cortex.2017.04.009

Dorsaint-Pierre, R., Penhune, V. B., Watkins, K. E., Neelin, P., Lerch, J. P., Bouffard, M., & Zatorre, R. J. (2006). Asymmetries of the planum temporale and Heschl's gyrus: Relationship to language lateralization. *Brain*, *129*(5), 1164–1176. https://doi.org/10.1093/brain/awl055

Dronkers, N. F., & Knight, R. T. (1989). Right-sided neglect in a left-hander: Evidence for reversed hemispheric specialisation of attention capacity. *Neuropsychologia*, *27*(5), 729–735. https://doi.org/10.1016/0028-3932(89)90118-8

Dronkers, N. F., Wilkins, D. P., Van Valin, R. D., Redfern, B. B., & Jaeger, J. J. (2004). Lesion analysis of the brain areas involved in language comprehension. *Cognition*, *92*(1–2), 145–177. https://doi.org/10.1016/j.cognition.2003.11.002

Durkin, M. W., Meador, K. J., Nichols, M. E., Lee, G. P., & Loring, D. W. (1994). Anosognosia and the intracarotid amobarbital procedure (Wada test). *Neurology*, *44*(5), 978–979. https://doi.org/10.1212/wnl.44.5.978

Dywan, C. A., McGlone, J., & Fox, A. (1995). Do intracarotid barbiturate injections offer a way to investigate hemispheric models of anosognosia? *Journal of Clinical and Experimental Neuropsychology*, *17*(3), 431–438. https://doi.org/10.1080/01688639508405134

Enriquez-Geppert, S., Huster, R. J., Scharfenort, R., Mokom, Z. N., Zimmermann, J., & Herrmann, C. S. (2014). Modulation of frontal-midline theta by neurofeedback. *Biological Psychology*, *95*, 59–69. https://doi.org/10.1016/j.biopsycho.2013.02.019

Farrer, C., & Frith, C. D. (2002). Experiencing Oneself vs Another Person as Being the Cause of an Action: The Neural Correlates of the Experience of Agency. *NeuroImage*, *15*(3), 596–603. https://doi.org/10.1006/nimg.2001.1009

Feinberg, T. E. (2000). Illusory limb movements in anosognosia for hemiplegia. *Journal of Neurology, Neurosurgery & Psychiatry*, *68*(4), 511–513. https://doi.org/10.1136/jnnp.68.4.511

Feinberg, T. E., Roane, D. M., Kwan, P. C., Schindler, R. J., & Haber, L. D. (1994). Anosognosia and Visuoverbal Confabulation. *Archives of Neurology*, *51*(5), 468–473. https://doi.org/10.1001/archneur.1994.00540170044015

Finlay, A. Y., & Marks, R. (1979). A history questionnaire for dermatology out-patients. *The British Journal of Dermatology*, *100*(3), 257–266. https://doi.org/10.1111/j.1365-2133.1979.tb06197.x

Fischer, S., Trexler, L. E., & Gauggel, S. (2004). Awareness of activity limitations and prediction of performance in patients with brain injuries and orthopaedic

disorders. *Journal of the International Neuropsychological Society*, *10*(2), 190–199. https://doi.org/10.1017/S1355617704102051

Fleming, J. M., Strong, J., & Ashton, R. (1998). Cluster Analysis of Self-Awareness Levels in Adults with Traumatic Brain Injury and Relationship to Outcome: *Journal of Head Trauma Rehabilitation*, *13*(5), 39–51. https://doi.org/10.1097/00001199-199810000-00006

Fleming, Strong, & Ashton. (2020). Self-Awareness of Deficits Interview (SADI). In R. L. Tate, *A Compendium of Tests, Scales and Questionnaires* (1. Aufl., S. 266–270). Psychology Press. https://doi.org/10.4324/9781003076391-69

Fordyce, D. J., & Roueche, J. R. (1986). Changes in perspectives of disability among patients, staff, and relatives during rehabilitation of brain injury. *Rehabilitation Psychology*, *31*(4), 217–229. https://doi.org/10.1037/h0091549

Formica, C., De Salvo, S., Corallo, F., Latella, D., Mìcchia, K., Bonanno, L., Quartarone, A., & Marino, S. (2022). Case report of anosognosia for hemiplegia: A fMRI study. *Medicine*, *101*(52), e32526. https://doi.org/10.1097/MD.0000000000032526

Fotopoulou, A. (2012). Illusions and delusions in anosognosia for hemiplegia: From motor predictions to prior beliefs. *Brain*, *135*(5), 1344–1346. https://doi.org/10.1093/brain/aws094

Fotopoulou, A. (2014). Time to get rid of the ‘Modular’ in neuropsychology: A unified theory of anosognosia as aberrant predictive coding. *Journal of Neuropsychology*, *8*(1), 1–19. https://doi.org/10.1111/jnp.12010

Fotopoulou, A. (2015). The virtual bodily self: Mentalisation of the body as revealed in anosognosia for hemiplegia. *Consciousness and Cognition*, *33*, 500–510. https://doi.org/10.1016/j.concog.2014.09.018

Fotopoulou, A., Pernigo, S., Maeda, R., Rudd, A., & Kopelman, M. A. (2010). Implicit awareness in anosognosia for hemiplegia: Unconscious interference without conscious re-representation. *Brain*, *133*(12), 3564–3577. https://doi.org/10.1093/brain/awq233

Fotopoulou, A., Rudd, A., Holmes, P., & Kopelman, M. (2009). Self-observation reinstates motor awareness in anosognosia for hemiplegia. *Neuropsychologia*, *47*(5), 1256–1260. https://doi.org/10.1016/j.neuropsychologia.2009.01.018

Fotopoulou, A., Tsakiris, M., Haggard, P., Vagopoulou, A., Rudd, A., & Kopelman, M. (2008). The role of motor intention in motor awareness: An experimental study on anosognosia for hemiplegia. *Brain*, *131*(12), 3432–3442. https://doi.org/10.1093/brain/awn225

Fourneret, P., & Jeannerod, M. (1998). Limited conscious monitoring of motor performance in normal subjects. *Neuropsychologia*, *36*(11), 1133–1140. https://doi.org/10.1016/S0028-3932(98)00006-2

Fowler, E. A., Della Sala, S., Hart, S. R., & McIntosh, R. D. (2018). Over- and underestimation of motor ability after a stroke: Implications for anosognosia. *Neuropsychologia*, *119*, 191–196. https://doi.org/10.1016/j.neuropsychologia.2018.08.007

Fried, I., Katz, A., McCarthy, G., Sass, K., Williamson, P., Spencer, S., & Spencer, D. (1991). Functional organisation of human supplementary motor cortex studied by electrical stimulation. *The Journal of Neuroscience*, *11*(11), 3656–3666. https://doi.org/10.1523/JNEUROSCI.11-11-03656.1991

Frith, C. (2002). Attention to action and awareness of other minds. *Consciousness and Cognition*, *11*(4), 481–487. https://doi.org/10.1016/S1053-8100(02)00022-3

Frith, C. (2005). The self in action: Lessons from delusions of control. *Consciousness and Cognition*, *14*(4), 752–770. https://doi.org/10.1016/j.concog.2005.04.002

Frith, C. D., Blakemore, S.-J., & Wolpert, D. M. (2000). Abnormalities in the awareness and control of action. *Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences*, *355*(1404), 1771–1788. https://doi.org/10.1098/rstb.2000.0734

Gainotti, G. (2012). Unconscious processing of emotions and the right hemisphere. *Neuropsychologia*, *50*(2), 205–218. https://doi.org/10.1016/j.neuropsychologia.2011.12.005

Gainotti, G. (2018). Anosognosia in degenerative brain diseases: The role of the right hemisphere and of its dominance for emotions. *Brain and Cognition*, *127*, 13–22. https://doi.org/10.1016/j.bandc.2018.08.002

Gainotti, G., Azzoni, A., Razzano, C., Lanzillotta, M., Marra, C., & Gasparini, F. (1997). The Post-Stroke depression rating scale: A test specifically devised to investigate affective disorders of stroke patients. *Journal of Clinical and*

Experimental Neuropsychology, *19*(3), 340–356. https://doi.org/10.1080/01688639708403863

Gallagher, S. (2000). Philosophical conceptions of the self: Implications for cognitive science. *Trends in Cognitive Sciences*, *4*(1), 14–21. https://doi.org/10.1016/S1364-6613(99)01417-5

Gandola, M., Bottini, G., Zapparoli, L., Invernizzi, P., Verardi, M., Sterzi, R., Santilli, I., Sberna, M., & Paulesu, E. (2014). The physiology of motor delusions in anosognosia for hemiplegia: Implications for current models of motor awareness. *Consciousness and Cognition*, *24*, 98–112. https://doi.org/10.1016/j.concog.2014.01.001

Gandola, M., Invernizzi, P., Sedda, A., Ferrè, E. R., Sterzi, R., Sberna, M., Paulesu, E., & Bottini, G. (2011). An anatomical account of somatoparaphrenia. *Cortex*, *48*(9), 1165–1178. https://doi.org/10.1016/j.cortex.2011.06.012

Garbarini, F., Rabuffetti, M., Piedimonte, A., Pia, L., Ferrarin, M., Frassinetti, F., Gindri, P., Cantagallo, A., Driver, J., & Berti, A. (2012). 'Moving' a paralysed hand: Bimanual coupling effect in patients with anosognosia for hemiplegia. *Brain*, *135*(5), 1486–1497. https://doi.org/10.1093/brain/aws015

Gasquoine, P. G. (2016). Blissfully unaware: Anosognosia and anosodiaphoria after acquired brain injury. *Neuropsychological Rehabilitation*, *26*(2), 261–285. https://doi.org/10.1080/09602011.2015.1011665

Geschwind, N. (1965). DISCONNEXION SYNDROMES IN ANIMALS AND MAN. *Brain*, *88*(2), 237–237. https://doi.org/10.1093/brain/88.2.237

Ghika-Schmid, F., & Bogousslavsky, J. (1997). Affective Disorders following Stroke. *European Neurology*, *38*(2), 75–81. https://doi.org/10.1159/000113164

Gialanella, B., & Mattioli, F. (1992). Anosognosia and extrapersonal neglect as predictors of functional recovery following right hemisphere stroke. *Neuropsychological Rehabilitation*, *2*(3), 169–178. https://doi.org/10.1080/09602019208401406

Gialanella, B., Monguzzi, V., Santoro, R., & Rocchi, S. (2005). Functional Recovery After Hemiplegia in Patients With Neglect: The Rehabilitative Role of Anosognosia. *Stroke*, *36*(12), 2687–2690. https://doi.org/10.1161/01.STR.0000189627.27562.c0

Gilmore, R. L., Heilman, K. M., Schmidt, R. P., Fennell, E. M., & Quisling, R. (1992). Anosognosia during Wada testing. *Neurology*, *42*(4), 925–925. https://doi.org/10.1212/WNL.42.4.925

Godfrey, H. P. D., Harnett, M. A., Knight, R. G., Marsh, N. V., Kesel, D. A., Partridge, F. M., & Heidi Robertson, R. (2003). Assessing distress in caregivers of people with a traumatic brain injury (TBI): A psychometric study of the Head Injury Behaviour Scale. *Brain Injury*, *17*(5), 427–435. https://doi.org/10.1080/0269905031000066201

Gold, M., Adair, J. C., Jacobs, D. H., & Heilman, K. M. (1994). Anosognosia for hemiplegia: An electrophysiologic investigation of the feed‐forward hypothesis. *Neurology*, *44*(10), 1804–1804. https://doi.org/10.1212/WNL.44.10.1804

Goldbeck, R. (1997). Denial in physical illness. *Journal of Psychosomatic Research*, *43*(6), 575–593. https://doi.org/10.1016/S0022-3999(97)00168-2

Goldberg, E., & Barr, W. B. (1991). Three possible mechanisms of unawareness of deficit. In *Awareness of deficit after brain injury: Clinical and theoretical issues.* (S. 152–175). Oxford University Press.

Gomez-Andres, A., Cunillera, T., Rico, I., Naval-Baudin, P., Camins, A., Fernandez-Coello, A., Gabarrós, A., & Rodriguez-Fornells, A. (2022). The role of the anterior insular cortex in self-monitoring: A novel study protocol with electrical stimulation mapping and functional magnetic resonance imaging. *Cortex*, *157*, 231–244. https://doi.org/10.1016/j.cortex.2022.09.008

Goodale, M. A., Jakobson, L. S., & Keillor, J. M. (1994). Differences in the visual control of pantomimed and natural grasping movements. *Neuropsychologia*, *32*(10), 1159–1178. https://doi.org/10.1016/0028-3932(94)90100-7

Green, J. B., & Hamilton, W. J. (1976). Anosognosia for hemiplegia: Somatosensory evoked potential studies. *Neurology*, *26*(12), 1141–1141. https://doi.org/10.1212/WNL.26.12.1141

Greicius, M. D., Supekar, K., Menon, V., & Dougherty, R. F. (2009). Resting-state functional connectivity reflects structural connectivity in the default mode network. *Cerebral Cortex (New York, N.Y.: 1991)*, *19*(1), 72–78. https://doi.org/10.1093/cercor/bhn059

Guggisberg, A. G., Dalal, S. S., Schnider, A., & Nagarajan, S. S. (2011). The neural basis of event-time introspection. *Consciousness and Cognition*, *20*(4), 1899–1915. https://doi.org/10.1016/j.concog.2011.03.008

Haddaway, N. R., Page, M. J., Pritchard, C. C., & McGuinness, L. A. (2022). PRISMA2020: An R package and Shiny app for producing PRISMA 2020-compliant flow diagrams, with interactivity for optimised digital transparency and Open Synthesis. *Campbell Systematic Reviews*, *18*(2), e1230. https://doi.org/10.1002/cl2.1230

Haggard, P. (2005). Conscious intention and motor cognition. *Trends in Cognitive Sciences*, *9*(6), 290–295. https://doi.org/10.1016/j.tics.2005.04.012

Haggard, P. (2017). Sense of agency in the human brain. *Nature Reviews Neuroscience*, *18*(4), 196–207. https://doi.org/10.1038/nrn.2017.14

Haggard, P. (2019). The Neurocognitive Bases of Human Volition. *Annual Review of Psychology*, *70*(1), 9–28. https://doi.org/10.1146/annurev-psych-010418-103348

Haggard, P., & Eimer, M. (1999). On the relation between brain potentials and the awareness of voluntary movements. *Experimental Brain Research*, *126*(1), 128–133. https://doi.org/10.1007/s002210050722

Haggard, P., Rossetti, Y., & Kawato, M. (1993). *Sensorimotor Foundations of Higher Cognition*. Oxford University Press. https://doi.org/10.1093/acprof:oso/9780199231447.001.0001

Hamilton, M. (1959). THE ASSESSMENT OF ANXIETY STATES BY RATING. *British Journal of Medical Psychology*, *32*(1), 50–55. https://doi.org/10.1111/j.2044-8341.1959.tb00467.x

Hamilton, M. (1960). A RATING SCALE FOR DEPRESSION. *Journal of Neurology, Neurosurgery & Psychiatry*, *23*(1), 56–62. https://doi.org/10.1136/jnnp.23.1.56

Hartman-Maeir, A., Soroker, N., & Katz, N. (2001). Anosognosia for Hemiplegia in Stroke Rehabilitation. *Neurorehabilitation and Neural Repair*, *15*(3), 213–222. https://doi.org/10.1177/154596830101500309

Hartman-Maeir, A., Soroker, N., Oman, S. D., & Katz, N. (2003). Awareness of disabilities in stroke rehabilitation—A clinical trial. *Disability and Rehabilitation*, *25*(1), 35–44.

Heilman, K. M. (2011). Anosognosia. In J. S. Kreutzer, J. DeLuca, & B. Caplan (Hrsg.), *Encyclopedia of Clinical Neuropsychology* (S. 179–183). Springer New York. https://doi.org/10.1007/978-0-387-79948-3_1264

Heilman, K. M., Barrett, A. M., & Adair, J. C. (1998). Possible mechanisms of anosognosia: A defect in self–awareness. *Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences*, *353*(1377), 1903–1909. https://doi.org/10.1098/rstb.1998.0342

Hibbard, M. R., Gordon, W. A., Stein, P. N., Grober, S., & Sliwinski, M. (1992). Awareness of disability in patients following stroke. *Rehabilitation Psychology*, *37*(2), 103–120. https://doi.org/10.1037/h0079098

Hier, D. B., Mondlock, J., & Caplan, L. R. (1983). Behavioural abnormalities after right hemisphere stroke. *Neurology*, *33*(3), 337–337. https://doi.org/10.1212/WNL.33.3.337

Hochstenbach, J., Prigatano, G., & Mulder, T. (2005). Patients' and Relatives' Reports of Disturbances 9 Months After Stroke: Subjective Changes in Physical Functioning, Cognition, Emotion, and Behavior. *Archives of Physical Medicine and Rehabilitation*, *86*(8), 1587–1593. https://doi.org/10.1016/j.apmr.2004.11.050

House, A., & Hodges, J. (1988). Persistent denial of handicap after infarction of the right basal ganglia: A case study. *Journal of Neurology, Neurosurgery & Psychiatry*, *51*(1), 112–115. https://doi.org/10.1136/jnnp.51.1.112

Invernizzi, P., Gandola, M., Romano, D., Zapparoli, L., Bottini, G., & Paulesu, E. (2013). What is Mine? Behavioural and Anatomical Dissociations between Somatoparaphrenia and Anosognosia for Hemiplegia. *Behavioural Neurology*, *26*(1–2), 139–150. https://doi.org/10.1155/2013/548467

Ito, M. (1970). Neurophysiological aspects of the cerebellar motor control system. *International Journal of Neurology*, *7*(2), 162–176.

Jeannerod, M., & Frak, V. (1999). Mental imaging of motor activity in humans. *Current Opinion in Neurobiology*, *9*(6), 735–739. https://doi.org/10.1016/S0959-4388(99)00038-0

Jehkonen, M., Ahonen, J.-P., Dastidar, P., Laippala, P., & Vilkki, J. (2000). Unawareness of deficits after right hemisphere stroke: Double-dissociations of

anosognosia: Unawareness of deficits after right hemisphere stroke. *Acta Neurologica Scandinavica*, *102*(6), 378–384. https://doi.org/10.1034/j.1600-0404.2000.102006378.x

Jehkonen, M., Laihosalo, M., & Kettunen, J. (2006). Anosognosia after stroke: Assessment, occurrence, subtypes and impact on functional outcome reviewed. *Acta Neurologica Scandinavica*, *114*(5), 293–306. https://doi.org/10.1111/j.1600-0404.2006.00723.x

Jenkinson, P. M., Edelstyn, N. M. J., Drakeford, J. L., & Ellis, S. J. (2009). Reality monitoring in anosognosia for hemiplegia. *Consciousness and Cognition*, *18*(2), 458–470. https://doi.org/10.1016/j.concog.2008.12.005

Jenkinson, P. M., Edelstyn, N. M. J., & Ellis, S. J. (2009). Imagining the impossible: Motor representations in anosognosia for hemiplegia. *Neuropsychologia*, *47*(2), 481–488. https://doi.org/10.1016/j.neuropsychologia.2008.10.004

Jenkinson, P. M., & Fotopoulou, A. (2010). Motor awareness in anosognosia for hemiplegia: Experiments at last! *Experimental Brain Research*, *204*(3), 295–304. https://doi.org/10.1007/s00221-009-1929-8

Jenkinson, P. M., & Fotopoulou, A. (2014). Understanding Babinski's anosognosia: 100 years later. *Cortex*, *61*, 1–4. https://doi.org/10.1016/j.cortex.2014.10.005

Jenkinson, P. M., Moro, V., & Fotopoulou, A. (2018). Definition: Asomatognosia. *Cortex*, *101*, 300–301. https://doi.org/10.1016/j.cortex.2018.02.001

Jenkinson, P. M., Preston, C., & Ellis, S. J. (2011). Unawareness after stroke: A review and practical guide to understanding, assessing, and managing anosognosia for hemiplegia. *Journal of Clinical and Experimental Neuropsychology*, *33*(10), 1079–1093. https://doi.org/10.1080/13803395.2011.596822

Kaplan, R. (1993). The role of nature in the context of the workplace. *Landscape and Urban Planning*, *26*(1–4), 193–201. https://doi.org/10.1016/0169-2046(93)90016-7

Kaplan-Solms, K., Solms, M., & Pfeffer, A. Z. (2018). *Clinical Studies in Neuro-Psychoanalysis: Introduction to a Depth Neuropsychology* (2. Aufl.). Routledge. https://doi.org/10.4324/9780429472992

Karnath, H.-O., Baier, B., & Nägele, T. (2005). Awareness of the Functioning of One's Own Limbs Mediated by the Insular Cortex? *The Journal of Neuroscience*, *25*(31), 7134–7138. https://doi.org/10.1523/JNEUROSCI.1590-05.2005

Katz, N., Fleming, J., Keren, N., Lightbody, S., & Hartman-Maeir, A. (2002). Unawareness and/or Denial of Disability: Implications for Occupational Therapy Intervention. *Canadian Journal of Occupational Therapy*, *69*(5), 281–292. https://doi.org/10.1177/000841740206900504

Katz, S. (1983). Assessing Self‐maintenance: Activities of Daily Living, Mobility, and Instrumental Activities of Daily Living. *Journal of the American Geriatrics Society*, *31*(12), 721–727. https://doi.org/10.1111/j.1532-5415.1983.tb03391.x

Kinsbourne, M., & Warrington, E. K. (1963). Jargon aphasia. *Neuropsychologia*, *1*(1), 27–37. https://doi.org/10.1016/0028-3932(63)90010-1

Kirsch, L. P., Mathys, C., Papadaki, C., Talelli, P., Friston, K., Moro, V., & Fotopoulou, A. (2021). Updating beliefs beyond the here-and-now: The counter-factual self in anosognosia for hemiplegia. *Brain Communications*, *3*(2), fcab098. https://doi.org/10.1093/braincomms/fcab098

Knecht, S. (2000). Handedness and hemispheric language dominance in healthy humans. *Brain*, *123*(12), 2512–2518. https://doi.org/10.1093/brain/123.12.2512

Kortte, K. B., McWhorter, J. W., Pawlak, M. A., Slentz, J., Sur, S., & Hillis, A. E. (2015). Anosognosia for hemiplegia: The contributory role of right inferior frontal gyrus. *Neuropsychology*, *29*(3), 421–432. https://doi.org/10.1037/neu0000135

Kortte, K. B., Wegener, S. T., & Chwalisz, K. (2003). Anosognosia and denial: Their relationship to coping and depression in acquired brain injury. *Rehabilitation Psychology*, *48*(3), 131–136. https://doi.org/10.1037/0090-5550.48.3.131

Kortte, K., & Hillis, A. E. (2009). Recent advances in the understanding of neglect and anosognosia following right hemisphere stroke. *Current Neurology and Neuroscience Reports*, *9*(6), 459–465. https://doi.org/10.1007/s11910-009-0068-8

Lamar, M., Swenson, R., Penney, D. L., Kaplan, E., & Libon, D. J. (2018). Digit Span. In J. S. Kreutzer, J. DeLuca, & B. Caplan (Hrsg.), *Encyclopedia of Clinical*

Neuropsychology (S. 1154–1160). Springer International Publishing. https://doi.org/10.1007/978-3-319-57111-9_1288

Langer, K. G. (2009). Babinski's Anosognosia for Hemiplegia in Early Twentieth-Century French Neurology. *Journal of the History of the Neurosciences*, *18*(4), 387–405. https://doi.org/10.1080/09647040802537064

Langer, K. G., & Bogousslavsky, J. (2020). The Merging Tracks of Anosognosia and Neglect. *European Neurology*, *83*(4), 438–446. https://doi.org/10.1159/000510397

Langer, K. G., & Levine, D. N. (2014). Babinski, J. (1914). Contribution to the Study of the Mental Disorders in Hemiplegia of Organic Cerebral Origin (Anosognosia). Translated by K.G. Langer & D.N. Levine. *Cortex*, *61*, 5–8. https://doi.org/10.1016/j.cortex.2014.04.019

Leritz, E., Loftis, C., Crucian, G., Friedman, W., & Bowers, D. (2004). Self-Awareness of Deficits in Parkinson Disease. *The Clinical Neuropsychologist*, *18*(3), 352–361. https://doi.org/10.1080/1385404049052412

Levine, D. N. (1990). Unawareness of visual and sensorimotor defects: A hypothesis. *Brain and Cognition*, *13*(2), 233–281. https://doi.org/10.1016/0278-2626(90)90052-P

Levine, D. N., Calvanio, R., & Rinn, W. E. (1991). The pathogenesis of anosognosia for hemiplegia. *Neurology*, *41*(11), 1770–1770. https://doi.org/10.1212/WNL.41.11.1770

Levine, J., Warrenburg, S., Kerns, R., Schwartz, G., Delaney, R., Fontana, A., Gradman, A., Smith, S., Allen, S., & Cascione, R. (1987). The role of denial in recovery from coronary heart disease.: *Psychosomatic Medicine*, *49*(2), 109–117. https://doi.org/10.1097/00006842-198703000-00001

Libet, B., Gleason, C. A., Wright, E. W., & Pearl, D. K. (1983). TIME OF CONSCIOUS INTENTION TO ACT IN RELATION TO ONSET OF CEREBRAL ACTIVITY (READINESS-POTENTIAL): THE UNCONSCIOUS INITIATION OF A FREELY VOLUNTARY ACT. *Brain*, *106*(3), 623–642. https://doi.org/10.1093/brain/106.3.623

Lindell, A. B., Jalas, M. J., Tenovuo, O., Brunila, T., Voeten, M. J. M., & Hämäläinen, H. (2007). Clinical Assessment of Hemispatial Neglect: Evaluation of Different Measures and Dimensions. *The Clinical Neuropsychologist*, *21*(3), 479–497. https://doi.org/10.1080/13854040600630061

Lu, L. H. (2000). Dissociation of anosognosia and phantom movement during the Wada test. *Journal of Neurology, Neurosurgery & Psychiatry*, *69*(6), 820–823. https://doi.org/10.1136/jnnp.69.6.820

Lu, L. H., Barrett, A. M., Schwartz, R. L., Cibula, J. E., Gilmore, R. L., Uthman, B. M., & Heilman, K. M. (1997). Anosognosia and confabulation during the Wada test. *Neurology*, *49*(5), 1316–1322. https://doi.org/10.1212/WNL.49.5.1316

Maier, F., Williamson, K. L., Tahmasian, M., Rochhausen, L., Ellereit, A. L., Prigatano, G. P., Kracht, L., Tang, C. C., Herz, D. M., Fink, G. R., Timmermann, L., & Eggers, C. (2016). Behavioural and neuroimaging correlates of impaired

self-awareness of hypo- and hyperkinesia in Parkinson's disease. *Cortex*, *82*, 35–47. https://doi.org/10.1016/j.cortex.2016.05.019

Marcel, A., Tegner, R., & Nimmosmith, I. (2004). Anosognosia for Plegia: Specificity, Extension, Partiality and Disunity of Bodily Unawareness. *Cortex*, *40*(1), 19–40. https://doi.org/10.1016/S0010-9452(08)70919-5

Marien, M. R., Colpaert, F. C., & Rosenquist, A. C. (2004). Noradrenergic mechanisms in neurodegenerative diseases: A theory. *Brain Research Reviews*, *45*(1), 38–78. https://doi.org/10.1016/j.brainresrev.2004.02.002

Marin, R. S., Biedrzycki, R. C., & Firinciogullari, S. (2014). *Apathy Evaluation Scale* [dataset]. https://doi.org/10.1037/t28570-000

Marková, I. S., & Berrios, G. E. (2014). The construction of anosognosia: History and implications. *Cortex*, *61*, 9–17. https://doi.org/10.1016/j.cortex.2014.09.011

McGlynn, S. M., & Schacter, D. L. (1989). Unawareness of deficits in neuropsychological syndromes. *Journal of Clinical and Experimental Neuropsychology*, *11*(2), 143–205. https://doi.org/10.1080/01688638908400882

Miall, R. C., Weir, D. J., Wolpert, D. M., & Stein, J. F. (1993). Is the Cerebellum a Smith Predictor? *Journal of Motor Behavior*, *25*(3), 203–216. https://doi.org/10.1080/00222895.1993.9942050

Miall, R. C., & Wolpert, D. M. (1996). Forward Models for Physiological Motor Control. *Neural Networks*, *9*(8), 1265–1279. https://doi.org/10.1016/S0893-6080(96)00035-4

Mograbi, D. C., & Morris, R. G. (2013). Implicit awareness in anosognosia: Clinical observations, experimental evidence, and theoretical implications. *Cognitive Neuroscience*, *4*(3–4), 181–197. https://doi.org/10.1080/17588928.2013.833899

Mograbi, D. C., & Morris, R. G. (2018). Anosognosia. *Cortex*, *103*, 385–386. https://doi.org/10.1016/j.cortex.2018.04.001

Monai, E., Bernocchi, F., Bisio, M., Bisogno, A. L., Salvalaggio, A., & Corbetta, M. (2020). Multiple Network Disconnection in Anosognosia for Hemiplegia. *Frontiers in Systems Neuroscience*, *14*, 21. https://doi.org/10.3389/fnsys.2020.00021

Moore, J. W. (2016). What Is the Sense of Agency and Why Does it Matter? *Frontiers in Psychology*, *7*. https://doi.org/10.3389/fpsyg.2016.01272

Monakow, C. (1914). *Die Lokalisation im Grosshirn und der Abbau der Funktion durch kortikale Herde*. JF Bergmann.

Morin, A. (2017). The “self-awareness–anosognosia” paradox explained: How can one process be associated with activation of, and damage to, opposite sides of the brain? *Laterality: Asymmetries of Body, Brain and Cognition*, *22*(1), 105–119. https://doi.org/10.1080/1357650X.2016.1173049

Morin, C., Pradat-Diehl, P., Robain, G., Bensalah, Y., & Perrigot, M. (2003). Stroke Hemiplegia and Specular Image: Lessons From Self-Portraits. *The International Journal of Aging and Human Development*, *56*(1), 1–41. https://doi.org/10.2190/F0G2-GW5C-4WG0-KBWL

Moro, V. (2013). The interaction between implicit and explicit awareness in anosognosia: Emergent awareness. *Cognitive Neuroscience*, *4*(3–4), 199–200. https://doi.org/10.1080/17588928.2013.853656

Moro, V., Besharati, S., Scandola, M., Bertagnoli, S., Gobbetto, V., Ponzo, S., Bulgarelli, C., Fotopoulou, A., & Jenkinson, P. M. (2021). The Motor Unawareness Assessment (MUNA): A new tool for the assessment of Anosognosia for hemiplegia. *Journal of Clinical and Experimental Neuropsychology*, *43*(1), 91–104. https://doi.org/10.1080/13803395.2021.1876842

Moro, V., Pernigo, S., Tsakiris, M., Avesani, R., Edelstyn, N. M. J., Jenkinson, P. M., & Fotopoulou, A. (2016). Motor versus body awareness: Voxel-based lesion analysis in anosognosia for hemiplegia and somatoparaphrenia following right hemisphere stroke. *Cortex*, *83*, 62–77. https://doi.org/10.1016/j.cortex.2016.07.001

Moro, V., Pernigo, S., Zapparoli, P., Cordioli, Z., & Aglioti, S. M. (2011). Phenomenology and neural correlates of implicit and emergent motor awareness in patients with anosognosia for hemiplegia. *Behavioural Brain Research*, *225*(1), 259–269. https://doi.org/10.1016/j.bbr.2011.07.010

Moro, V., Scandola, M., Bulgarelli, C., Avesani, R., & Fotopoulou, A. (2015). Error-based training and emergent awareness in anosognosia for hemiplegia. *Neuropsychological Rehabilitation*, *25*(4), 593–616. https://doi.org/10.1080/09602011.2014.951659

Morris, R. G., & Mograbi, D. C. (2013). Anosognosia, autobiographical memory and self knowledge in Alzheimer's disease. *Cortex*, *49*(6), 1553–1565. https://doi.org/10.1016/j.cortex.2012.09.006

Naito, E., Kochiyama, T., Kitada, R., Nakamura, S., Matsumura, M., Yonekura, Y., & Sadato, N. (2002). Internally Simulated Movement Sensations during Motor Imagery Activate Cortical Motor Areas and the Cerebellum. *The Journal of Neuroscience*, *22*(9), 3683–3691. https://doi.org/10.1523/JNEUROSCI.22-09-03683.2002

Nardone, I. B., Ward, R., Fotopoulou, A., & Turnbull, O. H. (2008). Attention and Emotion in Anosognosia: Evidence of Implicit Awareness and Repression? *Neurocase*, *13*(5–6), 438–445. https://doi.org/10.1080/13554790701881749

Nathanson, M. (1952). DENIAL OF ILLNESS: Its Occurrence in One Hundred Consecutive Cases of Hemiplegia. *A.M.A. Archives of Neurology & Psychiatry*, *68*(3), 380. https://doi.org/10.1001/archneurpsyc.1952.02320210090010

Nimmo-Smith, I. (2005). A diagnostic test of unawareness of bilateral motor task abilities in anosognosia for hemiplegia. *Journal of Neurology, Neurosurgery & Psychiatry*, *76*(8), 1167–1169. https://doi.org/10.1136/jnnp.2004.053744

Nurmi (Née Laihosalo), M. E., & Jehkonen, M. (2014). Assessing anosognosias after stroke: A review of the methods used and developed over the past 35 years. *Cortex*, *61*, 43–63. https://doi.org/10.1016/j.cortex.2014.04.008

Nyberg, L., Habib, R., McIntosh, A. R., & Tulving, E. (2000). Reactivation of encoding-related brain activity during memory retrieval. *Proceedings of the National Academy of Sciences*, *97*(20), 11120–11124. https://doi.org/10.1073/pnas.97.20.11120

Orfei, M. D., Assogna, F., Pellicano, C., Pontieri, F. E., Caltagirone, C., Pierantozzi, M., Stefani, A., & Spalletta, G. (2018). Anosognosia for cognitive and behavioural symptoms in Parkinson's disease with mild dementia and mild cognitive impairment: Frequency and neuropsychological/neuropsychiatric correlates. *Parkinsonism & Related Disorders*, *54*, 62–67. https://doi.org/10.1016/j.parkreldis.2018.04.015

Orfei, M. D., Caltagirone, C., & Spalletta, G. (2009). The Evaluation of Anosognosia in Stroke Patients. *Cerebrovascular Diseases*, *27*(3), 280–289. https://doi.org/10.1159/000199466

Orfei, M. D., Robinson, R. G., Prigatano, G. P., Starkstein, S., Rusch, N., Bria, P., Caltagirone, C., & Spalletta, G. (2007). Anosognosia for hemiplegia after stroke is a multifaceted phenomenon: A systematic review of the literature. *Brain*, *130*(12), 3075–3090. https://doi.org/10.1093/brain/awm106

Ownsworth, T., & Clare, L. (2006). The association between awareness deficits and rehabilitation outcome following acquired brain injury. *Clinical Psychology Review*, *26*(6), 783–795. https://doi.org/10.1016/j.cpr.2006.05.003

Ownsworth, T. L., McFarland, K., & Young, R. McD. (2000). Development and Standardization of the Self-Regulation Skills Interview (SRSI): A New Clinical Assessment Tool for Acquired Brain Injury. *The Clinical Neuropsychologist*, *14*(1), 76–92. https://doi.org/10.1076/1385-4046(200002)14:1;1-8;FT076

Pacella, V., Foulon, C., Jenkinson, P. M., Scandola, M., Bertagnoli, S., Avesani, R., Fotopoulou, A., Moro, V., & Thiebaut De Schotten, M. (2019). Anosognosia for hemiplegia as a tripartite disconnection syndrome. *eLife*, *8*, e46075. https://doi.org/10.7554/eLife.46075

Pacella, V., & Moro, V. (2022a). Anatomy and Disorders of Motor Awareness. In *Encyclopedia of Behavioral Neuroscience, 2nd edition* (S. 298–307). Elsevier. https://doi.org/10.1016/B978-0-12-819641-0.00009-8

Pacella, V., & Moro, V. (2022b). Motor awareness: A model based on neurological syndromes. *Brain Structure and Function*, *227*(9), 3145–3160. https://doi.org/10.1007/s00429-022-02558-y

Padovani, A., Pantano, P., Frontoni, M., Iacoboni, M., Di Piero, V., & Lenzi, G. L. (1992). Reversed laterality of cerebral functions in a non-right-hander: Neuropsychological and spect findings in a case of ‘atypical’ dominance. *Neuropsychologia*, *30*(1), 81–89. https://doi.org/10.1016/0028-3932(92)90016-F

Pascasio, A. (1963). Behavioural Changes in Patients Following Strokes. *Physical Therapy*, *43*(2), 143–143. https://doi.org/10.1093/ptj/43.2.143b

Patterson, J. (2011). F-A-S Test. In J. S. Kreutzer, J. DeLuca, & B. Caplan (Hrsg.), *Encyclopedia of Clinical Neuropsychology* (S. 1024–1026). Springer New York. https://doi.org/10.1007/978-0-387-79948-3_886

Pedersen, P. M., Jorgensen, H. S., Nakayama, H., Raaschou, H. O., & Olsen, T. S. (1996). Frequency, Determinants, and Consequences of Anosognosia in Acute Stroke. *Neurorehabilitation and Neural Repair*, *10*(4), 243–250. https://doi.org/10.1177/154596839601000404

Pia, L., Neppi-Modona, M., Ricci, R., & Berti, A. (2004). The Anatomy of Anosognosia for Hemiplegia: A Meta-Analysis. *Cortex*, *40*(2), 367–377. https://doi.org/10.1016/S0010-9452(08)70131-X

Polejaeva, E., & Woods, A. J. (2017). Behavioral Inattention Test (BIT). In J. Kreutzer, J. DeLuca, & B. Caplan (Hrsg.), *Encyclopedia of Clinical Neuropsychology* (S. 1–3). Springer International Publishing. https://doi.org/10.1007/978-3-319-56782-2_9134-1

Preston, C., Jenkinson, P. M., & Newport, R. (2010). Anosognosia for hemiplegia as a global deficit in motor awareness: Evidence from the non-paralysed limb. *Neuropsychologia*, *48*(12), 3443–3450. https://doi.org/10.1016/j.neuropsychologia.2010.07.027

Preston, C., & Newport, R. (2008). Misattribution of movement agency following right parietal TMS. *Social Cognitive and Affective Neuroscience*, *3*(1), 26–32. https://doi.org/10.1093/scan/nsm036

Prigatano, G. (2012). Anosognosia, denial, and other disorders of phenomenological experience. *The Official Journal of the Polish Neuropsychological Society*, *10*(3), 371–384. https://doi.org/10.5604/17307503.1023676

Prigatano, G., & Morrone-Strupinsky, J. (2010). Management and rehabilitation of persons with anosognosia and impaired self-awareness. *The Study of Anosognosia*, 495–516.

Prigatano, G. P. (1996). Neuropsychological rehabilitation after brain injury: Scientific and professional issues. *Journal of Clinical Psychology in Medical Settings*, *3*(1), 1–10. https://doi.org/10.1007/BF01989285

Prigatano, G. P. (1999). *Principles of neuropsychological Rehabilitation*. Oxford University PressNew York, NY. https://doi.org/10.1093/oso/9780195081435.001.0001

Prigatano, G. P. (2014). Anosognosia and patterns of impaired self-awareness observed in clinical practice. *Cortex*, *61*, 81–92. https://doi.org/10.1016/j.cortex.2014.07.014

Prigatano, G. P., & Klonoff, P. S. (1998). A Clinician's Rating Scale for Evaluating Impaired. Self-Awareness and Denial of Disability After Brain Injury. *The Clinical Neuropsychologist*, *12*(1), 56–67. https://doi.org/10.1076/clin.12.1.56.1721

Pyasik, M., Scandola, M., & Moro, V. (2022). Electrophysiological correlates of action monitoring in brain-damaged patients: A systematic review. *Neuropsychologia*, *174*, 108333. https://doi.org/10.1016/j.neuropsychologia.2022.108333

Ramachandran, V. S. (1995). Anosognosia in Parietal Lobe Syndrome. *Consciousness and Cognition*, *4*(1), 22–51. https://doi.org/10.1006/ccog.1995.1002

Ramachandran, V. S., & Blakeslee, S. (1998). *Phantoms in the brain: Probing the mysteries of the human mind* (1st ed). William Morrow.

Rasmussen, T., & Milner, B. (1977). THE ROLE OF EARLY LEFT‐BRAIN INJURY IN DETERMINING LATERALIZATION OF CEREBRAL SPEECH FUNCTIONS. *Annals of the New York Academy of Sciences*, *299*(1), 355–369. https://doi.org/10.1111/j.1749-6632.1977.tb41921.x

Raven, J. C. (2012). *CPM Raven's Coloured Progressive Matrices* [dataset]. https://doi.org/10.1037/t10910-000

Rizzolatti, G., Luppino, G., & Matelli, M. (1998). The organisation of the cortical motor system: New concepts. *Electroencephalography and Clinical Neurophysiology*, *106*(4), 283–296. https://doi.org/10.1016/S0013-4694(98)00022-4

Romano, D., Bottini, G., & Maravita, A. (2013). Perceptual effects of the mirror box training in normal subjects. *Restorative Neurology and Neuroscience*, *31*(4), 373–386. https://doi.org/10.3233/RNN-120273

Romano, D., Gandola, M., Bottini, G., & Maravita, A. (2014). Arousal responses to noxious stimuli in somatoparaphrenia and anosognosia: Clues to body awareness. *Brain*, *137*(4), 1213–1223. https://doi.org/10.1093/brain/awu009

Romano, D., & Maravita, A. (2019). The dynamic nature of the sense of ownership after brain injury. Clues from asomatognosia and somatoparaphrenia. *Neuropsychologia*, *132*, 107119. https://doi.org/10.1016/j.neuropsychologia.2019.107119

Ronchi, R., Rode, G., Cotton, F., Farnè, A., Rossetti, Y., & Jacquin-Courtois, S. (2013). Remission of anosognosia for right hemiplegia and neglect after caloric vestibular stimulation. *Restorative Neurology and Neuroscience*, *31*(1), 19–24. https://doi.org/10.3233/RNN-120236

Saj, A., Vocat, R., & Vuilleumier, P. (2014). Action-monitoring impairment in anosognosia for hemiplegia. *Cortex*, *61*, 93–106. https://doi.org/10.1016/j.cortex.2014.10.017

Schacter, D. L. (1990). Toward a cognitive neuropsychology of awareness: Implicit knowledge and anosognosia. *Journal of Clinical and Experimental Neuropsychology*, *12*(1), 155–178. https://doi.org/10.1080/01688639008400962

Schellig, D. (2012). *CORSI Corsi Block-Tapping-Test* [dataset]. https://doi.org/10.1037/t10909-000

Schilder, P. (2013). *The Image and Appearance of the Human Body* (0 Aufl.). Routledge. https://doi.org/10.4324/9781315010410

Seghezzi, S., Giannini, G., & Zapparoli, L. (2019). Neurofunctional correlates of body-ownership and sense of agency: A meta-analytical account of

self-consciousness. *Cortex*, *121*, 169–178. https://doi.org/10.1016/j.cortex.2019.08.018

Seghezzi, S., & Haggard, P. (2022). *Volition and "free will"*. https://doi.org/10.31234/osf.io/e46pb

Serrien, D. J., & Spapé, M. M. (2011). Motor awareness and dissociable levels of action representation. *Neuroscience Letters*, *494*(2), 145–149. https://doi.org/10.1016/j.neulet.2011.02.077

Sestieri, C., Tosoni, A., Mignogna, V., McAvoy, M. P., Shulman, G. L., Corbetta, M., & Romani, G. L. (2014). Memory Accumulation Mechanisms in Human Cortex Are Independent of Motor Intentions. *The Journal of Neuroscience*, *34*(20), 6993–7006. https://doi.org/10.1523/JNEUROSCI.3911-13.2014

Sherer, M., Bergloff, P., Boake, C., High, W., & Levin, E. (1998). The Awareness Questionnaire: Factor structure and internal consistency. *Brain injury : [BI]*, *12*, 63–68. https://doi.org/10.1080/026990598122863

Sherer, M., Hart, T., Nick, T. G., Whyte, J., Thompson, R. N., & Yablon, S. A. (2003). Early impaired self-awareness after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, *84*(2), 168–176. https://doi.org/10.1053/apmr.2003.50045

Sherer, M., Oden, K., Bergloff, P., Levin, E., & High, W. M. (1998). Assessment and treatment of impaired awareness after brain injury: Implications for community re-integration. *NeuroRehabilitation*, *10*(1), 25–37. https://doi.org/10.3233/NRE-1998-10104

Shergill, S. S., Bays, P. M., Frith, C. D., & Wolpert, D. M. (2003). Two Eyes for an Eye: The Neuroscience of Force Escalation. *Science*, *301*(5630), 187–187. https://doi.org/10.1126/science.1085327

Sirigu, A., Grafman, J., Bressler, K., & Sunderland, T. (1991). MULTIPLE REPRESENTATIONS CONTRIBUTE TO BODY KNOWLEDGE PROCESSING: EVIDENCE FROM A CASE OF AUTOTOPAGNOSIA. *Brain*, *114*(1), 629–642. https://doi.org/10.1093/brain/114.1.629

Solms, M. (1995). Is the brain more real than the mind? *Psychoanalytic Psychotherapy*, *9*(2), 107–120. https://doi.org/10.1080/02668739500700121

Spalletta, G., Girardi, P., Caltagirone, C., & Orfei, M. D. (2012). Anosognosia and Neuropsychiatric Symptoms and Disorders in Mild Alzheimer Disease and Mild Cognitive Impairment. *Journal of Alzheimer's Disease*, *29*(4), 761–772. https://doi.org/10.3233/JAD-2012-111886

Spielberger, C. (2021). *State-Trait Anger Expression Inventory*. https://doi.org/10.1037/t29496-000

Spinazzola, L., Pia, L., Folegatti, A., Marchetti, C., & Berti, A. (2008). Modular structure of awareness for sensorimotor disorders: Evidence from anosognosia for hemiplegia and anosognosia for hemianesthesia. *Neuropsychologia*, *46*(3), 915–926. https://doi.org/10.1016/j.neuropsychologia.2007.12.015

Starkstein, S. E., Fedoroff, J. P., Price, T. R., Leiguarda, R., & Robinson, R. G. (1992). Anosognosia in patients with cerebrovascular lesions. A study of

causative factors. *Stroke*, *23*(10), 1446–1453. https://doi.org/10.1161/01.STR.23.10.1446

Steward, K. A., & Kretzmer, T. (2022). Anosognosia in moderate-to-severe traumatic brain injury: A review of prevalence, clinical correlates, and diversity considerations. *The Clinical Neuropsychologist*, *36*(8), 2021–2040. https://doi.org/10.1080/13854046.2021.1967452

Stone, S. P., Halligan, P. W., & Greenwood, R. J. (1993). The Incidence of Neglect Phenomena and Related Disorders in Patients with an Acute Right or Left Hemisphere Stroke. *Age and Ageing*, *22*(1), 46–52. https://doi.org/10.1093/ageing/22.1.46

Stroop, J. R. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology*, *18*(6), 643–662. https://doi.org/10.1037/h0054651

Tagai, K., Nagata, T., Shinagawa, S., & Shigeta, M. (2020). Anosognosia in patients with Alzheimer's disease: Current perspectives. *Psychogeriatrics*, *20*(3), 345–352. https://doi.org/10.1111/psyg.12507

Thomas, A. K., & Loftus, E. F. (2002). Creating bizarre false memories through imagination. *Memory & Cognition*, *30*(3), 423–431. https://doi.org/10.3758/BF03194942

Tsakiris, M., Longo, M. R., & Haggard, P. (2010). Having a body versus moving your body: Neural signatures of agency and body-ownership. *Neuropsychologia*, *48*(9), 2740–2749. https://doi.org/10.1016/j.neuropsychologia.2010.05.021

Turnbull, O. (1999). Phantoms in the Brain: Human Nature and the Architecture of the Mind. *Neuropsychoanalysis*, *1*(2), 269–272. https://doi.org/10.1080/15294145.1999.10773270

Turnbull, O., Evans, C., & Owen, V. (2005). Negative Emotions and Anosognosia. *Cortex*, *41*(1), 67–75. https://doi.org/10.1016/S0010-9452(08)70179-5

Turnbull, O. H., Fotopoulou, A., & Solms, M. (2014). Anosognosia as motivated unawareness: The 'defence' hypothesis revisited. *Cortex*, *61*, 18–29. https://doi.org/10.1016/j.cortex.2014.10.008

Turnbull, O. H., Jones, K., & Reed-Screen, J. (2002). Implicit awareness of deficit in anosognosia? An emotion-based account of denial of deficit. *Neuropsychoanalysis*, *4*(1), 69–87. https://doi.org/10.1080/15294145.2002.10773381

Ullman, M. (1964). DISORDERS OF BODY IMAGE AFTER STROKE. *The American Journal of Nursing*, *64*, 89–91.

Ullsperger, M., & Von Cramon, D. Y. (2001). Subprocesses of Performance Monitoring: A Dissociation of Error Processing and Response Competition Revealed by Event-Related fMRI and ERPs. *NeuroImage*, *14*(6), 1387–1401. https://doi.org/10.1006/nimg.2001.0935

Vallar, G., Bottini, G., & Sterzi, R. (2003). Anosognosia for left-sided motor and sensory deficits, motor neglect, and sensory hemiinattention: Is there a relationship? In *Progress in Brain Research* (Bd. 142, S. 289–301). Elsevier. https://doi.org/10.1016/S0079-6123(03)42020-7

Venneri, A. (2004). Belief and awareness: Reflections on a case of persistent anosognosia. *Neuropsychologia*, *42*(2), 230–238. https://doi.org/10.1016/S0028-3932(03)00171-4

Vocat, R., Saj, A., & Vuilleumier, P. (2013). The riddle of anosognosia: Does unawareness of hemiplegia involve a failure to update beliefs? *Cortex*, *49*(7), 1771–1781. https://doi.org/10.1016/j.cortex.2012.10.009

Vocat, R., Staub, F., Stroppini, T., & Vuilleumier, P. (2010). Anosognosia for hemiplegia: A clinical-anatomical prospective study. *Brain*, *133*(12), 3578–3597. https://doi.org/10.1093/brain/awq297

Vuilleumier, P. (2004). Anosognosia: The Neurology of Beliefs and Uncertainties. *Cortex*, *40*(1), 9–17. https://doi.org/10.1016/S0010-9452(08)70918-3

Vuilleumier, P., & Vocat, R. (2010). Neuroanatomy of impaired body awareness in anosognosia and hysteria: A multi-component account. *Advances in the study of anosognosia*, 359–403.

Wechsler, D. (2012). *Wechsler Adult Intelligence Scale—Fourth Edition* [dataset]. https://doi.org/10.1037/t15169-000

Weinstein, E. A., & Kahn, R. L. (1955). *Denial of illness: Symbolic and physiological aspects.* Charles C Thomas Publisher. https://doi.org/10.1037/11516-000

Weiskrantz, L., Elliott, J., & Darlington, C. (1971). Preliminary Observations on Tickling Oneself. *Nature*, *230*(5296), 598–599. https://doi.org/10.1038/230598a0

Welman, A. J. (1969). RIGHT-SIDED UNILATERAL VISUAL SPATIAL AGNOSIA, ASOMATOGNOSIA AND ANOSOGNOSIA WITH LEFT HEMISPHERE LESIONS. *Brain*, *92*(3), 571–580. https://doi.org/10.1093/brain/92.3.571

Winkens, I., Van Heugten, C. M., Visser-Meily, J. M. A., & Boosman, H. (2014). Impaired Self-Awareness After Acquired Brain Injury: Clinicians' Ratings on Its Assessment and Importance for Rehabilitation. *Journal of Head Trauma Rehabilitation*, *29*(2), 153–156. https://doi.org/10.1097/HTR.0b013e31827d1500

Wolpert, D. M., Diedrichsen, J., & Flanagan, J. R. (2011). Principles of sensorimotor learning. *Nature Reviews Neuroscience*, *12*(12), 739–751. https://doi.org/10.1038/nrn3112

Wolpert, D. M., Ghahramani, Z., & Jordan, M. I. (1995). An Internal Model for Sensorimotor Integration. *Science*, *269*(5232), 1880–1882. https://doi.org/10.1126/science.7569931

Zapparoli, L., Seghezzi, S., Zirone, E., Guidali, G., Tettamanti, M., Banfi, G., Bolognini, N., & Paulesu, E. (2020). How the effects of actions become our own. *Science Advances*, *6*(27). https://doi.org/10.1126/sciadv.aay8301