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Emotional sequelae following acquired brain injury: a narrative review.

Conseguenze emotive a seguito di lesioni cerebrali acquisite: una revisione narrativa della letteratura

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Abstract

Le lesioni cerebrali acquisite (LCA), che comprendono traumi cranici (TBI) e ictus, comportano una rosa di conseguenze emotive che possono incidere profondamente sul funzionamento sociale e sulla qualità della vita dei pazienti. Questa revisione narrativa vuole riassumere le conseguenze emotive più di frequente associate alle LCA, tra cui ansia, depressione, apatia, anedonia, disinibizione, aggressività e deficit nella teoria della mente. La depressione è caratterizzata da pervasivi sentimenti negativi soggettivi e da una sofferenza emotiva, spesso accompagnata da una riduzione della motivazione e da una tendenza al ritiro dalle attività quotidiane. Al contrario, l'ansia può comportare una maggiore sensibilità agli stimoli interni ed esterni, che porta ad una persistente sensazione di tensione e attivazione fisiologica, che può anch'essa influire sulla motivazione. Sebbene apatia e anedonia possano sovrapporsi in parte con cambiamenti della motivazione, sono concetti distinti: l'apatia è definita da indifferenza emotiva e distacco affettivo, mentre l'anedonia si riferisce ad una capacità ridotta di provare piacere o interesse per attività precedentemente gratificanti. La disregolazione emotiva, come l'aumento dell'irritabilità, l'aggressività e i comportamenti impulsivi, può contribuire all'isolamento sociale. La disinibizione può manifestarsi attraverso comportamenti sociali inappropriati, complicando la reintegrazione sociale dei pazienti. Infine, i deficit nella teoria della mente, ovvero nella capacità di comprendere le emozioni e le prospettive altrui, possono ostacolare le interazioni e le relazioni sociali. Questa revisione narrativa della letteratura vuole evidenziare la natura pervasiva e multifattoriale delle conseguenze emotive risultanti dalle LCA. Tali disturbi sono spesso persistenti e complessi, si vuole quindi sottolineare la necessità di una maggiore attenzione e dell'implementazione di programmi di riabilitazione personalizzati per migliorare la qualità della vita dei pazienti.

Parole chiave

Lesione Cerebrale Acquisita; Conseguenze; Ictus; Trauma Cranico; Revisione

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Abstract

Acquired brain injuries (ABIs), including traumatic brain injuries (TBIs) and strokes, frequently result in a wide range of emotional sequelae that profoundly impact patients' quality of life and social functioning. This narrative review examines the emotional consequences that often follow ABIs, including anxiety, depression, apathy, anhedonia, disinhibition, aggression, and deficits in theory of mind. Depression is mainly characterized by pervasive negative subjective feelings and emotional suffering, frequently accompanied by reduced motivation and a tendency to withdraw from daily activities. In contrast, anxiety involves heightened sensitivity to internal and external stimuli, leading to a persistent sense of tension and physiological arousal, which can also affect motivation. While apathy and anhedonia may share some overlap with motivational changes, they are distinct constructs: apathy is defined by emotional indifference and affective detachment, while anhedonia specifically refers to a diminished ability to experience pleasure or interest in previously enjoyable activities. Emotional dysregulation, such as increased irritability, aggression, and impulsive behavior, contributes to interpersonal conflicts and social isolation. Disinhibition can manifest as inappropriate social behaviors, further complicating social reintegration. Additionally, deficits in theory of mind, or the ability to understand others' emotions and perspectives, impair social interactions and relationships. This narrative review aims to highlight the pervasive and multifactored nature of emotional consequences following ABI. These emotional disturbances are often persistent and challenging, underlying the need for increased awareness and tailored rehabilitation programs, to enhance patients' quality of life.

Keywords

Acquired Brain Injury; Consequences; Stroke; Traumatic Brain Injury; Review

Introduction

Acquired Brain Injury (ABI) refers to any form of brain damage that occurs after birth, excluding injuries related to congenital conditions, degenerative diseases, or birth-related trauma. ABI is categorized in two main types: Traumatic Brain Injury (TBI), caused by external physical forces, and Non-Traumatic Brain Injury (Non-TBI), resulting from internal processes such as strokes, tumors, infections, or anoxia. ABI disrupts neuronal structure and function, leading to impairments that vary in severity and duration, ranging from temporary deficits to lifelong disabilities (Goldman et al., 2022).

ABI is currently one of the leading causes of death and disability among young adults and is considered one of the most prevalent neurological disorders worldwide. Epidemiological data estimate a global incidence of 1 per 500 people, with the highest rates occurring in children under 4, adults under 30, and individuals over 65. A recent study of the Italian population from 2012 to 2021 estimates an ABI rate of 77.3% per 100,000 people. The primary causes include domestic accidents (33.1%) and road accidents (17.7%), predominantly involving males aged 15–24 and the elderly (60+) (Cardile et al., 2024). Individuals affected by ABI often face significant challenges reintegrating into society due to both psychological and physiological consequences, including impairments in motor function, language, cognition, sensory processing, and emotional regulation. Cognitive deficits following ABI often include impairments in arousal, attention, concentration, memory, language, and executive functioning. Memory loss may affect both verbal and nonverbal information, making it challenging to recall conversations, instructions, or visual cues. Executive dysfunction typically involves difficulties in planning, organizing, sequencing, and adapting to new tasks, often accompanied by compromised judgment and impulse control (Rao & Lyketsos, 2000).

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Physiologically, ABIs often involve lesions in central pathways that control both simple and complex motor functions. This can result in motor disturbances such as hemiplegia, spasticity, ataxia, and upper motor neuron syndrome, which present with various positive and negative signs affecting both static and dynamic motor skills (Zasler et al., 2012).

Risk factors for ABI are particularly high among males and the elderly, with common mechanisms of injury—including vascular damage, axonal injury, atrophy, and neuronal circuit disruption—shared by both TBI and Non-TBI. Clinical outcomes are influenced by factors such as age, genetics, socioeconomic status, and the specific nature of the injury, which also increases a risk of developing neurodegenerative diseases later in life (Cardile et al., 2024; Goldman et al., 2022). A higher cognitive reserve (CR)—described as the brain's capacity to sustain cognitive performance despite age-related changes or brain injury (Stern et al., 2023)—can mitigate cognitive decline, as it develops through engagement in cognitively stimulating and challenging life experiences (Oliva et al., 2024). Research also indicates that cognitive reserve serves as a protective factor across a variety of neurological conditions, including stroke and traumatic brain injury. These studies underscore the beneficial role of cognitive reserve in improving outcomes across a range of neurodegenerative and injury-related pathologies (Basagni et al., 2023).

Emotional disturbances following ABI are both common and complex, with individuals frequently experiencing disorders such as depression, anxiety, apathy, aggression, and emotional lability. It is essential to distinguish between depression and apathy, as these conditions may initially appear similar and can sometimes be mistakenly classified as the same disorder. The primary point of distinction relies on anhedonia—the inability to experience pleasure—which is a key characteristic of depression but is not necessarily associated with apathy. Apathy, by contrast, is linked to motivational deficits rather than mood disruptions, making it qualitatively different from depression, which typically involves feelings of sadness or hopelessness (Green et al., 2021; Bivona et al., 2019).

Emotional changes following ABI can significantly impact quality of life and present major obstacles to rehabilitation and social reintegration. Although research has historically focused more on cognitive and motor impairments, emotional sequelae have received increasing attention in recent years, due to their critical role in patient outcomes and long-term recovery. Clinicians and healthcare professionals should implement a combination of structured assessments alongside observational methods to effectively identify and monitor emotional and behavioral alterations following ABI. Tools such as the Neuropsychiatric Inventory (NPI, Cummings et al., 1994) and the Personality Assessment Inventory (PAI, Morey et al., 2008) are validated instruments that capture key neurobehavioral and emotional symptoms, including apathy, depression, and aggression. By regularly assessing these symptoms, clinicians can monitor symptom progression over time, allowing for more precise and timely adjustments to treatment plans.

This narrative review aims to summarize the most common emotional sequelae of ABIs, including anxiety, depression, apathy, anhedonia, aggression, theory of mind deficits, and disinhibition.

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Anxiety following ABI

Anxiety is a common and debilitating psychological condition that can arise following an ABI. This disorder can significantly affect patients' quality of life, interfere with the rehabilitation process, and further complicate clinical management. However, its prevalence and manifestations can vary significantly due to differences in research methods, diagnostic criteria, and the specific characteristics of the studied samples.

Anxiety is a common and debilitating consequence of TBI and cerebral stroke. Recognizing the associated risk factors and implementing tailored interventions are critical for enhancing patient recovery. Effective management of anxiety following TBI, or stroke requires a comprehensive and individualized approach, where targeted therapies are crucial for improving quality of life and promoting recovery.

Post-TBI Anxiety

TBI is associated with a range of long-term psychological complications, including anxiety disorders. The prevalence of anxiety disorders among TBI patients is notably high. A recent meta-analysis reported that 17.45% of TBI patients exhibit anxiety symptoms, with a relative risk (RR) of 1.9 compared to individuals without TBI (Dehbozorgi et al., 2024). Another meta-analysis indicated that 11% of TBI patients receive a diagnosis of Generalized Anxiety Disorder (GAD), while 37% report clinically significant levels of anxiety (Osborn et al., 2016). These findings underline the significant and persistent prevalence of anxiety in post-TBI patients, with a peak in symptoms occurring between 2 and 5 years after the injury.

The onset of anxiety after TBI is influenced by several factors. A previous history of anxiety disorders significantly increases the risk of developing anxiety post-TBI, with symptoms tending to persist and negatively impact quality of life (Lamontagne et al., 2022). A more specific study examined the pre-existing psychological variables of TBI patients as factors associated with the development of social anxiety. Among the various investigated variables (locus of control, self-esteem, self-efficacy, etc.), the feeling of stigma was identified as an independent predictor of social anxiety (Curvis et al., 2016). According to Mata-Bermudez et al. (2024), alterations in dopamine levels, its receptors, and the DAT transporter, along with changes in the neuronal circuits of brain areas involved in emotional processing, such as the prefrontal cortex, basal ganglia, and amygdala, are observed after a TBI. These alterations may result from both primary brain damage and the neuroinflammatory and metabolic processes that follow the trauma, suggesting that post-TBI depression and anxiety are multifactorial phenomena linked to a combination of dopaminergic dysfunctions and other neurobiological mechanisms.

One of the main challenges in managing post-TBI anxiety is the lack of consistency in the diagnostic methods and criteria across the literature, which complicates the accurate identification and management of these disorders. For example, Osborn et al. (2016) report significant variability in the prevalence of GAD among patients with TBI, with estimates ranging from 11% to 28%. The choice of diagnostic criteria (DSM-IV, DSM-III-R, ICD-10) substantially impacts these prevalence rates, with the ICD-10 yielding a notably lower prevalence (2%) compared to other

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systems. Additionally, the use of various diagnostic interviews (e.g., SCID-I, MINI, SADS-L) has further contributed to the variability in findings, with prevalence rates ranging from 2% to 28%.

Post-Stroke Anxiety

Among the psychological complications associated with stroke, anxiety is one of the most common conditions that has a substantial impact on both quality of life and recovery. Studies have shown that approximately 25% of stroke survivors experience acute anxiety, and 20% continue to exhibit anxiety symptoms 3-6 months after the event (Barker-Collo, 2007).

Recent studies have identified factors that influence the onset of post-stroke anxiety disorders, such as age, marital status, and education level. Younger patients and those with higher social deprivation scores tend to report higher levels of anxiety compared to older patients (Broomfield et al., 2015). Pre-existing psychological characteristics, such as neuroticism and pre-existing anxiety, are also significant risk factors for developing post-stroke anxiety and depression (Kootker et al., 2016). However, current studies are often limited by small sample sizes and a lack of homogeneity in diagnostic criteria.

An emerging aspect of post-stroke anxiety research concerns the role of sleep duration. A recent study found that a sleep duration of less than 6 hours per night significantly increases the risk of developing anxiety compared to a sleep duration of more than 7 hours. However, no significant differences were found between genders (Liu et al., 2021). These findings suggest the need for further exploration of how sleep quality and quantity may affect the psychological well-being of post-stroke patients.

Post-stroke anxiety can manifest in various forms, including generalized anxiety, panic attacks, and specific phobias. These psychological issues are a complex aspect of post-stroke recovery, with the primary challenge being the variability in assessment tools. The main tools used in the literature, such as the Beck Anxiety Inventory (BAI) (Beck et al., 1988; Barker-Collo, 2007), the State-Trait Anxiety Inventory (STAI) (Spielberger, 1989), and the Hospital Anxiety and Depression Scale (HADS) (Zigmond & Snaith, 1983; Curvis et al., 2016), assess different dimensions of anxiety but often fail to capture the full spectrum of psychological issues faced by stroke patients. Other tools, such as the Social Phobia Inventory (SPIN) (Connor et al., 2020), focus on specific phobias like social anxiety. However, in these cases as well, the patient's response may be influenced by their neurological condition, complicating the diagnosis. These disorders, in addition to being a response to the trauma experienced, can complicate the recovery process by interfering with rehabilitation and increasing the risk of further complications (Chun et al., 2018). Despite substantial rehabilitative interventions aimed at physical recovery, patients' perceived quality of life does not always improve, indicating that psychological support efforts may be insufficient (Ahlsio et al., 1984).

Treatment options for post-stroke anxiety remain limited and often inadequate to meet patients' specific needs. Some therapies, such as CBT, have shown promising results, but their efficacy must be confirmed by larger and better-designed studies. An investigation by Petrova et al. (2012) examined 198 patients with a first cerebral stroke, finding that 71.2% had affective disorders, including generalized anxiety and phobias. This study highlighted the importance of considering

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post-stroke anxiety disorders as relatively independent conditions, with specific risk factors and pathogenesis.

Depression in ABI

Depression is a mood disorder characterized by a persistent sense of sadness and diminished interest in daily activities. The DSM-5 (APA, 2013) classifies depressive disorders into several types: Disruptive Mood Dysregulation Disorder, Major Depressive Disorder, Persistent Depressive Disorder (dysthymia), Premenstrual Dysphoric Disorder, and Depressive Disorder due to Another Medical Condition. All depressive disorders share common symptoms, including feelings of sadness, emptiness, or irritability, along with physical and cognitive changes that significantly affect the individual's ability to function (APA, 2013).

Functional neuroimaging studies have shown that depression is associated with reduced basal metabolism in neocortical structures, particularly the prefrontal and frontal lobes (Sacher et al., 2012), alongside an increase in basal metabolism within the limbic system, including regions like the dorsal raphe nuclei, cingulate cortex, and left prefrontal cortex (Mayberg et al., 1999). Additionally, other studies have identified structural neuroanatomical changes correlated with major depression, such as enlarged lateral ventricles and reduced volumes in areas such as the basal ganglia, thalamus, hippocampus, and frontal lobe (Kempton et al., 2011).

Although depressive disorder diagnoses are established based on specific criteria in the DSM, additional tools are available for identifying and assessing these conditions. Depressive symptoms are usually assessed using tools such as the Beck Depression Inventory-II (BDI-II) and the Montgomery-Åsberg Depression Rating Scale (MADRS). These instruments evaluate key dimensions of depression, with the BDI-II offering a self-reported perspective and the MADRS providing clinician-administered insights. While both tools are widely recognized, their differing emphases—such as the BDI-II's inclusion of somatic and cognitive aspects versus the MADRS's focus on core affective symptoms—may influence reported prevalence rates and severity levels. This highlights the role of measurement choice in shaping study outcomes, particularly in heterogeneous conditions like ABI. (Hudgens et al., 2021; Montgomery et al., 1979).

There is a fair, albeit limited, literature that has explored the relationship between ABI and depression. This is largely due to the fact that early research in this area predominantly focused on the cognitive sequelae of ABI. However, from the 1970s, there has been a shift toward exploring the potential—and unfortunately common—psychopathological consequences of ABI. Among these, depression (diagnosed via the Structured Clinical Interview and the DSM-IV criteria) is one of the most prevalent conditions linked to TBI (Howlett et al., 2022). In a recent article, Iverson and Gardner highlight the well-established link between chronic traumatic encephalopathy (CTE) and various psychiatric disorders, particularly depression. These conditions are often diagnosed, like many affective disorders, using the World Mental Health Survey Initiative Version of the World Health Organization Composite International Diagnostic Interview (WMH-CIDI). This structured diagnostic tool produces diagnoses based on both the International Classification of Diseases, 10th Revision (ICD-10), and the DSM-IV. Their analysis also refers to a series of autopsy studies conducted on populations such as military veterans, professional athletes (including

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American football players and boxers). These studies identified widespread histological alterations in the central nervous system, which strongly correlated with the psychiatric symptoms exhibited by these individuals during their lives (Iverson & Gardner, 2021; Omalu et al., 2011; Baugh et al., 2012). Based on these findings, Montenigro and colleagues proposed a classification system for CTE, categorizing it into four types: behavioral/mood variant, cognitive variant, mixed variant, and dementia variant (Montenigro et al., 2014).

Another study by Bryant and colleagues (2010), on the other hand, further corroborates this association, by examining the prevalence of psychiatric disorders among patients admitted to the emergency room following head trauma. Using the Mini International Neuropsychiatric Interview, version 5.5, a brief structured diagnostic interview based on DSM-IV and ICD-10 criteria, their findings indicated that depression was the most common new-onset disorder, occurring in 16.3% of cases. The authors hypothesized that this incidence may be attributed to direct damage to the frontal lobes and subcortical pathways resulting from the injury.

Nevertheless, the study with the largest sample size was conducted by Choi et al., who collected data from the National Health Insurance Service-National Health Information Database (NHIS-NHID) in South Korea. Their results reported an incidence rate of 34.6% of depression among the North Korean population who have experienced head trauma (Choi et al., 2022). They also noted a higher incidence of depression in male patients, along with an apparent correlation between psychiatric symptomatology and the severity of head trauma.

A recent study conducted by Frank et al. (2022) examined the occurrence of depression following stroke, reporting a prevalence rate ranging from 20% to 60%. This finding establishes depression as the most common psychiatric comorbidity in this population. In this context, Feng and colleagues investigated the potential pathogenesis of post-stroke depression, identifying alterations in the frontal regions of the brain and the basal ganglia. However, they emphasized that this topic requires further investigation, as other etiopathological hypotheses suggest a complex interplay involving hyperactivation of the hypothalamic-pituitary axis, neuroinflammation, and immune system dysregulation (Feng et al., 2014).

Apathy following ABI

Apathy is a reduction in goal-directed behaviors and may result from reduced emotional reactivity, interference with motor planning, and failure to self-initiate actions (Levy et al., 2006).

Neuroimaging studies have found associations between apathy and structural and functional alterations in the frontal lobe, particularly in the anterior cingulate gyrus and also in subregions of the basal ganglia (Paul et al., 2005; Roth et al., 2004).

One of the most used tools for assessing apathy in ABIs is the Apathy Evaluation Scale (AES), which measures severity across self-reported (AES-S), informant-reported (AES-I), and clinician-administered formats (AES-C). This tool underscores the importance of considering how measurement tools affect the interpretation of emotional and motivational deficits in ABI population (Marin et al., 1991). The Apathy Scale (AS), a shortened version of the AES scale, is also widely used (Starkstein et al., 1992).

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Apathy can also be measured using the Dimensional Apathy Scale (DAS), a multidimensional tool. The DAS was designed to specifically assess three neurobehavioral subtypes of apathy through theoretical analysis and targeted item selection, making it the only scale that comprehensively measures these domains. It detects apathetic deficits related to planning, attention, or organization (Executive subscale), emotion integration (Emotional subscale), and self-generation of behavior or cognitive processes (Initiation subscale) (Radakovic et al., 2016).

Apathy following stroke

The pioneering empirical research on apathy in stroke patients has provided compelling evidence of its existence, showing that it may be more prevalent than previously believed. In this study of Starkstein et al. (1993) apathy was measured using the AS, which was administered by the examiner. They found that approximately 23% of a consecutive series of stroke survivors exhibited signs of apathy. Among those identified as apathetic, nearly half also displayed symptoms of depression. Additionally, this study highlighted that patients with apathy were significantly more likely to experience greater cognitive impairments and deficits in activities of daily living compared to those without apathy. In a study by Brodaty et al. (2005), apathy (measured with the AES) was diagnosed in 26.7% of stroke patients, in contrast to 5.4% in the control group. Subsequently, it has been estimated that the prevalence of apathy (assessed with the AS, using a cut-off score of 12) following a stroke, ranges from 20% to 25%, often correlating with cognitive decline and a chronic trajectory marked by progressive functional deterioration (Jorge et al., 2010). A recent study suggested that apathy after stroke is a common neuropsychiatric consequence, affecting approximately one in three patients. Furthermore, it has been estimated that the prevalence of pure apathy, without concurrent depression, is twice as high as that of pure depression, without accompanying apathy (Caeiro et al., 2013).

Apathy following TBI

In a study conducted by Kant et al. (1998) it was revealed that a significant number of patients with TBI exhibit apathy (assessed with the AES). Furthermore, the authors assert that apathy can occur independently of depression, suggesting that its underlying pathophysiological mechanisms may differ from those associated with depression.

Similarly, a study by Ciurli et al. (2011) confirmed that apathy (measured with the NPI), is the most prevalent neuropsychiatric symptom among subjects with TBI, identified in 42% of cases. Within the sample of participants with severe TBI, apathy was found to be the most common neuropsychiatric disorder. Consequently, it can be stated that apathy is a prevalent behavioral complication in TBI patients, affecting at least half of individuals at some stage during the post-TBI recovery period. While apathy in TBI is frequently associated with both depression and cognitive deficits, it can also manifest as an independent condition in numerous cases (Starkstein et al., 2014).

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Apathy following ABI

In patients with subcortical or hypoxic brain damage, as well as those with unilateral right hemisphere damage, apathy, assessed with the AES, has been found to be most severe as a result of ABI. The strong association between apathy and the location of the lesion suggests that the latter arises directly from the lesion and not from secondary psychological or socio-environmental problems. This finding points to an underlying dysfunction in the brain's motivational systems, supporting the classification of apathy in patients with acquired brain damage as a disorder of organic origin (Andersson et al., 1999).

This finding points to an underlying dysfunction in the brain's motivational systems, supporting the classification of apathy in patients with ABIs as a disorder of organic origin (Andersson et al., 1999).

Anhedonia and ABI

Anhedonia is defined as the inability to experience pleasure in activities that are usually considered enjoyable, such as physical exercise, hobbies, sexual activities, and social interactions, especially when compared to similar experiences that were previously perceived as pleasurable (Calabrò et al., 2012). According to the DSM-5, anhedonia manifests as a "markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day" (APA, 2013). On the other hand, the International Classification of Diseases (ICD-10) by the World Health Organization does not use the term "anhedonia" but refers to "loss of interest and pleasure" (World Health Organization, 2004).

In clinical practice, the term "anhedonia" encompasses a complex and multifaceted dysfunction related to the reward system, which becomes more evident compared to healthy subjects. However, a study by Ho and Sommers (2013) found that about 45% of studies measuring anhedonia do not provide a clear definition of the concept. The ability to experience pleasure is not limited to desire, reinforcement, and subjective pleasure but also includes cognitive aspects such as the ability to anticipate and predict rewards, the memory associated with these experiences, the association between value and relative cost, the effort required to obtain rewards, the integration of this information, the resulting decision-making process, and the self-motivation to engage in goal-directed behaviors (Der-Avakian & Markou, 2012). Consequently, reward processing is strongly influenced by executive functions (Berkman et al., 2011; Berkman et al., 2014).

Some authors have proposed a distinction between consummatory and anticipatory anhedonia to better describe the lack of a specific aspect of the pleasure process. Consummatory pleasure refers to the enjoyment felt during the execution of a pleasurable activity, whereas anticipatory pleasure relates to the expectation of pleasure deriving from future experiences. These concepts can explain the different behavioral, cognitive, and emotional experiences related to anhedonia. Other authors emphasize the need to distinguish between consummatory and motivational anhedonia, focusing on decision-making processes (Treadway & Zald, 2011).

From a neurobiological perspective, anticipatory pleasure is strongly, though not exclusively, correlated with the mesolimbic dopamine pathway, while the serotonergic and opioid systems

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appear to be more centrally involved in consummatory pleasure (Berridge & Robinson, 1998; Wise, 2008). Additionally, anticipatory pleasure is linked to motivational processes that promote behaviors aimed at achieving desired rewards (Schultz, 2002; Carver, 2001; Gard et al., 2007).

Anhedonia is linked to reduced reactivity and connectivity in neural networks involved in the perception and evaluation of rewarding or pleasurable stimuli, including the orbitofrontal cortex and ventral striatum/nucleus accumbens. It also involves disruptions in areas responsible for cost-benefit analysis and decision-making, such as the anterior cingulate cortex, ventromedial prefrontal cortex, and dorsolateral prefrontal cortex. Additionally, anhedonia affects reward processing and memory integration in the mesolimbic pathway (including ventral striatum/nucleus accumbens, ventral tegmental area/substantia nigra, and hippocampal connections), as well as salience attribution within the amygdala, and related limbic and paralimbic structures (Preda, 2014).

As the conceptualization of anhedonia has expanded, assessment tools have been developed to measure dimensions, such as anticipatory and consummatory pleasure, motivation, and effort across different reward domains. For instance, the Temporal Experience of Pleasure Scale (TEPS) (Gard et al., 2006) distinguishes between anticipatory and consummatory pleasure, while the Motivation and Pleasure Scale - Self-Report (MAP-SR), focuses on aspects of anticipation, motivation, effort and pleasure, mainly in the social domain (Llerena et al., 2013). Tools like the Specific Loss of Interest and Pleasure Scale (SLIPS) (Winer et al., 2014) and the Dimensional Anhedonia Rating Scale (DARS) (Rizvi et al., 2015) assess broader aspects of interest, motivation, effort, and pleasure. Similarly, the Positive Valence Systems Scale (PVSS) evaluates reward-related aspects specified in the positive valence systems (PVS) framework within the Research Domain Criteria (RDoC) (Khazanov et al., 2020). While these tools provide valuable insights into different facets of anhedonia, their varied emphasis on specific domains (e.g., physical versus social rewards) may influence how anhedonia is characterized and its prevalence. This underscores the importance of aligning tool selection with study objectives to ensure accurate interpretation of results.

Given the complexity of anhedonia, it is considered a symptom of various neuropsychiatric conditions, including depression (Pelizza & Ferrari, 2009), bipolar disorder (Tso et al., 2014), and ABIs of varying severity (Rao et al., 2007).

Anhedonia is a transdiagnostic symptom, in that it extends beyond major depression to social anxiety disorder and generalized anxiety disorder (Kashdan et al., 2011), as well as schizophrenia (Watson and Naragon-Gainey 2010) and substance abuse (Thomsen et al., 2015).

Anhedonia, assessed via low positive emotionality, has been shown to prospectively predict the development of both depression and anxiety, even when controlling for baseline symptoms (Kendall et al., 2015; Khazanov & Ruscio, 2016). Once disorders emerge, anhedonia serves as a strong predictor of a more unfavorable long-term course of major depression (Morris et al. 2009). It also forecasts poor psychosocial functioning following improvements in depressive mood (Vinckier et al., 2017) and increases the likelihood of depression recurrence (Wichers et al., 2010). Furthermore, anhedonia is a significant predictor of suicidal ideation and behavior (Winer et al., 2014; Ducasse et al., 2018), even after accounting for other cognitive and emotional symptoms of depression (Fawcett et al., 1990; Ballard et al., 2017) as well as other risk factors such as history of suicide attempts, childhood trauma, marital status, gender, and age (Ducasse et al., 2020).

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Anhedonia and Stroke

Motor, cognitive, and language deficits are common post-stroke consequences that limit the ability to perform daily activities, reducing participation in family, social, work, and community life (Hartman-Maeir et al., 2007; Tang et al., 2020). The reduction in functional autonomy in stroke survivors negatively affects their emotional well-being and quality of life, contributing to the onset of mood disorders (Patel et al., 2002; Marcheschi et al., 2018; Diamond et al., 2023). Specifically, it is estimated that between 25% and 79% of stroke patients suffer from post-stroke depression (PSD), a condition linked to the physical and psychological adversities they face (Whyte & Mulsant, 2002). Previous studies have shown that PSD has a negative impact on motor and cognitive recovery, contributing to a 15% increase in overall disability (Paolucci et al., 2019). This detrimental effect has been associated with deficits in the regulation of motivational behaviors (Gainotti et al., 2001).

Although many studies have focused on the negative impact of apathy on post-stroke rehabilitation (Marin, 1991; Sibon et al., 2012), motivational mechanisms and goal-directed behaviors can also be impaired by anhedonia, a condition still understudied in the stroke population. In this context, the disruption of dopaminergic networks following a stroke could trigger mood disorders and cognitive deficits, including memory dysfunction, contributing to the development of anhedonia (Piamarta et al., 2004; Terroni et al., 2015). Considering the crucial role of anhedonia in the motivational aspects of post-stroke recovery, a recent study by Ashaie et al. (2023) explored the association between three dimensions of PSD somatic symptoms, negative affect, and anhedonia. The results showed that anhedonia predicted an increase in somatic symptoms during the first year after discharge from rehabilitation, indicating its impact on physical distress rather than the direct effects of the stroke. Consequently, the authors emphasized the importance of examining specific dimensions of PSD, such as anhedonia, to better understand its development and etiology, allowing for targeted treatments and improved therapeutic outcomes (Ashaie et al., 2023).

A recent cohort study conducted by Segura et al. (2024) revealed a significantly higher prevalence of anhedonia among stroke patients (18.5-19.7%) compared to healthy controls (4.3%). Particularly, the study found that the levels of anhedonia among the patients were consistent across various factors, including stroke etiology, time since the event, affected hemisphere, lesion location, and motor and cognitive function levels. Additionally, anhedonia was associated with lack of motivation and increased negative emotional states, such as fatigue and anger, which adversely affected emotional well-being and quality of life, potentially leading to decreased participation in rehabilitation programs. Other studies have emphasized the role of anhedonia as a predictor of somatic symptoms of depression, such as fatigue, within the first year following rehabilitation discharge (Ashaie et al., 2023). It may also contribute to a self-perpetuating cycle of behavioral deterioration (Mallorqui et al., 2022). Furthermore, the literature indicates that older stroke patients face a heightened risk of experiencing long-term anhedonia. This increased vulnerability is attributed not only to a greater likelihood of social isolation (Yeh & Lo, 2004) but also to the neurophysiological damage resulting from brain injury.

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Anhedonia and TBI

From a neuropathological perspective, TBI is characterized by changes in various cortical areas, subcortical structures, and the white matter tracts that connect them. Recent evidence suggests that diffuse neuronal damage and cell loss can progress over weeks or months after the initial insult in selectively vulnerable regions of the prefrontal cortex, hippocampus, thalamus, striatum, amygdala, and forebrain nuclei. The resulting functional changes in neuronal circuits may constitute the neurological substrate of the cognitive and behavioral deficits frequently observed following TBI (Bigler et al., 2002; McIntosh et al., 1998).

Psychopathological disorders, particularly mood disorders, occur in the context of profound changes in cognitive and emotional processing following TBI. There is extensive literature regarding the cognitive changes observed after TBI. Depression occurs in approximately 25% of TBI patients (Fedoroff et al., 1992; Jorge et al., 1993). Feelings of loss, demoralization, and discouragement observed shortly after the injury are often followed by symptoms of persistent dysphoria. Fatigue, irritability, suicidal thoughts, anhedonia, lack of interest, and insomnia are observed in a considerable number of patients 6-24 months or even longer after TBI (Hinkeldy et al., 1990; Van Zomeran et al., 1985). In TBI patients, anhedonia reduces quality of life and may hinder rehabilitation (Jorge et al., 2005).

In a retrospective study (Lewis et al., 2015), data from 192 TBI participants from Phase III of the Vietnam Head Injury Study, a long-term follow-up study on veterans primarily with focal penetrating TBI (Raymont et al., 2011), were analyzed. Participants underwent neurological and psychiatric examinations, neuropsychological testing, and brain CT scans. The study aimed to examine the expression of anhedonic symptoms related to localized TBI. The primary finding was that anhedonic symptoms were more pronounced in individuals with damage to the right ventrolateral prefrontal region compared to those with injuries in other regions.

Disinhibition after ABI

The term "disinhibition syndrome" describes a wide range of behaviors that are inappropriate to the context and reflect the inability to control and repress some reactions. Different symptoms have as their common mechanism the lack of inhibition, such as impulsivity, inappropriate behaviors, aggressivity, and what Eslinger and Damasio (1985) called "acquired sociopathy", which is the loss of sensitivity, ability to respect social rules, and the account of consequences of aggressive actions on other people. The definition of disinhibition provided by Arciniegas and Wortzel (2014) is "socially and contextually inappropriate nonaggressive verbal, physical, and sexual acts that reflect a lessening or loss of inhibition and/or inability to appreciate social or cultural behaviors norms". Patients with disinhibition may have a normal level of cognitive intelligence and compromised emotional intelligence (Bar-On et al. 2003). According to the hypothesis of "somatic markers" (Damasio, 1994) a poor detection of somatic signals is the core feature that leads to disadvantageous choices for the people and for the way they relate to others. Blair and Cipollotti (2000) suggested an important mechanism for social cognition and control of aggressivity: the Social Response Learning. A lesion of the orbito-frontal cortex damages this system and can cause disinhibition and acquired sociopathy. This finding has been confirmed by

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recent studies using modern technologies like MRI. For example, Knutson et al. (2015) conducted a whole-brain analysis, and identified several brain regions involved in behavioral disinhibition, including the right orbitofrontal regions, bilateral insula, right temporal lobe, left frontal, precentral and postcentral regions, and bilateral gyrus rectus.

Further evidence comes from He et al. (2020), who analyzed structural MRI data from 361 healthy adults as part of the Human Connectome Project (HCP). They used the Connectome - based predictive model (CPM) to identify brain areas that are associated with inhibitory control. Their findings revealed a "high inhibitory control network" involving nodes in the prefrontal, orbitofrontal cortex, and inferior frontal gyrus, as well as a "low inhibitory control network", with nodes distributed across parietal, occipital, and limbic areas.

In a more recent study, Jenkins et al. (2024), found that disinhibition correlates with reduced global efficiency of Cognitive Control Network, and its connection with the Salience Network. Increasingly, studies of neural connectivity in disinhibition and other neuropsychiatric disorders are finding that these conditions are not the result of dysfunction in a single brain area. Instead, they may be better explained by widespread disconnection across distant brain regions.

Disinhibition in stroke.

Multiple studies indicate that between 5% and 76% of stroke patients show disinhibition at various time points ranging from 4 days to 4 years post-stroke (van Almenkerk et al., 2012; Angelelli et al., 2004; Aybek et al. 2005; Ghika-Schmid et al., 1999; Greenop et al. 2009; Mok et al., 2010; Buijck at al., 2012; Wong et al. 2014). However, these data are unreliable due to the frequent underdiagnosis and undertreatment of disinhibition. This presents a significant issue, as untreated disinhibition can result in prolonged stays in rehabilitation facilities and a slower recovery (Buijck et al., 2012). Ito et al. (2023) showed that patients with a MMSE score lower than 18 had higher Neuropsychiatric Inventory-Questionnaire Severity scores in disinhibition and Neuropsychiatric Inventory-Questionnaire Distress scores. This result shows that the severity and frequency of disinhibition is significantly higher in Subacute Stroke patients with severe cognitive impairment than in patients with mild cognitive impairment, while depression and apathy occur regardless of the severity of cognitive impairment. This study suggested that cognitive, behavioral and psychological symptoms should be early managed in stroke patients with cognitive impairment.

Disinhibition in TBI

There is considerable variability in studies regarding the prevalence of disinhibition among patients with TBI. Warriner et al. (2003), using the Minnesota Multiphasic Personality Inventory, reported that only 13% of TBI patients showed disinhibition, while Kelly et al. (2008) assessed the disinhibition through the Overt Behavior Scale (OBS, Kelly et al., 2006), and found a prevalence of 85.8%. This lack of agreement may be due to inconsistent definitions of the construct, the different assessment tools used, and variations in the severity of injuries within the samples (Osborne-Crowley and McDonald, 2018). Nonetheless, many studies have showed that disinhibition in TBI patients can lead to various long-term consequences, including caregiver

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burden (Brooks et al., 1986), family dysfunction (Douglas & Spellacy, 1996), reduced quality of life for patients (Koskinen, 1998), challenges in social reintegration (Simpson et al., 1999), and an increased risk of suicidal thoughts (Juengst et al., 2014).

Aggression after ABI

Aggression following ABI is typically characterized by disproportionate or inappropriate behaviors, which may be verbal or physical, and can be directed toward others, objects, or oneself. The manifestation of aggression can vary considerably; it depends on numerous factors, including the location and extent of the injury, as well as the individual's pre-injury personality (Neumann et al., 2015; Rao et al. 2009). Additionally, other co-occurring factors, such as cognitive impairment, emotional dysregulation, and environmental stressors, may exacerbate the aggressive behaviors. However, it remains unclear whether the varying expressions of aggression are to be considered as distinct syndromes or if they could be better understood as part of a continuum of symptoms (Neumann et al. 2015; Rao et al., 2009). Aggression following ABI can adversely impact family dynamics, social relationships, and community reintegration; furthermore, it can exacerbate the burden on families and may lead to the exclusion of the patient from treatment programs, thereby perpetuating a cycle of further isolation and diminishing access to essential social support systems (Hart et al., 2017). The *Overt Aggression Scale – Modified for Neurorehabilitation* (OAS-MNR) (Alderman et al., 1997) was developed as a standardized tool to assess aggression in neurorehabilitation settings using an operant framework. It categorizes aggression into four types—verbal aggression, and aggression toward objects, self, and others—each rated on a severity scale from mild to very severe based on observable behaviors. The OAS-MNR reliably records details of aggression episodes, including context and antecedents, and has proven useful in clinical practice, research, and program evaluation; it is one of the only tools, along with its extended version (Giles & Mohr, 2007), that has been specifically validated for individuals with ABI.

A variant, the *St Andrew's Sexual Behavior Assessment* (SASBA), assesses potentially inappropriate sexual behaviors. Both OAS-MNR and SASBA are considered "focal" measures, providing in-depth insights into specific behaviors. However, in cases where tracking every incident is impractical, broader "global" assessment tools, like the *Overt Behavior Scale* (OBS) and the *St Andrew's–Swansea Neurobehavioral Scale* (SASNOS), are valuable. These tools measure challenging behaviors, including aggression, in community settings for ABI populations. The OBS and SASNOS are easy to administer, providing a profile of strengths and weaknesses to support rehabilitation planning (Alderman et al., 2013).

The choice of tool can influence findings, with focal measures like the OAS-MNR capturing detailed episodic data, while global tools like the OBS and SASNOS provide a comprehensive behavioral overview. Selecting the appropriate tool is thus critical for aligning measurement with study objectives and ensuring accurate characterization of aggression in ABI populations.

Aggression after stroke

During the acute stage, stroke patients may exhibit aggressive behaviors, both physical and verbal, such as kicking, biting, pushing, throwing objects, screaming, cursing, and shouting.

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However, also in the subacute stage, patients often become more irritable, impulsive, and hostile, frequently displaying uncontrollable anger towards family members over minor issues. These symptoms are commonly referred to as post-stroke anger proneness (PSAP) (Kim, 2016). Such disruptive behavior during treatment or rehabilitation can cause significant distress to caregivers and result in communication breakdowns between patients and healthcare professionals, thereby hindering and delaying the rehabilitation and recovery process (Choi-Kwon et al., 2022).

The prevalence of aggressive behavior within the first week following a stroke is reported to range from 17% to 23%, increasing up to 32% between 3- and 12-months post-stroke. Aggression in stroke patients has been found to be correlated with factors such as younger age, a history of depression, and speech difficulties, (i.e., dysarthria). Additionally, it is often associated with concurrent depressive symptoms, impaired cognitive function, and motor dysfunction. (Choi-Kwon et al., 2022; Lau et al., 2017).

Numerous studies have attempted to establish a relationship between the locations of stroke lesions and PSAP; however, the findings have been inconsistent (Choi-Kwon et al., 2022). For instance, one study utilizing CT scans suggested that lesions in the left hemisphere and those situated closer to the prefrontal cortex were associated with aggressive behavior; however, they used a small sample size, which limits the reliability of this finding (Paradiso et al., 1996). In contrast, a study, conducted by Kim et al. using MRI, highlighted that PSAP was strongly linked to lesions in the fronto-lenticulo-capsular-pontine base area, while patients with lesions in other brain regions rarely exhibited PSAP (Kim et al., 2002). Conversely, other studies have reported no significant correlation between the location of stroke lesions and the occurrence of PSAP (Santos et al., 2005). Additional studies have indicated that PSAP may be linked to personality changes associated with frontal lobe lesions, which can lead to increased aggression or the amplification of preexisting aggressive tendencies. Furthermore, alongside the relationship between cognitive impairment and PSAP, patients may experience anger as a result of frustration related to impaired communication abilities, inappropriate responses to stimuli (such as pain or noise), or negative attributions regarding other's behaviors (Choi-Kwon et al., 2022; Neumann et al., 2015).

Aggression following TBI

Aggression is one of the most common consequences after TBI, with prevalence estimates ranging from 11% to 34% (Rao et al., 2009). The variation in reported prevalence rates across studies can be attributed to differences in study design, duration, population characteristics, and the criteria used to define and categorize aggression. Moreover, the complexity of post-TBI aggression, which may also be a symptom of delirium, mood disorders, or personality changes secondary to TBI, further complicates the ability to generalize findings (Visscher et al., 2010; Rao et al., 2009). Verbal aggression is typically the most prevalent subtype, in both the acute and the chronic settings (Roy et al., 2017).

Aggression is considered problematic following TBI due to the frequency, intensity, and apparent inability to regulate negative emotions, such as anger, and related behaviors. These emotional deficits are commonly described as being unpredictable, irrational, inappropriate, and disproportionate to the circumstances (Neumann et al., 2015). In fact, aggressive behaviors not

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only disrupt rehabilitation efforts but also impose a significant burden on both patients and caregivers. A comprehensive understanding of the correlates and predictors of aggression is crucial for effective prevention and treatment strategies, which could ultimately enhance rehabilitation outcomes during the critical early post-TBI period (Rao et al., 2009; Visscher et al., 2010).

In acute rehabilitation, aggressive behavior has been linked to a range of factors, including impaired language function, symptoms of post-traumatic stress disorder, frontal lobe injuries, a history of premorbid psychosocial issues and substance abuse, delirium, mood disorders (i.e., depression) and psychosis, late-onset partial complex epilepsy, a higher incidence of medical comorbidities, disorientation to place and time, and the use of anticonvulsant medications (Baguley et al., 2006).

The neuroanatomical mechanism underlying post-TBI aggression remains unclear, but it could be the result of an imbalance between inhibitory pathways in the frontal cortex and limbic structures that regulate impulse (Kim, 2002). For instance, in a study conducted by Tateno et al. (2003) frontal lesions were found to be associated with aggression; this would suggest that frontal lobe injury can cause damage to the ascending serotonergic pathways, which can contribute to the pathophysiology of both depression and aggressive behavior (Rao et al., 2009; Tateno et al., 2003).

Theory of mind and acquired brain injury

The term Theory of Mind (ToM) refers to the process of mentalizing, which is fundamental for social cognition and social interactions (Clausi et al., 2019). This mechanism is distinctly different from emotional contagion, an automatic process involving the recognition of others' emotions; ToM involves more complex processes, including the ability to attribute mental states different from one's own to others and to adopt another person's perspective in order to understand and predict their behavior. Conceptually, a distinction exists between "hot" functions, such as processing emotions, and "cold" functions such as understanding another's perspective. Among the hot functions, affective empathy refers to the ability to connect emotionally with the feelings of others while recognizing that these feelings are separate from our own. Emotion perception, another hot function, involves interpreting a person's emotional state based on cues like facial expressions or tone of voice. Cold functions, on the other hand, involve skills such as explaining the behavior of others by understanding their thoughts, appreciating others' points of view, and interpreting pragmatic inferences (McDonald, 2013).

It has been shown that patients with brain injuries may exhibit reduced empathy (Shamay-Tsoory et al., 2004), social isolation (Lezak et al., 2004), difficulties in understanding irony or sarcasm (Martin & McDonald, 2005; Channon et al., 2005), and poor social skills (Spatt et al., 1997). These factors can contribute to challenges in social reintegration.

Patients with brain lesions tend to fail tasks that require reasoning about mental states, making both ToM and mentalizing tasks predictors of deficits in social behavior. Various tasks are used to assess ToM, generally categorized into "first-order" tasks adapted from developmental psychology (e.g. the Sally-Anne test) and "second-order" tasks, with the "Faux Pas" test (Stone et al., 1998) being the most frequently used. This test involves presenting stories in which a character makes a faux pas, such as saying something inappropriate to the listener. To recognize the faux pas, the

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subject must understand that the protagonist of the story is unaware of making a social mistake (Clausi et al., 2019). The Sally-Anne test assesses understanding of false beliefs by asking participants to predict where a character will look for an object based on their perspective, rather than reality (Korkiakangas et al., 2016). (Korkiakangas et al., 2016). In the faux pas test, subjects are presented with stories where a character unintentionally says something socially inappropriate, requiring participants to recognize the social mistake and understand the protagonist's lack of awareness (Clausi et al., 2019). Participants answer questions designed to evaluate their ability to detect the faux pas, attribute intentions, and assess others' emotional states (Söderstrand & Almkvist, 2012). These tasks provide valuable insights into ToM impairments, particularly in populations with ABI, by assessing key cognitive and affective dimensions of social cognition. However, differences in task complexity, focus on verbal versus non-verbal cues, and reliance on executive functioning may influence findings, underscoring the importance of tool selection in capturing the full spectrum of ToM abilities.

A review by Abu-Akel & Tsoory (2010) highlighted that both cortical and subcortical regions form distinct, yet interacting, networks that support the cognitive and affective representation of mental states, both in relation to oneself and to others. The cognitive network involves the dorsomedial prefrontal cortex, the dorsal anterior cingulate cortex, and the dorsal striatum. The affective network involves the ventromedial prefrontal and orbitofrontal cortex, the ventral part of the anterior cingulate cortex, the amygdala, and the ventral striatum. Additionally, attention and selection systems, particularly the parietal-temporal junction, play a critical role in facilitating these processes. Neurochemically, the dopaminergic and serotonergic systems seem to play key roles in these functions.

ToM and TBI

The relationship between ToM and TBI has been extensively examined in the literature, consistently revealing deficits in ToM tasks among individuals with TBI. Most studies have used narrative stories (Bibby & McDonald, 2005; Martin & McDonald, 2005) or videos (McDonald & Flanagan, 2004; Turkstra et al., 2004) to assess ToM. However, Henry et al. (2006) adopted a less cognitively demanding test to minimize the influence of cognitive load. Despite the reduction in task complexity, TBI patients still showed considerable impairments, scoring significantly lower than the control group.

These findings align with broader research on the relationship between ToM and executive functioning. For instance, Apperly et al. (2007) demonstrated that both ToM tasks, (e.g. false belief tasks) and non-ToM tasks, such as "false photograph" tasks—where perspective-taking is not required—show deficits in individuals with brain damage. This suggests that, despite arguments for the specificity of ToM as a cognitive process (Frith & Frith, 2003; Saxe et al., 2004), efficient executive functioning is essential for performing mentalizing tasks. In the study by Apperly et al. (2007), impaired performance on mentalizing tasks correlated with deficits in a phonemic fluency task, a measure of executive functioning. This correlation was not found with a purely emotional task that required subjects to identify the emotion expressed on a face, although performance on this task was also impaired compared to controls. Overall, in addition to social cognition tasks,

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individuals with TBI show below-average performance in tasks assessing both emotional and cognitive empathy (de Sousa et al., 2010).

All these difficulties—in experiencing empathy, understanding others' mental states, and pure executive functioning—constitute significant barriers to full recovery after a brain injury and hinder the return to premorbid social life.

ToM and stroke.

Stroke has also been widely investigated in relation to ToM. Research in this area has predominantly examined the differences between lesions in the two hemispheres and their impact on both the cognitive (beliefs about beliefs) and emotional (beliefs about feelings) components of ToM. A study conducted by Yeh and Tsai (2014) compared the performance of subjects with right hemisphere lesions to those with left hemisphere lesions and compared these results with a control group of healthy individuals. The study involved tasks that assessed both cognitive and affective ToM in verbal (faux pas) and non-verbal modalities. The underlying hypothesis of this study primarily concerned empathy, suggesting that deficits in affective tasks are rooted in reduced empathic capacity caused by the lesion; therefore, tasks assessing empathy were also employed. The results showed that subjects with right hemisphere lesions exhibited poorer performance in both non-verbal cognitive ToM tasks and cognitive empathy tests compared to subjects with left hemisphere strokes. However, both groups were impaired compared to controls in both affective and cognitive ToM tasks.

Similar to what has been discussed for individuals with TBI, stroke patients also show poor performance on ToM tasks that correlate with tests assessing executive functioning, such as the Brixton Test (Hamilton et al., 2017). Therefore, it is crucial to consider the decline in cognitive flexibility, which may affect mentalizing tasks and, more broadly, the overall psychosocial functioning of individuals with brain lesions.

All these findings taken together, including those related to TBI and stroke, highlight the importance of administering tasks that assess ToM in cases of ABI. This approach is vital for developing rehabilitation programs that support social reintegration and promote the individual's complete recovery.

Therapeutic approaches and future directions

Improving the quality of life for patients with emotional sequelae following ABI requires a wide range of therapeutic approaches, yet achieving a clear consensus on optimal treatment strategies remains challenging. There is growing emphasis on multidisciplinary care, with an increase in advocating for a team-based approach that includes traditional healthcare providers, such as psychiatrists and neurologists, as well as specialists like occupational therapists, neuropsychologists, and psychotherapists. Such frameworks extend beyond hospital settings, actively engaging with community resources to provide comprehensive care.

Treatment options for post-stroke anxiety remain limited and often inadequate to meet patients' specific needs. While CBT has shown promise in reducing anxiety symptoms, further research is needed to confirm its efficacy in this population. Petrova et al. (2012) studied 198 patients with a

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first cerebral stroke and found that 71.2% experienced affective disorders, including generalized anxiety and phobias. Post-TBI anxiety is typically managed through both pharmacological and psychotherapeutic approaches. Studies have shown CBT to be effective in alleviating post-TBI anxiety and depression, with benefits lasting up to three months (Barua et al., 2024). However, pharmacotherapy remains the most common intervention, especially in cases of post-traumatic stress disorder, though mindfulness has proven helpful in youth with TBI, reducing anxiety and enhancing psychological functioning (Soo et al., 2019).

For patients with depression, cognitive behavioral therapy (CBT) has shown mixed efficacy, while repetitive transcranial magnetic stimulation (rTMS) has produced promising outcomes (Frank et al., 2022). Addressing apathy often includes pharmacological treatments like selective serotonin reuptake inhibitors (SSRIs), though these may only be effective when depressive symptoms coexist, and certain antidepressants may even impair motivation. Rehabilitative techniques, such as neuropsychological counseling, focus on restoring goal-directed behaviors through goal-setting, future planning, and self-monitoring, often complemented by CBT. Dopaminergic pharmacotherapy and rTMS have also shown some potential in treating apathy (Tay, 2021).

Treatment options for anhedonia often focus on reducing negative affect, but current psychotherapies, including CBT and mindfulness-based cognitive therapy, have limited impact on enhancing positive affect (Boumparis et al. 2016). Traditional therapies like cognitive therapy and antidepressants improve negative affect but only moderately increase positive affect levels, which remain below those in the general population. New approaches targeting positive valence systems show promise for effectively addressing anhedonia (Dunn et al., 2020; Sandman, 2022).

For aggression management following ABI, an integrative, multidisciplinary strategy is essential, starting with a thorough assessment. While pharmacological intervention may be required, particularly in acute settings, a more integrative approach should be adopted, incorporating cognitive-behavioral interventions tailored to individual needs. Disinhibition rehabilitation may benefit from behavioral management and self-management strategies, as well as electrical aversion therapy, though CBT alone has shown limited effectiveness in reducing disinhibition symptoms (Verberne et al., 2019).

Rehabilitation for ToM deficits can draw on methods used with children, such as conversation-based training. In this approach, participants discuss stories that highlight social nuances under the guidance of a trainer, who encourages perspective-taking through structured feedback and group discussion, followed by individualized exercises (Lecce et al., 2014; Caputi et al., 2020).

Overall, while traditional pharmacological and behavioral therapies continue to be widely used, the most promising outcomes may arise from a comprehensive, interdisciplinary approach that utilizes community resources and personalized care plans to address the complex needs of these patients.

Conclusions

This narrative review summarized the most common emotional sequelae after ABI such as depression, anxiety, aggression, anhedonia, apathy, disinhibition, and deficit in theory of mind.

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These emotional changes can arise from both direct neurological damage and the psychological stress of coping with the aftermath of the injury.

Despite the prevalence of emotional sequelae, it is only in recent times that growing attention has been given to improving early detection and intervention. The integration of neuropsychological assessments and interdisciplinary rehabilitation programs have been found to improve emotional outcomes. Therefore, tailored therapeutic strategies, including psychotherapy, pharmacological interventions, and cognitive rehabilitation, are vital for addressing both the emotional and cognitive consequences of ABI.

In conclusion, emotional changes following ABI can be pervasive and long-lasting, demanding more attention from clinicians and researchers. Future studies should focus on the long-term trajectory of these emotional sequelae and the development of more effective, individualized interventions to enhance recovery and improve patients' quality of life.

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