THE NEUROPSYCHIATRY OF ACQUIRED BRAIN INJURY

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Abstract

The psychiatric syndromes derived from acquired brain injury are the behavioural consequences of the combination of cognitive, emotional and volitional sequelae. Their impact on the quality of life of patients and relatives is far greater tan the impact of motor or sensory deficit. The description of these behavioural presentations and an attempt to understand the formation of the symptoms will be undertaken. Changes in awareness, empathy, emotional recognition and regulation are important mediating factors. Behavioural changes mainly take the form of disinhibition or apathy. Psychotic syndromes, major depression or bipolar disorders are rare. Some delusional ideas respond to changes in the perception of novelty or to changes in body perception.

Key words: Neuropsychiatry, brain injury, behavioural changes, anosognosia, apathy, disinhibition.

Resumen

Los síndromes psiquiátricos y los cambios conductuales que se derivan del daño cerebral son las consecuencias de la combinación de las alteraciones volitivas, emocionales y cognitivas. El impacto en la calidad de vida de los pacientes y de sus familiares es significativamente mayor que el impacto de las secuelas motoras o sensoriales. En este artículo se aborda la descripción de estos trastornos neuropsiquiátricos y se propone un acercamiento a la comprensión de cómo se gestan. Los cambios en conciencia de situación, empatía, reconocimiento y regulación emocional son factores mediadores importantes en la formación de los síntomas. Los cambios conductuales se presentan principalmente en forma de apatía o desinhibición. Los síndromes psicóticos, los trastornos bipolares o la depresión mayor son muy poco frecuentes. Por otro lado, las ideas delirantes responden a cambios en la percepción de la novedad/familiaridad de los estímulos o a cambios en la percepción del cuerpo que ocurren tras lesiones del hemisferio derecho.

Palabras clave: Neuropsiquiatría, daño cerebral, cambios conductuales, anosognosia, apatía, desinhibición.

Introduction

The aim of this chapter is to describe the specific mental changes presented by patients attending neurorehabilitation services. Traumatic brain injury and stroke are the two main etiologies; encephalitis, brain tumors and anoxia also pose rehabilitation challenges that entail psychiatric complications. When discussing the psychopathology of acquired brain injury (ABI), a reflection on the adequacy of our models of human psyche and our psychopathological tools will be included. ABI has not been systematically studied by psychiatrists; those of us that have spent our professional lifes caring for these patients have struggled trying to apply psychiatric nosology and psychopathological language to the psychological changes presented by this group of patients. This chapter will summarize that learning process and the considerations derived from it.

Psychopathology of ABI

Psychopathological language is based on a specific model or understanding of human psychological functions known as 'the trilogy of mind[1]. Kantian tripartite concept of mind and Faculty Psychology laid the foundations for western psychiatry to organise mental disorders as the weakening of one of these functions: volition, emotion and ideas or cognition. Berrios[2] explained the history of this process in detail and quoting Esquirol, Prichard and Bucknill & Tuke wrote: 'delusional, emotional and volitional insanities provided the template for groupings that lasted to this day'. Psychiatry has tended to define mental insanity on the basis of disorders of thinking, has seen the

decline of the concept of will since the end of the nineteenth century and has failed in the development of a rich psychopathology of emotions[3]. The result of these processes is an intellectualistic definition of mental illness.

Psychopathology, the language used to describe psychological and behavioral alterations, has been organized following these three groups of phenomena: disorders of will, emotion and reasoning. Symptoms, such as delusions, obsessions or hallucinations represent the derangement within the cognitive functions; sadness or elation are the prototypes of emotional disorders, while abulia, more recently apathy, compulsions and impulsivity represent volitional disorders.

The consequence of adherence to this model is that basic symptomatology has to fit in one of these conceptual categories, and symptoms that seem to share emotional, cognitive and volitional components are presented only with the more salient aspect (cognitive, emotional or volitional) as the only component of the symptom. Reality tends to be more complex and none of the classical neuropsychiatric symptoms can be fully described limiting the description exclusively to one of these psychological functions. Most symptoms in neuropsychiatry (and probably in general psychiatry as well), when looked at closely and described carefully, present a multiplicity of psychopathological faces to it. Irritability, one of the most frequent and neglected symptoms in patients with stroke or TBI, is a good example. Craig et al (2008) designed 'The irritability questionnaire' on the basis of the following definition of irritability: 'a mood that predisposes toward certain emotions (e.g. anger) certain cognitions (e.g. hostile appraisals) and certain actions (e.g. aggression)'.

Even classical psychiatric symptoms such as delusions cannot be separated from non-cognitive psychological changes: Capgras delusion, for example, only appears after the patient experiences an abnormal lack of familiarity accompanying the perception of a very well-known face; the same applies to reduplicative paramnesia, the strange idea appears after, or simultaneously, with an unexpected sensation or emotion, the lack of an expected familiarity (or the abnormal presence of a novelty perception). Abnormal feelings of novelty or familiarity are necessary components of what initially is categorized as a delusion, a pure disorder of reasoning. Apathy, the main representation of a disorder of volition, is another good example; it often presents the poverty of actions together with a lack of emotional life and with the abolition of the spontaneous generation of mental contents[5,6].

In regards to the psychopathology of ABI the first point to make is that psychotic symptoms (structured delusional ideas or repetitive hallucinations) play a minor role and that the classical affective disorders (retarded depression or mania) are also rare. On the other hand, mental changes, cognitive and non-cognitive are very prevalent and have enormous impact on quality of life of patients and families. Behavioural changes, sometimes conceptualized as organic personality disorders, are the most frequent psychopathological challenge, particularly in TBI, followed by different forms of emotional and affective disorders, frequently encountered in patients with stroke; both types of problems are usually combined with cognitive changes (attention, memory, executive disorders) that often play a role in the generation of the behavioural changes. The rest of the chapter will describe these types of disorders in greater detail, ac-

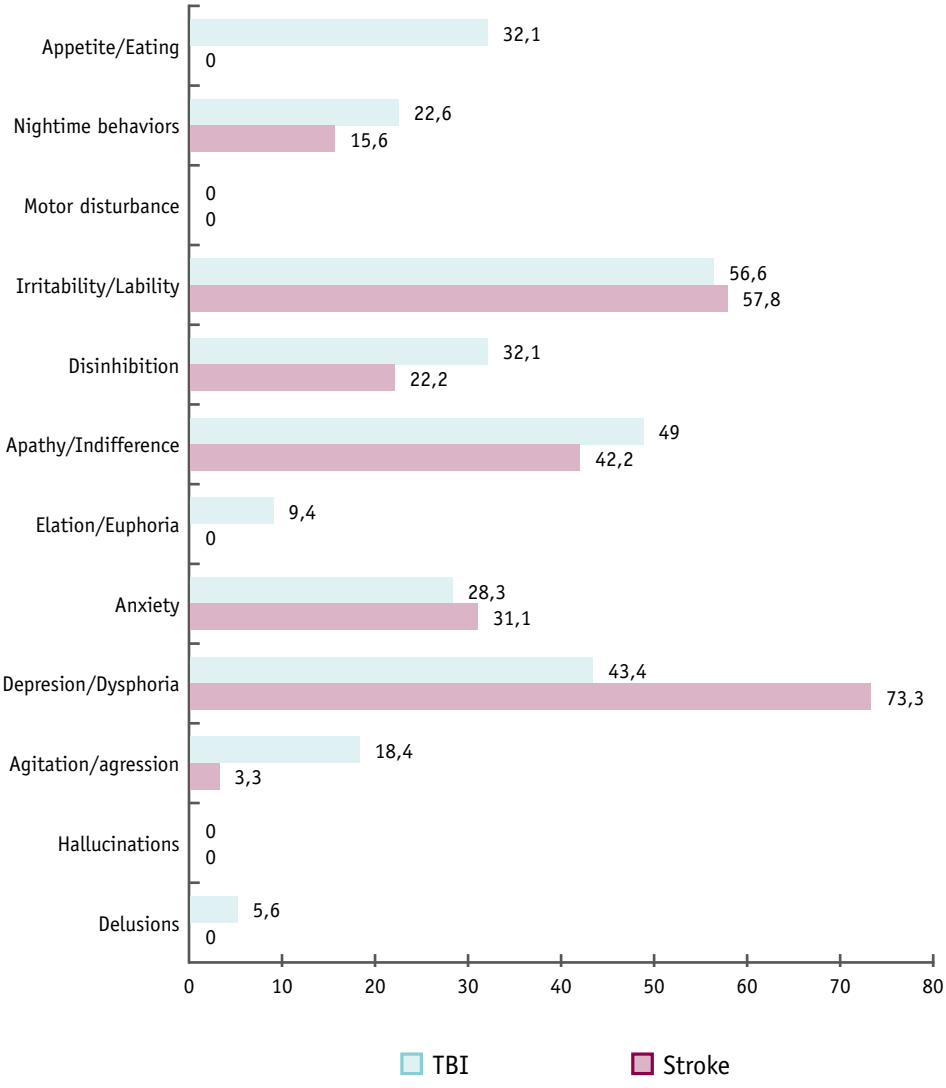
knowledging that the grouping is more didactic than real. The symptoms tend to be a combination of volitional, cognitive and/or affective/emotional changes that render a particular behavioural presentation.

Behavioural changes and organic personality disorder

One of the successful terms in the study of neuropsychiatry of ABI is '(neuro)behavioural changes' These changes can appear in the acute phase (post-traumatic amnesia or confusional states), accompany somatic or neurological complications (infections, hydrocephalus, epilepsy) or be part of a permanent change. Behavioural changes are loosely defined and they refer to the new appearance of behaviours that interfere with social functioning, or the absence of behaviours that are normally expected in that person according to the previous personality described by close relatives or friends. 'Personality' is a psychological construct that describes the permanent traits that underlie the patterns of emotional and behavioural reactions of an individual. When we describe permanent, repeated and signifit neurobehavioural changes in a patient with ABI, the diagnosis that follows is 'organic personality disorder' (OPD). The subtype of OPD depends on the most salient behavioural changes; DSM-5 includes labile, aggressive, apathetic, paranoid and disinhibited under the diagnosis of 'personality change due to another medical condition'.

We have explored these behavioural changes in three different PhD projects in Spain that are going to be used in the following paragraphs. Castaño[7] completed her PhD on the 'Neuropsychiatry of Traumatic Brain Injury'. The cross-sectional analysis of a chronic TBI sample is analyzed first[8]: 53 patients with severe TBI, between 2 and 8 years after trauma, young (mean age 35) and mainly male (85%). Only three patients were free of neuropsychiatric symptoms according to the NPI results; figure 1 summarizes the prevalence of psychiatric symptoms when the neuropsychiatric inventory (NPI) was used. The first result to highlight is the relative absence of classical psychotic symptoms: none of the subjects presented hallucinations and only three had delusions. On the other hand 56% of the patients presented with irritability/lability, 49% with apathy and 43% with depression; desinhibition and changes in eating pattern were detected in 32% of the sample.

Figure 1: Percentage of stroke and TBI patients scoring in items of the NPI (PhD thesis of Mimentza[10] and Castaño[7]).



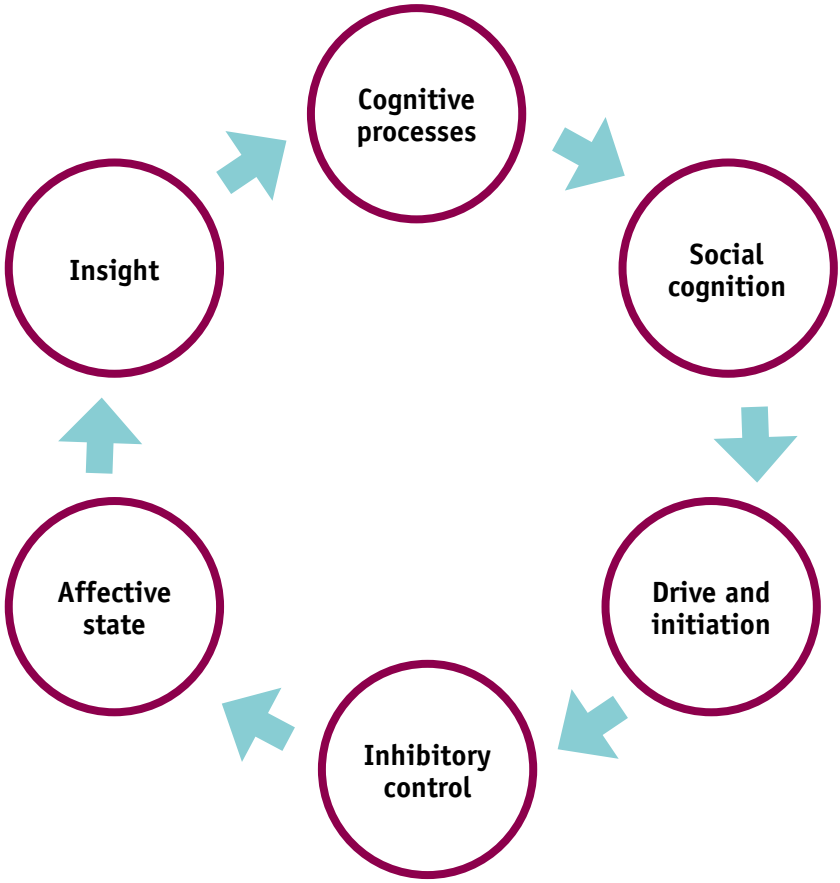
The same author compared subacute patients (n=50) and two samples of chronic patients (2 to 4 years, n=54 and 5 to 8 years n=52). Patients were mainly severe TBI (80%). Relatives reported personality changes in 80% of the patients in the three samples; a minority of the changes qualified as positive: more communicative, emotionally more expressive or less impulsive. In the three samples more than 90% of the patients presented psychopathological symptoms in the NPI with the same top three in the three different moments assessed: apathy, irritability/lability and desinhibition.

Sanchez-Cubillo[9] focused on the description and measurement of disinhibited behaviours in ABI. This research was carried out in a different hospital and city. The study included 93 patients attending a neurorehabilitation clinic, 70 TBI and 23 stroke, mean age of 36 and a majority of male subjects (72). 58 patients were diagnosed having an organic personality disorder (37 disinhibited type, 7 apathetic and 14 mixed type). The study included the development of a scale focused on the description of disinhibition. Out of the 31 items included in the scale the most prevalent were irritability, impulsivity, distractibility, lack of concern for future consequences and rigid behaviours. Despite the high prevalence of apathy as a symptom identified in the NPI and other neurobehavioural scales, a relative low proportion of patients are classified as OPD apathetic type. This probably reflects two facts: the high comorbidity of apathy and desinhibition and the salience of desinhibition when compared to apathy. Only the most severe cases of apathy, in the absence of aggressive or disinhibited behaviours, attract the diagnosis of OPD apathetic type.

Mimentza[10] focused upon the longitudinal analysis of the psychopathology of stroke patients. 45 stroke patients (mean age 60, 34 male) attending a neurorehabilitation inpatient clinic were assessed at different points in time. When assessed with the NPI three months after stroke none of them showed hallucinations or delusions, but 73% scored on depression, 58% on irritability/lability and depression and 42% on apathy (see figure 1).

Taking these three studies together we can confidently say that the most common behavioural changes in ABI are apathy, irritability, lability, desinhibition and depression. Each of these symptoms includes a wide collection of overt behaviours (childishness, overtalktiveness, inappropriate comments, lack of concern for others, lack of initiation, verbal abuse, hostile comments, crying) and a variety of subjective experiences (inner tension, sadness, indifference); limitation in the awareness of the behavioural changes and of psychological déficit is a very common accompanying phenomenon. Careful description of the clinical phenomena is a necessary initial step in clinical psychiatry; understanding symptom formation should follow in order to be able to design useful and rational therapeutic interventions. In the following paragraphs an attempt to list and discuss some of the psychological processes that lead to behavioural change are presented (see figure 2). It must be taken into account that the combined disruption of several of the processes will be more common than the isolated disorder of only one of them.

Figure 2: Psychological processes that can be altered in patients with neurobehavioural disorders.



Cognitive processes

The assessment of patients with ABI normally includes evaluation of attention, memory, language and executive functions. It is less common to find an explanation of how a problem with working memory, verbal fluency or planning translates to everyday behavior. The following examples can help to illustrate the relationship between cognitive functioning and change in social behaviour. A good recovery of an aphasic patient usually presents slowness in the processing of verbal information. Informal group interactions are characterized by a spontaneous sequence of interventions and changes of topic. Patients that have recovered functional communication often fail

to follow these group conversations and tend to remain silent or isolated. Relatives find difficult to realize the reason behind this retreat, get angry and frustrated, and confront the patient with the need to keep socially active. Social isolation and irritability are often the final behavioural presentation. A second example focuses on how poor working memory or poor short term memory will induce a change in leisure preferences. With poor memory it is no longer possible to follow the plot of a film or the argument of a book, while watching sports, comedy and nature documentaries can still be enjoyed, as they demand less capacity for the conservation and manipulation of information. Similarly, inability to follow the sequence of actions required to complete a complex task (repair the car, plan a holiday) can easily lead to frustration, sadness and irritable behavior.

Social cognition

Cases with severe brain injury, marked social difficulties and relative preservation of cognition pose the question of what kind of psychological functions underlie the social decline. The case of Phineas Gage is a classical example that has been extensively studied[11]: following a severe frontal TBI there was a good motor and cognitive recovery but marked personality change and social decline. Similarly, a number of neuropsychiatric disorders such as autism or schizophrenia display behavioural problems that do not seem to be fully explained by attention, executive or memory difficulties. Social cognition was introduced as a concept that would include all the psychological processes that need to be invoked to explain adequate social behavior, and that could therefore be used to understand behavioural disorders in the absence of cognitive deterioration. The boundaries and the content of this construct are not yet definitely settled, but theory of mind, social perception/knowledge and emotional processing are included in all the models proposed[12]; empathy is another key concept that has a wide overlap with theory of mind. The literature on rehabilitation of social cognition in brain injury pursues at least three different strategies[13]: (1) restoration of cognitive abilities such as speed of information processing, executive function or memory under the hypothesis that social abilities always make use of these common cognitive processes, (2) rehabilitation of specific abilities such as facial recognition of emotions or empathy; (3) finally the interventions on social skills that address the final behavior directly.

Inhibitory control and initiation

These two distinct capacities are sometimes incorporated in the models of executive function Norman and Shallice[14] listed five groups of functions included in their model of executive functions. One of them, 'Stopping behavior' is closely related with social disinhibition, while 'initiation and drive' and 'generative thinking' play a role in initiating behavior.

Inhibitory control is crucial in the understanding of disinhibited behaviours such as inappropriate comments, impulsive shopping, lack of social manners or sexual inadequate remarks or approaches. The inability to delay a response and give time to think and consider the characteristics of the social scenarios and possible alternative responses, results in a wide range of childish and disinhibited behaviours.

Apathy is the paradigm of the disorder in the initiation of action. The definition of apathy includes the reduction in expected self-initiated behaviours, together with an empoverishment in generative thinking and blunted affect. The relationship of these three groups of phenomena, behavioural, cognitive and affective, is far from clear. It can be argued that the core drive to action depends upon the preservation of a lively affective state or the capacity to generate and experience emotions. Alternatively we can place the generation of ideas as the primary function, or it can be hypothesized that both functions are necessary in the generation of social behavior, and that the absence or disorder of one or the other will give rise to a subtype of apathy. These are questions still open to research.

Insight and awareness

This cognitive ability is also incorporated in the more comprehensive models of executive functions. It is defined as the capacity to monitor and modify one's own behavior. Awareness can be applied to a very wide range of functions, from body functioning (sensory and motor control) to insight into one's actions and feelings. Unilateral neglect, denial of left hemiplegia or of blindness (Anton´s syndrome), unawareness of expressive aphasia, of behavioural change or of amnesia are some of the clinical presentations of the disorders of awareness in brain injury. Most of the models[15,16,17] propose that the understanding of unawareness includes a basic level that is related to the access to basic information but also other levels related to awareness of the implications of the deficit in everyday life and to mechanisms of psychological denial. Whatever the source or type of unawareness might be, it is not difficult to realize that these disorders will give rise to behavioural changes and social conflicts. Carers will often try to protect the patients from unnecessary risks that will often not be accepted by patients. Driving and returning to work are only two of the common situations that lead to conflicts.

Disorders of affect and emotion

Psychiatrists focus their efforts on the description of affective disorders while psychologists tend to study the nature of human emotions and the ways in which their recognition and regulation can be distorted in different groups of patients. What are the differences, if any, between affective states and emotions? Both sets of phenomena belong to the non-cognitive part of our psychic life, together with others such as drive, motivation or desire.

The literature tends to differentiate between emotions being a brief reaction (seconds or minutes) to external stimuli that elicit a specific facial expression and generate an adaptive behavioural response, and affective states, a long-lasting state (days or weeks), unrelated to external stimuli that biases thinking and does not always help with social adaptation. Neither of the definitions gives information on the specific nature of the experience which is where both concepts overlap. Let us take the case of fear (emotion) that can be a brief response to an external threat that disappears soon after the external situation changes, or, fear (affect) can be a more long-lasting state that biases the interpretation of neu-

tral stimuli and prompts a defensive way of thinking. While the 'cognitive revolution` helped to develop conceptual models of memory, language, attention or executive functions, the 'emotional revolution' has not arrived yet and we lack the conceptual sophistication that might help to understand the emotional and affective difficulties after brain injury.

Added to the difference between disorders of affect and emotion, we shall see disorders of a variety of 'contents' of affects or emotions. Within the realm of emotions there is, at present, a widely accepted categorization of six basic emotions: fear, anger, sadness, happiness, surprise and disgust[18]. The same categories have not been proposed for affect. Psychopathology invites clinicians to systematically assess the dimension sadness-elation, but we often encounter other forms of affective disorders such us persistent irritability, generalized anxiety or apathy. It is less common to consider abnormal absence or presence of familiarity (deja vu, jamais vu) as an affective disorder. The same could apply to the abnormalities in the experience of trust or certainty. Taking these limitations into account we shall summarize the literature on affective disorders after brain injury.

Post-stroke depression is the best known affective disorder in brain injury; it has been extensively studied in the last four decades[19]. Up to then, depression following stroke was interpreted as an emotional reaction to the brain lesion. Golstein´s 'catastrophic reaction' was a concept that followed this psychogenic view. Folstein et al[20] started to argue that depression could be a direct consequence of the brain disorder. Establishing the prevalence has not been easy because of the heterogeneity of samples (hospitalized, outpatient, community), the different scales and diagnostic tools used, the different moments of assessment and the exclusion of aphasics and demented patients. On the whole it is considered that one third of the patients with stroke will present significant depressive symptoms[21]. Robinson & Spalleta[19] established that 21% suffered minor depression and 19% presented major depression. House et al[22] presented a much lower incidence studying a community sample. The differential diagnosis of post-stroke depression includes 'fatigue', a poorly understood and frequently underdiagnosed syndrome that is present in up to half of all stroke survivors[23].Patients describe a subjective experience of lack of energy, or the need to exert a high level of effort in order to complete ordinary tasks, in the absence of sadness.

Pathological laughing and crying are the paradigm of the disorders of the expression of emotions. These syndromes can be found in a variety of neuropsychiatric conditions including stroke[24], multiple sclerosis, traumatic brain injury or pseudobulbar palsy. Minor stimuli provoke important emotional reactions that the patient judges as disproportionate and out of his control.

Psychosis

Psychosis is an uncommon complication of brain injury. Samples are very heterogeneous in a number of dimensions: diagnostic criteria and length of follow-up are two of the most important. Davison and Bagley[25] published the monograph entitled 'Schizophrenia like psychoses associated with organic disorders of the central nervous system'. They reviewed 14.385 cases with traumatic brain injury from 8 different series and the incidence of psychosis reported ranged from

1.7% to 9.08%. Achte et al[26] studied 3552 wounded soldiers, 42% with open head trauma and 30% with post-traumatic epilepsy, with a very long follow-up period: 22 to 26 years. They report the highest prevalence of a large sample with TBI: 317 cases (8,9%). The long follow-up period, the compensation associated to the diagnosis, the lack of temporal limitation between trauma and onset of psychosis (42% of the psychosis started 10 years later) and the imprecise use of the concept of psychosis (Korsakoff syndrome, dementia and confusion were included) help to explain the very high prevalence. The most recent large sample was collected in Belgium[27,28]: retrospective study of 530 patients with TBI with 18 psychosis being diagnosed, 3,4% of the sample. Most cases started soon after the trauma, 80% within a 6 month period. It is likely that a number of postraumatic confusional cases were included.

When all the samples are pondered, and post-traumatic confusional states excluded, it can be estimated that the prevalence of psychosis after traumatic brain injury ranges from 2 to 3%[29]. Unlike cognitive disorder, there is no need for a close temporal link between brain trauma and onset of psychosis. Some of the psychotic episodes appear soon after the injury while others do years after. Auditory hallucinations and delusional ideas are the most common symptoms with depressive symptomatology playing a relevant prodromal or comorbid role. Severity of trauma and temporal localization are important risk factors, though cases with mild TBI have also been reported.

Misidentification on syndromes after brain injury tend to be linked to right brain lesions, independent of the etiology. Right sided strokes present a number of psychotic syndromes related to body awareness: denial of ownership of the arm or leg, the belief that there are supernumerary limbs or somatoparaphrenia, the belief that a part of the body, usually the left arm, belongs to somebody else. Capgras syndrome, Fregoli syndrome and reduplicative paramnesia tend to appear after abnormalities in the experience of familiarity, commonly associated with right brain lesions.

Other mental symptoms that need to be differentiated from psychosis are confabulations and false memories. Confabulations are traditionally linked to Korsakoff syndrome following Wernicke´s encephalopathy, but a number of patients with severe TBI or stroke can also present these symptoms. Other forms of false memories or difficulties categorizing mental contents are sometimes referred by patients with ABI: the boundaries between the memory of a dream, an imagination or a real fact are not always clear to patients.

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