Published in : Annals of Physical and Rehabilitation Medicine (2016), n°59, pp. 18–22
DOI: 10.1016/j.rehab.2015.09.002
Status : Postprint (Author's version)

Neurobehavioral and self-awareness changes after traumatic brain injury: Towards new multidimensional approaches

A. Arnould [a,,b,c], E. Dromer [a,c], L. Rochat [b,d], M. Van der Linden [b,d,e], P. Azouvi [a,c]*
[a] Service de médecine physique et de réadaptation, hôpital Raymond-Poincaré, AP-HP, 104, boulevard Raymond-Poincaré, 92380 Garches, France
[b] Unite de psychopathologie et de neuropsychologie cognitive, université de Genève, Genève, Switzerland
[c] EA 4047, HANDIReSP, université de Versailles-Saint-Quentin-en-Yvelines, 78000 Versailles, France
[d] Swiss centre for affective sciences, université de Genève, Genève, Switzerland
[e] Unite de psychopathologie cognitive, université de Liege, Liege, Belgium

KEYWORDS:

Apathy
Impulsivity
Anosognosia
Traumatic brain injury
Multidimensional approach

ABSTRACT

Neurobehavioral and self-awareness changes are frequently observed following traumatic brain injury (TBI). These disturbances have been related to negative consequences on functional outcomes, caregiver distress and social reintegration, representing therefore a challenge for clinical research. Some studies have recently been conducted to specifically explore apathetic and impulsive manifestations, as well as self-awareness impairments in patients with TBI. These findings underlined the heterogeneity of clinical manifestations for each behavioral disturbance and the diversity of psychological processes involved. In this context, new multidimensional approaches taking into account the various processes at play have been proposed to better understand and apprehend the complexity and dynamic nature of these problematic behaviors. In addition, the involvement of social and environmental factors as well as premorbid personality traits have increasingly been addressed. These new multidimensional frameworks have the potential to ensure targeted and effective rehabilitation by allowing a better identification and therefore consideration of the various mechanisms involved in the onset of problematic behaviors. In this context, the main objective of this position paper was to demonstrate the interest of multidimensional approaches in the understanding and rehabilitation of problematic behaviors in patients with TBI.

Published in : Annals of Physical and Rehabilitation Medicine (2016), n°59, pp. 18–22
DOI: 10.1016/j.rehab.2015.09.002
Status : Postprint (Author's version)

1. Introduction

In order to illustrate the multidimensional aspect of behavioral changes following traumatic brain injury (TBI), this literature review focused on the manifestations of apathy, impulsivity and anosognosia, which constitute the most common behavioral changes associated with a TBI. This article presents a brief descriptive introduction of each problematic behavior followed by a synthesis of the most recent studies that have investigated the nature of these behaviors. Regarding the methodology for articles search, authors focused on these three main behavioral changes, which represented the most published studies reporting the psychological processes involved. Then, in PubMed, authors selected the original articles in order to illustrate the first studies and original definitions, followed by a selection of the most recent and significant articles describing the multidimensional nature of these manifestations.

Changes in behaviors and emotional attitudes are common symptoms often described in persons with TBI, regardless of its severity. These manifestations can be quite diverse such as irritability, apathy, impulsivity or intolerance to change, and they often represent the biggest barrier to rehabilitation in the acute phase as well as to socioprofessional reintegration on the long term. [1]. Furthermore, these problematic behaviors are most often associated to manifestations of anosognosia, which makes care management as well as social, professional and familial reintegration even more difficult. Moreover, these changes have been related to negative consequences on the quality of life of patients but also of their closed ones. Indeed, behavioral and emotional changes exhibited by patients are better predictors of the subjective caregiver burden than injury severity or cognitive impairments [2].

Recently, Ciurli et al. [3] sought to characterize neurobehavioral changes among a group of 120 individuals with severe TBI. Using the Neuropsychiatric Inventory [4], the authors found that family caregivers reported a wide range of neuropsychiatric symptoms such as: apathy (42%), irritability (37%), dysphoria/depression (29%), disinhibition (28%), eating disorders (27%), agitation/ aggressiveness (24%), sleep disorders (15%), delusions (14%), euphoria and mania (13%), aberrant motor behaviors (9%), hallucinations (8%), and anxiety (8%). These data highlight the important proportion of problematic behaviors post-TBI, as well as the diversity of these manifestations. In clinical practice, these manifestations are most often related to executive function impairments due to brain damage, but increasingly data from the literature report that these manifestations are underpinned by a variety of mechanisms related in part to the brain damage (i.e. cognitive, psychoaffective impairments) but also to the patient's personality traits and its environment. A precise identification and consideration of these multiple mechanisms are much needed to promote adapted and effective care management for these patients.

The objective of this article was to illustrate the multidimensional aspect of these behavioral changes through two types of manifestations: apathy and impulsivity. Furthermore, anosognosia, which is often associated to these problematic behaviors in patients with TBI, can be expressed in different ways, suggesting here also the involvement of multiple processes.

Published in : Annals of Physical and Rehabilitation Medicine (2016), n°59, pp. 18–22
DOI: 10.1016/j.rehab.2015.09.002
Status : Postprint (Author's version)

2. Manifestations of apathy

Apathetic manifestations are commonly described after TBI [5] and have been associated with major negative consequences, especially regarding patients' participation in rehabilitation [6,7], family life [8] and later social reintegration [9]. The complaint from patients or family members is often ''a lack of initiative, some passivity, disinterest towards oneself and others, lack of spontaneous conversation or even emotional blunting''. Conceptually, there is some agreement within the literature that apathy refers to a set of behavioral, cognitive and emotional features. More precisely, disorders of interest, action initiation and emotional reactivity are all dimensions of apathy and diminished goal- directed behavior is at the core of the disorder [10]. However, current conceptions of apathy are based on descriptive and categorical approaches, without taking into account the several processes involved in each apathy manifestation as well as the interactions between these manifestations [11]. Thus, according to the definition proposed by Mulin et al. [12], a diagnosis of apathy can be made in the presence of diminished motivation in comparison to the patient's previous level of functioning, and at least two of the three following domains of apathy, which must be present for at least 4 weeks:

- diminished goal-directed behavior;
- diminished goal-directed cognitive activity;
- diminished emotions.

However, different studies have underlined significant relationships between the different dimensions of apathy, and specifically between lack of goal-directed behaviors (i.e. lack of initiative) and lack of goal-directed cognitive activity (i.e. lack of interest) [13-15]. These data are not surprising since a lack of interest is closely related to a lack of initiative and on the other hand, a lack of initiated actions may gradually lead to a lack of interest. More specifically, a recent study [16] conducted in 68 patients with TBI showed the heterogeneity of apathetic manifestations, by using the caregiver version of the Apathy Inventory [17]. Cluster analyses precisely identified four subgroups of patients: a group with high scores on all apathy dimensions, a group with low scores on all dimensions, a group with major emotional blunting and a group with high scores on lack of initiative and lack of interest. These data clearly indicate that apathy is not an accumulation of isolated symptoms but rather a dynamic concept with various associations and dissociations between the symptoms.

Furthermore, recent studies have shown the implication of different psychological mechanisms in the various facets of apathy. Thus, in elderly subjects, lack of initiative has been associated with difficulties in the ability to run multiple tasks simultaneously (''multitasking'') [18], difficulties in prospective memory [14] and also low self-efficacy beliefs [19]. To be more precise, Esposito et al. [18] showed that the number of rules breaks on the modified Six Elements Test [20] was a significant predictor of lack of initiative in persons with a diagnosis of Alzheimer's disease. In addition, the multitude of mechanisms involved in apathetic manifestations is supported by the diversity of brain structures related to apathy in persons with TBI. A literature review reported the

Published in : Annals of Physical and Rehabilitation Medicine (2016), n°59, pp. 18–22
DOI: 10.1016/j.rehab.2015.09.002
Status : Postprint (Author's version)

implication of a number of cortical and subcortical brain structures in the occurrence of apathetic behaviors such as the ventromedial prefrontal cortex, the lateral prefrontal cortex, basal ganglia, anterior cingulate cortex, insula and amygdala [11]. Based on all these data, a multidimensional and integrative concept of apathy was recently proposed, taking into account on the one hand the diverse mechanisms involved in the various dimensions of apathy, including cognitive factors (e.g. executive functions), motivational factors (e.g. effort mobilization), emotional factors (e.g. negative mood), aspects related to the personal identity (e.g. self-esteem) and on the other hand, the direct relations (at a symptomatic level) between the different manifestations of apathy [11].

3. Manifestations of impulsivity

Impulsivity, generally defined as the tendency to express spontaneous and excessive behaviors, has been commonly described in persons with TBI [21]. One of the significant conceptual advances should be attributed to the work of Whiteside and Lynam [22]. On the basis of a factorial analysis performed on the data of 400 students who were administered several impulsivity assessments tools, they identified four dimensions of impulsivity: urgency (the tendency to experience strong reactions, frequently under conditions of negative affects), the lack of premeditation (the difficulty to think and reflect on the consequences of an act before engaging in that act), lack of perseverance (the difficulty to remain focused on a task that may be boring or difficult) and sensation seeking (the tendency to enjoy and pursue activities that are exciting and openness to trying new experiences). This multidimensional model of impulsivity has recently been confirmed in a sample of patients with TBI with a short form of the UPPS Impulsive Behaviour Scale, which was specifically designed by Rochat et al. [23-25] to assess impulsivity changes after TBI. This scale includes 16 items (4 items per dimension) with a pre- and post-TBI evaluation for each item in order to evaluate impulsivity changes since the head injury. Results from validation studies have shown that the dimensions ''urgency'', ''lack of premeditation'' and ''lack of perseverance'' increased significantly after TBI, whereas the dimension ''sensation-seeking'' decreased significantly according to the patients' significant others.

In terms of psychological processes, sensation-seeking has been associated with motivational processes, whereas the other three impulsivity dimensions (urgency, lack of premeditation, lack of perseverance) were more related to self-control processes [26,27]. Thus, Rochat et al. [25] showed that the urgency dimension was significantly associated with dominant response inhibition difficulties in patients with TBI. The more patients had dominant response inhibition difficulties, the more they exhibited the urgency facet of impulsivity. Lack of perseverance was specifically associated with difficulties in mental flexibility and sustained attention in elderly subjects as well as difficulties in resisting proactive interference in working memory in healthy subjects [28]. Furthermore, a study conducted on university students showed a significant relationship between a low level of premeditation and poor decision-making capacities [29]. Regarding the motivational aspect of impulsivity, several studies have highlighted significant links between sensation-seeking and reward sensitivity. Besides, these four impulsivity dimensions have been related to different

Published in : Annals of Physical and Rehabilitation Medicine (2016), n°59, pp. 18–22
DOI: 10.1016/j.rehab.2015.09.002
Status : Postprint (Author's version)

behavioral disorders and/or psychopathological states [24,30]. Indeed, Rochat et al. [24] recently reported significant correlations between the urgency dimension of impulsivity and a tendency to compulsive buying in a group of 74 patients with moderate to severe TBI. More specifically, results showed that urgency was a significant predictor of compulsive buying and that these two problematic behaviors significantly predicted the subjective burden perceived by the caregivers. This study illustrates the existing relationships between the different types of problematic behaviors and the potential implications of common psychological mechanisms in the various behavioral changes in patients with TBI.

4. Manifestations of anosognosia

Anosognosia is a disorder characterized by the difficulty in being aware of deficits consecutive to brain damage. The manifestations of anosognosia are diverse and several dissocia¬tions exist since this disorder can affect one or several domains and be expressed on different severity levels [31]. The first studies on anosognosia focused mostly on anosognosia for hemiplegia after ischemic stroke because of the easy evaluation of this motor component. Based on these observations, Levine [32] formulated a ''discovery theory'' of anosognosia for hemiplegia, according to which cognitive deficits might contribute to denial by preventing the detection of limb weakness when combined with proprioceptive or sensory loss. More precisely, Gold et al. [33] explained the denial behavior by referring to a cognitive model of motor control (''feedforward'' hypothesis). In this model, anosognosia is assumed to be related to deficits of a comparator system that has to match the congruity between the intended movement and the sensory consequences of the actually executed movement.

Later on, studies have focused on other aspects of cognition and particularly on anosognosia for cognitive disorders and behavioral changes. Thus, Sherer et al. [34] reported that anosognosia post-TBI can also concern behavioral, cognitive and emotional disorders. More precisely, it appears that visible or objective disorders (e.g. phasic disorders) are better identified by patients than more abstract disorders (e.g. attention disorders). In this context, global approaches to anosognosia have been proposed such as the one by Crosson et al. [35] who described three hierarchical level of deficit awareness:

- intellectual awareness which represents a patient's ability to recognize his/her deficits or impaired functioning;
- emergent awareness which represents a patient's ability to detect their difficulties as they emerge in daily life;
- anticipatory awareness which represents the awareness of long-term consequences enabling to build realistic projects for the future.

Further, Toglia and Kirk [36] proposed a model taking into account other variables: ''off-line'' awareness namely metacognitive knowledge, encompassing the knowledge one has of its own functioning and ''online'' awareness corresponding to the anticipation abilities and self-

Published in : Annals of Physical and Rehabilitation Medicine (2016), n°59, pp. 18–22
DOI: 10.1016/j.rehab.2015.09.002
Status : Postprint (Author's version)

monitoring of task execution (supported by executive control abilities and error-monitoring). These two models suggest that anosognosia of cognitive and behavioral difficulties might be supported by different psychological mechanisms. Among the cognitive process, Coben et al. [37] showed in 14 patients with TBI that the executive processes of cognitive estimation, planning and word generation accounted for 61% of the anosognosia score variations on the Cognitive Estimation Test [38]. Furthermore, Bogod et al. [39] conducted a study on 45 patients with TBI, and found a significant and positive correlation between anosognosia as measured with the SADI scale [40] and difficulties of dominant-response inhibition and mental flexibility during two tasks of error monitoring and go no-go. These data suggest that a deficit in one of the various executive functions could be involved in the occurrence of anosognosia in patients with TBI. Furthermore, studies have also showed the involvement of other types of cognitive mechanisms such as the capacity to take into account the mental state of another person and to detect false beliefs [41]. Finally, emotional processes could also be involved as suggested by Spikman et al. [42] who demonstrated that anosognosia for executive difficulties was significantly associated with difficulties to recognize facial emotions of anger and fear in patients with TBI.

Although these diverse data illustrate the multidimensional nature of anosognosia after TBI, there is to date no theoretical model of anosognosia for cognitive impairments and behavioral changes that takes into account the various mechanisms at play and their interactions. Interestingly, however, a multidimensional framework of anosognosia for hemiplegia was proposed by Vuilleumier [43], which accounts for different subtypes of unawareness. This model states that anosognosia may result from a combination of deficits in three different processes: appreciation of a deficit, verification of the information in case of uncertainty and modification of previous beliefs related to the impaired function. On the basis of this model, a recent study using a riddle test showed that patients with high level of anosognosia for hemiplegia presented high level of confidence for questions with few informative content about the target word. In addition, patients with anosognosia tended to perseverate with a first wrong answer even when faced with the incongruency of subsequent clues [44].

Finally, the analysis of neural correlates suggest the implication of a diffuse brain network in the onset of anosognosia after TBI, contrary to anosognosia for hemiplegia that was related to focal lesions of the right hemisphere [45,46]. Among the cerebral regions involved in anosognosia after TBI, studies have identified the insula [47], cingulate cortex, precuneus as well as the anterior subcortical and medial temporoparietal regions [48]. Furthermore, the connections between these different brain regions might be vulnerable to diffuse axonal injury frequently reported after TBI. Indeed, this type of injury could lead to disconnections between the different brain regions needed for awareness and information processing [49]. For example, Dehaene et al. [50] reported that being aware of a mental effort was possible with a unique global ''workspace'', involving white matter tracts, which mainly regulate the functions of memory, attentional, perception and evaluation processes. All these data contribute to a better understanding of the multidimensional nature of these manifestations of anosognosia following TBI.

Published in : Annals of Physical and Rehabilitation Medicine (2016), n°59, pp. 18–22
DOI: 10.1016/j.rehab.2015.09.002
Status : Postprint (Author's version)

5. Discussion

This review underlines the large variability of behavioral changes after TBI. Indeed, most definitions acknowledge that the clinical expression of each problematic behavior comprises multiple facets. For instance, four impulsivity-related dimensions were identified as well as three clinical manifestations for apathy. Furthermore, experimental and clinical data showed the existence of relationships between the various manifestations of a single behavior, as seen previously for apathy between the lack of initiative and lack of interest dimensions. Interestingly, these data also uncover significant links between facets of different types of problematic behaviors. To be more precise, a study reported that patients with TBI with challenging behavior usually display multiple types of challenging behavior rather than a single isolated behavior [51]. These associations were even found between apparently contradictory behaviors such as apathy and impulsivity. Indeed, various studies conducted in patients with Parkinson's disease showed positive and significant correlations between apathetic and impulsive manifestations [52]. Besides, problematic behaviors are often associated with anosognosia, which makes particularly difficult the awareness of behavioral changes as well as their consequences on daily-life. The manifestations of anosognosia are also very diverse insofar as they depend on different levels of awareness.

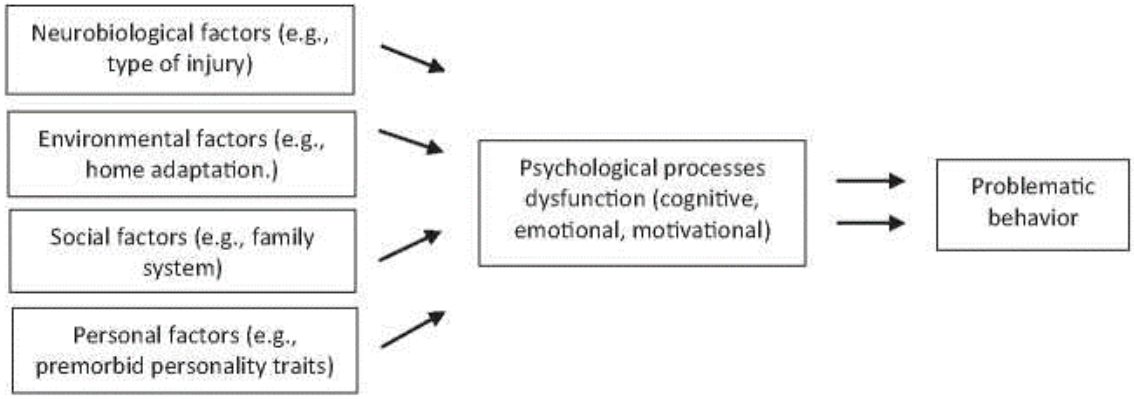

Fig. 1. *Diversity of factors involved in the occurrence of problematic behavior after traumatic brain injury.*

Based on the diversity of behavioral disturbances and the complexity of their interactions, the initial hypotheses involving a unique factor in the occurrence of these manifestations are clearly inadequate. Indeed, as seen previously, recent neuropsychological and neuroanatomical studies conducted in patients with apathy, impulsivity or anosognosia converge towards a multidimensional nature of these problematic behaviors, involving different cognitive, emotional, or motivational processes, but also aspects related to personal identity (Fig. 1). These findings also underline that some behavioural changes can result in similar overt behaviours while their etiologies may be heterogenous. All these data have led to the development of multidimensional models of neurobehavioral and self-awareness disorders that, contrary to categorical and symptomatic approaches, take into account the complexity and diversity of these changes. Such

Published in : Annals of Physical and Rehabilitation Medicine (2016), n°59, pp. 18–22
DOI: 10.1016/j.rehab.2015.09.002
Status : Postprint (Author's version)

new approaches have the potential to enable the development of more specific evaluation tools for a precise identification of these changes as well as the development of targeted and effective rehabilitation, focused on the person, at an early stage of rehabilitation.

Additional studies are required to refine these models, by identifying all the psychological mechanisms involved in each manifestation, as well as the interactions between these different mechanisms [11]. Furthermore, the exploration and consideration of other factors such as social, environmental and premorbid personality characteristics, seem important to better understand these problematic manifestations. Indeed, some studies showed that premorbid personality traits namely neuroticism or extraversion were significant predictors of post-TBI behaviors and could represent moderators between the severity of the TBI and interpersonal relationships [53-55]. In this sense, biopsychosocial approaches should be encouraged for the management of neurobehavioral difficulties, since these approaches apprehend behavioral changes as a result of a complex and dynamic interaction between neurobiological (e.g., type and severity of injury, time since injury), social (e.g. psychosocial history, family context), personal (e.g., medical history, personality traits, previous and current coping strategies, education-related beliefs) and environmental factors (e.g., problematic and anxiety-inducing situations related to the brain injury) relative to each person [56,57]. These approaches also take into account the family context, which can be quite disrupted due to behavioral and awareness changes [58-60]. In this context, it seems important to analyze the impact of neurobehavioral difficulties on the subjective burden perceived by family members in order to guide the patients' rehabilitation and promote in parallel adaptation strategies for family members.

Disclosure of interest

The authors declare that they have no competing interest.

References

[1] Meulemans T, Van der Linden M, Seron X, Juillerat AC. Évaluation des conduites émotionnelles, de la personnalité et de la motivation. In: Seron X, Van der Linden M, editors. Traité de neuropsychologie clinique, Tome1. Marseille: Solal; 2000. p. 301-17.

[2] Kreutzer JS, Gervasio AH, Camplair PS. Patient correlates of caregivers' distress and family functioning after traumatic brain injury. Brain Inj 1994;8:211-30.

[3] Ciurli P, Formisano R, Bivona U, Cantagallo A, Angelelli P. Neuropsychiatric disorders in persons with severe traumatic brain injury: prevalence, phenomenology, and relationship with demographic, clinical, and functional features. J Head Trauma Rehabil 2011;26:116-26.

[4] Cummings JL, Mega M, Gray K, Rosenberg-Thompson S, Carusi DA, Gornbein J. The neuropsychiatric inventory: comprehensive assessment of psychopathology in dementia. Neurology 1994;44:2308-14.

[5] Lane-Brown AT, Tate RL. Measuring apathy after traumatic brain injury: psychometric properties of the Apathy Evaluation Scale and the Frontal Systems Behavior Scale. Brain Inj 2009;23:999-1007.

Published in : Annals of Physical and Rehabilitation Medicine (2016), n°59, pp. 18–22
DOI: 10.1016/j.rehab.2015.09.002
Status : Postprint (Author's version)

[6] Gray JM, Shepherd M, McKinlay WW. Negative symptoms in the traumatically brain-injured during the first year post-discharge, and their effect on rehabilitation status, work status and family burden. Clin Rehabil 1994;8:188-97.

[7] Kant R, Duffy JD, Pivovarnik A. Prevalence of apathy following head injury. Brain Inj 1998;12:87-92.

[8] Marsh NV, Kersel DA, Havill JH, Sleigh JW. Caregiver burden at 1-year following severe traumatic brain injury. Brain Inj 1998;12:1045-59.

[9] Mazaux JM, Masson F, Levin HS, Alaoui P, Maurette P, Barat M. Long-term neuropsychological outcome and loss of social autonomy after traumatic brain injury. Arch Phys Med Rehabil 1997;78:1316-20.

[10] Marin RS. Apathy: a neuropsychiatric syndrome. J Neuropsychiatry Clin Neurosci 1991;3:243-54.

[11] Arnould A, Rochat L, Azouvi P, Van der Linden M. A multidimensional approach to apathy after traumatic brain injury. Neuropsychol Rev 2013;23:210-33.

[12] Mulin E, Leone E, Dujardin K, et al. Diagnostic criteria for apathy in clinical practice. Int J Geriatr Psychiatry 2011;26:158-65.

[13] Sockeel P, Dujardin K, Devos D, Deneve C, Destee A, Defebvre L. The Lille apathy rating scale (LARS), a new instrument for detecting and quantifying apathy: validation in Parkinson's disease. J Neurol Neurosurg Psychiatry 2006;77:579-84.

[14] Esposito F, Rochat L, Juillerat Van der Linden AC, Van der Linden M. Apathy and prospective memory in aging. Dement Geriatr Cogn Dis Extra 2012;2:456-67.

[15] Esposito F, Rochat L, Juillerat Van der Linden AC, Lekeu F, Charnallet A, Van der Linden M. Apathy in aging: are lack of interest and lack of initiative dissociable? Arch Gerontol Geriatr 2014;58:43-50.

[16] Arnould A, Rochat L, Azouvi P, Van der Linden M. Apathetic symptom presentations in patients with severe traumatic brain injury: assessment, heterogeneity and relationships with psychosocial functioning and caregivers' burden. Brain Inj 2015;1-7.

[17] Robert PH, Clairet S, Benoit M, et al. The apathy inventory: assessment of apathy and awareness in Alzheimer's disease. Parkinson's disease and mild cognitive impairment. Int J Geriatr Psychiatry 2002;17:1099-105.

[18] Esposito F, Rochat L, Van der Linden. et al. Apathy and executive dysfunction in Alzheimer disease. Alzheimer Dis Assoc Disord 2010;24:131-7.

[19] Esposito F, Gendolla GHE, Van der Linden M. Are self-efficacy beliefs and subjective task demand related to apathy in aging? Aging Ment Health 2014;18:521-30.

[20] Wilson BA, Alderman N, Burgess PW, Emslie H, Evans JJ. Behavioural assessment of dysexecutive syndrome. Saint-Edmunds, UK: Thames Valley Test Company; 1993.

[21] McAllister TW:. Neurobehavioral sequelae of traumatic brain injury: evaluation and management. World Psychiatry 2008;7:3-10.

[22] Whiteside SP, Lynam DR. The Five Factor Model and impulsivity: using a structural model of personality to understand impulsivity. Pers Individ Dif 2001;30:669-89.

[23] Rochat L, Beni C, Billieux J, Azouvi P, Annoni JM, Van der Linden M. Assessment of impulsivity after moderate to severe traumatic brain injury. Neuropsychol Rehabil 2010;20:778-97.

Published in : Annals of Physical and Rehabilitation Medicine (2016), n°59, pp. 18–22
DOI: 10.1016/j.rehab.2015.09.002
Status : Postprint (Author's version)

[24] Rochat L, Beni C, Billieux J, Annoni JM, Van der Linden M. How impulsivity relates to compulsive buying and the burden perceived by caregivers after moderate-to-severe traumatic brain injury. Psychopathology 2011;44: 158-64.

[25] Rochat L, Beni C, Annoni JM, Vuadens P, Van der Linden M. How inhibition relates to impulsivity after moderate to severe traumatic brain injury. J Int Neuropsychol Soc 2013;19:890-8.

[26] Bechara A, Van Der Linden M. Decision-making and impulse control after frontal lobe injuries (research support, NIH, extramural review). Curr Opin Neurol 2005;18:734-9.

[27] Billieux J, Rochat L, Van der Linden M. Une approche cognitive, affective et motivationnelle de l'impulsivité. In: Van der Linden M, Ceschi G, editors. Traite de psychopathologie cognitive, TomeI. Marseille: Solal; 2008. p. 137-52.

[28] Gay P, Rochat L, Billieux J, d'Acremont M, Van der Linden M. Heterogeneous inhibition processes involved in different facets of self-reported impulsivity: evidence from a community sample. Acta Psychol (Amst) 2008;129:332-9.

[29] Zermatten A, Van der Linden M, d'Acremont M, Jermann F, Bechara A. Impul- sivity and decision-making. J Nerv Ment Dis 2005;193:647-50.

[30] Miller J, Flory K, Lynam D, Leukefeld C. A test of the four-factor model of impulsivity-related traits. Pers Individ Dif 2003;34:1403-18.

[31] Vuilleumier P. Anosognosia. In: Bogousslavsky J, Cummings JL, editors. Behavior and mood disorders in focal brain lesions. Cambridge, UK: Cambridge University Press; 2000. p. 465-519.

[32] Levine DN. Unawareness of visual and sensorimotor defects: a hypothesis. Brain Cogn 1990;13:233-81.

[33] Gold M, Adair JC, Jacobs DH, Heilman KM. Anosognosia for hemiplegia: an electrophysiologic investigation of the feed-forward hypothesis. Neurology 1994;44:1804-8.

[34] Sherer M, Boake C, Levin E, Silver BV, Ringholz GM, High WM. Characteristics of impaired awareness after traumatic brain injury. J Int Neuropsychol Soc 1998;4:380-7.

[35] Crosson B, Barco PP, Velozo CA. Awareness and compensation in postacute head injury rehabilitation. J Head Trauma Rehabil 1989;4:91-6.

[36] Toglia J, Kirk U. Understanding awareness deficits following brain injury. Neuro Rehabil 2000;15:57-70.

[37] Coben RA, Boksenbaum SI, Kulberg AM. Cognitive determinants of unawareness of deficits: the importance of specific frontal-mediated executive functions. Arch Clin Neuropsychol 1995;10:309-10.

[38] Coben RA, Boksenbaum SI, Kulberg AM. The cognitive estimation test: a potential new measure of frontal-mediated executive functioning. Arch Clin Neuropsychol 1995;10:310-1.

[39] Bogod NM, Mateer CA, MacDonald SW. Self-awareness after TBI: a comparison of measures and their relationship to executive functions. J Int Neuropsychol Soc 2003;9:450-8.

[40] Fleming J, Strong J, Ashton R. Self awareness of deficits in adult with traumatic brain injury: how best to measure? Brain Injury 1996;10:1-15.

[41] Bivona U, Riccio A, Ciurli P, et al. Low self-awareness of individuals with severe traumatic brain injury can lead to reduced ability to take another person's perspective. J Head Trauma Rehabil 2014;29:157-71.

Published in : Annals of Physical and Rehabilitation Medicine (2016), n°59, pp. 18–22
DOI: 10.1016/j.rehab.2015.09.002
Status : Postprint (Author's version)

[42] Spikman JM, Milders MV, Visser-Keizer AC, Westerhof-Evers HJ, Herben-Dekker M, van der Naalt J. Deficits in facial emotion recognition indicate behavioral changes and impaired self-awareness after moderate to severe traumatic brain injury. Plos One 2013;8:1-7.

[43] Vuilleumier P. Anosognosia: the neurology of beliefs and uncertainties. Cortex 2004;40:9-17.

[44] Vocat R, Staub F, Stroppini T, Vuilleumier P. Anosognosia for hemiplegia: a clinical-anatomical prospective study. Brain 2010;133:3578-97.

[45] Feinberg TE, Haber LD, Leeds NE. Verbal asomatognosia. Neurology 1990;40:1391-4.

[46] Desmurget M, Reilly KT, Richard N, Szathmari A, Mottolese C, Sirigu A. Movement intention after parietal cortex stimulation in humans. Science 2009;324:811-3.

[47] Karnath HO, Baier B, Nagele T. Awareness of the functioning of one's own limb mediate by the insular cortex? J Neurosci 2005;25:7134-8.

[48] Ham TE, Bonelle V, Hellyer P, et al. The neural basis of impaired self-awareness after traumatic brain injury. Brain 2013;137:586-97.

[49] Geschwind N. Disconnexion syndromes in animals and man. Brain 1965;88: 237-94.

[50] Dehaene S, Kerszberg M, Changeux JP. A neuronal model of a global workspace in effortful cognitive tasks. Proc Natl Acad Sci U S A 1998;95:14529-34.

[51] Kelly G, Brown S, Todd J, Kremer P. Challenging behaviour profiles of people with acquired brain injury living in community settings. Brain Inj 2008;22: 457-70.

[52] Sinha N, Manohar S, Husain M. Impulsivity and apathy in Parkinson's disease. J Neuropsychol 2013;7:255-83.

[53] Sela-Kaufman M, Rassovsky Y, Agranov E, Levi Y, Vakil E. Premorbid personality characteristics and attachment style moderate the effect of injury severity on occupational outcome in traumatic brain injury: another aspect of reserve. J Clin Exp Neuropsychol 2013;35:584-95.

[54] Malec JF, Brown AW, Moessner AM. Personality factors and injury severity in the prediction of early and late traumatic brain injury outcomes. Rehabil Psychol 2004;49:55-61.

[55] Rusk BK, Malec JF, Brown AW, Moessner AM. Personality and functional outcome following traumatic brain injury. Rehabil Psychol 2006;51:257-64.

[56] Zasler ND, Martelli MF, Jacobs HE. Neurobehavioral disorders. In: Barnes M, Good D, editors. Handbook of neurology. New York: Elsevier; 2013. p. 377-88.

[57] McAllister TW. Neuropsychiatric aspects of TBI. In: Zasler N, Katz DI, Zafonte RD, editors. Brain injury medicine. New York: Demos Medical Publishing, Inc.; 2007. p. 815-33.

[58] Jumisko E, Lexell J, Soderberg S. Living with moderate or severe traumatic brain injury: the meaning of family members' experiences. J Fam Nurs 2007;13:353-69.

[59] Serio C, Kreutzer J, Gervasio A. Predicting family needs after traumatic brain injury: implications for intervention. J Head Trauma Rehabil 1995;10:32-45.

[60] Ennis N, Rosenbloom BN, Canzian S, Topolovec-Vranic J. Depression and anxiety in parent versus spouse caregivers of adult patients with traumatic brain injury: a systematic review. Neuropsychol Rehabil 2013;23:1-18.