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Emotion regulation after acquired brain injury

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His absorption in the momentary impression is practically complete, so that the emotional response to it also unfolds to its maximum, unhampered by any competing mental content.

Hanfmann, Rickers-Ovsiankina & Goldstein,

Case Lanutti: Extreme concretization of behavior due to damage of the brain cortex

I propose that thoughts should be regarded as prior to the apparatus for using thoughts.

Wilfred Bion, *Elements of Psychoanalysis*

Contents

Abstract

Emotion dysregulation is a common phenomenon after brain injury, often compromising socio-emotional adjustment and participation. Nevertheless, there has been little research exploring the mechanisms by which brain damage impacts emotion regulation [ER]. In contrast, outside the field of neuropsychology, the study of ER has matured during the last decade, generating a robust body of evidence on the strategies that people use to modulate their feelings. The main goal of this thesis is to bring together, for the first time, these two fields of knowledge. Chapter two presents three articles touching key conceptual issues, such as the description of self-regulation and self-other regulation problems after brain injury, the relationship between neuropsychological profiles of impairment and ER strategies deficits, and the impact of concrete behaviour on emotional experience. Chapter three explore the problem of emotion elicitation and emotional reactivity. In two articles, the efficacy of internal and external forms of elicitation is explored on a student sample [$n = 40$], as well as compared between people with right hemisphere [RH] damage and matched healthy controls [RH: $n = 10$, HC: $n = 15$]. The main finding of both studies is that internal elicitation procedures generate higher levels of subjective reported emotion across populations of different age. In addition, patients with RH damage present similar levels of emotional reactivity compared to controls. Chapter four explores how specific ER strategies are compromised by focal brain injury. In the first study, people with RH frontal lesions [$n = 10$] were compared to healthy control [$n = 15$] on a response modulation task. It was found that RH patients were impaired voluntarily manipulating emotional facial expressions, and that a subgroup of RH patients was unable to inhibit emotional displays. The second study explored the impact of unilateral lesions in the capacity to reappraise [RH: $n = 8$, LH: $n = 8$, HC: $n = 14$]. Individuals with RH and LH lesions were equally slow, compared to controls, generating reappraisals. However, when time was not considered, both groups were equally productive. Finally, Chapter five uses a single case study methodology to explore the mechanism by which ER, and particularly reappraisal, is disrupted after left prefrontal lesions. Here, two articles offer important insight into how concreteness and executive impairment are associated to emotion dysregulation, and the mechanisms by which such dysregulation can be externally compensated.

Chapter 1: Introduction

1. Introduction

In the last decade the field of neuropsychological rehabilitation has notably turned towards models that emphasize socio-emotional adjustment and participation (Bowen, Yeates, & Palmer, 2010; Wilson, 2008; Wilson, Gracey, Evans, & Bateman, 2010). This paradigm shift appears to respond to the increased awareness of the limits of 'cognitive' rehabilitation (e.g. Wilson, 1997), a greater understanding of the relevance of emotion in rehabilitation (e.g. Mateer, Sira, & O`Connell, 2005), a better comprehension of how positive activities shape identity reconstruction (e.g. Ylvisaker & Feeney, 1998, 2000), and the recognition that family environment modulates cognitive impairment (e.g. Yeates, Henwood, Gracey, & Evans, 2007) and can be key to emotional well-being (e.g. Bowen et al., 2009; Hammond, Davis, Cook, Philbrick, & Hirsch, 2012).

In this context, the study of how brain injury compromises socio-emotional capacities has acquired greater relevance, focusing on changes to emotional *perception* and *expression* (e.g. Borod, Bloom, Brickman, Nakhutina, & Curko, 2010), as well as deficits in *emotional reactivity* and *empathy* (e.g. Bramham, Morris, Hornak, Bullock, & Polkey, 2009; Shamay-Tsoory, Tomer, Goldsher, Berger, & Aharon-Peretz, 2004; Sousa, Mcdonald, & Rushby, 2012). In addition, an important literature on the neuropsychiatric effects of brain injury has developed, describing how lesions to diverse brain areas may generate a wide range of psychiatric syndromes (e.g. Chemerinski & Levine, 2006; Robinson, 2006).

It is interesting that, in this scenario, research on ER has been so sparse[1]. Particularly, considering that ER problems have been widely reported as a common impairment after focal and diffuse brain damage (e.g. Abreu et al., 2009; Bechara, 2004; McDonald, Hunt, Henry,

[1] A PsycINFO 'all time' search of publications with the terms emotion regulation + brain injury/brain damage/traumatic brain injury [in abstract], showed only three scientific articles.

Dimoska, & Bornhofen, 2010; Obonsawin et al., 2007; Wilson, Alderman, Burguess, Emslic, & Evans, 1996). Such state of the art appears to be the consequence of two problems. The first one is the absence of a comprehensive theoretical framework on ER to explore changes after brain injury. There is only one publication, a book chapter (Beer & Lombardo, 2007), which considers well known ER models -used in experimental psychology- to explain emotion dysregulation after brain damage. The second problem is the predominance of a neuropsychiatric perspective in understanding emotion dysregulation (e.g. Cattran, Oddy, & Wood, 2011). The limitation of such a perspective is that it tends to reduce emotion dysregulation to the presence of psychiatric symptoms. This, as it will be discussed in chapter 5, is not always the case. Emotion dysregulation events may occur in people with acquired brain injury, even when they do not meet any psychiatric criteria. A key proposition of this thesis is that a better comprehension of the psychological mechanisms that underlie emotion dysregulation, and how they are related to neuropsychological impairment, is a more useful way to understand ER change after brain damage.

Outside the field of neuropsychological and neuropsychological rehabilitation, the concept of ER has acquired vast popularity [see Fig 1], mostly because of a robust set of evidence linking it to psychopathology (e.g. Aldao, Nolen-Hoeksema, & Schweizer, 2010; Werner & Gross, 2010) and well-being (e.g. Nyklícek, Vingerhoets, & Zeelenberg, 2011).

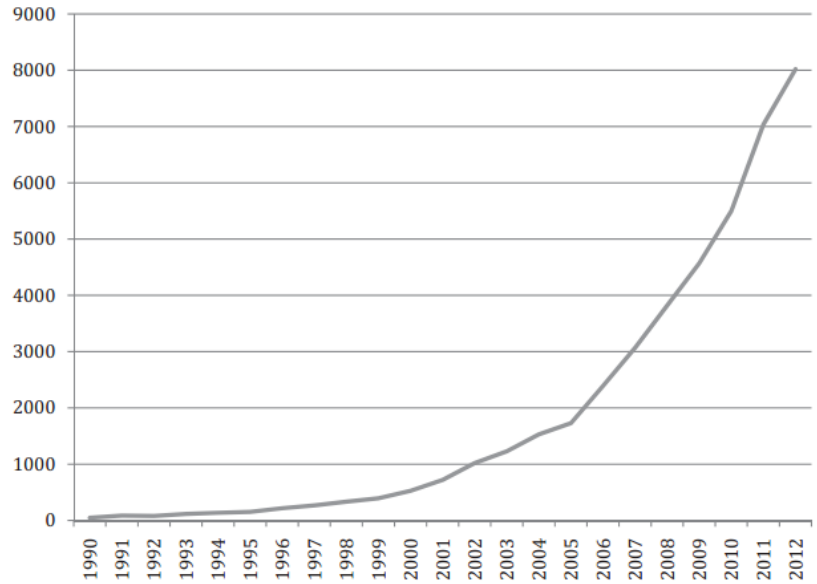

Fig. 1 Number of publications containing the exact phrase emotion regulation in Google Scholar, from 1990-2012 (Gross, 2013).

In this context, ER has been defined as the set of processes by which individuals can influence *which* emotions they have, *when* they have them, and *how* they experience and express those emotions (Gross, 1998). This definition refers mainly to the *intrapersonal* processes [intrinsic ER] that people use to modulate how they feel [see Fig 2]. However, a set of *interpersonal* processes [extrinsic ER], that individuals use to mutually regulate their emotional states has also been described (Gross & Thompson, 2007; Niven, Totterdell, & Holman, 2009). Both forms of ER [intrinsic and extrinsic] appear to be highly relevant when exploring emotion dysregulation after brain injury (Salas, 2011). Nevertheless, the literature using Gross' model of ER in patients with acquired brain injury is almost non-existent (Beer & Lombardo, 2007; Gillihan et al., 2010; McDonald et al., 2010; Salas, Gross, Rafal, Viñas-Guasch, & Turnbull, 2013).

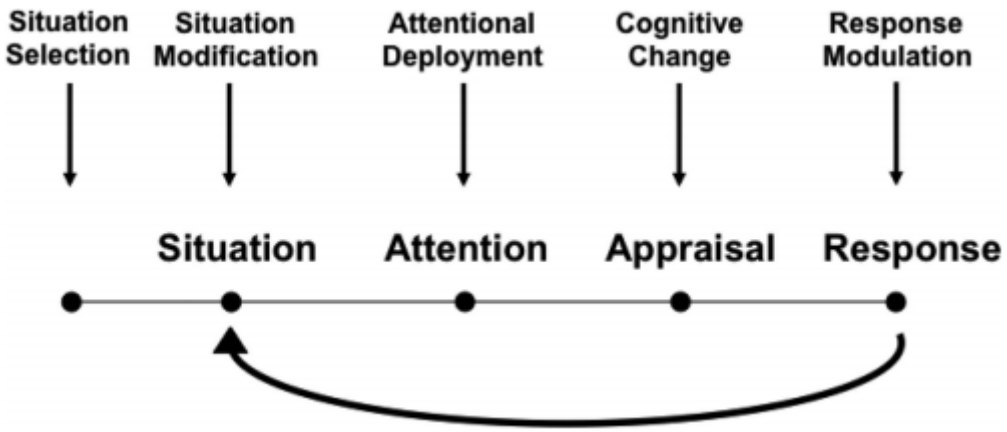

Fig 2. James Gross' models of Emotion Regulation. The five emotion regulation strategies proposed by the model are presented at the top of the figure. The first four ER strategies [from left to right] are often called *antecedent focused*, for they are implemented before the emotional response is triggered. The last ER, response modulation, is considered as *response focused*, for its main goal is to modulate an already triggered emotional reaction.

The main goal of this thesis is to bridge the *conceptual* and *experimental* gap between the fields of neuropsychology [particularly neuropsychological rehabilitation] and ER. This task is complex, and presents important challenges, for these two fields have never been systematically integrated. The structure of this thesis reflects such complexity, and constitutes an attempt to capture the problem from different perspectives.

As a main strategy, this thesis has adopted a multilevel approach [see Fig 2], where theory development, group studies, and single case studies, are equally important and inform each other. Such an approach has been a characteristic feature of neuropsychological research, particularly when charting new psychological processes (Shallice, 1988). Theorico-clinical studies, and case studies, allow the formulation of hypotheses on how ER and brain injury may relate. Theorico-clinical studies draw such hypotheses from clinical observations of brain injured patients, and also from a robust body of evidence on the neuropsychological consequences of focal brain damage. Single case studies, on the other hand, generate hypotheses based on the detailed report of how ER is disrupted in one single subject, together

with the description of the neuropsychological mechanisms that appear to underlie such impairment. The design of group studies is influenced by these two sources of information. The main goal of the group approach is to test whether theoretical predictions, and case observations, are replicated and generalized to the brain injury population. A final dimension of this multilevel approach are the methodological studies, which explore how the tools used to elicit and measure emotion may be influenced by brain injury. This is particularly relevant in order to understand the relationship between emotion reactivity and emotion regulation.

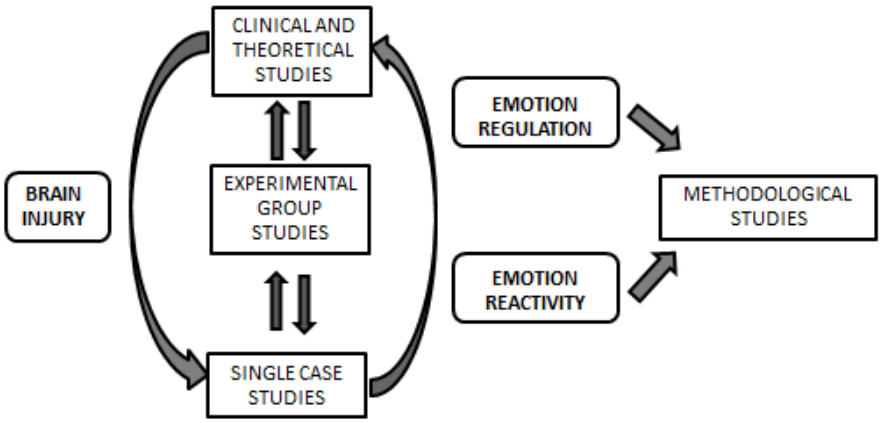

Fig 2. Multilevel approach to the study of ER after acquired brain injury.

The structure of this thesis reflects the complexity of a multilevel approach. There are four chapters, each of them corresponding to a different methodology to address the problem of ER and brain injury [Review Articles, Groups Studies, Single Case Studies and Methodological Articles]. In addition, each chapter consists of a set of related articles. Most

of the articles in this thesis have already been published in scientific journals, while others are under review or in preparation.

Chapter 2 [*Review Articles*] addresses several theorico-clinical issues of the brain injury/ER interface. The first paper of this section (Salas, 2012) describes how intrinsic and extrinsic regulatory processes are intimately related in brain injury, specifically in the modulation of the well-known catastrophic reaction (Goldstein, 1995). Another theoretical problem addressed in this chapter is whether specific subgroups of patients with focal lesions [e.g. ventromedial PFC damage] may be selectively impaired in a particular ER strategy [e.g. situation selection] (Salas, Gross, & Turnbull, submitted). Finally, the third paper (Salas, Vaughan, Shanker, & Turnbull, 2013) describes the often neglected problem of concrete behaviour, and the challenges that it poses to neuropsychological rehabilitation and the use of psychological therapies. This article offers a conceptual background for a case study on concrete behaviour and emotion regulation impairment (see section 5, Salas et al., 2013)

Chapter 3 [*Methodological articles*] addresses a key problem for ER research: emotion elicitation (Coan & Allen, 2007). The first article (Salas et al., 2011) compares the effectiveness of two mood induction procedures [MIPs]: film clips and autobiographical recall, in a student sample. The effectiveness of such MIPs is crucial to ER research, for ER can only be measured when there is an emotional response that has been triggered, and requires modulation. A related methodological problem is treated in the second article of this chapter. It explores whether people with unilateral right hemisphere lesions experience lower levels of positive and negative emotion [as measured by self-reports], when using two types of MIPs [film clips and autobiographical recall]. This issue is relevant because a decrease in emotional reactivity would impact emotion regulation, for lower levels of emotional intensity would require less regulatory effort.

Chapter 4 [*Group studies*] focuses on the neuroanatomical and neuropsychological basis of two emotion regulation strategies: response modulation and reappraisal. By doing this, two hypotheses proposed in the Review Articles section [chapter 4] and in the Single Case studies section [chapter 5] are tested. The first article explores the role of the right prefrontal cortex in ER, specifically in relation to one well known ER strategy: response modulation. This article is the first to compare the performance of a group of patients with right prefrontal lesions with a group of matched healthy controls on a response modulation task. In addition, the relationship between response modulation performance and neuropsychological measures of inhibition is examined. The second article addresses a related issue, which is the role of laterality in reappraisal generation capacity. This article explores this issue by comparing reappraisal generation capacity in participants with unilateral damage to the left and right hemisphere, and healthy controls. The relationship between reappraisal generation and specific cognitive control abilities is also described here.

Chapter 5 [*Single case studies*] expands upon the theory developed in chapter 2, and the group studies presented in chapter 4, particularly in relation to the use of reappraisal. However, each of the case studies addresses the problem from a different perspective. The first article (Salas et al., 2013) analyses in detail the performance of a participant with a left frontal stroke [Mrs M], who presented with a remarkable inability to generate reappraisals [and was one participant in a group study, chapter 4]. This case is especially interesting because it discusses the potential impact of concreteness, or the inability to disengage from immediate experience, in the use of reappraisal. The second case (Salas, Radovic, Yuen & Turnbull, in preparation) offers a different viewpoint of the problem. It describes how executive impairment [particularly the difficulty to generate thoughts] modifies emotional reactivity and emotion regulation, this in a man who suffered a left fronto-parietal stroke [Professor F]. An interesting feature of this case study is that it uses a mixed methodology to

assess changes in several emotional processes. This article also describes, and discusses, the interpersonal mechanisms used by Professor F to modulate his negative emotional states.

The final section of the thesis [Discussion] will bring together the diverse contributions of each article, synthesising the major themes and strands of evidence. By doing this, it is expected to offer a more comprehensive view of how emotion regulation and brain injury are related. The theoretical and practical implications of this research will also be discussed.

Chapter 2: Review Articles

2.1. Surviving Catastrophic Reaction after brain injury: the use of Self-regulation and Self-Other regulation[2].

2.1.1. Abstract

Catastrophic reactions are usually observed after brain injury. Although Kurt Goldstein described this concept more than fifty years ago, it has not been adequately incorporated in the theoretical and clinical field of neuropsychological rehabilitation. In the following paper a number of cases where patients experience catastrophic reactions are illustrated, as well as the intrapersonal and interpersonal mechanisms that facilitate the regulation and reorganization of the self. It is proposed that catastrophic reaction activates attachment mechanisms by default, mobilizing the patient to look for a significant other to regulate himself from outside. This paper also describes how high levels of arousal, triggered by a catastrophic reaction during interpersonal conflict, impact on mentalizing, which is a self-regulatory function that generates representations of our own and other's mental states. Finally, it is suggested that lesions to different brain areas might impair specific Self-regulatory and Self-Other regulatory components, requiring the adaptation of psychotherapeutic technique to these particular profiles. Examples of the modification of psychotherapeutic technique with the patient and the family are provided.

[2] Salas, C. E. (2012). Surviving Catastrophic Reaction after Brain Injury: The use of Self-Regulation and Self-Other Regulation. *Neuropsychoanalysis*, 14(1): 77-92

"The most devastating loss of all becomes a loss of a meeting of the minds"

Paula Freed

2.1.2. A peculiar early memory

Two years after his stroke, Professor F, a 70-year-old renowned academic, arrived for his weekly psychotherapy session. He sat on the couch and said he wanted to read something he had written. His occupational therapist had suggested that he train his right hand (affected by two large left frontal-parietal strokes) by writing stories. "At first I thought about writing stories -he said- so my sons could tell my stories to their sons, as I did by telling them stories about my father. But I have decided to write the story of my accident, and I'd like you to listen about the first memory I had. When I opened my eyes I found myself in a room. Now I know it was a hospital's room. Now I know I was there because something in my head was broken, but at that time I couldn't think about it, *I couldn't think about anything at all*. It is almost impossible to describe that experience; I just can say that my mind was in silence, like a 'pap'". At that moment I asked Professor F whether that 'pap' meant feeling his mind empty. "No –he replied- neither I could say it was a void, it was just a sensation in my head, a 'pap'. If I say it was a silence, I am not sure the word 'silence' is right, or if the sensation I had then is related to something known. My second memory is seeing my whole family standing beside my bed; my kids, my wife. That *calmed me down* and *I felt a great serenity*. Everything was going to be all right".

Accessing so clearly to the early experience of a brain injury survivor is something unusual. Cognitive deficits frequently impede remembering or reconstructing the initial events (Grotta & Bratina, 1995). This makes Professor F's report very interesting. Moreover, his testimony is relevant because it illustrates the interaction between the insufficiency of cognitive resources to deal with environmental demands –*I could not think about anything at*

all- and the spontaneous search for others' support –*seeing my family calmed me down and I felt a great serenity*.

Brain injury survivors usually describe the experience of cognitive deficits as having a "disordered" mind (Prigatano, 1991), a situation that is emotionally lived as a disorganized inner state or a psychological catastrophe (Goldstein, 1995 [1965]). A further consequence of having a "disordered mind" is that the mechanisms that allow recovery from intensely negative emotional states, in other words, self-regulatory mechanisms, are compromised (Freed, 2002). In more tragic circumstances, brain damage may also impair the capacity to use others as a source of external regulation, for example by compromising the ability to emotionally connect to others (Bowen, Yeates & Palmer, 2010). The aim of this article is to draw a preliminary theory of these regulatory processes after brain injury, thus informing the assessment and design of interventions in rehabilitation settings.

2.1.3. Catastrophic Reaction

Let's consider the following situations frequently observed in acquired brain injury (ABI) survivors. When I ask Mrs. N, a vascular dementia patient, to tell me where she is now, and what the date is, she becomes restless. Her body starts shaking on the couch and her face is distorted as she tries to organize her thoughts among confabulations and disperse ideas. A couple of minutes later the agitation increases and she desperately asks to leave the room, mumbling that she has something to do or that she needs to go to the toilet. Mr. C, a young man with large strokes in the middle cerebral and left anterior arteries, cannot transform his thoughts into words. Mr. C gets agitated; his face is deformed by the anxiety caused by my questions and he constantly turns his head toward his wife looking for help. He says, "I don't… I don't know…I have it on the tip of my tongue… no…no…I can't". Or consider Professor F and how he writes down his ideas before attending to a work meeting,

saying that by doing so he will avoid forgetting what he has to say and becoming confused. These are stories of patients with widespread localized brain injuries and heterogeneous cognitive deficits. However, they share a common factor, the kind of emotional reaction they experience when they fail to respond to environmental demands. In the first two cases confusion and anguish take over. In the last one, anticipatory anxiety and preparation predominate, as a way of avoiding confusion and anguish.

Kurt Goldstein (1995 [1965]) called these anguish and confusion responses to environmental demands as catastrophic reactions:

> "In catastrophic reaction the individual enters into a *disorganized inner state*, experiencing a physical and mental shock, feeling unfree, buffeted and vacillating. But this shock does not just impact his own person; it also provokes a *collapse* in his experience of the surrounding world. The state of anxiety caused by the catastrophic reaction impacts the survivor's functioning for a long period of time, also affecting tasks that he/she could accomplish easily under other circumstances. On the contrary, under an organized situation, the organism's responses are constant, correct, and adequate to the organism to which they belong, as well to its specific circumstances. The individual himself experiences them with a feeling of smooth functioning, unconstraint, well-being, adjustment to the world and satisfaction. In other words, behavior has a clear order, a global pattern that aligns all the organism's components –somatic and psychic- in a particular task" [pp. 48-49].

Goldstein's conceptualization of catastrophic reaction is rooted in the profound understanding of how brain injury may shatter the coherence and continuity of the Self. According to Goldstein, *coherence* refers to the proper alignment of the organism's somatic and psychological components during a task. Coherence may be disrupted after brain injury, compromising the integration of different levels of functioning, as in patients with prefrontal lesions, who struggle to "do" what they "know" (Stuss et al., 2002; Stuss & Alexander,

2007). On the other hand, *continuity* denotes the uninterrupted [and fluid] updating of representations over time (Modell, 1993; see also Edelman, 1992, 2006). This property may also be disrupted after brain injury, generating "delays" and "fissures" in the coupling between subject and environment.

Contemporaneous authors, such as Ben-Yishay (2000), have approached the catastrophic reaction in a similar fashion to Goldstein, defining it as the "behavioral manifestation of a threat to the person's very existence" (p. 128). On the contrary, the neuropsychiatric literature has tended to use the term in a much-reduced way, narrowing it to inadequate outbursts of frustration, depression or anger (Carota et al., 2001; Chemerinski & Robinson, 2000; Starkstein et al., 1993). This trend to understand the catastrophic reaction as a psychopathological syndrome appears to disregard the original meaning of the concept. Goldstein approach to the catastrophic reaction is not neuropsychiatric, but highly phenomenological, *describing* how survivors experience themselves and the world when their *whole* organism is under a disorganized functioning (Goldstein, 1959). This emphasis on understanding the patient's experience, versus placing it on a specific nosology, is a familiar point of view for rehabilitation clinicians, who try to comprehend patients from *inside* as the first step of the rehabilitation process (Prigatano, 1999). Such clinical approach has led many psychotherapists to adopt a Self-Psychology perspective, rather than a neuropsychiatric one, preserving catastrophic reaction original meaning as a *disruption* (loss of coherence and continuity) in the sense of self (Klonoff, Lage & Chiapello, 1993; Klonoff, 2010; Prigatano, 1999; Salas & Turnbull, 2010).

2.1.4 Self-regulation strategies used to deal with catastrophic reaction after brain injury.

But how does a survivor deal with the persistent threat of entering into a disorganized state? In Mrs. N's case she seems to feel safe only when she is close to her sister, whom she is able to recognize and look for actively. Her sister comments that the only place where Mrs. N feels calmed is in her bedroom, reading the same magazines and doing the same routine over and over. Professor F, who still possesses numerous capacities despite his moderate executive dysfunction, exhibits more complex behaviors, although the goal remains identical. Each visit to an unknown place is carefully prepared and supported by his family. And after several repetitions, without many variations, a sense of control and calmness is finally conquered. However, there is a persistent look of alarm in his face, an expression of the difficulty he has reacting flexibly to environmental variations.

In light of these clinical observations it would not seem strange to suggest that survivors deal with catastrophic reaction's threat through modification of environmental demands. These modifications are made using two main strategies: changes in the patient's behavior and the search for cognitive/emotional support from significant others. Regarding the first strategy, Goldstein (1995 [1965]) states that the survivor will try to stay away from situations that could generate catastrophic reactions (see Riley et al., 2004 for a list of these situations in Traumatic Brain Injury), passively isolating himself from the world, or actively avoiding it, according to certain basic criterion extracted from successive exposure to the environment. In some situations the patient may escape exhibiting 'defensive' behaviors, which would be nothing more than a way of keeping the situation under control. Although these behaviors (watching TV for hours, staying away from other people, compulsively asking to be taken to the toilet) might look stereotyped, or rigid to an external observer, they

always play a crucial role in keeping environmental demands under control, and thus preserving a basic sense of mastery. As Goldstein himself has affirmed, "at a certain stage of disintegration these substitute actions are the last resource, the only means by which existence can be maintained. In this sense they are meaningful; they enable the organism to come to terms with the environment, at least in some way" [p. 52].

Another behavioral strategy brain injury survivors use to avoid catastrophic reactions is the *tendency toward orderliness*. Disorder demands choice of alternatives, change of attitude, and rapid transition from one behavior to another, exactly what is difficult or impossible for survivors to do (Goldstein, 1995 [1965]). Thus, a conversation among three or more people becomes a hard and frustrating task for Professor F; sudden topic changes, unexpected interruptions and jumps between present, past and future scenarios are experienced as a confusing and exhausting chaos. However, when you are alone with Professor F he manages to communicate in a smart and sensible way. This especially takes place when he feels calm and knows that people will wait until his ideas *emerge*. He tries very hard to keep his discourse ordered and linear, as a way of preserving the words that may allow him to describe how he feels. To an external observer his careful and detailed narrative would reflect nothing more than concreteness and lack of flexibility. But the truth is that his evident order is also a way of protecting his valued, but scarce thoughts, which occasionally refuse to emerge, or when they do, rapidly fade away. Goldstein points out that in order to avoid the anguish caused by disorganized situations, patients hold tenaciously to orderliness in a seemingly primitive, rigid and compulsive fashion. This is Professor F's order, an order that stands as the only way of preserving the possibility of looking at himself. An order that when lost generates a rather visceral and non-represented type of experience (e.g. somatic pain, heightened arousal), as well as mental states described by himself as *turbid* and *perplexing*. In those disorganizing moments Professor F refers to feeling paralyzed,

experiencing something he does not understand, something that is so intense that it even blocks the ability to comprehend what is going on. Catastrophic reaction ultimately means a *disruption* in the continuity of existence, a falling, a displacement from the center.

2.1.5. Self-Other regulation: Attachment system activation as the search for inner world's order.

So far, I have suggested that ABI survivors organize their external environment to avoid experiencing the internal disorganization that the catastrophic reaction entails. It seems necessary to elaborate further on what the subjective experience of *disorganization* is and may mean. Goldstein himself associates the catastrophic reaction with *severe* anxiety (Goldstein, 1959), an observation that has been supported by more contemporaneous authors. For example, Freed (2002) has pointed out that the anxiety experienced by survivors is not the kind of anxiety that emerges as a signal of danger or conflict (signal anxiety according to Freud, 1926 [1959]), but a more primitive one, referred in the psychoanalytical literature as *fragmentation anxiety* (Klonoff, Lage & Chiapello, 1993; Kohut & Wolf, 1978; Winnicott, 1996 [1962]). Here, mental states, which are sustained by global patterns of brain activation that underlie the brain's cohesive functioning (Siegel, 1999), are disrupted. In some patients the emergence of this primitive anxiety can be observed as a loss of reality testing capacity (Mr. D in Kaplan-Solms & Solms, 2002). In others, who have more residual resources, it may be experienced as boundless panic (e.g. patient V.O. in Dewar & Gracey, 2007; patient P. in Gracey et al., 2007).

Defining catastrophic reaction as the absence of a unifying mental state which allows smooth transactions between organism and world, or the disruption of the self's coherence and continuity, forces us to consider the regulatory mechanisms by which the organism recovers its homeostasis. I have described above how survivors manage to regulate

themselves by ordering their behavior and environment, now I will illustrate the interpersonal mechanisms that allow the same goal.

In adult human beings, the self-regulatory function of psychosomatic states is a consequence of early interpersonal exchanges (Self-Other regulation) that are later internalized as regulatory affective-cognitive schemas (Calkins & Hill, 2007; Schore, 2003; Thompson & Meyer, 2007). At these early stages, it is another human being who initially regulates us through his/her attunement with our own inner states, -which reflects needs and emotions (Bion, 2007 [1962]; Fonagy et al., 2004). If we also keep in mind that this self-regulatory capacity appears to be related to the normal functioning of the frontal lobes (Quirk, 2007; Quirk & Beer, 2006; Ochsner & Gross, 2007; Schore, 2003) it would not seem surprising that acquired brain injury survivors may show deficits in self-regulation (Abreau et al., 2009; Beer & Lombardo, 2007; Bramham et al., 2008; Robertson & Knight, 2008) requiring that caregivers, or significant others, play an auxiliary role in regulating their inner states from outside. This type of regulation has been usually referred in the developmental literature as external or extrinsic regulation (Fox & Calkins, 2003; Gross & Thompson, 2007)

Although there is a limited literature regarding attachment and emotional adjustment in ABI, authors such as Prigatano (2008) have recognized its relevance to the development of a working alliance between patients and rehabilitation teams. A substantial literature has described the attachment system as innate (Bowlby, 1969; Panksepp, 1998), organizing physiological and brain regulation (Schore, 2003; Fonagy, 2008) and playing a key role in the search for proximity (Bowlby, 1973), as a mean for reestablishing safety and reducing previous states of tension (Fonagy, 2008). In children the search for proximity allows protection in the face of danger (hunger, adverse temperature changes, attacks from others and predators, separation) and *modulation of disorganizing mental states*. In adults, especially in moments of stress, attachment system activation is observed in the search for availability

of specific attachment figures (parents, partners), as a source of comfort, advice and support (Siegel, 1999). To Mary Main (cited in Siegel 1999) the inner experience of an activated attachment system is associated with feelings of anxiety and fear and can be triggered by diverse situations. Consequently, it is possible to suggest that ABI survivors, when experiencing catastrophic reactions, will attempt to regulate their inner states from outside, through the use of others' minds-brains (Salas, 2008). Due to the disorganization produced by the insufficiency of cognitive resources to cope with environmental demands (which tend to be cortically localized and highly vulnerable to brain injury), the organism functionally returns to early patterns of behavior (predominantly subcortical and automated). From here it is possible to understand Mrs. N's persistent search for proximity towards her sister, or Mr. C looking at his wife when he can't find words, or even Professor F's serenity after seeing his family by his side. The relevant point here, under the light of attachment theory, is that the search for the external environmental order referred by Goldstein is finally an effort to reestablish the *inner world's* order.

2.1.6. A regression of interpersonal processes: The use of other's minds.

According to Paula Freed (2002), deficits in memory, perception, problem solving, reasoning and abstraction cause a regression in affective and interpersonal processes. In this context, regression means a *temporary* return to evolutionary previous ways of functioning, when the organism does not yet have adequate resources to deal by itself with inner and outer demands, requiring the use of others' psychological apparatus to regulate its somatic states (i.e. extrinsic regulation). Such regression of emotional processes usually implies a reduction in the capacity to direct attention to the organism's somatic states, integrate somatic states with symbolic –and verbal representations, relate affective states to complex environmental situations, down-regulate emotional responses by using reappraisal or taking into account

other people's intentions or emotions. A consequence of this regression is the persistence of basic and primitive emotional states during moments of disorganization, which appraise stimuli simply as positive (proximity) or negative (avoidance) (Davidson, 1990). Another implication is the supremacy of automatic anxiety over signal anxiety, where the survivor cannot mobilize coping, or defensive resources, to prepare himself (Freud, 1926 [1959]). A regression in interpersonal processes implies also a *temporary* return to asymmetric patterns of interpersonal exchange and reciprocity, because certain mental functions that sustain mutuality, such as mentalizing and empathy, are damaged or abolished. Survivors' dependency on their caregivers will also elicit the activation of their own early interaction patterns related to care and safety. Consequently, an old scene is re-interpreted, where an organism requires the use of a significant other's mental functions to be regulated. This is something that caregivers know, and usually complaint about, when they say that "it is like having a new child". Some caregivers are capable of providing the environmental and interpersonal order needed to avoid and modulate catastrophic reaction, perhaps because they have internalized positive affective regulation experiences early in their own childhood. However, not all caregivers are able to do this and in such cases, there is the potential for having one catastrophe on top of another; the loss of the meeting of the minds, the *worst loss* of all.

2.1.7. A Disencounter of Minds

Mr. L is a kind and well-mannered man who fell off a roof whilst supervising a building construction. As a consequence he had a severe traumatic brain injury affecting most of his right frontal and right parietal cortices. When I met him he was in his wheelchair, hemiplegic and drowsy. He also had a significant difficulty perceiving stimuli from his left side and evaluating realistically his physical and cognitive problems. It was a classic case of

anosognosia or impaired self-awareness. In addition, he had a disruptive delusional jealousy, which usually appeared as a perseverative topic at the beginning of each session:

C.S: How is everything going Mr. L?
Mr. L: Things are wrong in my life. I'm thinking about moving out.
C.S: Why? (I wonder also how he would manage to do so considering his motor impairment)
Mr. L: My wife. She keeps receiving calls from a man. They don't tell me who he is, but I know she is after something. I know it (He remain silent for a while and then becomes drowsy).
C.S: How are you so certain that she is cheating on you?
Mr. L: She usually goes out (…) and she does not want to have sex with me anymore. I know it.

Mr. L was able to create an infinite number of stories regarding how his wife cheated on him, which usually were hooked to a real stimulus from the environment. For example, a card where his daughter wrote "I love you" to her mother, was convincing proof of his wife's unfaithfulness. A call from the taxi driver, telling them that he was about to pick them up, and take them to the rehabilitation center, was also another piece of evidence that confirmed his suspicions. Absolutely every event that he experienced could be used as evidence to confirm his conviction; a TV advert, something that was said on the news, a line from a soap-opera. Anything! Even I was considered (for a long period of time) as a possible accomplice.

Mr. L: Can I ask you something? Did my wife ask you to convince me that nothing is going on? Are you covering her up?
C.S: No, she hasn't. But if I say no would you believe me?
Mr. L: I would like to believe you. You have been nice to me and helped me.
C.S: But I imagine you are not completely sure I'm telling the truth.
Mr. L: yes.

Obviously something in Mr. L's reality testing was wrong, but curiously it was usually restricted to his wife faithfulness (like an encapsulated delusion). Far away from that topic

Mr. L was a reasonable, good humored and peaceful man. However, his wife, a woman that loved him deeply, had numerous complaints:

> Mrs. L: (with tears in her eyes). I don't know if I can take it any longer. A couple of days ago he was watching TV and suddenly he started yelling 'you don't love me' and saying other very hurtful things.
> C.S: do you have any idea why he started saying that?
> Mrs. L: I don't know. We were watching a soap opera and perhaps he picked up something from the show. Sometimes it starts out of nothing. I really don't know.
> C.S: so what did you do?
> Mrs. L: I tried to talk to him. To tell him that I do really love him, that this is the reason why I'm taking care of him. You know. I don't care if he does not recover completely. I know he won't. I just want him to love me as he used to. I can take care of the house and get the money, but this (…) I don't know if I can live like this.
> C.S: so trying to reason with him did not work at all?
> Mrs. L: no. Finally I ended up crying and feeling very anxious. He kept accusing me and I became angrier, as if he really hated me. I don't understand why this happens. So I just left. But I had to come back, and go to bed later with him. I hate that.

Mr. L insulted her when he believed he had found some proof of her infidelity, and such hostility could last for several hours, impenetrable to any argument. At nights he insisted in having sexual intercourse, as a way of testing her love. And she accepted this on many occasions, but his daily accusations exhausted her. To Mr. L's wife he was not the same man anymore. He had become a violent and selfish person, who no longer cared about her feelings; the sadness and anger he caused by his insults. But to Mr. L none of that was evident. For him, certainty existed only in what he was able to see, and understand in a concrete fashion, for example, whether or not his wife agreed to have sexual intercourse. When emotionally aroused by his jealousy, Mr. L was blind to his beloved's inner states, a dimension too abstract or subtle. Although Mr. L and his wife loved each other, their situation was an example of what Paula Freed has described as the disencounter of the minds, *the worst loss of all*.

Interpersonal relationships are probably the most complex situations human beings must face, partly because their core element is unpredictability (Goldberg, 2001). Human relationships demand flexibility and continuous adaptation. If catastrophic reactions are triggered by situations that require choosing between alternatives and switching from one set of behavior to another (Goldstein, 1995 [1965]), it is not a surprise that interpersonal conflict become a source of severe anxiety and confusion to ABI survivors. Mr. L's fear of abandonment (as he abandoned his first wife to move in with his current wife, his infidelity) was a feeling of such intensity that it caused huge failures in his reality testing. But he had no choice. To get out of those delusions, by himself, was impossible.

Perhaps someone could say that Mr. L's case is a rather extreme example of how brain injury can generate a disencounter of minds. So let's consider something more ordinary, such as Mrs. A's arguments with her husband. Mrs. A is a 57 years old housewife, with a severe dysexecutive syndrome caused by a large left frontal-parietal stroke. She is upset because her son has invited some friends over to swim at the pool at night. She thinks (later on) this is a sign of lack of respect, because no one asked her permission. So she impulsively goes to the backyard and throws them out of the house. Her husband gets angry with her and asks why she is behaving so selfishly. She does not have a clue, at that moment she just feels perplexed without even knowing how she got into the fight. However, she is able to perceive that her husband is annoyed, which causes her to become even more anxious. Her mind gets disorganized because of the anxiety and negative arousal, and she is not capable of finding reasons to justify her behavior. Her mind shuts down and she can just yell: "because I don't want to… because I don't want to". Finally he walks away, while she stays still, captured by a mixture of indecipherable sensations, which are turbid and somatically painful. She does not understand what has just happened, neither what she feels, or why. Only after a couple of sessions we are able to come up with an explanation. She feels anger, guilt, helplessness and

abandonment. At home she has always 'called the shots', and feeling that she is not able to decide or control anything anymore fills her with frustration. She feels she is not herself any longer, that she has changed and nobody cares about what she thinks or comprehends what she experiences. Mrs. A's fight with her husband is another example of the disencounter of the minds caused by catastrophic reaction, where a patient is unable to represent, and communicate, her feelings and intentions. To her husband, her childish and impulsive behavior is equally incomprehensible, a sad confirmation that Mrs. A is not the same anymore.

According to Freed (2002), in order to sustain interpersonal relationships ABI survivors must regain certain cognitive reintegration. They instinctively search for others, with whom they have had relationships with in the past, to provide this integrative function. However, because of feelings of *discontinuity* with the survivor, some relatives fail to provide this aid. The reasons of this experience of discontinuity have been illustrated by Feigelson (1993), who suggests that relatives often feel the neurological insult as a "personality death", as the emergence of a "double", a "ghost", someone physically familiar but emitting a continual sense of unreality. Relatives also experience intense anxieties when observing the patient's struggle to tolerate catastrophic reaction. Consequently, relatives usually respond in a negative fashion to these needs, which intensify the survivor's internal disorganization even more, activating feelings of loss, rejection, abandonment, and reinforcing his/hers already diminished capacity to relate to others (Freed, 2002; p. 62). As a result, in response to the instinctive search for coherence through the use of others, the survivor finds an abyss; the impossibility of using other's minds as a source of psychic compensation and inner regulation. Activation of the attachment system is not enough to modulate survivors' catastrophic reactions if there is no one capable of mentalizing the survivor's inner states or of tolerating and symbolizing the anxiety produced by their emotional demand.

2.1.8. Beyond thinking about other people's minds: the self-regulatory role of Mentalizing.

Usually referred to in the cognitive literature as Theory of Mind (Gallagher et al., 2002; Gallagher & Frith, 2003), mentalizing refers *mostly* to a preconscious imaginative mental activity (Fonagy, 2008), where we generate a representation, or mental scenario, about what other people, and ourselves, could be thinking and feeling. In the last decade several studies have tried to explore mentalizing deficits in people with acquired brain injury (Martín-Rodríguez & Leon-Carrión, 2010, for a review) and traumatic brain injury (Bibby & McDonald, 2005; Henry et al., 2006; Muller et al., 2009), using a traditional Theory of Mind (ToM) paradigm.

One of the main limitations of this line of research appears to be that it overlooks emotion as a core component of mentalizing, a tendency that has slowly changed with the incorporation of concepts such as affective ToM (Shamay-Tsoory & Aharon-Peretz, 2007) or cognitive and affective empathy (Shamay-Tsoory et al., 2004; Shamay-Tsoory et al., 2009). Perhaps, the difficulty of addressing the emotion-cognition link is related to the obvious restrictions of using laboratory procedures (TOM-like tasks), which often involve simplified repetitive tasks that do not reflect the experience of spontaneous mentalizing in everyday life (Spiers & Maguire, 2006). On the contrary, in natural settings (observing patient-relative or patient-psychotherapist interactions), the impact of emotion on cognition is striking (for a detailed description of these interactions see Kaplan-Solms & Solms, 2002). Patients who perform quite well on neuropsychological batteries during experimental testing may struggle with handling a simple interpersonal argument, understanding what they feel, imagining other people's stance or down-regulating emotions by finding alternative interpretations of negative events.

What appears to be missing in ToM-like tasks is the *regulatory* role of mentalizing, which is its core function according to psychoanalytic theory. From this background, mentalizing is understood as an internalized interpersonal process (Fonagy & Target, 1997), which allows the adjustment of affective states (Fonagy & Target, 2006; Fonagy et al., 2004) and the regulation of the self (Fonagy, 2008; Fonagy et al., 2004), by means of tolerating and containing negative emotion through the generation of representations (Fonagy et al., 2004). This interpersonal aspect of mentalizing is of the upmost importance when working with brain injury survivors, because a catastrophic reaction (and the primitive anxieties it generates) dampens mentalizing (Fonagy, 2008), which then impairs the regulation of catastrophic reaction. In consequence, as I have suggested, patients will instinctively look for significant others to regulate themselves from outside (Self-Other regulation). However, this search for others may imply a further weakening of mentalizing abilities, because the activation of a system that moves us towards the establishment of a bond with another human being appears to demand that we do not think about (evaluate or doubt) his/her inner states (Bartels & Zeki, 2004).

2.1.9. Dynamic localization approach to Self-regulation and Self-Other regulation deficits

A question that needs to be addressed is how diverse localized lesions may generate rather specific 'signatures' of Self-regulation and Self-Other regulation deficits. Let's consider the following clinical examples to illustrate how mentalizing (a Self and Self-Other regulatory ability) may be differently impaired, mobilizing patients to recruit different mentalizing components from other people's minds.

Because of the negative arousal generated by the discussion with her husband, Mrs. A is not able to represent the reasons behind her outburst, as a way of sharing her feelings of frustration and worthlessness. Mr. L's case is quite different. It seems that the failures in mentalizing are not produced by the disorganizing impact of negative arousal on an already compromised cognition, but by the dominance of inner reality when imagining his wife's intentions. When arguing with her husband, Mrs. A's capacity to "think" or represent internal states is impaired. On the contrary, when accusing his wife, Mr. L's reasoning appears too sharp, too well articulated, but lacking common sense and judgment.

Another striking difference between these two cases is related to how they use other people's minds, and how permeable they are to the regulatory influence of others. This issue is crucial to design psychotherapeutic interventions. Mrs. A is eager to understand what happened during the argument with her husband and makes huge efforts to explain the events, correcting herself when she feels I do not understand. In her mind, the use of my mind (my advice) could be of some help. In her mind my mind exists, and has contents that might differ from hers. And when I help her building a cognitive-emotional representation, from her scattered impressions (e.g. "so what you tell me sounds as if you felt frustrated"), she amazingly regains coherence (e.g. "That's the word, that is how I felt!") and anxiety fades away. The most impressive finding here is that, the *temporary* use of my capacity to link a somatic experience to a language representation seems enough to regain some sense of coherence. Even more, when such a cognitive-affective schema is generated, and elaborated over and over, she is able to integrate it and use it later (e.g. "The other day this happened… and I felt... I felt like you said once… remember that word you used?"), internalizing some sort of self-regulation.

The interpersonal exchanges with Mr. L were quite different. I always felt that he was distant, lost in the mist of his drowsiness, and emerging from time to time only when something powerful enough was able to pull him back into the world (as his jealousy). To him, I existed only when I presented myself concretely in front of him, or when someone at home recalled something we had agreed to do (e.g. some behavioral strategy we had decided to implement). He never asked for help spontaneously, and he showed signs of psychic conflict only when I confronted him. Although we developed a very solid working alliance, and he trusted me by the end of our work, he could not proactively use my mind as a source of permanent compensation. In other words, to him, my mind existed only when *I* was able to *hold it* in front of him, generating enough noise to attract his attention outside. However, when we were able to tune ourselves, true emotional contact used to appear allowing addressing his fears or worries. However, any symbolic intervention regarding his emotional difficulties (e.g. "perhaps your fear about your wife cheating on you is related to how you cheated on your first wife") was useless. These interventions made sense to him at those moments, but he could not internalize them, and use them later to regulate himself.

I hope these clinical reports help to portray how lesions to diverse areas of the brain may generate very different profiles of mentalizing deficits. This working hypothesis follows Luria's proposal that complex psychological functions (here, mentalizing) depend on the concerted work of different, and sometimes distant, neurological components (1966; 1973). A similar claim has been raised by neuroimaging findings on ToM (Carrington & Bailey, 2009) and also by the psychoanalytic literature on mentalizing (Fonagy, 2008). A working hypothesis like this one may offer valuable guidelines in the assessment of patients' capacity to Self-regulate, and use Self-Other regulation strategies, by detecting which mentalizing components are impaired. This may allow also designing individually tailored interventions to address such deficits.

If we consider the clinical cases described above (Mrs. A, Professor F, and Mr. L) a question regarding the lateralization of mentalizing components arises. With no doubt, Mr. L's inability to Self-regulate, and use others as a source of regulation, is greater than Mrs. A's or Professor F's, suggesting a predominant role of right hemisphere structures in mentalizing. Although there is a large body of literature that associates right-sided lesions (Griffin et al., 2006; Happe et al., 1999; Siegal et al., 1996; Surian & Siegal, 2001; Wimmer et al., 1998), and particularly right frontal lesions (Stuss et al., 2001), with deficits in ToM-like tasks, there is no general consensus yet regarding the specific roles of right and left components.

Although the role of right-sided structures seems relevant to comprehend the dynamic nature of mentalizing, it would be overly simplistic to reduce all of Mr. L's difficulties to a laterality effect. In consequence a more detailed consideration of his neuropsychological deficits is needed. A behavioral observation that seemed appropriate to start with is the fact that he is constantly "self-absorbed", and other people's minds seem to exist only when they make enough "noise" to generate moments of shared attention, transactions that need to be constantly *supported* from outside. This failure in *joint attention*, the capacity to coordinate attention with a social partner (Mundy & Newell, 2007), might be related to the damage of the so-called Reorienting System (Corbetta et al., 2008), a right ventral fronto-parietal network that allows *switching between internally and externally directed activities*. Another possible explanation is that Mr. L's right-sided lesion compromised the *processing of eye gaze*, a basic capacity for the assessment of other's people mental states (Baron-Cohen 1995), that has been associated to right posterior (Superior Temporal Sulcus) activation (Haxby et al., 2000; Pelphrey et al., 2005; Wicker et al., 2003). Consequently, it is possible to suggest that components that allow flexible transitions between internal and external mental activity, and social engagement of attention, are impaired.

Damage to Mr. L's right somato-sensory cortex may have also contributed to the "blindness" toward his wife's inner states. This hypothesis seems supported by studies that have associated lesions to the right somato-sensory cortex with impairment in the perception of emotions from faces (Adolphs et al., 2000). These findings have led some authors to suggest that, in order to access other people's minds, we must first *internally simulate* what it would feel like to be the other person (Adolphs, 2001; Gallese, 2003; Gallese et al., 2004). In other words, Mr. L's lesion may have impaired a core mentalizing component, which generates a direct (non-explicit) bodily-form of experiential understanding of others, known also as *intentional attunement* (Gallese, 2006).

Another relevant behavior that needs to be explained here is Mr. L's delusion of infidelity, also known as Othello Syndrome (Todd & Dewhurst, 1955). The neurological literature has related this type of delusion to right frontal lesions (Nakamura et al., 2006; Richardson et al., 1991), although there is no clarity regarding the underlying pathophysiological or psychological mechanisms. The co- occurrence of other delusional syndromes –e.g. somatoparaphrenia- after right frontal lesions (Feinberg et al., 2005; Feinberg et al., 2010; Feinberg & Keenan, 2005) have led some authors to suggest that the right frontal lobe is not associated specifically with one type of delusion, but with a more general role in preserving the equilibrium between internal and external reality (Feinberg, 2010). Salas & Turnbull (2010) have suggested a possible role for the right frontal lobe in the regulation of negative autonomic arousal, proposing that when this process fails, automatic cognitive-affective operations (primitive defense mechanisms) are generated to deal rapidly with disorganizing anxiety, evacuating completely any trace of negative arousal. In other words, Mr. L's use of massive projection (Othello Syndrome) may be understood as the consequence of a failure in the *down-regulation of autonomic arousal*, thus impeding the use

of mature defenses, which require *tolerating* a minimum level of distress to generate psychic conflict.

I have suggested that Mr. L's deficits may be better understood by considering which mentalizing components appear to be impaired (Reorienting System, social engagement, simulation and arousal regulation). What about the left-sided patients I have described? Which mentalizing components are compromised here? In Mrs. A and Professor F's cases the most evident difficulty is the presence of a vicious cycle where high levels of anxiety impair mentalizing, and mentalizing deficits hamper the regulation of anxiety. During moments of high negative arousal they can't make sense of what they experience, feeling perplexed, paralyzed and turbid. They seem unable to describe what they feel; think about it or even understand its causes. A consequence of this Self-regulatory failure appears to be a tendency to 'get trapped' in negative affect, which is experienced for extended periods of time. This tendency may underlie the reported higher frequency of neuropsychiatric 'catastrophic reactions' (Starkstein et al., 1993) and depressive symptoms (Narushima et al., 2003; Robinson, 2006; Robinson et al., 1984) after left-sided lesions. This emotional persistence (Thompson, 1990), or emotional inflexibility (Bonanno et al., 2003; Coifman & Bonanno, 2010), may be associated with the impact of negative affect on cognitive flexibility (Davis & Nolen-Hoeksema, 2000) and problem solving (Gasper, 2003), cognitive abilities that are necessary for the overt reasoning that mentalizing entails (Damasio et al., 2003). In sum, mentalizing deficits after frontal left-sided lesions appear to be characterized by a failure of an executive component that allows the *generation* and *flexible manipulation* of verbal representations, which are crucial to regulating behavior (Luria, 1966; 1973; Winsler, Fernyhough & Montero, 2009), "buffering" (Feldman et al., 2001) and "digesting" emotional experience (see concept of Mentalized Affectivity in Fonagy et al., 2004).

Cases of agrammatic aphasic patients, after left hemisphere damage, where ToM abilities are preserved despite language difficulties (Varley & Siegal, 2000; Varley et al., 2001) are of extreme interest when drawing a possible role of left frontal structures in self-regulation and self-other regulation. In Mrs. A case, although she struggles to find the words that denominate what she feels, and has great difficulties in reasoning about her own intentions, she is capable of perceiving and somehow understanding her husband's emotions. She knows he is mad at her, although she cannot figure out why. In other words, even though she is not able to verbally represent her experience, and reflect upon it, she can emotionally attune to her husband's anger. Perhaps this is possible because the *simulation* component (right somato-sensory cortex) remains undamaged.

A final distinction between Mrs. A and Mr. L needs to be discussed here. Mrs. A's right frontal lobe is intact. However, she experiences persistent levels of negative affect. Salas & Turnbull (2010) have addressed the issue of defense mechanisms and laterality, suggesting that in patients with right frontal lesions the inability to tolerate negative arousal may triggers the protective, but primitive, recruitment of language and reasoning (e.g. delusional confabulation). By using these primitive mechanisms patients appear to "evacuate" negative emotional states, quickly dampening any trace of negative arousal, and, in some cases, even placing themselves in positive scenarios instead (e.g. Fotopoulou et al., 2004; Turnbull et al., 2004).

In contrast, patients with left frontal lesions are more able to tolerate negative autonomic arousal, hence, primitive defenses are not activated and negative emotion is still experienced. In consequence, left-sided patients are trapped in the *concreteness* of negative emotion, are unable to "evacuate" through primitive defense mechanisms or down-regulate emotion by using symbolic and reasoning functions (e.g. rationalization), which are compromised by left frontal injury. This impairment in the use of language to regulate

behavior seems supported by evidence that relates reappraisal (a self-regulation strategy based on generating alternative meanings to emotional events) with left frontal activation (Goldin et al., 2008). Another source of evidence that appears to support this hypothesis comes from the so-called gateway hypothesis (Burguess, Dumontheil & Gilbert, 2007, Burguess, Gilbert, Okuda & Simons, 2006), which proposes that the rostral prefrontal cortex, BA 10, is crucial for the self-generation and maintenance of thoughts and representation (stimulus-independent cognition). It is possible to suggest that the higher frequency of catastrophic reaction found after lesions to the left polar frontal cortex –BA 10- (Robinson, 2006; Starkstein et al., 1993), might be associated to some extent to a difficulty regulating emotions through the use of thinking.

These cases are intended to illustrate how diverse brain lesions may impair mentalizing (a Self and Self-Other regulatory ability) differently. By describing how these deficits unfold during patient-relative and patient-therapist interactions, I wish to emphasize the interpersonal, or 'relational', nature of these processes. It is my proposition that the same 'relational' domain that is damaged may be also a source of psychological compensation and emotional encounter.

2.1.10. Addressing Self and Self-Other regulatory deficits in psychotherapeutic settings.

One of the main issues in the use of psychotherapy with brain injury patients is how to adapt the available tools to the specific cognitive and emotional peculiarities of this population. Although this idea has been explored in relation to cognitive deficits (Judd & Wilson, 2005), the emotional and regulatory difficulties remain largely unattended. The theoretical background presented in this paper follows the efforts of several authors to integrate cognitive, emotional (Freed, 2002; Gracey et al., 2007; Mateer et al., 2005; Prigatano, 1991)

and interpersonal (Bowen et al., 2010; Feigelson, 1993; Freed, 2002; Lewis, 1999; Yeates, 2009; Yeates et al., 2008) domains in the design of therapeutic interventions. However, this paper develops and works on this work by exploring in detail how patients might use other people's minds to regulate themselves from outside.

If we consider the clinical cases presented, several differences appear regarding how diverse lesions impact on Self and Self-Other regulation. It seems that patients with right-sided lesions will display difficulties in more basic components of social cognition, such as joint attention and intentional attunement. If the lesion is large, and also includes the right frontal lobe, even reality testing might be impaired. In cases like this (Mr. L) a classic psychotherapeutic approach is not the first choice. However, the *external scaffolding* of the impaired interpersonal function is a potential intervention. An example of this is the intervention designed in collaboration with Mr. L's wife. The aim of this intervention was to bypass these difficulties, allowing some kind of re-connection between them, at least temporarily. Clinically speaking, this was extremely relevant because she wanted to take care of him, but at the same time she felt that he was ungrateful and didn't care about what she was feeling. For her, the most painful change caused by his brain damage was not physical, or economical, but the loss of intimacy and connection (for a detailed description of intimacy and connection problems after brain injury see Bowen, Yeates and Palmer, 2010). In other words, the interpersonal conflicts were eroding the same emotional bond that was a source of motivation to take care of him.

The first goal of the intervention was to modify her expectations regarding Mr. L's spontaneous consideration for her inner states. This goal was very hard to attain, and we had to spend quite a long time talking about it, explaining to her the neurological reasons why he couldn't do so, and supporting the mourning process this loss implied. This strategy also had an implicit intention: to avoid a negative circuit of frustration and disappointment caused by

unfulfilled expectations. Secondly, an intervention was designed whereby every time he started showing disrespectful behaviors (accusing her of cheating or telling her that he will abandon her because of this) she was instructed not to enter into an argument. Instead, she had to describe in simple words what she was feeling, face-to-face, as a way of capturing his attention and concretely informing him about her inner experience: 'when you accuse me of that… I feel sad', 'what you say really hurts me', 'I feel you don't love me anymore'. When Mr. L's wife was able to deliver this kind of emotional feedback Mr. L's behavior changed. It was as if by seeing his wife's pain, concretely, her inner states momentarily acquired an *emotional salience* and *clarity*. She reported:

> Mrs. L: it has worked. Not always, but generally. It is very difficult, because my first reaction is anger and disappointment. But then I think of what we have talked about, that his brain injury does not allow him to think clearly. So I try, I try to stay calm.
> C.S: can you describe how it was the last time?
> Mrs. L: I think everything exploded because I received a phone call or maybe because I had to go out. He hates it when I go out. He thinks I'm going out to see someone. So he started saying I was going out to meet someone, a man. And he became so angry about it. So I sat in front of him and said 'it really makes me sad that you don't trust me', 'it makes me sad that you destroy our family by doing this'. I was very sad at that point. I think he was confused at first, but then he became less angry. I did not get into denying his accusation, as you suggested. I just described how I felt. It usually works. However, it is emotionally so draining….

Left-sided patients present a very different profile of difficulties, basically because the joint attention and intentional attunement components appear to be spared, preserving an implicit capacity to connect to others. In consequences, when experiencing a catastrophic reaction they will spontaneously look for others as a source of compensation. The problem with these patients is more related to the adaptation of psychotherapeutic tools according to their language and executive deficits. In these cases the use of concrete materials, like drawing (Kaplan-Solms & Solms, 2000), identity maps (Ylvisaker & Feeney, 2000) or sand

play (Hirao et al., 2008) have proved to be a useful method of bypassing language, working memory and abstraction deficits.

In a previous paper (Salas, 2008), it was suggested that patients with frontal left sided lesions tend to use the mind of the therapist to connect ideas with words, connect somatic states and affects with mental representations, connect present experience with elements from the future and past, thread narratives, generate metaphors from disperse elements and also to scaffold fantasy generation. In my opinion, all these possible uses of the therapist's mind are ways to compensate for deficits in the capacity to *generate* and *manipulate* mental representations.

In the case of Professor F, he commonly used my mind to generate a continuous narrative that made sense of the disorganizing bodily sensations, which tended to further impair his executive functions. For instance, after returning to his job, he started to dislike being at his office and reporting feelings of 'restlessness'. He did not have clarity about what he felt, or understood why he felt that way, but remembered that now he had difficulties getting to the first floor of the building (his office was in the ground floor), because the stairs did not have a rail to lean on. When I asked him about the first floor he said 'everything happens upstairs, most of the people from the department work there and usually share a cup of coffee during the breaks'. Two ideas came into mind. First, I remembered how much Professor F enjoyed being with other people. Then a football field appeared as an image (Professor F was a football fan), and the thought of watching a football game from the sideline appeared [*fantasy scaffolding*]. So I suggested to him that it is like watching a game from the side line'. He smiled and said 'yes, something like that'. However, although he was smiling, I noticed a subtle change in my mood and started to feel melancholic. So I told him 'now, when we are talking about this, I feel a little bit sad. I know that you told me you felt restless and uncomfortable in your office, but I wonder if it is because you feel sad in there'

[*connecting somatic states with verbal representations / Linking different emotional states*]. For a minute or so he tried to organize the ideas in his mind and finally said 'I think so, now that you mention it, it makes sense (…) maybe is that I miss being with other people'. I remembered a previous conversation where he told me that his former colleagues, the ones from his generation, were all dead or retired [*connecting present and past experience*]. While I remembered this I felt even more melancholic. A couple of minutes passed and suddenly his face changed, as if he has grabbed a precious idea, 'it is not that I miss other people because of the work you usually do with them, the projects…you know… to be active… to do stuff… is like I miss people because I need them. I haven't realized until now how important people are to me'. At that point, thinking about what he has just said, he felt moved. Then I asked him 'so, how was it before the accident?' [*generating a narrative / connecting past and present experience*]. He smiled again 'you got me there… before the accident I was too worried about my duties and I always thought that I care about people only because it was part of my role as a manager'. He kept trying to grasp something in his mind for a couple of minutes, like searching for an object inside an untidy drawer. A second wave of associations came later: 'the door of my office was always closed; now it is always open, as if I were expecting them to enter'. The image of an opened door resonated strongly in my mind and became a persistent image for several minutes. Then he started to describe different interpersonal situations (family, students, and colleagues) where he enjoyed just sharing a moment with other people, as if he was more sensitive now, and in more contact with his feelings. So I said 'I have been thinking about that opened door you mentioned, and I wonder if it means that now you feel more than before [*connecting present and past experience*]. Maybe because of the accident, or maybe because you also have changed, you are not so logical anymore. So you feel more, more often and more intensely. As if you are open to others, not closed, as before [*generating a metaphor to compact meaning*]'. He

smiled again and thought for a while 'I like that image, the open door (…) and is true, I feel more like an open door now. Maybe that's why now I miss other people more, why I feel sad sometimes when I'm alone. Maybe I was closed before and didn't feel anything of this at all'.

Relatives of patients like Professor F can also be trained in managing catastrophic reactions, especially considering that new life events will generate states of disorganization that need to be 'digested'. The training usually involves two key steps. First, when the patient feels disorganized (because of a new event, a conflict or problem he doesn't understand or know how to solve it) it is important not to minimize his experience, by saying things like 'you don't have to worry, everything will be all right'. This is a usual defensive maneuver used by relatives to reduce their own anxiety, which also prevent patients from regaining a sense of agency over their own experience and problems. Professor F recognizes this: 'that is why I prefer talking to you…my wife gets anxious when she sees me like this and starts telling me what to do…when I don't have a clue about what is s going on'. The second step is related to addressing the confusion that catastrophic reaction entails, and is based in the fact that is not possible to solve a problem that has not been cognitively and emotionally formulated yet. In other words, patients like Professor F need help to generate a sense of coherence and continuity by assembling a mental schema that links sensations, feelings and thoughts. Once a schema such as this is generated, and all the elements are available, the patient may judge by himself the meaning of his experience and later reflect upon his problems with a renewed sense of mastery.

2.1.11. Concluding remarks

In the last few decades neuropsychological rehabilitation has moved away from a paradigm centered on the remediation of cognitive deficits, towards the consideration of emotion and identity reconstruction. This theoretical turn appears to be related to a major emphasis on *participation* problems than deficits or impairments. A consequence of such a holistic, and person-centered approach, is to focus on how brain injury survivors may return to meaningful activities in spite of cognitive and behavioral difficulties. The main goal of this paper was to describe the cognitive and emotional difficulties that brain injury survivors face when they try to resume significant interpersonal relationships, and use interpersonal relationships as a source of support and psychological compensation.

Perhaps the main thesis of this work is that ABI survivors use other people's minds to regulate themselves from outside. When experiencing catastrophic reactions, due to the impossibility of coping with environmental demands, patients instinctively search for other's support. However, it appears to be that not all patients use other people's minds in the same way, and that the type and localization of brain lesion might have relevance regarding which components of other people's minds they use. I have put forward the hypothesis that lesion to the anterior and posterior regions of the right hemisphere impair more basic Self and Self-Other regulatory components, such as *tolerating negative arousal* and *intentional attunement*, respectively. Lesions to the left anterior portion of the brain appear to compromise the capacity to *generate* and *manipulate* mental representations, necessary components for Self-regulation but not for Self-Other regulation.

A limitation of this proposal is that it is mainly based on anecdotal evidence, hence this ideas need to be tested in more rigorous experimental settings. However, I believe that this work is a relevant first step in exploring systematically how brain injury impairs

interpersonal processes differentially, and also how we can use the same interpersonal relationships as therapeutic tools.

Finally, it is extremely important to differentiate between Self-regulation and Self-Other regulation when assessing brain injury patients, because the distinction is relevant to the design of individual and family tailored interventions. In the future it might be even possible to manualise procedures to address specific regulatory deficits, which might also improve the effectiveness and outcome of our rehabilitation programs. In the case of left anterior patients the adaptation of psychotherapeutic tools would be a fruitful path. As for the right anterior and posterior patients, family interventions and environment modifications appear to be promising lines of research.

2.2. The Neuropsychology of Emotion Regulation Strategies: Insights from Patients with Focal Brain Lesions[3].

2.2.1. Abstract

In the past decade, there has been a notable increase in the amount of research on the neural bases of emotion regulation [ER] strategies. Virtually all of the findings have been in the domain of functional imaging, which is surprising given the well-established role in neuropsychology of evidence from patients with focal brain lesions. This article attempts to build a theoretical bridge between the ER literature and the literature on patients with focal lesions. Specifically, this article focuses on the way in which well-known neuropsychological deficits might impair one or more ER strategies delineated by the process model of emotion regulation, including: situation selection, situation modification, attentional deployment, cognitive change, and response modulation. The evidence reviewed confirms many of the neuroimaging findings, suggesting that ER strategies rely on a set of frontal lobe-related processes. In addition, this review offers novel insights into the way ER strategies may depend on *specific* and *shared* neuropsychological components, and suggests novel components not usually considered, such as inner speech. This review also discusses the importance of these findings for neuropsychological rehabilitation, where issues of ER are of great clinical relevance.

[3]Salas, C.E., Gross, J.J., & Turnbull, O.H. (submitted). The Neuropsychology of Emotion Regulation Strategies: Insights from Patients with Focal Brain Lesions *Neurobehavioral Reviews*. This paper has been re-submitted for review.

2.2.2. Introduction

There is a large neuropsychological literature addressing emotional dysfunction after brain injury (Carson et al., 2000; Gainotti, 1997, 2000, 2001; Heilman and Satz, 1983; Heilman, Blonder, Bowers and Crucian, 2000; House et al., 1989; Narushima et al., 2003; Robinson, 2006; Starkstein et al., 1987), especially in relation to both emotional *perception* (Borod et al., 1992; Borod et al., 2002; Borod et al., 1986; Harciarek et al., 2006; Mandal et al., 1993) and *expression* (Blonder et al., 1993; Blonder et al., 2005; Bloom et al., 1992; Borod and Madigan, 2000; Borod et al., 2002; Kazandjian et al., 2007; Weddell et al., 1990). This literature has been a key source of evidence unpacking the neural basis of a number of potentially discrete emotional processes (Adolphs, 2007; Demaree et al., 2005; Gainotti, 1972). However, a trend in the last decade has been to move beyond these basic emotional processes and explore how brain injury may modify more complex aspects of emotional life, such as its subjective *experience* (Gillihan et al., 2010; Hornak et al., 2003), *social* function (e.g. Beer et al., 2006; Bramham et al., 2009; Shamay-Tsoory et al., 2008) and *regulation* (Beer and Lombardo, 2007; Hilz et al., 2006; Roberts et al., 2004; Woolley et al., 2004).

Emotion regulation (ER) is usually defined as the set of process by which individuals influence *which* emotions they have, *when* they have them, and *how* they experience and express those emotions (Gross, 1998). It has been suggested that five strategies may be used to regulate emotion at different points during the emotion generation process [see figure 1]: situation selection, situation modification, attentional deployment, cognitive change, and response modulation (Gross, 1998). The first four strategies can be considered *antecedent-focused*, for they occur before emotional responses are triggered, while response modulation may be seen as a *response-focused* strategy, because it is often implemented after the emotional response is generated (Gross and Muñoz, 1995).

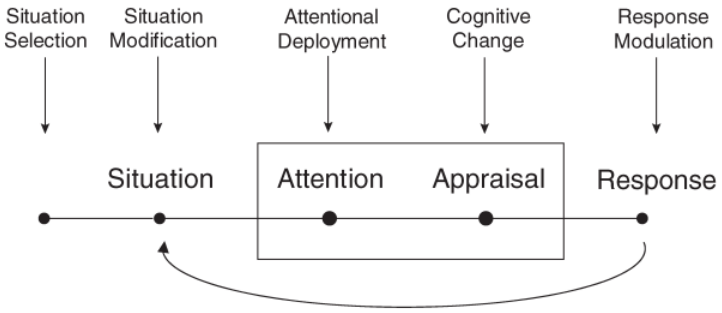

Fig 1. The Process Model of Emotion Regulation. [from Gross & Thompson, 2007].

It has been suggested that brain damage may impair ER, influencing coping and further socio-emotional adjustment after the injury (Abreau et al., 2009). Unfortunately, the available evidence is mostly based on ER problems in neurodegenerative diseases (Goodkind et al., 2010; Henry et al., 2009) or after adult traumatic brain injury (McDonald et al., 2010; Obonsawin et al., 2007; Rochat et al., 2009, Tate, 1999) and paediatric traumatic brain injury (e.g. Ganesalingam et al., 2006, 2007; Tonks et al., 2007), with few studies exploring such difficulties in patients with focal brain lesions (e.g. Bechara, 2004; Beer, 2007; Beer et al., 2003; Mathiesen et al., 2004; Roberts et al., 2004; Woolley, 2004). Furthermore, the existing studies on brain injury and ER have tended to address emotion dysregulation from a neuropsychiatric point of view (e.g. Cattran et al., 2011; Chemerinski and Levine, 2006; Gainotti et al., 1999; Robinson, 2006), ignoring the psychological *mechanisms* by which thoughts and feelings are regulated.

During the past decade, knowledge about the mechanisms by which people regulate their emotions has increased significantly, offering a solid theoretical background (Gross, 2007). In fact, there is a large literature on the relationship between ER, mental health (Gross and Muñoz, 1995; John and Gross, 2004), well-being (i.e. Nyklicek et al., 2011) and psychopathology (Aldao and Nolen-Hoesksema, 2010; Aldao et al., 2010; Kring and Sloan,

2010; Kring and Werner, 2004; Taylor and Liberzon, 2007). Behavioural studies have begun to unpack the psychological components involved in several ER strategies, by investigating the relationship between those strategies and discrete cognitive processes (Gyurak, Goodkind, Madan, Kramer, Miller and Levenson, 2009; McRae, Ciesielski and Gross, 2011; McRae, Jacobs, Ray, John and Gross, 2011). These studies have suggested that certain psychological processes are particularly involved in some ER strategies, as is the case of verbal fluency in response modulation (Gyurak, Goodkind, Kramer, Miller and Levenson, 2012), and working memory and set shifting in reappraisal (McRae et al., 2011). Moreover, in a rapidly growing literature using neuroimaging, the neural correlates of several ER strategies have been described (Goldin et al., 2008; McRae et al., 2010; Ochsner and Gross, 2005; Ochsner et al., 2004; Ochsner et al., 2002; Wager et al., 2008), allowing the construction of a *preliminary* neural architecture of ER (Green and Malhi, 2006; Ochsner and Gross, 2007).

One limitation of exclusively using a neuroimaging approach to disentangle the neuropsychological and neural architecture of ER is that it does not allow us to establish which brain areas are 'necessary' for a specific ER component. Several authors have made a similar claim regarding other emotional and cognitive processes (Adolphs, 2007; Price and Friston, 2002; Shallice et al., 2008b; Stuss et al., 2002). In consequence, it seems justified to study patients with focal lesions from an ER perspective, and explore how damage to a range of brain regions might impair specific neuropsychological components that are necessary for successfully implementing a particular ER strategy. Such an approach could not only inform our knowledge of the *neuropsychological* basis of these strategies, but also would contribute to the assessment and design of interventions in rehabilitation settings (Salas, 2012).

Two obstacles to this approach must be noted, however. One obstacle is that research on some ER strategies, such as situation selection and situation modification, is almost non-existent; even though both concepts appear to have substantial face validity. As a

consequence, compared with attentional control, cognitive change, and response modulation, there is little understanding of the basic components involved in situation selection and modification, limiting the dialogue with findings from lesion studies. One way to address this challenge is to look at under-investigated ER strategies (e.g., situation selection and modification) through the novel lens of neuropsychological lesion studies, making it possible to unpack the underlying components. This opens numerous possibilities for future research, such as improving the design of tasks that might isolate core ER mechanisms, or identifying specific groups of neurological patients that can be studied using existing ER paradigms.

A second major obstacle is that each emotion regulation strategy, from a dynamic localization point of view (Luria, 1966), is a complex mental process that relies on several neuropsychological components (e.g. memory, language, executive functioning, etc.), which obviously depend on a widely distributed network of brain structures (Ochsner and Gross, 2007). Furthermore, it is likely that the same brain area (e.g. ventrolateral PFC) might be relevant for several ER strategies (e.g. envisioning emotional future events, and affective shifting). This point is especially relevant because a skeptical reader might suggest that, considering the complexity of emotion regulation strategies, damage to *any* part of the brain might compromise their function. However, evidence from neuroimaging (Ochsner and Gross, 2007 for a review) and lesion studies (Beer and Lombardo, 2007 for a review) appear to suggest that ER strategies are not compromised by damage to *all* brain areas. As will be reviewed in this paper, a survey of the literature suggests that the principal effects are found after lesions to various prefrontal structures associated with the *regulation of behaviour*, and damage to subcortical structures related to the *generation of emotion*. In contrast, there seems to be no compelling evidence to suggest that many other brain areas [e.g. temporal-parietal junction, occipital cortex] have a critical role in ER. One way to address this challenge is to arbitrarily select a single psychological component (e.g. *foresight*, or the capacity to imagine

future scenarios), which is at the core of a specific emotion regulation strategy (e.g. situation selection), and from which there is sufficient neuropsychological evidence from studies of patients with focal lesions to draw reliable conclusions. On the basis of the criterion of relevance, and sufficient evidence, this review will focus on several neuropsychological processes. These are: foresight (*situation selection*), cognitive and affective flexibility (*situation modification*), sustained attention (*attentional deployment*), cognitive control and language (*reappraisal*), motor expressive control, response inhibition and interoception (*response modulation*).

The aim of this article is to draw a theoretical bridge between the growing field of ER and our current understanding of cognitive and emotional processes after focal brain lesions. To our knowledge there is only one previous review that has attempted a similar task (Beer and Lombardo, 2007), although these authors addressed only a limited number of ER strategies. The goal of the present review is to describe how specific *neuropsychological deficits* (e.g. episodic amnesia) may impair the use of one or more regulatory strategies defined by the process model.

2.2.3. Situation selection

Situation selection is, in temporal terms, one of the antecedent-focused ER strategies. It has been usually defined as taking actions that make it more (or less) likely that we will end up in a situation we expect will produce desirable (or undesirable) emotions (Gross and Thompson, 2007, p. 11). It has also been referred to as people's capacity to predict the trajectory of their emotional experience into the future (Werner and Gross, 2010, p. 21). Situation selection is unquestionably a complex psychological process; however research on it is almost non-existent (for an exception see Rovenpor, Skogsberg and Isaacowitz, 2012). Nevertheless, based on the available definitions of situation selection, it is possible to suggest that it

depends, at least, on two main components: 1) the capacity to *generate* an array of hypothetical future scenarios; and 2) the ability to *decide* amongst them based upon their potential emotional impact. Two groups of brain injured patients are of interest here, and will be described in detail in this section: patients with hippocampal damage exhibit impairment in the *generation* of future scenarios, while patients with ventromedial prefrontal cortex lesions present deficits in the *selection* of possible scenarios, based on their potential emotional value.

2.2.3.1. Situation selection and foresight impairment after hippocampal lesions

The capacity to mentally pre-experience, or imagine, a personal event that may occur in the future has been variably labelled as episodic foresight (Suddendorf and Corballis, 2007), episodic future thought (Atance and O'Neill, 2001, Szpunar, 2010), projection (Okuda et al., 2003), prospection (Buckner and Carroll, 2007), or simulation (Schacter and Addis, 2007). Interestingly, it has been proposed that the ability to simulate future scenarios depends on similar mechanisms than the capacity to recall personal events from the past (Addis et al., 2007; Suddendorf, 2009 for a review). In other terms, what allows humans to mentally project themselves backwards in time to re-live, or forward to pre-live events (Suddendorf and Corballis, 1997), appears to depend on a common ability to mentally travel in time (Szpunar, 2010). Such mental time travel would allow individuals to endlessly recombine raw elements from the past to construct and imagine possible events (Addis et al., 2007; Okuda et al., 2003; Schacter and Addis, 2007; Suddendorf and Corballis, 2007; Szpunar 2010), thus increasing behavioural flexibility in the achievement of long term plans (Suddendorf and Corballis, 2007). From this point of view, situation selection would imply the recruitment of memory systems that allow humans to predict the emotional trajectory of hypothetical situations, based on previous experience.

The neuropsychological basis of episodic future thought, and hence situation selection, could productively be investigated in patients with severe episodic amnesia. The study of amnesic patients has been an especially relevant piece of evidence supporting the hypothesis that episodic future thought depends on some shared mechanisms with episodic memory (Atance and O'Neill, 2001). In 1985 Endel Tulving described the case of N.N. [later known as K.C], a patient with severe episodic amnesia after bilateral hippocampal damage, who also presented with impairment in his capacity to imagine future events. In the same way that N.N. was unable to remember events from the past, when asked about what he might be doing tomorrow, he seemed unable to imagine a future scenario. Tulving suggested that N.N. suffered from incapability to experience *extended* subjective time, and depicted him as living in a "permanent present". From then on, data from several cases on episodic amnesia have supported the relationship between episodic memory and episodic future thought (Hassabis et al., 2007; Klein et al., 2002; Levine et al., 1998). Further support of this relationship has come from neuroimaging studies, where a shared neural architecture of episodic memory (medial prefrontal cortex, posteromedial parietal cortex and the medial temporal lobes) is recruited when people simulate future personal episodes (Addis et al., 2007; Botzung et al., 2008; Okuda et al., 2003; Szpunar et al., 2007; Buckner and Carroll, 2007; Hassabis and Maguire, 2007; Schacter and Addis, 2007, 2009; Spreng et al., 2009).

2.2.3.2. Situation selection and foresight impairment after vmPFC lesions

A second element in the situation selection process is related to the motivational components that guide it. It has been suggested that the generation and selection of future scenarios appears to be driven by previous emotional experiences (D'Argembau and Van der Linden, 2007), perhaps sharing some of the basic mechanisms attributed to emotion-based decision-making (Loewenstein, 2007). Some authors have stressed this link by proposing that it is the

affective state associated to mental simulations of positive and negative outcomes, which is consciously represented, which motivates us to engage in some activities and avoid others (Bechara and Damasio, 2005; D'Argembau and Van der Linden, 2007). In other words, situation selection will not only imply the *projection* of the self into the future, by using elements from the past, but also the *selection* of possible scenarios based on the emotional value of potential future events

The study of patients with lesions to the ventromedial prefrontal cortex (vmPFC) has been a further important source of evidence in understanding the neurobiological basis of associating affective states with mental simulations of positive and negative outcomes (D'Argembau and Van der Linden, 2007). The available evidence suggests that patients with damage to the vmPFC present a shortened future time perspective (Fellows and Farah, 2005b) and exhibit difficulties anticipating physiologically the onset of aversive stimuli (Bechara et al., 2000 for a review; Bechara et al., 1996; Damasio et al., 1990; Roberts et al., 2004) and also unlearning reward and punishment association (Fellows and Farah, 2003, 2005a; Rolls et al., 1994). This impairment in the capacity to generate, and flexibly modify, emotional association to future events appears to lead them to take disadvantageous choices (Bechara et al., 1997; Bechara et al., 2000) by neglecting future consequences (Bechara et al., 1994; Bechara et al., 2000). Of relevance to the neurological basis of situation selection, it has been suggested that the anterior portion of the vmPFC (BA 10, 11) is recruited to a greater extent than posterior areas (BA 25), when envisioning emotional events in the far future compared to the near future (D'Argembau et al., 2008).

Although most of what is known about the relevance of vmPFC to situation selection comes from work on emotion-based decision-making, several studies have directly explored autobiographical memory and mental time travel capacity in this population. For example, Levine (2004) reported that patients with vmPFC lesions recalled fewer internal details

(thoughts and feelings) from biographical episodes than patients with dlPFC lesions and controls, although the recollection of external details (personal and public facts) did not differ from healthy participants. Levine interpreted this reduction in internal episodic specificity as 'reflecting a state of consciousness partially stripped of the benefits of mental time travel, and relatively constrained to the here and now' (p. 61). This report is extremely interesting if we consider the available evidence associating episodic memory and episodic future thought (Addis, Wong and Schacter, 2007; Suddendorf, 2009 for a review). It suggests the possibility that the impairment of patients with vmPFC lesions to travel back in time could extend to traveling forward into the future as well. This assumption seems supported by a study from Fellows and Farah (2005b) where patients with brain lesions were asked to think about future events that may happen in their lives. The main finding was that participants with lesions to the vmPFC showed significantly less content, and shorter time perspective, than patients with dlPFC lesions and controls.

In sum, the available evidence on mental time travel and decision-making suggests that lesions to the medial temporal cortex and vmPFC may impair situation selection differentially. Damage to the vmPFC appears to impair the capacity to generate, and flexibly associate, emotional value to events, thus guiding selection of optimal environments. On the other hand, lesions to the medial temporal cortex appear to preserve emotional experience (Feinstein et al., 2010), and emotional learning (Turnbull and Evans, 2006), but compromise people's ability to use raw elements from the past to spatially construct virtual scenarios (Hassabis et al., 2007) and travel forward in time.

2.2.4. Situation modification

Situation modification is a second form of antecedent-focused ER, whose main goal is typically described as being to *modify the external environment* in order to make emotional

responses more manageable (Gross and Thompson, 2007, p. 12). Theoretically speaking, and in contrast to situation selection, which requires individuals to travel forward in time, situation modification occurs mostly in the present moment or immediate future, constraining individuals to rapidly and *flexibly* generate alternative actions that may change the course of a situation. Commonly cited examples of situation modification are: generating physical distance by turning away one's head or body; displaying an affect to engender an emotional impact on another person; or telling a joke to manipulate the listener's emotional state (Werner and Gross, 2010, p. 23).

As these examples make clear, situation modification is a rather non-specific ER strategy and, in the same way as situation selection, it has not yet been an object of substantial experimental research. Because individuals can modify a situation in an almost infinite number of ways, several neuropsychological components might be considered as playing a role in this complex psychological process. Situation modification is likely to require: 1) the *awareness* of the potential link between a situation and its emotional impact; 2) the *generation* of an alternative version of the situation; and 3) the *execution* of responses (in real time) to change the situation. The available evidence on how brain injury may impair the ability to modify behaviour in accordance with environmental contingencies differs substantially across lesion site. This section will only focus on two groups of patients that present different forms of behavioural inflexibility: affective inflexibility [ventromedial PFC damage] and cognitive inflexibility [lateral PFC damage]. Even though it is not the intention of this review to reduce the complexity of situation modification solely to this type of neuropsychological impairment, we believe that data from these two groups of patients offer substantial information to understand a core component of situation modification, which is the capacity to *flexibly* modify behaviour according to environmental demands.

2.2.4.1. Situation modification and affective flexibility after lesions to the vmPFC cortex

As described above, in relation to situation selection, patients with damage to the vmPFC exhibit difficulties in personal and social decision-making situations, often taking disadvantageous choices (Damasio et al., 1997; Bechara et al., 2000). It has been suggested that these difficulties are related to an impaired capacity to use emotions while navigating complex situations and events (Damasio et al., 1991; Damasio and Anderson, 1993), where reinforcement contingencies change rapidly (Rolls, 2000). A dramatic example of this failure in emotional decision making can be observed in the patient Elliot (Damasio, 1994), who presented severe personality changes after a meningioma extraction, which caused bilateral damage to the vmPFC. Damasio reports that after the operation Elliot's life drifted apart and he 'could not sustain a regular employment (…) did business with a disreputable character despite family warnings and married to a woman nor approved neither by family or friends (…) his ability to reach decisions was impaired, as was his ability to make an effective plan for the hours ahead of him, let alone to plan for the months and years of his future' (pp. 36-37).

A number of theories have been proposed to account for these difficulties (for a detailed description see Beer, 2007). One of them suggests that lesions to the vmPFC impair *affective shifting*, the ability to adapt associative learning when an initially rewarded stimulus is no longer rewarding (extinction), or when the reward and punishment value of two stimuli switch (reversal learning) (Fellows and Farah, 2003). *Affective shifting* seems likely to be a key component of situation modification, especially considering that during social situations reinforcing stimuli are constantly exchanged, and their value needs to be continually updated (Rolls, 2004).

Evidence to support the role of vmPFC in affective shifting comes from a range of sources. For example, Rolls et al., (1994) studied patients with and without vmPFC lesions on

a reversal and extinction task, where participants had to accumulate points by touching patterns on a screen, and stop touching them once they were no longer rewarded (extinction), or its value was reversed to punishment (reversal learning). The results showed that subjects with and without vmPFC lesions were able to learn the initial criteria of the task. However, when contingencies where changed, only the vmPFC group tended to perseverate, favouring the previously rewarded stimuli. In a related study, Fellows and Farah (2003) compared the performance of patients with lesions of the vmPFC with a group with dlPFC damage (and controls) on a card gambling reversal learning task. Their findings were similar to those of Rolls and colleagues (1994). Neither form of prefrontal damage compromised initial learning of the emotional associations, but the ventromedial group was specially impaired in modifying their behaviour when the task contingencies where changed.

Neuroimaging studies of healthy population have also implicated vmPFC in reward processing, especially when circumstances are uncertain or changing (Elliott et al., 2000; O'Doherty, et al., 2001; Rogers et al., 1999). Finally, lesion studies with animals have supported a similar role of the vmPFC in emotional learning, associating damage to this area to "emotional perseveration" (Morgan et al., 2003). In sum, the evidence supporting a role for affective shifting, and the vmPFC, in situation modification seems fairly robust.

2.2.4.2. Situation modification and cognitive flexibility after lateral prefrontal lesions

An important observation from the above mentioned studies is that lesions to the lateral portions of the frontal lobes do not appear to impair *affective* flexibility. However, although these patients may be able to flexibly modify emotional associations according to environmental changes, they do present with difficulties managing their behaviour, especially in complex and ambiguous situations that require choice (Burgess, 2000; Burgess and Shallice, 1996; Burgess et al., 2000; Fortin et al., 2003). For example, they typically show

difficulties consciously representing sequences of speech and behaviour, or initiating and executing such sequences in an orderly fashion (Anderson and Tranel, 2002; Fuster, 2001; Luria 1966). A classic example of this impairment is the reported dissociation between 'knowing' and 'doing' (Stuss et al., 2002; Stuss and Alexander, 2007), where patients seem able to know what *should* be done, but struggle to formulate a plan of action according to goals, to select appropriate routines to accomplish those goals, or evaluate the final outcome (Luria, 1966).

Some authors have interpreted this failure to achieve known goals (Duncan, 1986) as an impairment in the use of information to intentionally guide behaviour (Barcelo and Knight, 2002; Milner, 1964), or a deficit in the capacity to acquire and use behaviour-guiding rules (Miller and Cohen, 2001). A related consequence of this difficulty is that patients may become disorganized or inflexible in their behaviour or ideas (Podell, 2009; Sandson and Albert, 1984; Stuss and Benson, 1990). This disorganized or inflexible quality of behaviour, a form of cognitive inflexibility, may prevent patients from voluntarily modifying their actions, and thus altering the course of a situation. In consequence, it seems justified to suggest that situation modification can be compromised not only by a deficit in re-learning emotional associations (after vmPFC lesions), but also by an impairment in the use of cognition to guide and control behaviour (after dlPFC lesions). If situation modification implies the *generation* of alternative behaviour and the *execution* of new responses, lesions to the dlPFC may seriously compromise this 'cold' executive component.

There is a large literature relating prefrontal cortex (Karnath and Wallesch, 1992; Miller and Cohen, 2001), and especially dorsolateral prefrontal cortex (Alexander et al., 2007; Anderson and Tranel, 2002; Derrfuss et al., 2004; Derrfuss et al., 2005; Fletcher and Henson, 2001; Petrides, 2005) with cognitive control. Most of the available knowledge about the role of the lateral prefrontal cortex on cognitive control comes from studies on *cognitive*

flexibility after brain injury, the ability to shift avenues of thought and action in order to perceive, process and respond to situations in different ways (Eslinger and Grattan, 1993).

Such cognitive flexibility has been extensively explored in people with acquired brain injury, especially using a task-switching paradigm (Shallice et al., 2008). The Wisconsin Card Sorting Test (Heaton, 1993) is perhaps the most widely used task-switching test, and its performance has long been linked to dorsolateral PFC functioning in both neurologically lesioned (see Demakis, 2003 for a meta-analysis; Lombardi et al., 1999; Milner, 1963; Stuss et al., 2000) and healthy control groups (Berman et al., 1995; Monchi et al., 2001). However, the interpretation of findings in task-switching studies is not straightforward, and performance seems to depend on different components, which appear to have a diverse neural substrate (Aron et al., 2004; Shallice et al., 2008b; Stuss and Alexander, 2007; Stuss et al., 2000).

Several studies have found that lesions to the *left* ventrolateral prefrontal cortex (vlPFC), especially BA 44-45, generate impairments in *task setting*, a specific component of task switching (Alexander et al., 2007; Shallice et al., 2008; Stuss and Alexander, 2007). Patients with left vlPFC lesions show a greater error rate in the initial stage of switching paradigms (Alexander et al., 2005; Stuss and Alexander, 2007), where a new criterion needs to be formed, or a new schema generated, in order to complete a task. According to Stuss and Alexander (2007) damage to the left PFC compromises the patient's ability to use task instructions to direct behaviour (verbal regulation of behaviour) even when there is a comprehension of their meaning. From an emotion regulation perspective, it might be suggested that patients with left vlPFC lesion might have difficulties in the transition between old and new sets of behaviour, impairing the flexibility required for the on-line modification of a situation.

Lesions to the right lateral PFC (rlPFC) generate a completely different profile of

"switching difficulties" (Stuss and Alexander, 2007). Patients with damage in this area present errors of all kinds (Aron et al., 2004), and during the whole task, even when instructions are offered to assist performance (Stuss et al., 2000). Stuss and Alexander (2007) have interpreted this finding as a failure in *monitoring*; the process of checking the task for quality control and adjustment of behaviour. This interpretation is consistent with other authors who have suggested a similar role of the rlPFC in monitoring and checking (Shallice 2002, Stuss and Alexander, 2000). If situation modification implies flexibly executing a new set of behaviours, in order to modify the stimulus that is the source of dysregulation, *monitoring* impairments may seriously hamper the timing of actions, occurrence of errors, or awareness of discrepancies between the intended behaviour and its real effect. In other words, lesions to the rlPFC may severely compromise the proper adjustment of behaviour according to environmental feedback.

In sum, lesions to lateral and ventromedial PFC may generate a specific 'signature effect' (Shallice et al., 2008b) in situation modification impairments. Perhaps vmPFC lesions generate the most pervasive deficits, compromising the capacity of flexibly adjusting learned emotional associations, which allow the organism to shift towards more favourable situations. Lesions to the lateral PFC appear to spare affective learning, but compromise the ability to guide behaviour according to plans (i.e. Bechara et al., 1998) and inhibit the repetition of ineffective responses (Anderson and Tranel, 2002). Damage to the left vlPFC may specifically impair the initial transition from one situation to another, generating a disorganized behaviour that will, however, improve with practice and external support. Lesions to the right lateral PFC, on the contrary, may generate a more pervasive difficulty in modifying the environment, particularly because the capacity to perceive errors, and used them to re-direction behaviour, is impaired.

2.2.5. Attentional deployment

Attentional deployment refers to a strategy whereby individuals regulate emotion by *changing* the focus of attention (Gross and Thompson, 2007). It has been suggested that attentional deployment might be considered to be an *internal* version of situation selection, where people choose more favourable internal scenarios to focus on (Werner and Gross, 2010). A paradigmatic example of attention deployment is *distraction* (Gross and Thompson, 2007), where the internal focus is moved towards thoughts and memories that are inconsistent with the undesirable present emotional state (Watts, 2007), or directed to a non-emotional aspect of the situation (Rothbart and Sheese, 2007). A less investigated form of attention deployment is *concentration*, whereby cognitive resources are fully utilized in one activity, so that attention is focused on positive objects or thought (Gross, 1998).

There is a modest literature on attentional deployment describing the psychological components of this ER strategy. Nevertheless, it has been suggested that attentional deployment recruits: 1) *sustained attention*, particularly when using concentration, to *maintain* an state of alert and focus in one activity or mental content over time (Sturm, 1996); and 2) *selective attention*, in order to *limit* (or inhibit) the extent to which the emotionally evocative aspect of an event is attended (McRae et al., 2010), by *switching* the focus of attention towards a neutral (Rothbart and Sheese, 2007), or a positively-valenced (Watts, 2007) object or thought.

It is surprising that, although attentional problems are a frequent deficit after brain injury, and a common target for rehabilitation (for reviews see Manly, 2003; Michel and Mateer, 2006; Park and Ingles, 2001), no study has directly explored attentional deployment in this population from an emotion regulation point of view. Nevertheless, there is a large literature on the impact of brain injury on attentional systems (for a review see Leclercq and Zimmermann, 2002), which can be used to formulate some preliminary hypotheses. This

section will mainly focus on the impact of right hemisphere damage on *sustained* attention, without addressing in detail selective attention, a form of 'executive attention' (Petersen and Posner, 2012; Posner and Rothbart, 2007; Posner et al., 2006), which is extensively treated in sections 3 (Situation Modification) and 5 (Cognitive Change).

2.2.5.1. Attentional deployment and attentional deficits after right hemisphere lesions

Attentional deficits (slowness, selectivity, sustained attention and shifting) are reported as more *frequent* and *disruptive* after right than left-sided lesions (Leclercq et al., 2002). The best known attentional impairment after right hemisphere damage is that of hemi-inattention or neglect (for a review see Corbetta and Shulman, 2011). However, such type of deficit, compared to other non-spatial attentional deficits that arise from right hemisphere lesions, do not appear to play a central role in emotion regulation.

Deficits in sustained attention tasks have also been widely investigated after right hemisphere damage (Anderson et al., 2005; Robertson et al., 1995; Robertson et al., 1997; Wilkins et al., 1987), specifically after lesions to right prefrontal cortex (Molenberghs et al., 2009; Rueckert and Grafman, 1996; Stuss et al., 2002; Picton et al., 2006; Wilkins et al., 1987; Shallice et al., 2008a). This set of evidence is interesting in view of neuroimaging studies suggesting that the voluntary manipulation of attentional focus appear to rely on right prefrontal structures (Mayer et al., 2007; McRae et al., 2010).

Lesions to the right prefrontal cortex have long been known to produce an increase in distractibility (Woods and Knight, 1986), whereas more basic forms of involuntary attention, such as orienting responses, appear pathologically enhanced, and more voluntarily ones weakened (Homskaya, 1973), generating a form of "exaggerated attentiveness" (Goldstein, 1936a, 1936b), where patients passively *drift* from one stimulus to another. Luria (1973) offers an illustrative example of distractibility when describing a patient who performed a

task alone with no difficulties, but failed as soon as a stranger entered into the room or the person in the next bed whispered something to the nurse. Typically, the patient ceased to perform the task, and transferred his gaze to the newcomer, or joined in the conversation with his neighbour (p. 275). A similar pattern of difficulties has been also described by Scott (2011), who notes that patients with attentional impairment often report problems completing projects, because they initiate another project before finishing the current one (p. 149).

Inability to sustain attention during a task also appears to compromise *divided* attention, or people's capacity to perform more than one task at the same time. For example, Evans (2009) described the case of a patient [David] with a right internal capsule stroke who tended to be easily distracted, and also experienced difficulties doing more than one thing at once. He could not watch films for more than 20 minutes, where he would start finding it hard to concentrate. At that stage he described having to force himself to 'stay with' the film, an internal struggle that added further distraction, and made the activity even harder to accomplish (p. 94). David also reported that after the injury he had difficulties resuming a hobby of his, painting miniature military figures, while listening to the radio. He felt unable to do these two tasks at the same time, often becoming frustrated and thus abandoning the activity (p. 93).

More recent lesion studies seem to support the role of right PFC in attention, suggesting that damage to the right dorsolateral PFC generates 'monitoring' deficits, where patients are less able to sustain attention to a rule-set, in other words to *maintain* the selection of a determined schema -so that consistent target selections are made, and responses to competing targets are inhibited (Picton et al., 2006; Shallice et al., 2008a; Stuss et al., 2002). In addition, evidence from neuroimaging studies has supported brain injury data on the role of the right PFC in sustained attention (Cohen et al., 1992; Pardo et al., 1991; Lewin et al., 1996; Manly et al., 2003; O'Connor et al., 2003).

In sum, patients with right hemisphere damage, particularly right frontal lesions, appear to be less able to voluntarily sustain (as in concentration) and change (as in distraction) the focus of attention, thus probably compromising the capacity to voluntarily manipulate attention in order to modulate emotional experience. For example, it is possible that patients with right hemisphere damage might find it difficult to *concentrate* on neutral or positive stimuli when facing a negative situation, because their attention is captured by the emotional saliency of events. To our knowledge, there is only one case study that has proposed a possible interaction between impaired attentional function and the regulation of negative arousal after brain injury (Gracey et al., 2007). In that report, the authors suggested that attentional deficits play a role in understanding anxiety symptoms, especially regarding the abnormal 'hypervigilance' to potential triggers of panic-like attacks.

Another possibility that needs to be considered is that attentional impairment might *enhance* attentional deployment ability. This hypothesis relies on the above mentioned "exaggerated attentiveness" (Goldstein, 1936a, 1936b), which describes patient's tendency to drift involuntarily from one stimulus to another. It is conceivable that "exaggerated attentiveness" has the unexpected consequence of *distraction*, not only from events which one would like to attend to (as we commonly understand it) but also from events which one might *not* wish to attend towards. In other words, "exaggerated attentiveness" may enhance the ability to resist rumination, because the focus of attention is easily captured by novel distractors. As a consequence, attention does not persist on negative mental contents, but is involuntarily shifted away, or passively *drifts* away from them. In this sense, "exaggerated attentiveness" after right hemisphere lesions might be considered the neurological inverse of phenomena such attentional inflexibility, which is often observed in depressed patients (Davis and Nolen-Hoeksema, 2000; Whitmer and Banich, 2007). In passing, we note that several other cases where brain damage may not only impair function, but also enhance some

psychological processes, appear to offer support for this hypothesis (see the concept of *lesion facilitation* in Kapur, 2011).

2.2.6. Cognitive Change

A fourth ER family is cognitive change, which involves changing one's thinking in order to change one's emotions. One particularly well researched form of cognitive change is reappraisal, which refers to changing a situation's *meaning* in a way that alters its emotional impact (Gross and Thompson, 2007). It involves modifying the way that we *think* about a situation (hence cognitive change), usually reframing the meaning of an aversive event in less negative or more positive terms (McRae et al., 2011; Ochsner and Gross, 2007).

Reappraisal has been suggested to be a complex multistep process, requiring the orchestration of several cognitive abilities. It has been suggested that, in order to reappraise we need to: (1) keep the automatic appraisal of a negative situation in mind (*working memory*), (2) decrease the salience of that appraisal (*inhibition*), (3) generate alternative interpretations (*working memory manipulation* and *verbal ability*), (4) identify and engage in the interpretation that is the most appropriate (*set shifting*), (5) keep the new appraisal in mind (*working memory maintenance*), and (6) keep track of the success of the regulation process (*monitoring*) (McRae et al., 2011). This section will focus on patients with lesions to the left lateral PFC, whom often present with cognitive control and language impairments.

2.2.6.1. Reappraisal and cognitive control impairment after lesions to the left lateral prefrontal cortex

Cognitive control can be broadly considered as the ability to coordinate thoughts or actions in relation to internal goals (Koechlin et al., 2003) and environmental contingencies (Stuss et al., 1995). It has frequently linked to reappraisal (Banich et al., 2009; Ochsner and Gross, 2005), mostly because reappraisal appears to recruit core cognitive control processes, such as inhibition and set shifting (Miyake et al., 2000). Neuroimaging studies have supported this claim by showing that reappraisal ability appears to be closely associated with activation of cognitive control and language areas, particularly in the left prefrontal cortex (Goldin et al., 2008; Green and Mahli, 2006; Kim et al., 2011; Ochsner and Gross, 2005, 2007; Ochsner et al., 2002; Kalisch, 2009; Ochsner et al., 2004; Vanderhasselt et al., 2012).

Patients with lesions to the left lateral PFC (BA 44, 45) have been described as exhibiting cognitive control deficits (Alexander et al., 2007; Goldberg and Bilder, 1987). A particular type of cognitive control deficit, widely studied in these patients, is *task setting*. As reviewed before in section 3, task setting is usually conceptualized as a component of task switching, which allows disengaging from a previous activity, and the initiation of a new one. Prototypically, patients with task setting impairment present with a greater error rate in the initial stage of switching paradigms, when they are establishing a new criterion or mental schema (Alexander et al., 2005; Shallice et al., 2008; Stuss and Alexander, 2008b). This difficulty can often be observed as perseverative thoughts or as an inability to generate new thoughts.

Although the link between task setting problems and reappraisal ability seems theoretically coherent, to the best of our knowledge only one case study has described how task setting impairment after brain damage might compromise reappraisal ability (Salas et al., submitted). In this paper, a patient (Mrs M) with marked concrete thinking, after a left frontal

stroke, was studied in terms of her ability to generate positive reinterpretation from pictures that elicited negative emotions (*International Affective Picture System*). Compared to patients with no concrete thinking, and normal controls, Mrs M presented with a virtual inability to spontaneously generate positive reappraisals from the pictures. When analysing her performance in detail, it was possible to observe that her thinking process was often captured by the emotional consequences of the pictures, or by irrelevant details. In other words, and from a task setting point of view, Mrs M appeared unable to inhibit the initial [emotional] appraisal, in order to generate a new set of cognitions. Instead, and despite being directly requested to generate a positive reinterpretation, she presented with frequent perseverative thoughts related to the negative aspect of the pictures.

2.2.6.2. Reappraisal and language impairment after lesions to the left lateral prefrontal cortex

Language has been proposed as a necessary cognitive ability to reappraise (McRae et al., 2011). The rationale seems to be that, in order to reappraise, an alternative narrative about events has to be generated. Evidence from neuroimaging studies has supported this assumption, showing activation of language related areas in the left prefrontal cortex during reappraisal tasks (Goldin et al., 2008; Ochsner and Gross, 2005; 2007; Ochsner et al., 2002; Ochsner et al., 2004; Vanderhasselt et al., 2012).

Lesion studies have offered substantial evidence relating the role of the left PFC in different aspects of language (Binder et al., 1997). However, for the purpose of this review, we will only focus on two aspects often compromised after damage to this area: cognitive control and inner speech.

Language has a key role in cognitive control, coordinating sensory and semantic processes from posterior areas of the brain, and accommodating moment-by moment shifts in

goals and strategies (Binder et al., 1997). It has been described, for example, that subjects with language impairment are compromised in task switching because, in order to suppress the internal interference from a recently activated task set, *verbal* representations of the upcoming task are necessary (Chiou and Kennedy, 2009; Mecklinger et al., 1999). A review of task switching and Stroop paradigms has suggested that the left inferior frontal cortex (BA 6, 8, 44) may function as a convergence zone, where language information is integrated with premotor and working memory information, thus allowing the updating of task representations (Derrfuss et al., 2005).

The case of Mrs M might also be used to illustrate how language impairment may play a role in task switching. After her stroke, the main presenting complaint was a moderate expressive language difficulty, which evolved into a simple anomia at the time of testing, thirty five months later. Her neuropsychological profile confirmed this language problem, emphasizing a marked difficulty in verbal fluency tasks (phonological and semantic), which was consistent with her damage to BA 44, a brain area related to the generation of verbal representations that guide task switching (Derrfuss et al., 2005). In the reappraisal task this language impairment was reflected in responses that were informative (she was able to report the main themes of picture), but lacked fluidity and synthesis. She also struggled to name objects from pictures, for which she compensated by substitution or circumlocution. In sum, it can be argued that Mrs M's deficit might be partially accounted for by impairment in using language to facilitate the process of shifting from one behaviour to another.

A second aspect of language, which has not been a focus in the context of reappraisal, is inner speech. This is typically considered to be a form of inner verbal thought, usually described as fully internal and silent (Winsler, 2009). We propose that inner speech might have a role in reappraisal, firstly because reappraisal is a form of intrinsic emotion regulation, where emotion is regulated *within the self* (Gross and Thompson, 2007). In addition, the

process of reappraisal appears to require covertly talking to ourselves, in order to identify or engage with alternative interpretations of events (McRae et al., 2011). Furthermore, it has been suggested that inner speech is related to a set of abilities necessary to reappraise, such as self-observation, problem solving, and self-awareness (Morin, 2005, for a review). Unfortunately, although inner speech has long been linked to the development of self-regulation (Vygotsky, 1934/1987; Sokolov, 1972; Tullet and Inzlicht, 2010) and executive control (Emerson and Miyake, 2003; Gruber and Goschke, 2004; Miyake et al., 2004), current emotion regulation theory has not yet suggested it as a possible language-related ability.

However, there are good grounds for offering this suggestion. For example, internal speech impairment has been long related to left PFC damage (Luria, 1959, 1966, 1973), compromising self-talk and language-based thinking (Morin, 2009; Moss, 1972). This peculiar state of mind has been referred to by patients as a "dramatic silence" (Taylor, 2006) or an "impossibility to think" (Salas, 2012). In everyday situations this impairment may be observed, for example, in patients' inability to use inner speech as a guide to internally keep track of the steps of a home routine (e.g. cooking), forcing them to speak their thoughts aloud in order to hear themselves (for an example of this problem in children with traumatic brain injury see Rees and Skidmore, 2011). In a recent lesion study by Geva and colleagues (2011) it was reported that damage to the left inferior frontal gyrus (BA 44) was associated with poor performance on a conscious inner speech task. This lesion-based evidence is congruent with neuroimaging studies that have also pointed to the role of left inferior frontal areas in inner speech (see Jones, 2009, for a review).

Taken together, evidence from patients with lesions to the left PFC seems to support the relevance of cognitive control and language in reappraisal. Patients with damage to this area are often impaired initiating new sets of behaviour and using language to internally

represent goals or even think covertly. Such types of problems are likely to compromise reappraisal, which depends on being able to shift avenues of thought, and which is predominantly (as far as we know) a verbal exercise.

2.2.7. Response modulation

Response modulation is typically defined as the diverse attempts to influence emotion-response tendencies, once they have already been elicited (Gross, 1998; Gross and Thompson, 2007). Two strategies are usually proposed to sub-serve this modulatory function: *suppression* and *amplification* (or exaggeration). Emotional suppression refers to the conscious *inhibition* of one's own emotional expressive behaviour, while affectively aroused (Gross and Levenson, 1997). In contrast, amplification denotes the behavioural *augmentation* of an already initiated emotion (Demaree et al., 2004; Henry et al., 2009).

Facial expression is a common target of response modulation (Gross, 1999). In order to *suppress* facial expressions, muscular contraction has to be inhibited, so facial muscles remain relaxed. Suppression might also take the form of camouflage of the face, by *adding* facial movements with a different emotional valence [e.g. smiling when you are angry] (Ekman and Friesen, 2003). In contrast, amplification requires increasing the number of facial areas involved, its intensity and the duration of the expression. When no emotion is strongly felt, amplification understood to rely on simulation, which requires remembering the sensation of how each emotional expression felt on the face, so it can be voluntarily adjusted (Ekman and Friesen, 2003).

Response modulation strategies appear to rely on several processes. In order to suppress: (1) *Motor control* is needed to voluntarily manipulate facial expression according to contextual demands (e.g. showing sadness when someone else is sad) (Beer and Lombardo, 2007); and (2) *Response inhibition* is also required, to withhold the behavioural display

triggered by the felt emotion (Gross, 1999; Gross and John, 2003; Gross and Thompson, 2007; Kühn et al., 2011). The distinction between motor expressive control and response inhibition is based on evidence suggesting a dissociation of these two components. For example, damage to motor control pathways can generate a dysregulation of emotional expression, when no emotional experience is present. In contrast, response inhibition implies a felt emotion, which cannot be properly withheld [see sections below for further examples]. Finally, in order to modulate emotional responses (3) *Interoceptive and Emotional Awareness* are needed, to monitor and adjust the exterior state of the body, as well as the internal emotional experience (Giuliani et al., 2011). Three groups of patients, that present selective impairment in different response modulation components, will be described in this section: motor control pathway damage, right prefrontal damage and insula damage.

2.2.7.1. Response modulation and impairment of motor control pathways

An essential aspect of response modulation is the capacity to voluntarily manipulate the 'motor output' of an emotion; for example, by relaxing the facial musculature when suppressing, or increasing the number of facial areas involved when amplifying (Ekman and Friesen, 2003). Compared to other emotion regulation strategies, such as attention selection or even reappraisal, motor control is a unique feature of response modulation (Beer and Lombardo, 2007). Most response modulation experimental paradigms take this capacity for granted, when asking healthy controls to 'hide' or 'exaggerate' what they are feeling, instead focusing onto the executive abilities involved in the task, such as working memory, inhibition or abstraction (Gyurak et al., 2009, 2012). However, evidence from patients with brain injury suggests that the motoric control of expressions also needs to be properly considered.

It has been noted before (Beer and Lombardo, 2007) that patients with brain injury may experience difficulties producing facial expressions, thus compromising response

modulation strategies that depend on it. For example, observations of patients with damage to the motor control pathways have offered valuable information regarding dissociation between *volitional* and *emotional* (spontaneous) facial movements (Rinn, 1984). Lesions to the motor strip and its corticobulbar projections compromise voluntary movements in the face (hemi-paralysis), but notably spare spontaneous facial expression [e.g. smiling when something amusing occurs]. In contrast, damage to the extrapyramidal motor system (especially the basal ganglia) impairs spontaneous emotional movements, preserving voluntary control to verbal command.

More recent accounts on the role of the motor cortex in the control of facial movements have suggested that *voluntary* movements are mediated by lateral cortical structures (primary motor cortex and ventrolateral premotor cortex), both in the vascular territory of the middle cerebral artery. *Emotional* movements, on the contrary, appears to depend on midline cortical structures (rostral cingulate motor cortex, caudate cingulate motor cortex), both irrigated by the anterior cerebral artery and receiving widespread input from limbic areas (Morecraft et al., 2001; Morecraft et al., 2004).

This set of lesion studies suggests that damage to midline and lateral cortical structures may compromise response modulation strategies differentially. For example, it is possible that patients with impaired voluntary expression, although able to emotionally react to stimuli, will experience difficulties suppressing, because suppression requires the voluntary manipulation of facial muscles that are usually understood to act as 'controls' (e.g. hiding a smile by rising down the corners of the mouth) (Keltner, 1995; Keltner and Buswell, 1997). On the other hand, patients with impaired spontaneous expression might have difficulties facially reacting to the stimulus, and perhaps also enhancing or amplifying their facial responses when required.

A final group of patients with motor control impairment that deserves attention are

subjects with pathological laughing and crying (PLC). PLC has been defined as a dysregulation of emotional *expression*, often characterized by outbursts of emotions which are unrelated, or out of proportion, to the underlying feelings of happiness and sadness (Parvizi et al., 2009; Poeck, 1985, Wilson, 1923). Patients, for example, may cry in response to a joke, or laugh in response to a frustrating test failure (for an example, see patient C.B. in Parvizi et al., 2001). This dissociation between emotional experience and expression is perhaps best portrayed by one of the many terms coined for the phenomenon: *involuntary* emotional expression disorder (Cummings, 2007).

Reviews of the neuroanatomical basis of PLC have suggested that lesions disrupting the cerebro-ponto-cerebellar pathways appear to compromise the cerebellum's capacity to automatically adjust the execution of laughter and crying to the cognitive and situational context in which a triggering stimulus appears (Parvizi et al., 2009; Parvizi et al., 2001). In other words, PLC can be considered to be a form of affective dysmetria (Miller et al., 2011), a discoordination, or pathological dissociation, between the behavioural response and its affective and cognitive context (Parvizi, 2012).

The study of patients with PLC might contribute to a better understanding of the neural basis of response modulation, especially regarding the role of subcortical structures mediating suppression or the inhibition of facial displays. The most observable impairment in these patients is a lack of voluntary control over the *onset* of the attacks, and a long latency period until some type of control over the expressive behaviour is regained (see patient C.B. in Parvizi et al., 2001). An interesting question that remains open is whether suppression and amplification are partially preserved in these patients in other situations when they are not 'captured' by the attack. This possibility seems feasible and deserves further study, considering that some patients with PLC appear able to voluntarily express *appropriate* laughter and crying at least in some social contexts (Parvizi, 2012).

2.2.7.2. Response modulation, response inhibition, and right prefrontal damage

A core aspect of response modulation, and especially suppression, is the ability to *inhibit* a behavioural display while affectively aroused (Gross and Levenson, 1997). This capacity has been usually labelled in the literature as *response inhibition,* which encompasses the variety of processes aimed at controlling behaviour, particularly the suppression of unwanted, prepotent, or reflexive actions (Aron et al., 2004; Dillon and Pizzagalli, 2007).

Response inhibition has been largely explored using experimental paradigms where a motor response has to be withheld, as in *go-no go*, or *stop-signal* tasks. Consistently, neuroimaging findings (for reviews see Aron, 2007; Aron et al., 2004; Dillon and Pizzagalli, 2007; Chambers et al., 2009; Forstmann et al., 2008; Levy and Wagner, 2011; Rubia et al., 2003) and transcranial magnetic stimulation studies (Chambers et al., 2006, 2007) have suggested that response inhibition is mediated by a right hemisphere network, where the inferior frontal gyrus is a critical area -although there is some evidence that the dorso-medial PFC is also implicated in response inhibition (Floden and Stuss, 2006; Picton et al., 2006).

A limitation of the available evidence on response inhibition is that the experimental paradigms used have not involved the suppression of *emotional* behaviour but only motor responses. Nevertheless, there is one neuroimaging study (Lee et al., 2008) that has used an emotion interference paradigm, assessing the ability to inhibit emotional facial expressions. In the task, such facial expressions were evoked by looking at pictures of people showing emotional displays, an automatic phenomenon of 'contagion'. In some of the trials subjects were instructed to inhibit their spontaneous facial expressions, simulating an emotion of opposite valence (e.g. frowning when watching someone smiling). The authors found that activation in a set of areas (supplementary motor area, superior temporal sulcus and right insula), including the right ventrolateral prefrontal cortex area (BA 47), that has been previously associated to volitional response inhibition in motor tasks. More importantly, BA

47 and superior temporal sulcus activation was positively correlated with suppression scores from an Emotion Regulation Questionnaire (Gross & John, 2003). This finding is in line with the evidence reviewed above, and suggests that inhibition of the motor response associated with emotional behaviour is dependent, amongst other structures, on the right inferior frontal cortex.

Difficulties inhibiting emotional behaviour have been long reported after brain injury, but have not been explored using ER paradigms. Of especial interest are lesions to right prefrontal cortex, which has been associated with impairment in several domains of emotional life. For example, right frontal lobe damage has been related to greater severity of emotional (Tranel et al., 2002) and emotion-based decision making deficits (Manes et al., 2002; Tranel et al., 2002), difficulties inhibiting self-perspective (Samson et al., 2005), disinhibition of sympathetic centres (Hilz, et al., 2006), a higher frequency of delusional pathology (Feinberg et al., 2010) and manic disorders (Cumming and Mendez, 1984). This set of evidence appears to suggest that the right frontal lobe has a special role in emotion modulation, integrating cognition and affect (Stuss and Alexander, 1999, 2000).

It is therefore of some interest to see whether the lesion studies literature is consistent with data from brain imaging. Three lesion studies have explored the association between lesions to the right PFC and response inhibition. In one, the relationship between damage to the right inferior frontal gyrus and response inhibition performance was explored (Aron et al., 2003). The authors compared 18 patients with right frontal lobe lesions to 16 healthy controls in a stop-signal inhibition task. They found that, compared to other right frontal regions of interest (medial frontal, orbital frontal and superior frontal), the right inferior frontal gyrus (especially BA 44, 45) was critical for response inhibition; presenting negative associations with stop signal reaction time.

An extension of this study explored the impact of unilateral lesions to the PFC on

response inhibition, using a task switching paradigm (Aron et al., 2004). The authors reported that subjects with right PFC damage, specifically to the *pars opercularis* of the inferior frontal gyrus, were significantly more impaired in suppressing inappropriate responses. Finally, a third study investigated the effect of frontal lobe lesions on a response inhibition go-no go task (Picton et al., 2006). It was reported that patients with supplementary motor area lesions (BA 6) exhibited the largest number of response inhibition errors, and that patients with lesions to the right vlPFC (BA, 44, 45, 47) also presented an increase in the variability of response. Taken together, these data appear to support the role of the right PFC in general, and especially its ventrolateral surface, in motor response inhibition. In relation to response modulation, these data seem to suggest that patients with right ventrolateral damage might present difficulties withholding emotional expressions (suppression) when situational contingencies require it. This is clearly an interesting area for future research.

2.2.7.3. Response modulation, the insula, interoceptive and emotional awareness

It has been suggested (Giuliani et al., 2011) that, in order to manipulate emotional behaviour, *interoceptive* and *emotional awareness* are required. This capacity allows us to monitor and adjust the external body in relation to emotional internal experience: How much emotion am I showing? How am I feeling now? Do I need to keep suppressing or amplifying? Interoceptive awareness has been described as a sensory capacity for the on-going status of all the tissues and organs of the body, including skin, muscle and viscera (Craig, 2008). It has been suggested that these body sensations provide the basic ingredients for what is ultimately perceived as a 'feeling' (Bechara and Naqvi, 2004; Critchley et al., 2001; Critchley and Nagai, 2012; James, 1884) and, in consequence, interoceptive awareness is likely to have a crucial role in emotional awareness and the capacity to consciously recognize *what* we feel (Craig, 2010; Barret, Quigley et al., 2004; Wiens, 2005).

During the last decade a number of neuroimaging studies have suggested that the insular cortex may be a key neural structure for interoceptive and emotional awareness (Craig, 2009, 2010b; Critchley et al., 2004; Simmons et al., 2012; Zaki et al., 2012). Furthermore, it has been proposed that, specifically, the *anterior* insula is essential for the subjective awareness of emotion, through the cortical representation of the interoceptive image of the body's physiological condition (Coen et al., 2009; Craig, 2008; Critchley et al., 2004).

In addition to the issue of emotional experience, there is also a growing literature on the role of the anterior insula and emotion *regulation*. For example, it has been suggested that the joint activation of ACC and the right anterior insula not only generates a representation of emotional awareness (the experiential aspect), but also enables motivation to *modulate* (the regulatory aspect) subjective feelings (Craig, 2008; Medford and Critchley, 2010). This regulatory role of the insular cortex has received support from studies with clinical populations that exhibit emotion dysregulation problems, such as posttraumatic stress disorder (Simmons et al., 2009) or anxiety disorders (Paulus and Stein, 2006). Neuroimaging studies using suppression paradigms have also offered valuable data, for example demonstrating insula involvement in tasks that require the suppression of natural urges [spontaneous blinking] (Lerner et al., 2008), suppression of imitative emotional expressions (Lee et al., 2008) and suppression of emotions themselves (Hayes et al., 2010; Goldin et al., 2008). In addition, anterior insula volume has also been correlated to expressive suppression usage (Giuliani et al., 2011).

In view of the neuroimaging evidence relating the insula to interoceptive and emotional awareness, as well as to ER, it seems reasonable to expect that patients with insular damage would be impaired in such capacities. Unfortunately, despite repeated calls to carry out in depth studies of patients with focal lesions to the insula (Bechara and Naqvi, 2004;

Jones et al., 2010), we still know very little. However, from the available literature on neuropsychological deficits after insular damage (for reviews see Ibanez et al., 2010; Jones et al., 2010) it seems that lesions to this structure do not compromise emotional *perception* (Couto et al., 2012), but impair to a certain extent *interoceptive* awareness. Studies have reported a decrease in addictive behaviours (Naqvi et al, 2007), bluntness towards risk (Clark et al, 2008; Weller et al., 2009) and decision making impairment (Bar-On et al., 2003). Lamentably, in relation to emotional awareness and ER, it is not possible to draw firm conclusions, given the limited extent of this literature.

To our knowledge there is only one case study, recently published (Damasio et al., 2012), that attempts to explore the link between emotional changes and insular damage. The report is quite remarkable in that it summarizes observations for over two decades, from a patient (Patient B.) with extensive brain damage as result of encephalitis (bilateral insula, bilateral amygdala system, medial and middle sections of the oPFC cortices and polar and mesial temporal cortex). A relevant aspect of the patient's neuropsychological presentation is his dense amnesia, being unable to remember any factual items for more than 45seconds (anterograde amnesia), or recall any events from the two decades prior to the neurological insult (retrograde amnesia).

Based on behavioural observations (from researchers, relatives and blind observers), psychological evaluations (Rorschach inkblot test, Thematic Apperception Test), self-awareness tasks, and emotional learning tasks, the authors concluded that all aspects of B.'s feelings were intact, though insular cortex had been entirely destroyed bilaterally. B. was able to manifest 'his likes and dislikes, comfort and discomfort, pleasure and pain' (Damasio et al., 2012, p. 2). The main proposition of the authors is that the insular cortex does not appear to be a *necessary* brain area for the experience of feelings. Instead, the authors suggest that other brain structures, which are preserved in B, such as the somatosensory cortex, the brain

stem complex and the basal ganglia, can account for the generation of "feeling maps and feeling states with an hedonic component" (p. 12).

If we consider B.'s case, from the point of view of the insula and its relevance for *interoceptive* awareness, the evidence presented by the authors appears to challenge the neuroimaging findings. Clearly B. does not present the 'absence of emotional salience' suggested by Craig (2008), or the 'lack of appropriate affective response' suggested by Jones, Ward and Critchley (2010), as a consequence of insula damage. On the contrary, B is described as able to experience a wide array of interoceptive (e.g. pain, itch, tickle) and emotional (e.g. happiness, apprehension, irritation, caring) feelings. Unfortunately the type of data offered by the authors is largely based on the observation of B's behaviour and B's self-report, which makes difficult to link these findings to the experimental paradigms on which the interoceptive role of the insula has been forged, such as heart beat detection (Critchley et al., 2004), emotional response to pain (for a review of studies see Craig, 2008) or even decision making (Clark et al, 2008; Weller et al., 2009).

A second point that needs to be addressed refers to B.'s emotional *awareness*. If we use the multilevel operationalization of emotional awareness proposed by Lane and Schwartz (1987), it is clear that B. is able to experience physical sensations, that he exhibits action tendencies based on them, and that he can recognize and label basic emotions. However, it is difficult to infer, either way, from the report about B.'s *higher* levels of emotional awareness, which require a reflective capacity (Monsen and Monsen, 1999), in order to recognize and describe blends of emotions, or even blends of blends of emotional experience (Lane and Schwartz, 1987). It is interesting here to note that Damasio et al., (2012) do mention a major psychological change on B., but without further elaborating on its relevance for emotional life. This they describe as a 'shallowness of intellect (p. 2)' and also as 'shallowness of his mental contents (p. 2)'. Unfortunately, the authors do not explore this in great detail, or

attempt to formally investigate the nature of this change. Two questions arise from this. Is this 'shallowness of mental contents' exclusively a consequence of the amnesia and hippocampal damage, or of the insula lesion, given its proposed integrative function between cognition and affect (Craig, 2009; Kurth et al., 2010; Simmons et al., 2012)? Is it possible that this 'cognitive shallowness' impairs the access to higher levels of emotional awareness, which require representational ability to reflect upon feelings? Studies in acquired concrete thinking after brain injury (Hanfmann et al., 1944; Salas et al., in press) seem to support this point, especially in relation to impairment in the capacity to manipulate ideas and thoughts (Goldstein, 1936, 1944; Goldstein and Scheerer, 1941).

In conclusion, although Damasio et al., (2012) add extremely important data to debate the role of the insula in interoceptive awareness, B's case may not completely refute the related hypothesis that the insula might have a role in the awareness of emotional feelings, especially in higher order levels of awareness. Unfortunately, the presence of dense amnesia is a major confounding variable, suggesting that future studies might explore the same phenomena in patients with more circumscribed lesions, and more sensitive instruments to capture latent differences (see *Affect Consciousness Interview*, Lech et al., 2008).

In relation to ER, although Damasio et al., (2012) do not offer any direct observation on B.s capacity, it is possible to infer some changes. For example, the authors describe 'a pattern of diminished influence of negative feelings in the elaboration of meaning of complex social situations (p. 8)', which appears to be associated to 'a slight upward change in the experience of positive emotion' and a 'lack of concerns about the future (p. 3)'. One possibility to explain such phenomena is that B's lesion generated changes in emotional *reactivity*, so positive emotions are more promptly experienced and negative emotions are lived less intensively. A dampening in reactivity to negative emotions would imply a decrease in the frequency of negative situations that require regulation. Nevertheless, it would

not be possible to attribute these changes to insular damage, especially considering clinical reports describing a decrease of negative emotions and an increase in positive feelings in acquired amnesia (Talland, 1968; Tate, 2002; Ogden, 1996; O'Connor et al., 1995; Corkin et al., 1997; Kaushall et al., 1981).

In addition, it remains an open question how B. would deal with events that require him to modulate (e.g. suppress, amplify) what he is feeling. So far the authors suggest that his 'affective life became the most salient aspect of his behaviour (p. 2)', which seem to imply that he became more direct in expressing likes and dislikes. In this sense it is possible to think that perhaps B. would present difficulty to suppress or amplify emotional behaviour, because response manipulation requires decreasing the *salience* of spontaneous emotional responses. However, this is merely speculative and limited by the confounding effect of deep amnesia and its impact in 'time travelling' ability (see section 2).

In sum, evidence from patients with focal lesions seems to support the relevance of motor control and behavioural inhibition in response modulation, particularly for suppression. The available data from damage to the insula is less conclusive, although it appears to challenge assumptions regarding its role in interoceptive awareness. Future studies exploring emotional changes in more restricted insular lesions are urgently needed, as is the use of experimental paradigms that allow for a cross talk with the existing evidence from the neuroimaging literature.

2.2.8. Conclusions

During the past decade, there has been a growing interest in the neural basis of ER strategies but, surprisingly, most of the evidence has been gathered from neuroimaging methods, ignoring the possibilities offered by the lesion study approach. The main goal of this article has been to link existing *neuropsychological* knowledge from lesion studies with recent

evidence on the core psychological mechanisms underlying different ER strategies. To incorporate lesion studies in the field of ER would offer a powerful 'convergence of methods' (Gonsalves and Cohen, 2010).

This review has described sub-groups of brain injured patients (e.g. right frontal damage) that are impaired in particular *psychological processes* (e.g. sustained attention), which have been suggested as key elements of specific ER strategies (e.g. attentional deployment). This review is the first [c.f. Beer and Lombardo, 2007] to explicitly adopt a *process model* of ER (Gross and Thompson, 2007), addressing *all* five proposed ER strategies. In addition, this review approaches ER strategies from a dynamic localization point of view (Luria, 1966), which holds that complex psychological process, as in the case of ER strategies, rely on several neuropsychological components and associated distributed neural systems.

An important limitation encountered in the task of review has been the remarkably unbalanced degree of attention that each ER strategy has received by the scientific community. The basic psychological mechanisms of some ER strategies, such as response modulation or cognitive change, appear to be far better understood than, for example, situation selection or situation modification, where research is almost non-existent (for an exception see Rovenpor, Skogsberg and Isaacowitz, 2012). The gap is therefore larger for the earlier *antecedent-focused* ER strategies. As a consequence, the exploration of these under-investigated strategies is urgently needed, to offer a more complete understanding of ER, and indeed to address applied issues such as rehabilitation after brain injury.

A further conclusion is that the available data from lesion studies seem to support the idea that *voluntary* ER comprises a set of neuropsychological processes highly dependent on frontal lobe functions (Davidson, Fox and Kalin, 2007; Ochsner and Gross, 2005, 2007; Ochsner et al., 2004; Quirk, 2007; Quirk and Beer, 2006; Zelazo and Cunningham, 2007).

Even though this link has been suggested by several studies in relation to cognitive change (Goldin et al., 2008; Kalisch, 2009; Kim et al., 2011; McRae et al., 2011; Ochsner et al., 2002) and suppression (Goldin et al., 2008; Gyurak et al., 2009; Kühn et al., 2011; Lee et al., 2008; Levesque et al., 2003; Ohira et al., 2006; Phan et al., 2005), the data presented here offer additional support regarding other, less investigated, strategies, such as situation selection, situation modification and attentional deployment.

In addition, evidence from brain injured patients suggests a number of other non-frontal brain structures that are necessary to sustain key ER processes. This includes the temporal lobes [see section 2.1 on foresight, situation selection and amnesia] and the cerebellum [see section 6.1 on response modulation and motor control]. In sum, evidence from lesion studies suggests that ER strategies probably depend on a wide network of cortical and subcortical brain structures, where prefrontal regions, because of their relevance for the behaviour regulation, appear to have a critical role. Nevertheless, this is merely an initial, and preliminary, sketch of the neuropsychological processes that may underlie ER, focusing on the components that present the most robust current set of evidence. In the future, the contribution of other less studied neuropsychological components, and brain areas, will need to be considered.

An additional observation is that, although some psychological abilities (e.g. time travel) appear to be more closely related to one specific ER strategy (e.g. situation selection); others seem to be recruited by several [see Table 1]. For example, *situation modification* and *cognitive change* both rely on *task setting*; a process commonly impaired after left vlPFC lesions (Alexander et al., 2007; Shallice et al., 2008a; Stuss and Alexander, 2007). Nevertheless, although task setting appears to have a key role in *both* strategies, it may well be employed in slightly different ways. In situation modification it allows the generation of alternative *actions*, while in cognitive change it facilitates the shifting between alternative

interpretations. Another example of overlap in function is inhibition. This has a key role in response modulation, where emotional behaviour needs to be withheld (Gross and Levenson, 1997). However, inhibition seems to also have a role in attention deployment, by limiting the extent to which an emotionally evocative aspect of an event is attended (McRae et al., 2009). The fact that ER strategies rely, at the same time, on *specific* and *shared* neuropsychological components, is consistent with the idea of a common 'functional architecture' for ER, sufficiently flexible to support multiple types of control strategy (Ochsner and Gross, 2005).

The complex landscape of these neuropsychological findings can be summarized by two principles: a) a single ER strategy may be compromised by damage to different brain areas, each lesion impacting the strategy with its particular neuropsychological signature. For example, *situation selection* can be altered by hippocampal damage (episodic future thought impairment) and also by vmPFC lesions (emotional learning impairment). In addition, b) damage to a specific brain area (e.g. left vlPFC lesions), with its associated signature deficit (cognitive control impairment), may compromise more than one ER strategy (e.g. situation modification and cognitive change). The consideration of these two principles, when exploring ER from a lesion study paradigm [c.f. Luria 1966], is a fundamental guide to progressively build a map of the neuropsychological components that underlie different ER strategies.

A further advantage of the lesion study approach is that it allows the investigation of components that have been suggested by psychological models, but that are difficult to capture when testing healthy controls, or even when assessing patients with psychiatric problems. For example, the study of subjects with concrete behaviour [see section 5] offers a unique opportunity to observe how reappraisal might rely on the capacity to disengage from, or inhibit, initial emotional appraisals. Even though this component has been suggested in the literature on cognitive change (McRae et al., 2011), the assessment of such capacity in a

healthy population has not been confirmed when reinterpreting negative events.

The study of brain injured patients also offers valuable opportunities to enrich the psychological models on ER, by proposing *new* neuropsychological components. Inner speech is a case in point. This form of inner verbal thought has been long been associated with self-talk (Morin, 2009; Moss, 1972), self-regulation (Vygotsky, 1934/1987; Sokolov, 1972; Tullet and Inzlicht, 2010) and executive control (Emerson and Miyake, 2003; Gruber and Goschke, 2004; Miyake et al., 2004). Lesions to the left inferior frontal gyrus appear to compromise the capacity to use inner speech (Geva et al., 2011). It is interesting that psychological models of reappraisal have not considered the relevance of inner speech, though they acknowledge a general role for verbal ability (McRae et al., 2011). Perhaps a reason why inner speech has been overlooked is that experimental designs tend to take this capacity for granted, and perhaps because in healthy controls such ability appears to be universally present.

Other examples of neglected psychological components might be episodic future thought, envisioning emotional future events, motor control, interoception, etc. The benefits of investigating brain injured population is therefore that, in these 'extreme cases', such basic capacities are often compromised, opening an atypical window on behaviour. In future, it might be possible to derive further insight from this patient population, refining the operationalization of ER strategies and developing more challenging measures of how ER components might be tested in healthy controls. This could be especially relevant for the comprehension of ER strategies that have been poorly operationalized, as is the case of situation selection and situation modification.

Emotion Regulation Strategy	*Neuropsychological Component*	*Lesion*
Situation Selection	Mental travel (episodic memory and episodic future thought)	Medial temporal cortex
	Emotional learning (envisioning emotional future events)	Ventromedial Prefrontal cortex
Situation Modification	Affective Shifting	Ventromedial Prefrontal cortex
	Cognitive Shifting (Task Setting)	Left Ventrolateral Prefrontal cortex (BA 44, 45)
	Cognitive Shifting (Monitoring)	Right Ventrolateral Prefrontal cortex
Attentional Deployment	Sustained Attention and Selective Attention	Right Hemisphere (Prefrontal)
Cognitive Change	Cognitive Shifting (Task Setting)	Left Ventrolateral Prefrontal cortex(BA 44, 45)
	Language (Cognitive Control)	Left Inferior Prefrontal cortex (BA 6, 8, 44)
	Language (Inner Speech)	Left Inferior Prefrontal cortex (BA 44)
Response Modulation	Motor Control of Facial Expressions (Voluntary)	Motor Strips and Corticobulbar Projections; Primary Motor Cortex and Ventrolateral Motor Cortex
	Motor Control of Facial Expressions (Spontaneous)	Extrapiramidal Motor System, Basal Ganglia; Rostral cingulate motor cortex, caudate cingulate motor cortex
	Coordination of Facial Expressions with Affective-Cognitive Context	Ponto-Cerebellar pathways
	Response Inhibition	Right Ventrolateral Prefrontal cortex (BA 44, 45, 47)
	Interoceptive Awareness and Emotional Awareness	Insula

Table 1. Summary of Emotion Regulation Strategies, neuropsychological components and related neural structures

This review has focused on the study of patients with focal brain lesions. Nevertheless, other populations, with more *widespread* or *non-focal* damage, for example degenerative disease or traumatic brain injury, might also be sources of valuable insight. An obvious limitation, of course, is that such patients have more diffuse lesions, thus limiting the inferences that can be drawn about particular brain areas. However, this limitation should not overshadow the many possibilities offered by such populations. Emotion dysregulation is a key feature of some neurological degenerative conditions (Goodkind et al., 2010; Henry et al., 2009; Stocchi and Brusa, 2000; Sturm et al., 2006) and traumatic brain injury (McDonald et al., 2010; Obonsawin et al., 2007; Rochat et al., 2009, Tate, 1999). Interestingly, such disorders present, as a population, with a reasonably consistent neuropsychological profile dominated by impairments to executive abilities (Fork et al., 2005; Huey, 2009; Millis et al., 2001; Stopford et al., 2012), which are key for the many cognitive control processes required in ER (Ochsner and Gross, 2005, 2007; Ochsner et al., 2004; Quirk, 2007; Quirk and Beer, 2006).

A limitation of this review is that it focuses exclusively on ER strategies from a process model point of view (Gross, 1998). Even though this model is perhaps the most comprehensive framework currently available, it does not consider all the psychological mechanisms that have been related to the regulation of negative affective states. For example, there is a growing set of evidence suggesting that the *acceptance* of emotional experience, defined as the openness to internal experience and willingness to remain in contact with those experiences (Hayes, Luoma, Bond, Masuda and Lillis, 2006), may be even more effective than classic ER strategies [such as suppression] in reducing negative affect (Campbell-Sills, Barlow, Brown and Hofman, 2006). In addition, acceptance-based therapies have been suggested as useful tools managing emotional difficulties in patients with acquired brain injury, whom often struggle cognitively re-constructing meaning, because of difficulty

manipulating thoughts (Coetzer, in press). Future studies might address this ability, exploring how it relates to different psychological components, and also how lesions to different brain areas may compromise it selectively.

A final issue of enormous importance is the applied aspect of these ideas. A better understanding of the way in which voluntarily ER breaks down after brain damage is also of great relevance for neuropsychological rehabilitation. In recent years the field has progressively moved towards an emphasis on socio-emotional functioning and social participation (Mateer et al., 2005; McGrath, 2004; Wilson, 1997; 2003; 2008; Wilson et al., 2010). In this sense, the rehabilitation professionals are well aware of changes in the capacity to manage feelings exhibited by their patients. However, they typically lack rigorous understanding of the ways in which these can be classified within an emotional regulation framework, the ways in which these map into lesion sites and the ways in which such an understanding might shape neuro-rehabilitation assessment and intervention. Thus, an understanding of the neuropsychology of ER can be of direct clinical importance.

2.3. Stuck in a moment: Concreteness and Psychotherapy after Acquired Brain Injury[4].

2.3.1. Abstract

This paper surveys the issue of concrete thinking after brain injury –a phenomenon which is widely recognized clinically, but under-investigated in formal research settings. Through the lens of the classical work of Kurt Goldstein the paper outlines the diverse clinical manifestations of concreteness, and the barriers which this might present to the psychotherapeutic process. However, the paper also outlines the way in which preserved psychological functions in highly concrete patients, especially the capacity to focus on immediate reality, and experience emotions in present time, can be used as a lever for psychotherapeutic interventions. The paper concludes with a range of practical suggestions which may aid the psychotherapist in reaching out to this challenging patient group.

[4] Salas, C.E., Vaughan, F.L., Shanker, S., & Turnbull, O.H. (2013). Stuck in a moment: concreteness and psychotherapy after acquired brain injury. *Neuro-Disability & Psychotherapy*, 1(1): 1-38.

> "Closer examination shows that in order to readjust itself to the world, the injured organism has withdrawn from more or less numerous points of contact with it and has thus attained a re-adaptation to a shrunken environment"
>
> Kurt Goldstein, *The modification of behaviour consequent to cerebral lesions*.

2.3.2. Introduction

Concreteness, sometimes used synonymously with the term impaired abstraction, is a common phenomenon after brain injury. It is quite usual to hear, among rehabilitation professionals, phrases such as "this intervention is unlikely to work, because this patient is too concrete", or "he cannot completely understand what he is going through, because of his concreteness". If we listen to the way professionals use this term, when speaking about patients, one might suggest that: a) therapists are able to perceive something *characteristic* in the mode these patients function (an observable fact), which they later *label* as concreteness; b) this mode of functioning is experienced as a challenge to the use of therapeutic tools, and the achievement of rehabilitation goals, including eventual generalisation and habituation of therapeutic gains. However, when professionals are explicitly asked about how they understand concreteness, and how they address it technically, their responses are usually imprecise, with a vague use of terminology, and with a rather poor grasp to how concreteness maps into specific impaired functions, or particular sites of brain lesions. The purpose of this article is to address this conceptual gap, by offering a theoretical account of concreteness, and drawing preliminary guidelines on how psychotherapeutic tools could be adapted, to facilitate emotional adjustment in this population.

2.3.3. What is Concreteness

Concreteness, or the difficulty to orient our actions by a conceptual point of view (Goldstein, 1942; Goldstein & Scherer, 1941), is not a popular concept today in neuropsychology. This is evident if we consider the remarkably modest amount of research that has been published in the last few decades addressing concreteness after brain injury [see Fig. 1]. The principal reason may well be that, compared to other neuropsychological processes broadly related to frontal lobe function, such as working memory, planning, or set-shifting, concreteness seems vague and lacking neuroanatomical specificity.

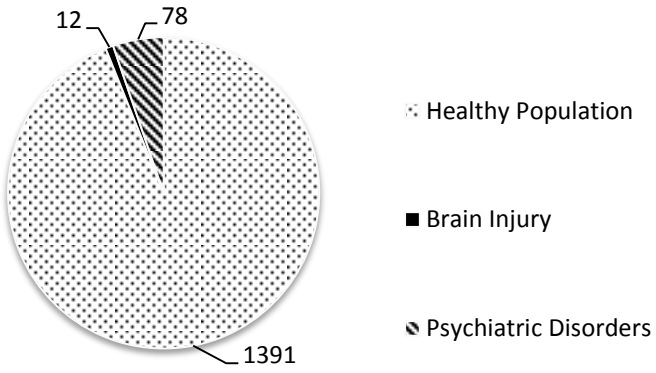

Fig. 1: Number of publications on concreteness and brain injury. An all-time literature search in PubMed was performed using the following keywords: concreteness, concrete attitude, concrete thinking and brain injury. From a total of 1481 relevant articles, only 12 were related to brain injury.

Based on clinical observations, different authors have suggested that concreteness is not an homogeneous deficit (Goldstein & Scherer, 1941, Prigatano, 1989) and that it is frequently associated with diffuse lesions (Judd, 1999), or with widespread damage to the frontal lobes (Judd, 1999; Klonoff, 2010; Ogden, 1996), particularly on the left (Goldstein, 1942). Given that concreteness [or impaired abstraction] has long been associated with diffuse lesions to the frontal lobes (Yang, Fuller, Khodaparast & Krawczyk, 2010), and with a growing interest of actual neuropsychology in fractionating frontal lobe functions (Miyake et al., 2000; Stuss, 2011; Stuss & Alexander, 2000; 2007), is no surprise that concreteness has potentially become a rather archaic concept. Interestingly, a different historical trajectory has occurred in the more applied field of neuropsychological rehabilitation, where concreteness as a concept which can inform practice, has survived among clinicians that work with people who experience a brain injury. This paper will focus, and develop, the *clinical* value of concreteness.

Authors in neuropsychological rehabilitation have used the concept of concreteness in different forms. For example Cicerone and Giacino (1992) refer to the phenomenon as a failure appreciating the *abstract* or *symbolic* qualities of ideas, comments or things. Ponsford, Sloan and Snow (1995) have suggested that concrete patients struggle to *generalize* from one event, or in *distilling the essence* of a situation, which impairs their capacity to learn from experience. Another frequent reported feature of concreteness is a difficulty thinking beyond present thoughts, or seeing beyond one's own perspective (Judd, 1999), which may even translate into difficulties with empathy (Eslinger, 1998), or self-centeredness (Judd, 1999; Obonsawin, Jefferis & Lowe, 2007).

It is interesting to note that most of the above mentioned definitions are in close alignment with the early work of Kurt Goldstein, who coined the term "concrete attitude". He described it as a "*realistic* attitude, where behaviour is confined to the *immediate* [not

reflective] apprehension of a given thing or situation" (Goldstein, 1936b; 1942; Goldstein & Scherer, 1941).When addressing concreteness, most authors refer more or less directly to this feature; a basic difficulty in the organism's ability to *detach* itself from the immediate environment, in order to generate a flexible response. Clinically speaking this is usually portrayed as a difficulty using abstract thought or discursive reasoning to guide behaviour. As a consequence, behaviour may become perseverative or extremely susceptible to the varying stimuli in the environment (Goldstein & Scherer, 1941, p. 3).

However, an important trend in recent accounts is the tendency to reduce concreteness to a *cognitive* impairment, equivalent to other neuropsychological deficits caused by brain injury (e.g. aphasia, impulsivity, inattention). This interpretation may be misleading, and certainly does not reflect Goldstein's original idea. He explicitly specified that concreteness was not equivalent to a cognitive deficit, but rather reflected an *attitude* of the *total* personality:

> "The abstract and the concrete attitudes are not acquired mental sets or habits of an individual, or special isolable aptitudes, such as memory, attention, etc. They are rather *capacity levels of the total personality*. Each furnishes the basis for all performances pertaining to a specific plane of activity. In other words, each attitude constitutes one definite *behavioural range* which involves a number of performances and responses. These latter, when taken individually at their surface value, may appear to be discrete entities of quite a diversified nature (e.g. attention, recall, retention, recognition, synthesizing, symbolization, etc.)" (Goldstein & Scherer, 1941, p.1)

What seems to be essential in Goldstein's definition, and absent in recent accounts, is that concreteness refers to a radical change in the way that the *whole* personality, or the *Self*, constructs internal and external experience. When elaborating this point, Goldstein explains that, by *attitude*, he implies a form of *readiness* for response. In assuming an abstract or

concrete attitude, he claims, the individual *as a whole* gears himself toward a specific direction of activity, a *mode* of functioning (Goldstein & Scherer, 1941, p. 2). This emphasis on the Self, which we believe is crucial for holistic rehabilitation, is difficult to grasp when concreteness is considered as a cognitive impairment. The intention of this article is to move away from such neuropsychological reductionism, adopting a self-psychology perspective to brain injury (Klonoff, Lage & Chiapello, 1993; Prigatano, 1999; Salas & Turnbull, 2010; Salas, 2012).

In this regard it is interesting to note the similarities between Goldstein's proposition and more recent theoretical accounts of how behaviour may change after frontal lobe damage (see Table 1). Mesulam (1986; 2002), for instance, has suggested that large frontal lesions may allow the *resurgence* of a *default mode*, a realm of neural function where inflexible stimulus response linkages (the 'realistic attitude' of Goldstein) remain impervious to modification by context or experience (p. 14-15). Here the shared notion of "mode of functioning" is notable, because it stresses the idea that all the organism's abilities are aligned under one single pattern of functioning (a *mode* in Mesulam and an *attitude* in Goldstein).

Concrete Attitude **(Goldstein & Scherer, 1941)**	***Default Mode*** **(Mesulam, 2002)**
Realistic Attitude (unreflective and confined to the immediate apprehension of an object, idea or situation)	Horizon of consciousness confined to here and now and set reflexively in a stimulus-bound mode
Thinking and acting are directed by the immediate claims made by one particular aspect of the object, or the outer world situation.	Automatic reactions to salient events and immediate gratifications are guided by prevailing motivational states
Rigidity and lack of shifting (but also abnormal fluidity)	Presence of repetitive responses although they may not fit with environmental demands
Impairment in the manipulation and operation over ideas and thoughts	Options for alternative interpretations are not encouraged
Cannot assume an attitude towards the "mere possible" and to think or perform symbolically	Appearance cannot be differentiated from significance

Table 1: Comparison between Goldstein's Concrete Attitude and Mesulam's Default Mode.

A final theoretical comment needs to be made regarding concreteness as a neuropsychological deficit. There is no doubt that frontal lesions may impair many cognitive capacities that allow an abstract stance (e.g. set shifting, working memory, monitoring, etc.), however, this *loss* of abstract abilities should not be considered as an equivalent of concreteness itself. In other words, concreteness cannot be reduced to the *absence* of an abstract attitude, to some kind of abstraction "blindness". Following Mesulam's idea of a default mode, concreteness may be better explained as a *re-surgence* of a primary mode of functioning, which has been progressively obscured by the development of abstract capacities throughout ontogeny, and which is preserved in patients despite extensive frontal lobe damage. This view of concreteness, as *preservation*, not as a deficit, has been described previously by Sacks (1986) and has radical consequences to how we approach concrete patients, and recognize them as experiencing beings:

> "Much easier to comprehend, and altogether more natural, is the idea of the preservation of the concrete in brain damage –not regression *to* it, but preservation *of* it, so that the *essential personality* and *identity* and *humanity*, the being of the hurt creature, is preserved" (Sacks, 1986, p. 165).

We have briefly revised the concept of concreteness, hoping that by clarifying it theoretically, its practical relevance in rehabilitation and psychotherapy would become evident. By moving away from a definition that reduces concreteness to a specific cognitive deficit, we have adopted a broader view, highlighting how brain damage modifies the organism's *relationship* with its outer and inner worlds. This distinction is indispensable to understand, for example, how functioning in a concrete *mode* might transform, but not abolish, subjective experience after the injury (see Prigatano, 1991).

2.3.4. Why concreteness is relevant to Neuropsychological Rehabilitation

We believe that the concept of concreteness has survived among rehabilitation professionals because of its clinical value. Concreteness is useful because, as a concept, it fits well with three main principles that guide rehabilitation practice. Firstly, concreteness underlines how brain injury modifies the experiencing Self, a proposition that is in accordance with the first principle of rehabilitation: the clinician must begin with the patient's subjective or phenomenological experience, in order to reduce frustration and confusion as means to engage him in the rehabilitation process" (Prigatano, 1999, p. 3). Goldstein's model of concreteness offers a detailed description on how *phenomenological experience* may change after brain injury, allowing a deeper understanding of these transformations and, in consequence, helping clinicians to grasp the disorganized mind and to attune to the wounded soul.

Secondly, we know that neuropsychological rehabilitation has progressively evolved from an interest on cognitive deficits, and retraining, to an increased emphasis on socio-emotional functioning and participation (Mateer, Sira & O'Connell, 2005; McGrath, 2004; Wilson, 1997; 2003; 2008; Wilson, Gracey, Evans & Bateman, 2010). This movement is, for example, illustrated by the growing literature on identity change (Carroll & Coetzer, 2011; Cloute, Mitchell & Yates, 2008; Gracey et al., 2008; Yeates, Gracey & McGrath, 2008; Ylvisaker & Feeney, 2000; Segal, 2010) and interpersonal relationships (Bowen, Yeates & Palmer, 2010; Bowen et al., 2009; Feigelson, 1993) after brain injury. Concreteness, as we have defined it, taps directly into these issues. It usually implies some kind of *shrinkage* of the *temporal* and *representational* domains of the Self, which radically transforms the way in which the Self relates to itself [Self as an object] and to others [Self-Other]. A deeper understanding of concreteness may, therefore, guide the assessment and design of rehabilitation interventions addressing these emotional and interpersonal issues.

Thirdly, concreteness, often associated with frontal lobe damage, compromises elaborated thinking and the use of organising schemas. As a consequence, patients may experience difficulties creating new and adequately organised mental models of the self after the injury (Ylvisaker & Feeney, 2000; Prigatano, 1989; Ylvisaker, Mcpherson, Kayes & Pellet, 2008), compromising a central goal for rehabilitation (Biderman, Daniels-Zide, Reyes & Marks, 2006; Klinger, 2005; Nochi, 1998).

2.3.5. Why concreteness is relevant to psychotherapeutic approaches to brain injury

The influence of holistic paradigms in neuropsychological rehabilitation has generated renewed interest on the use of psychotherapeutic tools to address emotional (Coetzer, 2004; 2007; Dewar & Gracey, 2006; Freed, 2002; Gracey, Olsham & Kritzinger, 2007; Klonoff, 2010; 2011; Lewis, 1999; Prigatano, 1986; Psaila & Gracey, 2009; Salas, 2008) and interpersonal (Bowen, 2007; Bowen, Yeates & Palmer, 2010; Salas, 2012; Yeates et al., 2008) difficulties after brain injury. Interestingly, lack of insight and inflexible thinking, two features usually associated to concreteness, have been reported by therapists as common challenges in their work with people who acquired a brain injury (Judd & Wilson, 2005). Unfortunately very few authors have addressed how concreteness may impact the use of psychotherapeutic tools, and the necessary technical modifications that working with these patients entail (Judd, 1999; Klonoff, 2010; Miller, 1993; Prigatano, 1986, 1994; Salas, 2008b; Ylvisaker & Feeney, 2000).

Concreteness is relevant to psychotherapy for several reasons. First, and most obvious, because it implies a change in how the subject relates to his internal world, thus altering the same territory in which psychotherapy takes place. Second, the *realistic attitude*

may impact different dimensions of the therapeutic process[5], setting theoretical and technical challenges that therapists must deal with. For example, concreteness might compromise patients' capacity to spontaneously report problems or conflicts to the therapist (*Therapeutic operations*), reflect about themselves during personal (*Self-relatedness*) and interpersonal interactions (*Therapeutic bond*) or link emotions with external events or situations (*In-session impacts)*.

2.3.6. Challenges to psychotherapeutic work with concrete patients

The *concrete* or *realistic attitude*, described by Goldstein, can be considered as contraction in temporal (*presentness*) and representational (*meaning generation*) dimensions of the Self (see figure 2). In this section we will describe how these transformations may challenge the use of classic psychotherapeutic tools, requiring theoretical and technical adaptations.

2.3.6.1. Concrete attitude as Presentness

According to Goldstein, the concrete or realistic attitude implies that we are confined to the *immediate apprehension* of a given object or situation, in its particular uniqueness. Thinking and acting are thus directed by the *immediate claims* which one particular aspect of the object or of the outer world situation makes (1941, pp. 2-3; 1942, pp. 89-90). At a behavioural level, this feature of concreteness can be observed as a difficulty of voluntarily shifting from the experience of a present stimulus, or also as a passive drifting from one stimulus to another (exaggerated attentiveness). In both cases the object, or situation, acquires an *abnormal preponderance,* and the individual is thus *forced* by the stimulus (Goldstein, 1936a; 1936b). In practice, patients may come across as "inflexible" or "rigid".

[5] see Coetzer, 2007, for a description of different dimensions in a generic model of psychotherapy for traumatic brain injury

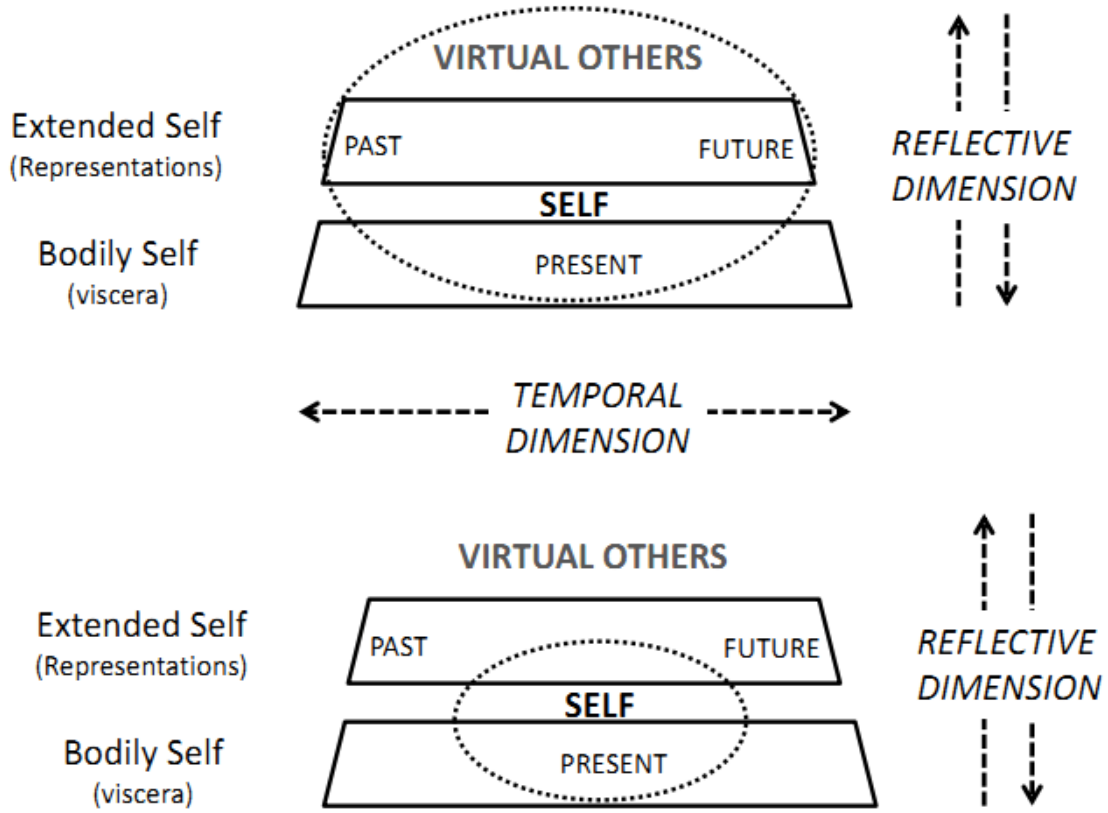

Figure 2

Figure 2: Changes generated by concreteness in *temporal* and *reflective* dimensions of the Self. The figure represents how concreteness contracts the Self (dotted circle line) along these two dimensions. *Temporal* changes modify the Self's capacity to move forwards and backwards in time (time travel), while *Reflective* changes modify the self's capacity to detach from immediate experience, a basic requirement for self-observation and self/other-observation. Visceral experience (Bodily Self), or core experience, is largely preserved despite representational deficits (Extended Self)

The degree to which an individual can be *forced* by the stimulus may vary. At the most extreme end of the continuum, patients may exhibit *utilization behaviours* (Lhermitte, 1983; 1986; Lhermitte, Pillon & Serdaru, 1986), where the visuo-tactile presentation of an object elicits its use or grasping, even though no instructions are given to do so (Shallice et al., 1989). Such patients open doors that are in front of them, get dressed after seeing a shirt in the closet, or lie down if their attention is taken by the bed. In milder cases, individuals may preserve the capacity to inhibit motoric outputs in relation to objects, but their cognition can be "captured" by immediate object/situation claims, as in difficulties *sustaining* a cognitive set, or *shifting* to another (Shallice, Stuss, Picton, Alexander & Gillingham, 2008a; 2008b). For example, when instructed to say words that begin with the letter "s", with the exception of names, a concrete patient may say: "the only things I can think of now are names".

Even though Goldstein primarily described concreteness in behavioural terms, he also acknowledged its relevance for subjective experience (see chapter on values in his Lanutti case; Hanfmann, Rickers-Ovsiankina & Goldstein, 1944). Thus, if a person is constantly *forced* by stimuli, his/hers capacity to detach from immediate experience is compromised, becoming somehow stuck in the experiencing moment. We would like to expand Goldstein's conceptualization by suggesting that concreteness can change the temporal dimension of the Self; usually understood as a capacity to move backwards into past experiences (hindsight), or forward into possible future scenarios (foresight), an ability typically described in the literature as 'time traveling '(Suddendorf & Corballis, 1997; 2007).

2.3.6.2. Presentness and Therapeutic Operations

According to Orlisnky's (2009) model of generic psychotherapy (see Table 2), one dimension of the psychotherapeutic process relates to the so called *therapeutic operations*, where patients present information or complaints to the therapist, usually related to problematic feelings, symptoms or life situations. This is the first step of any psychotherapeutic exchange cycle, where patients bring *into* the session difficulties that they have experienced *outside* the consulting room.

Psychotherapy Dimensions	Definition
Therapeutic contract	Mutual understanding between therapist and patient regarding goals of the collaboration, methods to be used, modality (e.g. individual, couple therapy, etc.) and norms governing participants' behaviour in their role as therapist or patient.
Therapeutic operations	Technical or instrumental aspect of the process. Four cyclic steps can be described: a) Presentation of complaints and information about problematic feelings, symptoms or life situations; b) Construction by the therapist of an "expert" understanding of the real problem underlying patient's complaints; c) Offering a therapeutic intervention by the therapist; d) The intervention evokes co-operation dynamics (collaboration or resistance) that offers further information for a new cycle to start.
Therapeutic bond	Interpersonal aspect of the therapy process. It encompasses task-teamwork and empathic resonance.
Self-relatedness	Intrapersonal aspect of the process, defined as the way in which a person perceives and responds to him while interacting with those around him.
In-Session Impacts	It refers to positive (insight, emotional relief and sense of hope) and negative (confusion, anxiety and discouragement) results attained during sessions
Temporal patterns	Different configurations that the five previously mentioned dimensions acquire as the therapy process change over time as micro-events within therapy sessions and macro-events over the course of treatment

Table 2: Different dimensions of a psychotherapeutic process according to the Model of Generic Psychotherapy (Orlinsky, 2009)

Concrete patients may struggle presenting "material" to the therapist because the emotional noise of a conflictive event may rapidly fade once new events force the organism's attention in another direction. As a result, patients may appear indifferent (or even happy!), with no visible trace of any distress that might become material to initiate a therapeutic cycle. In many cases this untroubled stance may differ from the therapist's internal experience of something conflictive that is left outside, or the relative's distress when describing family issues. The therapist might be tempted then to point out events or situations that deserve to be attended to by the patient, usually with no success. At best, the patient may rationally acknowledge the existence of such events or problems, but without emotional congruence or signs of conflict. In more severe cases, the patient may experience the situation interpreted by the therapist as something completely alien. Consider the case of Mrs I, a patient seen by one of the authors (FV).

> Mrs I is a 54 year old woman who sustained a severe traumatic brain injury in a road traffic accident. She also has a large right frontal infarction, caused by carotid dissection during the same accident. She has a dense left hemiplegia and a wide range of cognitive (mainly executive), emotional and behavioral impairments. In most sessions, Mrs. I experiences low mood, and expresses a great deal of anger and frustration about her circumstances and the difficulties she experiences. These usually relate to the control of her finances, the ongoing adaptation of her home and arrangements for her care and support. She has a strong tendency to blame others for difficulties that arise and she has little insight into her impairments and their consequences. In a few other sessions, Mrs. I appears to be entirely content and happy. During these sessions and at moment of positive affect, it is impossible to engage her in a discussion about her brain injury, or any of the difficulties she commonly complains about. On these occasions, Mrs. I no longer feels that she has a brain injury. She reports that she is content because she will soon be able to move into her new home. Mrs. I is

convinced that the move into the new accommodation will be problem-free and that life in the new house will be perfect.

The case of Mrs. I illustrates how concreteness can compromise the capacity to use events from the past as psychotherapeutic material in the present. In her situation, past negative events (e.g. difficulties in the house conversion or managing finances), that usually are a source of distress, and appear to not exist when she is *captured* [in the present] by circumstances that elicit positive emotions. In other words, during these moments of positive emotion it seems as if she exists entirely within her contentment and could not relate to any other emotional experience. She attributes her previous frustration and anger to a relatively minor factors (e.g. the builders weren't making good progress) and her current happiness to its reversal. When asked about some of the important factors that usually underpin her sense of catastrophe, she dismisses them as no longer important because the house will be finished soon. She refuses to accept that the difficulties that accompany any move into recently built accommodation will occur, and will inevitably be disappointed and enraged when they do.

It is interesting to note that Mrs I's difficulties do not exclusively involve returning to a past scenario to generate conflict, but also to consider possible future events as potential sources of distress. This observation is in agreement with the idea that psychotherapy does not exclusively work through the elaboration of material from past events, but also deals with the emotional impact of "possible" scenarios [this is striking in the treatment of anxiety disorders, for example]. This evaluation of "future consequences" relies on the Self's capacity to project into the future, detaching from present moment and its current emotional valence. Concrete patients, being forced by present stimulus or situations, can show difficulties in the use of future scenarios to activate a manifest conflict.

A similar observation has been made by Freed (2002) when describing that patients with traumatic brain injuries have difficulties using bodily signals of negative value (*signal*

anxiety in psychoanalytic language) to anticipate, and prepare, for negative future events. Evidence from the study of frontal lobe and the envisioning of emotional events in the future (D'Argembau, Xue, Lu, Van der Linden & Bechara, 2008), and data from prospective memory deficits after traumatic brain injury (Shum, Fleming & Neulinger, 2002; Shum, Valentine & Cutmore, 1999; Potvin, Rouleau, Audy, Charbonneau & Giguere, 2011) and frontal lobe lesions (Umeda, Kurosaki, Terasawa, Kato & Miyahara, 2011; Volle, Gonen-Yaacovi, de Lacy Costello, Gilbert & Burguess, 2011), appears to offer additional support to this hypothesis.

2.3.6.3. Concreteness, representational meaning and abstraction

Changes in the *reflexive* dimension of the Self are of great relevance for emotional adjustment and potential to utilize and gain from psychotherapy. This dimension refers to a capacity to detach from direct experience, by verbally reflecting upon our own (or other's) behaviour. We specifically relate this dimension to the generation of *representational* meaning, because it involves the *manipulation* of ideas and thoughts about experience. Here is interesting to note that Goldstein himself emphasized that the process of disintegration towards the concrete does not reduce the *arousal* of ideas and thoughts as such, but affects, and modifies the way of *manipulating* and *operating* with ideas and thoughts (Goldstein & Scherer, 1941). This distinction is extremely important because highlights that concreteness does not compromise primary or core experience, but instead the way we built reflective meaning upon these elements. Consider the case of Mr A to illustrate these matters:

> "Mr A is a young man in his thirties that suffered a severe traumatic brain injury in a car accident. After two years of successful rehabilitation he was finally working in his former position, as executive of an important bank. Even though he still experienced some cognitive difficulties related to effort tolerance and

multitasking, he was able to perform his job without major problems. One day Mr A's wife called his psychotherapist (CS) in a state of crisis. She told him that, for several days, Mr A was acting strangely. He was restless, and complained of headaches. He also kept saying that he did not want to go to the office. Mr A's wife found this quite unusual, because headaches were frequent, and Mr A never stopped working because of this complaint. She commented to the therapist that she tried to talk to him, to find out if there was something bothering him, in order to understand his avoidance to work, but without success. However, she mentioned to the therapist that perhaps Mr A was behaving this way because of the arrival of someone new to the office, which has been placed in a very similar position than Mr A. This made sense to the therapist, and he remembered that Mr A had talked about this new staff member weeks previously. However, Mr A did not looked explicitly conflicted by this event at that moment. Indeed, he made jokes and appeared to look patronizingly towards the new employee. During the next session the therapist tried to explore Mr A's understanding of this crisis and his need to avoid work. Mr A was not able to link the events with any external circumstance or emotional state. He simply stated that he felt ill, and that this did not mean anything beyond that. It took several sessions to progressively link these bodily feelings to the changes that have taken place at work. It took even longer to begin exploring more complex emotions associated to the threatening presence of the new staff member. Mr A realized the new employee was smart, and perhaps more skilful than him after the accident. He expressed irritation to how this new arrival was trying to win over his boss. He also referred feelings of anger at his accident and himself for making things hard at work, thus limiting his chances for a promotion."

Mr A's case is interesting for several reasons. It illustrates a common difficulty of concrete patients to "make sense" out of conflicting situations, by reflecting upon their experience as a coping mechanism (e.g. I feel threatened by this new employee who is smarter than me). It has been described that, in such situations, concrete patients tend to avoid the source of conflict (Krpan, Levine, Stuss & Dawson, 2007; Krpan, Stuss & Anderson, 2011), as a way of down-regulating the emotional disorganization it produces (Riley,

Brennan & Powell, 2004). The case of Mr A is also an example on how concrete patients preserve a capacity to guide behaviour based on *somatic* sensations (e.g. headaches, restlessness), which mobilize the organism in a rather basic approach or withdrawal fashion.

Difficulty reflecting on experience appears to be closely related to abstraction deficits[6]. Several studies have reported that patients with brain injury, and especially those involving the frontal lobes, such as traumatic brain injury, may present with impairments in the ability to structure and synthetize abstract concepts (Elmore & Gorham, 1957; Hagen, 1984; Yang, Fuller, Khodaparast & Krawczyk, 2010) as in proverbs, irony and metaphor (Groher, 1983; Hagen, 1984; Levin, Benton & Grossman, 1982; Towne & Entwisle, 1993). This type of impairment would compromise the patient's capacity to look beyond the most salient elements of experience, in order to generate a more broad understanding of situations that encompass multiple perspectives (see Table 3).

Abstraction capacities appear to have a central role in a psychotherapeutic process. It has been proposed that abstraction is a key component in the generation of new mental structures during psychotherapy (Schneider, 1983, in Mergenthaler, 1996). In addition, the temporal coincidence of abstraction and emotion has been suggested to be related to therapeutic change (Mergenthaler, 1996).

A paradigmatic impairment of abstraction in TBI patients is the difficulty to comprehend metaphor or, in simple words, that the map is not always the territory (which is a metaphor on itself that we are using to synthetize an idea). By using metaphors we compact meaning (Ylvisaker & Feeney, 2000) and transfer information regarding internal states more effectively to others (e.g. I felt like hiding my head in the sand). As the reader can imagine, metaphor has an important role in psychotherapy and hence, metaphoric comprehension

[6] Deficits in the use of abstraction capacities to generate representational meaning can also be caused by excessive amounts of arousal or negative emotion, which dampens higher order cognition. This has been described, for example, by Fonagy et al., (2004) in relation to how anxiety compromises the capacity to think about our own and other people's mental states.

deficits can obstruct the use of metaphor to: a) access to personal memories and feelings (Frank & Frank, 1993); b) facilitate the entry for the therapeutic dyad into the patient's inner world (Angus, Levitt & Hardtke, 1999); c) construct narratives that generate meaning (Sarbin, 1986; Schafer, 1992).

Stimuli	Concrete patient (left prefrontal stroke)	Healthy Subject
Fork /Spoon	You…(gestures bringing something into the mouth) put it in your mouth	Cutlery
Socks/Shoes	For your feet…warmth	Clothing
Yellow/Green	Colours	Colours
Dog/Lion	Bark?	Animals
Coat/Suit	Here…here puts the suit (puts her hands on her chest)…warmth (keeps doing the gesture on her chest) to fasten…wool.	Clothing
Orange/Banana	Fruit.	Fruits
Eye/Ear	On your face…one is for sight and the other one for hearing	Senses
Boat/Car	Transport	Transport
Table/Chair	For…good for eating	Furniture
Work/Play	They are both good for you…you work and you play.	Activities people do
Steam/Fog	Same really. Steam is by man and fog is here (points to the sky)… rain. Steam…you press a button and comes out.	Made of water, misty.
Egg/Seed	They are both the same really… more generation	Beginning of life
Democracy/Monarchy	One has a king and the other one does not	Ways of ruling
Poem/Statue	Statue you see it and the poem is for you to say	Forms of art

Table 3: Comparison of a concrete patient and a matched healthy control on the Similarities task (Wechsler Adult Intelligence Scale III).

2.3.6.4. Concreteness, Insight and emotional awareness

Another aspect of the psychotherapeutic process that can be challenged by concreteness is the capacity to generate insight (Judd, 1999). Insight refers to an *awareness* and understanding of one's own behavioural patterns and motivations (Luborsky, Crist-Cristoph, Mintz, & Auerbach, 1988), and has been considered as a type of "therapeutic realization" that occurs during sessions and leads to change (Orlinsky, 2009). Insight is a necessary capacity in the emotional adjustment of patients with traumatic brain injury (Coetzer, 2007), especially when addressing questions about normality or the meaning of life after brain damage (Prigatano, 1986; 1991; 1999). The case of Mr A can illustrate this point in terms of the difficulties he experienced in understanding how his somatic states (headache and restlessness) were connected to environmental triggers (arrival of the new staff member). To him, nothing else existed besides the immediate distress that forced him to act. His capacity to detach from direct experience, and to reflect upon his behaviour, was somehow shattered -especially when in more challenging states of arousal. Here, it was not an external stimulus that *forced* Mr A' to function in certain direction, but an internal and urgent state of catastrophe (see Salas, 2012).

The case of Mrs I illustrates a slightly different quality of concreteness, which is the influence of emotion on cognitive ability and awareness. Although Mrs. I usually has little awareness of her neuro-behavioural impairments, she can occasionally become more accepting of the idea that her emotions, behavior and cognitive abilities have been altered and impaired by the TBI. However, these moments of insight are both transient and critically dependent upon Mrs. I having a strong sense of being heard and validated within the current session. In these moments, when Mrs. I is calmer and soothed, she seems more able to detach herself from direct experience, and adopt a slightly more reflective, and less defensive, position in relation to her difficulties. She can, for example, give clear descriptions of some

profound changes, such as being unable to inhibit overfamiliar responses with strangers. For further discussion on this point, see section on *building mental schemas and manipulating negative arousal*.

If insight refers to an awareness and understanding of one's own motivations (Luborsky, Crist-Cristoph, Mintz, & Auerbach, 1988), and is closely linked to emotional relief (Orlinsky, 2009), the relationship between emotional experience and abstraction deficits needs to be further understood. Goldstein himself briefly wrote on this topic, suggesting that patient's emotional responses (e.g. dulling of emotions or great excitability) were determined by their capacity to grasp the essentials of a situation, and not to a primary defect in emotional experience per se (Goldstein 1936b). Modern authors have proposed a similar idea regarding patients with frontal lesions, suggesting that disruption of emotion is highly dependent on context. For example, patients may show diminished emotional responses to issues that are *abstract* or not *immediately present*, together with exaggerated emotional responses to immediately present stimuli (Anderson, Barrash, Bechara & Tranel, 2006). Consider the example of Mr R, a patient who suffered a large left frontal and parietal stroke, to illustrate how concreteness may *narrow* emotional life to the present moment (Goldstein, 1936a). As part of an experiment Mr R had to watch short film clips, which were intended to elicit sadness. After each of the clips he had to report how he felt during the films by rating emotional words (e.g. how shaky did you feel while watching the clip?). When asked "how much lonely did you feel?" he replied, perplexed: "why should I feel lonely if I'm here in this room with you?"

Mr R's behaviour and verbal report is interesting. If we consider his facial behaviour during the clip, it is clear that he was able to experience sadness. However, when he was asked to offer some insight about the recently passed emotional experience he struggled to grasp the essence of the question. Although this type of question seems quite straightforward,

answering it would require him to: (a) detach from the present moment and move backwards in time (while watching the clip); (b) activate an "as if" emotional experience of a non-present situation (how did I feel then?); (c) infer abstract information related to the emotional experience (how much?). Mr R's answer reflected difficulties performing these operations, and was instead forced by the salient elements of the present situation (he was, in fact, not alone in the room, at that moment) and the emotional states associated with it (he did not currently feel lonely).

A further understanding of how concreteness compromises emotional awareness, and a key capacity for psychotherapy, can be obtained from research on alexithymia and TBI. Alexithymia, a clinical concept that has been widely related to mental health, has striking similarities with temporal and reflective changes involved by concreteness. For example, alexithymia has been defined as comprising a: (a) difficulty identifying and describing emotions; (b) a concrete communication style; (c) an externally oriented style of thinking; and (d) limited imaginal capacity (Taylor, Bagby & Parker, 1997). In addition, it has also been suggested that alexithymia is linked to a failure elevating emotions from pre-conceptual to conceptual levels of mental organization (Taylor, Bagby & Parker, 1997), a core feature of concrete thinking.

In TBI, there are reports that suggest a high level of incidence of alexithymia (Becerra, 2002; Henry et al., 2006; Wood & Williams, 2007; 2010), which does not appear to be explained by low level emotion perception deficits (McDonald et al., 2011). TBI patients who exhibit alexithymic traits tend to present more physical symptoms (Wood, Williams & Kalyani, 2009), which appear to function as ways of dealing with emotional distress (Williams et al., 2001). This piece of evidence seems to support the idea that concreteness is characterized by a representational [but not somatic] difficulty generating meaning (as

portrayed in Mr A's case). Unfortunately, to our knowledge, no research program has explored the link between concreteness (as described by Goldstein) and alexithymia.

2.3.6.5. Concreteness and the Therapeutic Bond

Changes in the reflective and temporal dimensions of the Self not only influence the use of therapeutic tools (e.g insight), but also the interpersonal space where psychotherapy unfolds, often known as *therapeutic bond* (Orlinsky, 2009). Evidence associating the quality of the therapeutic bond with therapy outcome (Martin, Garske & Davis, 2000; Orlinsky, Ronnestad & Willutzki, 2004) appears to justify the need to comprehend how concreteness may compromise such a relevant dimension.

According to Orlinsky (2009) two aspects of the therapeutic bond can be distinguished. *Task team-work* refers to how much patients and therapists are able to invest in their respective roles, and how they can coordinate positions of control and initiative. *Personal Rapport* denotes the level of empathic resonance that patient and therapist may accomplish, by attuning to one another. It also refers to the emotional climate generated by their emotional responses to one another.

As reviewed above, in relation to therapeutic operations, concreteness can compromise patients' capacity to present problems or difficulties, which are the basic material for therapeutic work. This difficulty can influence the balance of control and initiative inside the dyad, forcing the therapist into a more pro-active or leading attitude. This particular team-work configuration is not negative *per se,* but it can potentially endanger the patient's sense of agency, which is based on the experience of control and proactivity. To consider this is of extreme relevance, especially in view that a main goal for psychotherapy with brain injured patients is helping them regaining some sense of agency in their life, despite the difficulties set by cognitive deficits.

In relation to the second aspect of the therapeutic bond, *personal rapport*, several ideas need to be considered. Firstly, it is necessary to clarify that concreteness does not appear to compromise patients' capacity to react emotionally to others or to establish new emotional bonds, which are basic abilities required to found a therapeutic relationship. Nevertheless, concrete patients may present difficulties functioning in more abstract or representational levels of interpersonal life[7]. For example, concrete patients might struggle to generate emotionally-driven cognitions about the therapist (so called *phantasies* in psychoanalytic language), which are often a source of conflict, as well as material for the process. In other words, patients will react emotionally to the therapist according to past experiences (*e.g. feeling negative during the week and deciding not to call the therapist*), but will be less able to produce the associated affective-mental scripts about his/her relationship with the therapist (e.g. *I will not call him now that I feel troubled; I do not have to bother him. I will wait until our next session. I should sort this out by myself. Yes! He has his own life. Why should he care for me? You have to do it by yourself! Or are you not able to? Before this damn accident you never asked for any help! Why should it be different now?)*. The exploration of such mental scenarios is a key element for psychotherapy, where affective-mental scripts that guide behaviour, progressively become more conscious, thus facilitating its voluntary regulation. However, affective-mental scripts that involve the therapist are especially relevant because they allow an *in situ* exploration, capturing the emotional intensity of *present* experience. The 'archaeological' task of putting together, in the simplest possible way, the different elements that compose affective-mental scripts is a fundamental step in the process of identity reconstruction after brain injury (Salas, 2009; Ylvisaker & Feeney, 2000).

[7] Here the distinction between Bottom-up and Top-down emotion generation described by Ochsner et al (2009) is extremely relevant to critically read evidence suggesting that TBI patients present a decrease in emotional reactivity.

Another core aspect of *personal rapport* is the emotional climate generated by patient-therapist interaction. Here we would like to put forward two basic ideas in line with previous work on countertransference and brain injury (Coetzer, 2006; Lewis, 1999; Klonoff, 2010; Pepping, 1993; Salas, 2008). The first one relates to the therapist's *experience* of the patient's concrete mind, which may trigger diverse feelings and sensations. For example, patient's difficulty generating meaning beyond what is tangible can be felt by the therapist as 'impenetrability', or an inability to access the subtleties of subjective life. Impairments in idea generation can be also experienced by the therapist as 'inertia', a sort of resistance of the mind to move in any direction. These sensations are, at the same time, obstacles in the therapeutic process and 'samples' of the patient's inner experience of his own mind. Even though they can trigger feelings of frustration, they are valuable hints about the nature of the patient's concrete world.

This raises a second observation, which relates to the impact of the concrete mind of the patient, on the abstract mind of the therapist. This is not a phenomenon exclusively associated with concreteness, but one that can be widely observed when working with brain injured patients with different profiles of cognitive deficits, and which can be referred as '*organic* countertransference[8]'. For example, when working with dysexecutive patients, therapists may exhibit problems finding words or organizing thoughts, thus effectively mimicking the patients' deficits. The basic idea here is that, in the same way that therapists (or relatives) influence, and enhance, patient's cognitive performance (Bowen, Yeates & Palmer, 2010; Freed, 2002), patients' neuropsychological deficits can also impact therapists' (and relatives) mental functioning. This is a relational axiom that needs to be kept in mind when addressing the impact of concreteness on personal rapport, especially regarding the capacity to attune to each other. The main point here is that, when working with brain injured

[8] The term *organic* countertransference is used to emphasize that some of the feelings and sensations experienced by the therapists may not be related to the patient's conflicts or personality traits, as is often seen in psychiatric populations, but to the peculiar way in which his 'disorganized mind' works.

patients we do not just attune emotionally but, perhaps more importantly, also neuropsychologically.

A specific difficulty involved in attuning to concrete patients is that therapists try to accomplish such goals using their abstract mind, which by default adds multiple layers of complexity to the simpler experience of concrete patients. Where there is pure vivid and intense experience, the abstract mind steps back, looking for connections, patterns or regularities. Thus the abstract mind is constantly complicating, diluting and unifying experience (Sacks, 1986). This is a challenge, perhaps one of the most important challenges, for any therapist working with concrete patients. Its relevance is manifold. No access to the phenomenological experience of the patient (Prigatano, 1999) is possible without the therapist becoming concrete. Similarly, no guidance can be offered to relatives without teaching them how to relate to the concrete.

2.3.7. Technical modifications in psychotherapeutic work with concrete patients

2.3.7.1. The starting point: preservation of [present] emotional experience

After considering the challenges that concreteness sets to psychotherapy, the reader might wonder: to what purpose, given that these patients seem poor candidates for psychotherapy? This type of reaction by clinicians, which is a mixture of discouragement and disbelief on the suitability of our psychotherapeutic tools, for meaningful and effective work with brain-injured persons, on many levels makes complete sense. Indeed, feelings of frustration are quite common among psychotherapists who work with this population (Judd & Wilson, 2005).

It is the experience of the authors that the only way out from this position of powerlessness is by focusing not exclusively on what is lost, but also on what is preserved. As mentioned above, in concreteness, "the personality, identity and humanity, the being of the hurt creature, is preserved" (Sacks, 1986, emphasis added). An idea such as this might seem counterintuitive, considering the massive transformations in psychological functioning that abstraction loss entails. However, while abstraction might be disrupted, the *sentient* self of concrete patients remains intact. Although this may be hard to picture, especially after our detailed description of concreteness as a deficit, the case of Mr J might be of help to illustrate more practically this point.

Mr J is a 37 year old man who sustained a severe TBI after a traffic accident. As a consequence he experienced deficits in cognitive domains such as sustained attention and divided attention, prospective memory, working memory, set shifting and fluidity. In addition, he also showed awareness difficulties in terms of "indifference" towards these cognitive difficulties when they were not concretely evident. A striking fact about Mr J was that, despite his cognitive deficits, he remained someone capable of experiencing, and expressing, emotional states during interpersonal interactions. Furthermore, his emotional experience had the same features described by Sacks (1986); it was vivid, intense, detailed yet simple. This is observable in the following example, where Mr J was asked to perform a fluidity task:

> *Task 1*: Write five different sentences with the word "rock". 1) There is a seagull over the rock; 2) There is a sea lion sleeping on the rock; 3) I sit on the rock to watch the sea; 4) The rock is very hard; 5) Sea stars are stuck onto the rock. *Task 2:* Write five different sentences with the word "sky". 1) The sky is blue; 2) In the sky there are white clouds; 3) In the sky there are many stars; 4) If you look up into the sky at night you see only stars; 5) The sky is illuminated by stars.

Mr J's answers in the task present some common features of concreteness. They are highly detailed, and mainly based on sensory information. It is also possible to observe some tendency to perseverate in the topic or scene (e.g. a rock in the sea) without shifting to other different uses, or possible contexts, for the target object. Both elements, the high sensory level of detail, and the capture by a single situation or scenario, are examples of what we have described above as 'presentness', or 'being forced' by stimuli. However, it is interesting to note that Mr J's answers do not exclusively portray the impact of abstract deficits in thought processes, but also suggest richness of emotional experience in the present. His answers have, arguably, a genuine sense of depth, as if the detailed depiction of the scene could transmit the experiencing moment with unusual vividness. The apparent paradox between abstract loss and preservation of personality seem here to dissipate. Emotional experience always occurs in present time, and concreteness is presentness. Even though the capacity of the self to project into past or future scenarios adds invaluable layers of complexity to emotional life, emotional experience always unfolds in present time (feel*ing* sad), even if it is triggered by memories from the past (I lost some one I love) or phantasies about the future (I will be left alone).

It is exactly the preservation of these basic emotional capacities what have led authors to suggest that is not only the disorganized mind that needs to be addressed in rehabilitation (e.g. cognitive retraining), but also the wounded 'soul' (Prigatano, 1991). Concrete patients, like all patients with brain injury, need to regain a sense of meaning and identity in order to cope better with the psychosocial problems they face (Prigatano, 1986; 1994). This need has been perhaps underestimated by psychotherapists (Judd & Wilson, 2005), who have tended to assume that cognitive impairments prevent these patients engaging with and benefitting from psychotherapy (Jude & Wilson, 1999; 2005; Prigatano, 2003). This paper proposes a different point of view. We believe that by acknowledging, and comprehensively understanding, these

impairments, it is possible to adapt the way we do psychotherapy (see Prigatano, 1986), and to work more effectively as a result. We also believe that this challenge has an ethical connotation, based on the recognition that concrete patients clearly experience psychological suffering (Goldstein, 1959; 1995 [1965], Hanfmann, Rickers-Ovsiankina & Goldstein, 1944, Salas, 2012), and often attempt to understand their situation, despite deficits of abstraction (Prigatano, 1986) and, at some point, they actively seek for help in order to gain such understanding (Freed, 2002; Salas, 2012). Consider Mr J as an example:

> *Task 3*: write 5 things you do after you get out of bed. 1) Well…I get up and brush my teeth; 2) I get up and go wash my teeth; 3) I get up and feed the dog; 4) I get up and take a shower; 5) I get up…and you will have to help me. *Task 4*: write five things you do before you go to bed. 1) Well I get up and I don't know what to do; 2) I get up and don't know what I would do; 3) I get up and don't know what to do'; 4) I have never fall in despair; 5) I have never…

2.3.7.2. Addressing presentness in psychotherapy: using external reality.

It has been described that presentness implies a contraction in the temporal dimension of the self. Such transformation may challenge the initiation of a therapeutic cycle, which is usually characterized by patients proactively bringing into a session complaints related to problematic life situations. This difficulty has been noted by different authors (Klonoff, 2010; Prigatano, 1986), who have stressed the need to increase the frequency of sessions [to repeat and rehearse information] or include external aids [such as notebooks or lists of potential discussion topics] in order to manage abstraction and memory problems. From Orlinky's model of generic psychotherapy (2009), presentness would compromise the generation of temporal patterns, which are the articulation, over the course of treatment, of the different dimensions of the psychotherapeutic process (explanations of the problems offered by the therapist, insights, emotional relief, etc.).

Perhaps the most important idea to have in mind, when addressing presentness, is that external reality is the most powerful elicitor of emotional reactions. This is a fact that can be easily observed, for example, during the transition from inpatient to outpatient rehabilitation, where contact with the real world violently strikes patients' perceptions of their abilities. Before this encounter, and due to difficulties triggering emotional states based on possible future scenarios, concrete patients are unable to emotionally size up this impact. In other words, it appears that, for concrete patients, problems only exist when they *are* occurring, not much before, and not much after.

The implications of this particular state of mind for psychotherapy are manifold. For example, psychotherapy should carefully seek to identify real life events that generate discrepancies between patient's abilities and expectations and the environment, and use them as *breaches* to access subjective experience. This might seem obvious and simple, but is not. The case of Mr A. is an example of how a real life event (arrival of a new colleague) generated a crisis which is not detected by the therapist or reported by the patient. In this case is thanks to the close collaboration of Mr A's wife that this event is spotted, and then addressed with the patient. The use of these breaches, generated by real life events, requires the flexible management of session frequency, in order to "strike while the iron is hot". A close coordination with relatives and key colleagues at work is vital to catch these events.

A second implication of dealing with presentness is how to artificially generate breaches that allow access to subjective life. In other words, how it may be possible to mimic real life situations in the therapeutic setting, as a means to activate (and then address) conflicts. Two possibilities must be considered here. One is the use of couple or family sessions, alternated with individual sessions. Of special interest here are approaches developed to address "intimacy" problems after brain injury (Bowen, Yeates & Palmer, 2010) and dementia (Balfour, 2011). By including significant others in the sessions, it is possible to

engage the patient in real life interactions, which will arouse emotional reactions more effectively and bring interpersonal conflicts into the session. Another useful method to generate breaches is to move out from the consulting room into the patient's own environment. This approach allows the therapist to experience, with the patient, real life situations (at home, at work) which can be later used as source of therapeutic material. The work of Mark Ylvisaker and Timothy Feeney on identity reconstruction is perhaps the best example of doing psychotherapeutic interventions in ecological contexts (Salas, 2009; Ylvisaker & Feeney, 2000).

2.3.7.3. Making sense: building mental schemes and manipulating negative arousal.

Making sense out of the experience of acquiring a brain injury, and the changes it implies, has been described as one of the main goals of psychotherapy after brain damage (Prigatano, 1986; 1994). However, deficits in abstraction can compromise patients' capacity to reflect upon emotional experience and generate meaningful interpretations of what they are going through. Several technical modifications can be implemented to bypass or compensate such abstraction impairment.

One suggestion is that insight regarding emotional or interpersonal difficulties can be best accomplished through continued repetition and generalization (Prigatano, 1986). In other words, compared to patients without neurological lesions, concrete patients require a prolonged exposure to information in order to fully incorporate it as part of a mental schema or mental category (which can be later used to guide behaviour and decision making). In some cases information may take months, or even years, to sink in -and when it does, is often in the context of an external event that has generated some type of internal disorganization. Somehow, this information appears to have been "rehearsed" inside sessions, and is then meaningfully (emotionally) connected with the event, and a new mental schema is formed.

The subjective experience that accompanies such situations can be a mixture of surprise and fulfilment. Professor F, a patient of one of the authors (CS) arrived in a state of puzzlement to one session, 4 years after the stroke:

> So this is what you meant… this is the problem that we keep talking about…how my head doesn't work sometimes…when it gets messy, disorganized…when I'm too nervous. Well, I was at work and received this call asking to finish this document…and I did not expect that. My head went blank and I did not know what to do, where to start…I was frozen. And there I remembered…and I thought…this is what we have talked about…the executive thing. In a weird way that calmed me down a little bit.

Once a mental schema has been internalized, it needs, ideally, to be generalized to other similar situations, and progressively brought under the same explanatory category. In the case of Professor F, many other life situations (e.g. booking a flight, sorting out bills) that were experienced as mind-disorganizing, were explained under the same basic model: "Dealing with unexpected events makes you feel confused, and when you feel confused your head feels messy and it doesn't obey you". This basic model was progressively enriched through personal metaphors that he spontaneously used to explain his experience. For example, in moments of confusion, when he felt paralyzed, he would describe his mind as being in a state of *inertia*, without any thinking activity.

Generalization also implies that insights need to be shared by significant others who may be present when the patient experiences these events, or are actively sought after for help. It is extremely important that significant others clearly understand the schema, so they can help the patient without diminishing his sense of agency. For example, Professor F spontaneously started calling his wife during moments of confusion, as a means to extract

himself from the mental inertia. He did not need help deciding what was best to do, but could not initiate the process of thinking by himself. That was all he needed.

An additional problem to the generation of mental schemas is concrete patients' difficulty in tolerating psychological conflict -where a somatic response generates an impulse to act which is somehow counter-regulated by higher order processing[9]. By thinking about somatic experience we detach ourselves from a perception-action mode of functioning (doing), by adopting a reflective stance (thinking). Because abstraction is compromised in concrete patients, somatic responses can be experienced without a coherent representational correlate, only as a tendency to act (see previous section on Concreteness, representational meaning and abstraction). In Mr A's case, for example, a negative event from the environment triggered a basic experience of 'threat' which was quickly regulated by concretely avoiding the source of discomfort (work).

The basic question that needs to be answered then is how psychotherapy can be shaped to address difficulties in the generation of representational meaning from somatic experience. Several considerations can be of use here. The first is that such patients' representational capacity (abstraction) might fluctuate according to situational demands, and perceived availability of support. This is not a new idea. In fact, Bowen, Yeates and Palmer (2010) systematized a significant amount of evidence suggesting that performance in cognitive functions of different sort is heavily dependent on context. In addition, there is substantial literature on the impact of negative emotion (or arousal) on executive abilities (Davis & Nolen-Hoeksema, 2000; Demanet, Liefooghe & Verbruggen, 2011; Gasper, 2003; Oei et al., 2011; Schoofs, Preuss & Wolf, 2008; Smallwood, Fitzgerald, Miles & Phillips, 2009) or the capacity to think about mental states (Fonagy, Gergely, Jurist & Target, 2004). A key point is that, as psychotherapists, it is possible to influence patients' representational abilities by

[9] See Salas & Turnbull (2010) for a discussion on conflict, defence mechanisms and brain injury.

manipulating their level of negative arousal. This is of special relevance when working with couples/families, or when the patient-therapist relationship is the focus of the conflict (e.g. alliance rupture).

Another source of negative arousal, that requires modulation, is the patient's struggle using his/her mind during sessions. Often, concrete patients can experience difficulties generating ideas, finding words or understanding therapists' interventions. The experience of these deficits can generate vicious cycles where negative emotion impairs cognitive function further. The therapist's stance toward these difficulties is crucial. He has to be available enough to act as an auxiliary ego when, for example, the patient is unable to find a word. Nevertheless, at the same time he has to offer the patient enough space to use, and reclaim, his own mind, tolerating the tendency to "fill in the gaps" that is caused by mutual frustration. A patient of one of the authors, Professor F, commented on this *therapeutic attitude* when writing about his brain injury:

> The weekly work with my psychotherapist has been very important: supporting without *replacing*, confirming without *imposing*, helping to understand without rushing into conclusions, week by week I'm learning a new way of relating.

A first step in the generation of meaning, or the building of mental schemas, is helping patients to learn that somatic responses refer to something important, disregard the exact meaning they may have. We have denominated this process as "flagging", because it is not about understanding what is going on, but simply about marking the somatic response to events as something relevant, so it can be brought in for later discussion. In the case of Mr A. he started paying more attention to headaches, and flagging whenever they occurred. At first he was not able to understand by himself their meaning, but collaborative exploration allowed the generation of a set of "typical causes" which he could later test as possible explanations.

2.3.7.4.Making sense: using symbols to generate and compact meaning.

We have described above how concrete patients struggle abstracting the essential from different elements (see table 3). A similar problem may occur during the process of synthetizing insights and awareness of changes after the injury. Although it may seem paradoxical, considering the research on metaphor comprehension impairment and TBI (e.g. Towne & Entwisle, 1993), several authors have suggested that the use of metaphor or symbols can help bypass these abstraction problems (Prigatano, 1986; 1989; 1999b; Salas, 2009; Ylvisaker & Feeney, 2000; Ylvisaker, Mcpherson, Kayes & Pellet, 2008). A possible explanation for this apparent contradiction is that metaphors used in experimental settings, compared to those used in psychotherapeutic interventions, are not chosen by patients themselves, and therefore, they are not personally compelling or emotionally meaningful (see Teasdale & Barnard, 1993 for an approach to metaphor based on affect and action tendencies). In addition, experimental designs actively avoid the overexposure of participants to stimuli, while in psychotherapeutic settings with concrete patients repetition is a key element in the generation of new mental schemas.

Although different authors emphasize diverse aspects of metaphor in their use with concrete patients, they tend to agree that metaphor facilitates the understanding of complex ideas (Prigatano, 1986; Ylvisaker, Mcpherson, Kayes & Pellet, 2008), integrates cognition and affect (Prigatano, 1989), helps articulating identity issues (Prigatano, 1999b; Ylvisaker & Feeney, 2000) and can be presented as tangible graphic material (e.g. drawings or identity maps).

For Prigatano (1986), symbols address the core issue with few words and powerful imagery, helping patients with information processing deficits to grasp abstract ideas. He

suggests that, paradoxically, metaphors and symbols are often necessary to help patients understand common experiences after acquiring a brain injury, such as the loss of normality (Prigatano, 1989). Even more importantly, symbols (e.g. the journey of the hero; birth/death) offer guidance in coping with such transformations (Prigatano, 1989), facilitating the process of rebuilding a meaningful life (Pepping & Prigatano, 2003). Thus him the use of symbols related to basic human activities, such as work, love and play, can help patients to integrate thoughts and feelings, re-establishing a new sense of self (Prigatano, 1989).

Mark Ylvisaker and Timothy Feeney approach the use of metaphors from a slightly different point of view. Metaphors are, for these authors, effective instruments compacting information and generating positive versions of the self that can be used to create meaning, motivate and regulate behaviour (Ylvisaker & Feeney, 2000; Ylvisaker, McPherson, Kayes & Pellet, 2008). Through the use of metaphor, for example by choosing a personal heroic figure (e.g. a sportsman), different values (e.g. strength of character), goals (e.g. be respected), feelings (e.g. being in control) and action strategies (e.g. careful preparation), can be emotionally unified into an identity schema, making it more accessible to memory with only one unit of thought (Ylvisaker & Feeney, 2000). Usually the construction of these schemas is supported by external graphic organizers, which allow concrete patients to elaborate, organize, remember and express thoughts more effectively.

2.3.7.5.Becoming concrete: concrete patients need concrete therapists

When it comes to addressing the therapeutic challenges posed by concreteness, the modifications suggested in the section above can be assumed to be useful. For example, the generation of affective/mental scripts of the patient-therapist relationship can be facilitated by the use of metaphors and graphic organizers, as well as the basic principles of repetition and generalization. We will not develop this point further in order to focus on another often

unattended problem, which is how to access these patients' inner experience from an abstract mind point of view.

We believe that a first step in this task is to familiarize with a non-conceptual form of experience. Mark Ylvisaker, for example, described this as "talking from the guts" (Salas, 2009), suggesting that concreteness is related to a rather visceral level of processing information, also called implicational meaning (Teasdale, 1997, in Ylvisaker & Feeney, 2000). In practical terms this would require therapists to push themselves into a bottom-up therapeutic stance, where staying in the present moment, and attending to somatic and affective responses are the main points of orientation. This approach is not new. In fact, coming from a psychoanalytic background, Wilfred Bion (1963; 1967) suggested that the basic clinical attitude was to confront every session 'without memory, desire, or understanding'; because they might cloud the therapist's capacity to grasp what *is* happening during a session. Interestingly, a similar idea has been put forward by Mindfulness based therapies, which attempt to develop the capacity for 'sustained moment-to-moment awareness' (Siegel, Germer & Olendski, 2009).

Becoming concrete does not imply that the therapist should remain concrete, or completely abandon an abstract stance. As we have suggested before, that is impossible, for we are constantly distancing ourselves from experience in order to make sense of it. Becoming familiar with the world of the concrete is simply an initial step which offers first hand material with which to comprehend the patient's suffering, and the experience that close ones might also have of his/her new way of relating to them. An ideal therapist should be able to immerse himself in a concrete mode of functioning, so he can later emerge from it to reflect upon such experience. This constant shifting between the two modes of functioning is perhaps the heart of the therapeutic process.

We would like to offer a final observation on the emotional impact of treating concrete patients. Therapists often arrive at working with brain injured patients after being trained to work with normal or psychiatric populations. One of the consequences of such 'career path' is that therapist frequently spend quite a long time adjusting their theoretical and technical tools. This period of fine-tuning often involves a grieving process too, where the therapist mourns for the patient he will not have, that one he was trained for, the one who will get cured by gaining insight about his problems and history, the abstract patient. In a way, part of the therapist's identity is endangered during such transition: what am I? Am I *just* a neuropsychologist? Am I *just* a counsellor? What can I do if my tools don't work? To address these emotional difficulties is perhaps one of the most important tasks when working with concrete patients.

2.3.8. Closing remarks

Concreteness is a frequently observed phenomenon in rehabilitation settings, causing important obstacles to teams in helping patients to resume a productive and meaningful life. Concrete patients, as any other human being, experience emotional difficulties adapting to the drastic changes that a brain injury inevitably results. Overcoming these difficulties is especially demanding for these patients, because their "thinking" ability, a core capacity for psychotherapy, is compromised. In this paper we have tried to described in detail how concreteness transform different dimensions of the Self and how these changes may challenge the use of psychotherapeutic tools. We have also described technical modifications to address these changes (see Table 4 for a summary).

The observation of concrete patients in therapeutic settings is also important for theoretical motives. It sheds light into several neuro-philosophical problems, such as the neural basis of reason and emotion, time and consciousness and the construction of meaning.

After considering the evidence that these "experiments of nature" present, when the veil of abstraction (and its illusions) is removed, our idea of what is *fundamentally* human seem to change. We are organisms closely connected to our environment, and our behaviour, is the consequence of this incessant flux. Consciousness echoes this flux, always unfolding in an ever changing now. And emotion, visceral emotion, is the core that generates meaning, a felt meaning. This is the 'world of the simple' described by Oliver Sacks, or the 'shrunken environment' portrayed by Kurt Goldstein. Several authors have addressed these same questions from other points of view. We hope this article will contribute by motivating psychotherapists and researchers to further explore and understand the world of the concrete.

Concreteness Features	***Impact on Psychotherapeutic Operations and Tools***	***Theoretical and technical modifications***
Changes in the TEMPORAL dimension of the Self	*Initiation of a Therapeutic Cycle:* difficulty using events from the past and possible future scenarios, as session material.	Use real life events as 'breaches' to access subjective experience of conflict
		Close coordination with relatives and work colleagues to spot events of emotional significance
		Flexible management of sessions' frequency to capture events of emotional significance
		Alternate individual sessions with couple and family sessions to mimic real life situations
		Incorporate field trips to explore, with the patient, real life situations
	Therapeutic Bond, Task team-work: difficulty coordinating positions of control and initiative	Enhancing patient's sense of agency by calibrating when and how much help is needed
Changes in the REFLECTIVE dimension of the Self	*Positive In-session impact, Insight:* difficulty generating awareness of motivations and making sense of emotional responses	Teaching patient to 'flag' somatic responses to events
		Collaborative generation of a set of 'typical' causes, or events, that trigger somatic responses
		Prolonged exposure to information or repetition
		Generalization of insight to other situations and use of significant others to reinforce new mental schemas
		Use of personally compelling metaphors
		Use of external graphic organizers (e.g. identity maps)
	Negative In-session impact: difficulty tolerating negative arousal when exploring	Monitoring and manipulating patient's level of negative arousal

psychological conflict	
	Facilitate cognitive processing without compromising patient's sense of agency
Therapeutic Bond, Personal Rapport: difficulty generating emotionally-driven cognitions about the patient-therapist relationship	Use of graphic organizers, metaphors and repetition/generalization principles
Therapeutic Bond, Personal Rapport: difficulty attuning to a concrete mode of experience	Adopting a Bottom-up therapeutic stance: staying in the present moment and attending to somatic and affective responses
	Flexible shifting between concrete and abstract modes of functioning
	Elaborate mourning feelings associated to working with non-abstract patients.

Table 4: Summary of technical modifications when working with concrete patients.

Chapter 3: Methodological Articles

3.1. Inside-out: comparing internally generated and externally generated basic emotions[10].

3.1.1. Abstract

A considerable number of mood induction (MI) procedures have been developed to elicit emotion in normal and clinical populations. Although external procedures (e.g. film clips, pictures) are widely used, a number of experiments elicit emotion by using self-generated procedures (e.g. recalling an emotional personal episode). However, no study has directly compared the effectiveness of two types of internal versus external MI across multiple discrete emotions. In the present experiment, 40 undergraduate students watched film clips (*external* procedure) and recalled personal events (*internal* procedure) inducing four basic emotions (fear, anger, joy, sadness) and completing later a self-report questionnaire. Remarkably, both internal and external procedures elicited target emotions *selectively,* compared to non-target emotions. When contrasting the *intensity* of target emotions, both techniques showed no significant differences, with the exception of Joy, which was more intensely elicited by the internal procedure. Importantly, when considering the overall level of intensity, it was always greater in the internal procedure, for *each* stimulus. A more detailed investigation of the data suggests that recalling personal events (a type of internal procedure) generates more negative and mixed blends of emotions, which might account for the overall higher intensity of the internal mood induction.

[10] Salas, C. E., Radovic, D. & Turnbull, O. H. (2011). Inside-out: Comparing internally generated and externally generated basic emotions. *Emotion.* Advance online publication. doi: 10.1037/a0025811

3.1.2. Introduction

The study of emotion is today a mature field, with its own standardized measures, induction procedures, data analytic techniques, sub-disciplines and core theoretical debates (Coan & Allen, 2007). A number of mood induction (MI) procedures have been devised to elicit and manipulate emotional response, using a diverse range of techniques such as self-referential statements (Kenealy, 1986; Velten, 1968), posing facial expression (Ekman, 2007; Laird, Wagener, Halal & Szegda, 1982), autobiographical recall (Averill, 1982; Barlett, Burleson & Santrock, 1982; Brewer, Doughtie & Lubin, 1980, Camras & Allison, 1989; Izard, 1972, 1977; Schaefer & Philippot, 2005; Scherer, Walbott & Summerfiel, 1986; Shaver, Schwartz, Kirson & O'Connor, 1987; Turnbull, Evans & Owen, 2004), imagery (Wright & Mischel, 1982), film clips (Gross & Levenson, 1995; Hagemann et al., 1999; Hewig, Hagemann, Seifert, Naumann & Bartussek, 2005; Philippot, 1993; Rottenberg, Ray & Gross, 2007; Schaefer, Nils, Sanchez & Philippot, 2010), music (Eich, Ng, Macaulay, Percy & Grebneva, 2007; Vastfjall, 2000), positive and negative performance feedback (Farmer et al., 2006; Isen, Clark, Shalker & Karp, 1978), social interactions (Roberts, Tsai & Coan, 2007) or a combination of the above mentioned (Berna et al., 2010; Furman, Hamilton, Joorman & Gotlib, 2010; Harkness, Jacobson, Duong & Sabbagh, 2010).

The development and validation of these MI procedures is of special relevance since different scientific related fields rely on them, for instance, to elicit and measure emotion in clinical populations (Gemar, Segal, Sagrati & Kennedy, 2001; Hunt & Forand, 2005; Scher, Ingram & Segal, 2005; Van der Does, 2002), or to map the neurological basis of affective processes (Damasio et al., 2000; Kimbrell et al., 1999; Najib, Lorberbaum, Kose, Bohning & Mark, 2004; Posse et al., 2003). Until now emotional pictures (Bradley & Lang, 2007; Lang, Bradley & Cutbert, 2008) and film clips (Gross & Levenson, 1995; Hewig et al., 2005;

Rottenberg et al., 2007) have been the best standardized and the most widely used tools. However, when comparing the *effectiveness* of different MI procedures in eliciting target emotions, the evidence is rather scarce, and at least 15 years old. In one of these reports, Martin (1990) observed that the use of self-referential techniques (the so called Velten procedure), and the facial expression technique, showed on average 50% of success eliciting a target emotion. In contrast, imagination, music, films and autobiographical recall reached levels of 75%. According to the Westerman, Spies, Stahl and Hesse (1996) meta-analysis of MI procedures, the most effective technique for inducing elated and depressed mood is films/histories, paired with explicit instructions to the participants during the induction process.

Although the available evidence seems to suggest that films are a robust MI procedure (Rottenberg et al., 2007, for a review), several studies have moved towards the more internally generated approach of autobiographical recall, as a means of eliciting emotions (Damasio et al., 2000; Harrison et al., 2008; Phan, Wager, Taylor & Liberzon, 2002; Turnbull et al., 2004). The rationale behind this choice appears to lie in the assumption that internally-generated approaches have more ecological validity, since it uses personal events with individual meaning. Such a claim seems supported by evidence that relates higher emotional intensity to particular types of autobiographical memory (Philippot, Schaefer & Herbette, 2003), and also from literature suggesting that some discrete emotions (e.g anger, fear) may not be as intensely elicited when using films (externally generated) compared to self-generated stimuli (Harmon-Jones, Amodio & Zinner, 2007; Levenson, 2007; Lobbestael, Arntz & Wiers, 2008; Rottenberg, Ray & Gross, 2007). A distinction of this sort, between internal and external MI procedures, has been proposed before by Gerrards-Hesse, Spies and Hesse (1994), who suggest that MI procedures such as autobiographical recall are based on the "free mental" generation of emotional states, while in film clips the emotion inducing

material is "presented" from the outside. It is clear that a substantial limitation of this literature is the absence of empirical findings that allow us to directly compare the internal-external dimension.

One of the main problems in evaluating the relative merits of induction procedures is the shortage of articles that use or compare more than one technique (for exceptions see Chartier & Ranieri, 1989; Clark, 1983; Isen & Gorgoglione, 1983; Lobbestael, Arntz & Wiers, 2008; Martin, 1990). Although not directly comparing different MI procedures in the same experiment, the available meta-analyses have suggested that film clips (with no instruction) and autobiographical recall show no difference in their efficacy to induce *negative* emotions, while films clips are more effective generating *positive* emotional states (Gerrards-Hesse et al., 1994; Westerman et al., 1996). Bearing in mind the well-established position of film clips, but the increased usage of self-generated procedures, and with the assumption that internally-sourced stimuli have an advantage over externally-sourced stimuli, it seems timely to compare these two internal (autobiographical recall) and external (film clips) procedures in terms of their efficacy in eliciting specific emotions.

Another relevant issue when comparing the efficacy of MI procedures in eliciting emotions is the extent to which experimental designs include more than one emotion. Twenty years ago, Martin (1990) reported that most of the MI procedures have not investigated systematically the full range of basic emotions. For instance, there were no studies inducing fear or anger through autobiographical recall, or experiments that used film clips to elicit anger. In the last decade this panorama has changed, and several studies have progressively targeted a wider range of basic emotions using autobiographical recall (Damasio et al., 2000; Ray, Gross & Wilhem, 2008; Rivers, Brackett, Katulak & Salovey, 2007; Wenzel, Jackson, Brendle & Pinna, 2003), film clips (Rottenberg et al., 2007, for a review) and mixed procedures (Mayer, Allen & Beauregard, 1995). However, we are not aware of studies that

have directly compared external (e.g. films) and internal (e.g. autobiographical recall) procedures across multiple discrete emotions. The need to assess multiple basic emotions simultaneously is extremely relevant to the issue of *selectivity*. In other words, when we intend to elicit a specific emotion (e.g. fear), do we only generate fear, or are other emotions also generated (e.g. anger or sadness)? An approach of this sort seems vital, given that several authors have suggested that MI procedures may elicit more than one emotional state (Izard, 1972; Polivy, 1981; Westerman et al., 1996), and that basic emotions (e.g. sad/anger) may correlate (Zelinski & Larsen, 2000).

In sum, there are a number of questions in the emotion elicitation literature that are of considerable methodological interest, and remain unanswered. Our study had two main goals: to compare the *selectivity* of two types of internal and external MI procedures eliciting target emotions (e.g. does 'fear' elicit only fear?) and to compare the *intensity* levels of emotional experience between these two internal and external MI procedures (e.g. is internally-generated fear more intense?).

3.1.3. Method

3.13.1. Participants

The participants for this study were 33 female and 7 male psychology undergraduates, who received course credits for their participation. The subjects were between 18 to 28 years old (M = 19.7, SD = 1.8). Individuals who were not English native speakers were excluded, to avoid the confounding effect of anxiety and emotional distress caused by language demands of the Affective Story Recall task.

Although this sample was imbalanced on male/female proportion, we decided not to exclude males in order to avoid a significant decrease in the power of the study. To explore the effect of gender imbalance in the sample, a similar analysis to the one that is described in

the result section was performed, excluding the 7 male participants (n=33). The results of this analysis showed a similar trend to the one observed considering both sex groups (n=40).

3.1.3.2. Stimuli

3.1.3.2.1. Film Stimuli

Four emotional clips, and two neutral clips were used, each of them matching one of the four basic emotions described by Panksepp (1998). These clips have been previously validated regarding their capacity to elicit specific and discrete emotions (Gross & Levenson, 1995; Hewig et al., 2005; Rottenberg, Ray & Gross, 2007). The neutral clips, *Alaska's Wild Denali* (Hardesty, 1997), showed Alaska's landscapes and wild life, and both were 150s in length. The Joy film, *Bill Cosby Himself* (Cosby, 1983), showed a standup comedy performance by Bill Cosby (121s). The Sadness clip, *The Champ* (Zeffirelli, 1979), showed a young boy facing the sudden death of his father after a boxing match (171s). The Fear clip, *The Shining* (Kubrick, 1980), showed a young boy alone in the hall of a Hotel before he opens the door of a room, whilst anxiety-inducing music plays in the background (122s). Finally, the anger clip, *My Bodyguard* (Bill, 1980), showed a young man who is bullied and humiliated (246s).

3.1.3.2.2. Affective Story Recall Stimuli

In the Affective Story Recall task (ASR), developed by Turnbull et al. (2004), participants were asked to recall a series of personal experiences, from events in their life, related to specific emotions. In this study we used the same discrete emotions as in the film clips (anger, joy, sadness and fear). Two neutral recall conditions (going shopping and fixing a meal) were added at the beginning and end of the task, to calculate initial and final baselines. Each recall was prompted with the following phrase: *"Try to recall an event in your life that has caused you to feel… (e.g. anger). Try to be very detailed about the way you feel"*. Each

participant had a maximum of 3 minutes to describe the event, although they could extend this if they felt they have not finished yet the story after the three minutes, or even finish before if they felt they have nothing else to say. A minimum of time was not set in order to avoid that task instructions could activate negative emotions by adding pressure and anxiety to the recollection[11].

3.1.3.3. Measures

To generate several emotional measures after each clip and story, we designed a shortened version of the PANAS-X (Watson & Clark, 1994), the PANAS-XS. The PANAS-X is a 60 items self-report that assesses specific emotional states. It has a general dimension of positive and negative affect, but also includes specific scales for discrete emotions. Participants are asked to rate each of the 60 emotional words (e.g. cheerful, hostile, shaky) indicating the extent to which they felt each emotion according to a 5-point scale (very slightly or not at all, a little, moderately, quite a bit and extremely). The adaptation of the PANAS-X to the PANAS-XS is justified by the need for a shorter version to use when emotional states need to be assessed in the same experiment several times, as is the case when reporting the subjective states of neutral conditions and four basic emotions during the film clips and ASR. The adaptation was produced by taking the elicitor of each basic emotion addressed in this study from the PANAS-X, and asking 20 male and 20 female university students to rate them according to how representative they felt each word was of each discrete emotion. The two words with the highest, and the word with the lowest overall score, were selected. 12 items

[11] The average time in seconds for each emotion recollection was: Joy (M = 89.90, SD = 38.88), Anger (M = 105.42, SD = 45.13), Fear (M = 105.11, SD = 44.86), Sadness (M = 111.989.90, SD = 54.70). Only Joy was significantly different from the three negative emotions, in the average leght participants used to recall the story ($F_{(3, 37)}$ = 7.326. p < .001). The range of duration was: Joy 40s-177s; Anger 32s-187s; Fear 38s-198s; Sadness 40s-236s.

finally compose the PANAS-XS: happy, joyful, and energetic (joy); sad, downhearted and alone (sadness); angry, hostile and disgusted (anger); scared, afraid and shaky (fear).

3.1.3.4. Procedure

Participants individually attended a 90-minute assessment session. They were told that they would be asked to recall personal events in relation to certain emotions. In addition they would have to watch clips from movies in the screen of a computer and report how they made them feel. Finally they were also informed that they would have to complete two questionnaires between tasks [see appendix 1].

ASR and film clip tasks were counterbalanced in their order of appearance across participants to avoid possible order effects. In the ASR task participants sat in front of the experimenter. Next to the experimenter there was a computer that displayed a digital chronometer, so they could check the time left for each story (3 min max.). Before beginning the task the researcher explained the ASR task in some detail, and asked for a neutral episode, as a way of familiarizing the participant with the task, and obtaining a baseline measure. The order of the emotional clips was counterbalanced to control for possible order effects. Thus, four conditions were generated, considering that we were interested in observing the three possible ways in which negative emotions might interact, and the three possible ways in which positive and negative emotions might also interact (N1-F-S-J-A-N2; N1-S-A-F-J-N2; N1-A-J-S-F-N2; N1-J-F-A-S-N2). Participants were randomly allocated to each of them. After the first neutral episode, participants were introduced to how to fill in the PANAS-XS, asking them specifically to rate the highest amount of each emotional state (PANAS-XS words) they felt while recalling the personal event. They were also asked to register any other emotion they could have felt, but which was not described in the PANAS-XS. The same

procedure was repeated after each story recall. When the ASR was finished, participants took five minutes to answer two questionnaires.

Before starting the Film Clips task, participants were told that they would watch clips from movies, and that they would be asked to report how each clip made them feel. The order of exposure to the film clips was counterbalanced using the same four conditions as in the ASR. Instructions were displayed on the screen, asking to fix their gaze on the screen and watch the clips carefully. After each clip an instruction asked them to answer the PANAS-XS. This procedure was repeated after each video. Once each of the PANAS-XS was completed, an instruction asked the participants to press a key to continue. A debriefing procedure followed the test.

3.1.3.5. Score Data Analysis

The average score of each PANAS-XS subscale (Fear, Sadness, Joy and Anger) was calculated, this for each eliciting stimulus (Neutral 1, Fear, Sadness, Joy, Anger and Neutral 2), on both tasks (ASR and FC). To test the *Selectivity hypothesis* these averaged scores were compared, using a repeated measures design. In the cases where the sphericity assumption was not respected, Greenhouse-Geisser correction was used (for Greenhouse-Geisser's Epsilon between 0.9 and 0.7), or multivariate Pillai's Trace statistic was reported (for Greenhouse-Geisser's Epsilon less than 0.7). The differences between the target emotion score and the other emotions were calculated using a simple contrast, with the target emotion as reference. The *Intensity hypothesis* was assessed for each stimulus using a paired sample t-test, first for the *average* of emotional intensity, and then for the *target* emotion intensity for both instruments. To explore further this hypothesis the average scores of the positive and negative non-target emotions were also compared using paired-sample t-test. In addition,

association measures between target and non-target emotions were calculated, using Pearson's bivariate correlations.

3.1.4. Results

The present study tested two hypotheses: a) The internal and external elicitation procedures will elicit the target emotion with greater intensity than any other basic emotion (*Selectivity*) b) The emotions elicited by the self-generated procedure will show greater intensity than the ones generated by the external procedure (*Intensity*).

3.1.4.1.Selectivity

In relation to the *Selectivity* hypothesis, both MI procedures elicited each of the four target emotions with higher levels of intensity than the other three emotions, demonstrating that internal (Affective Story Recall) and external (Film Clips) procedures are equally effective inducing an emotion selectively. The PANAS-XS subscale's average intensity, for each of the four eliciting emotions, in both tasks, is illustrated in Figure 1.

As Figure 1 shows, differences in the average intensity between the target emotion and the other emotions, across the four emotional stimulus, are observed in the internal (Fear: v= .67, $f_{(3, 37)}$= 25.03, p< .01; Sadness: $f_{(2.61, 101.86)}$= 88.44, p< .01; Joy: v= .92, $f_{(3, 37)}$= 148.91, p< .01; and Anger: $f_{(3, 99.13)}$= 43.79, p< .01) , and external (Fear: v= .76, $f_{(3, 37)}$= 39.45, p< .01; Sadness: v= .89, $f_{(3, 37)}$= 95.85, p< .01; Joy: v= .85, $f_{(3, 37)}$= 67.02, p< .01; and Anger: $f_{(2.64, 102.88)}$= 100.20, p< .01) procedure. Thus, the comparison between the target emotion and the other three emotions was statistically significant for *each* stimulus, and for both tasks, showing higher levels of intensity for the target emotion in each case (see Table 1), with large effect sizes.

3.1.4.2.Intensity

In relation to the Intensity hypothesis data was analyzed in three steps. Firstly, the intensity of the target emotion was compared between both MI procedures (e.g. when eliciting fear, are there differences in the score of fear between the internal and external procedures?). Using this approach, only Joy appeared significantly higher in the story recall than in the film clips. When eliciting sadness, anger or fear, it appears to make no difference whether we use internal or external MI procedures (see Table 2).

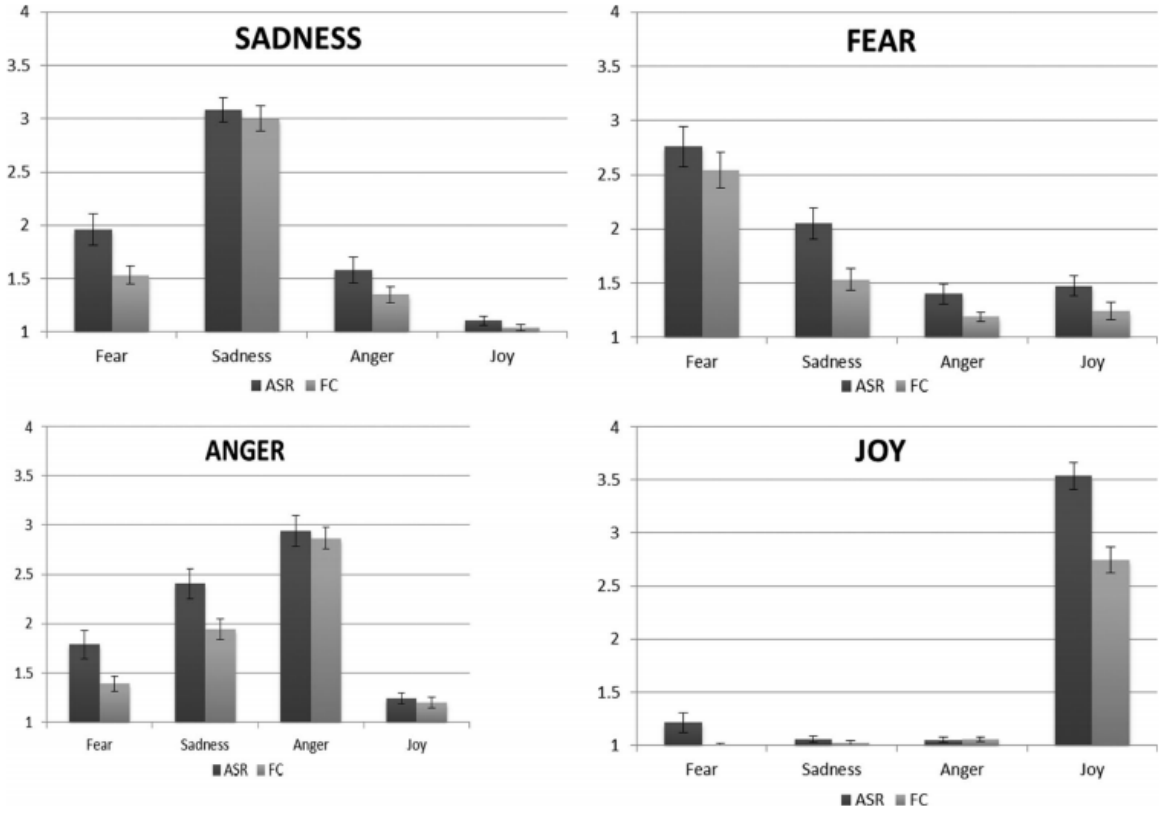

Figure 1: PANAS-XS scores across four basic emotions, for internal (Affective Story Recall) and external (Film Clips) stimuli. In each case the internal and external procedure selectively induce the target emotion.

Target emotion	Non target emotion	Affective Story recall				Film Clips			
		Paired Dif	*F*	*p*	*d*	Paired Dif	*F*	*p*	*d*
Fear	Joy	1.28	28.83	< .01	1.84	1.30	48.07	< .01	2.68
	Anger	1.36	75.14	< .01	1.61	1.35	99.99	< .01	2.68
	Sadness	0.71	33.08	< .01	0.74	1.01	71.03	< .01	1.40
Anger	Joy	1.69	107.7	< .01	2.68	1.67	216.67	< .01	3.71
	Fear	1.15	42.91	< .01	1.41	1.48	157.64	< .01	2.56
	Sadness	0.54	12.36	< .01	0.60	0.93	94.61	< .01	1.45
Joy	Anger	2.48	451.2	< .01	4.12	1.68	189.54	< .01	3.52
	Fear	2.32	221.7	< .01	3.53	1.73	204.37	< .01	3.61
	Sadness	2.48	364.8	< .01	4.15	1.72	187.13	< .01	3.56
Sadness	Joy	1.98	262.31	< .01	3.89	1.96	248.85	< .01	3.68
	Fear	1.13	90.03	< .01	1.48	1.47	216.34	< .01	2.27
	Anger	1.50	202.5	< .01	1.73	1.65	277.19	< .01	2.58

Table 1: Difference between Target emotion and other emotions in internal (Affective Story Recall), and external (Film Clips), eliciting procedures. Internal and external procedures elicit selectively the target emotion.

	Affective Story Recall		Film Clips		Paired Differences			
Target	Mean	(SD)	Mean	(SD)	ASR-FC	*t*	*p*	*D*
Fear in FEAR	2.76	1.18	2.54	1,02	0.21	0.98	0.33	0.20
Sadness in SAD	3.08	0.73	3.00	0.74	0.08	0.56	0.58	0.11
Joy in JOY	3.53	0.78	2.74	0.77	0.79*	6.51	< 0.01	1.02
Anger in ANGER	2.94	0.99	2.90	0.71	0.04	0.30	0.77	0.05

Table 2: Target emotion intensity comparison between internal (Affective Story Recall), and external (Film Clips), eliciting procedures. Only Joy is significantly higher on the internally generated procedure.

As a second step, a *general intensity level* was calculated averaging the PANAS-XS score of all emotions (fear, sadness, anger, joy), and then comparing them between procedures (e.g. when eliciting fear, is the average intensity of the 4 basic emotions different between internal and external procedures?). As a result we obtained that the internal procedure average emotional intensity was significantly higher than the external procedure (see Table 3) in each of the four stimuli.

	Affective Story Recall		**Film Clips**		**Paired Differences**			
Stimuli	Mean	(SD)	Mean	(SD)	ASR-FC	*t*	*p*	*d*
Fear	1.92	0.58	1.63	0.44	0.29*	3.10	.004	0.57
Sadness	1.93	0.53	1.73	0.36	0.20*	2.21	.033	0.45
Joy	1.72	0.26	1.46	0.20	0.26*	7.07	< .001	1.11
Anger	2.09	0.58	1.85	0.40	0.23*	2.75	.009	0.47

Table 3: Average emotional intensity comparison between internal (Affective Story Recall) and external (Film Clips) mood induction procedures. When all PANAS-XS scores are averaged in each stimulus, the internal procedure (Affective Story Recall) is significantly higher.

Because the target emotion did not differ significantly between internal and external procedures (with the exception of joy), but the general intensity level was higher in the internal one, differences in the associated emotions between procedures were explored. Thus, two scores were generated for each stimulus, one for the non-target positive emotion and another for the non-target negative emotions (e.g. when eliciting fear, is the average intensity of the non-target negative emotions [sadness and anger], or positive emotion [joy], higher in the internal procedure?). As a result, when eliciting *any* of the negative emotions (e.g. fear), the average score of the non-target negative emotions (e.g. anger & sadness) was always significantly higher in the internal procedure (Fear $t_{(39)}$=3.59, p< .001; Sadness $t_{(39)}$=2.65, p= .01; Anger $t_{(37)}$=3.15, p< .001) with a medium effect size (Fear d= 0.65; Sadness d= 0.53; Anger d= 0.62). As for the positive non-target emotion (joy), only when eliciting fear it was significantly higher ($t_{(39)}$=3.30, p< .001) in the internal procedure, with a small effect size (d= 0.42). An unexpected finding was that when eliciting a positive emotion (joy), the negative

non-target emotions (anger, sadness, fear) were also significantly higher ($t_{(39)}$= 2.30, p= .03) with a medium effect size (d= 0.47). Considering these findings, it is not possible to conclude that the higher general level of intensity generated by the internal procedure is explained exclusively by higher scores on the valence-like (positive or negative) non-target emotions.

Finally, to explore even further the relationship between target and non-target emotions, associations between the target and non-target emotions were calculated for each stimulus, in both tasks (See Table 4). For the internally-generated procedure, when a negative stimulus was presented, the target emotions always showed positive associations with the non-target negative emotions. In the externally generated procedure, negative target and non-target emotions were also positively associated, with the exception of Anger (target) – Fear (non-target). Regarding the association of positive and negative emotions, the only significant association found was in the internal procedure, where Joy (target) was negatively associated to Anger (non-target).

	Anger		**Sadness**		**Fear**		**Joy**	
Target	ASR	FC	ASR	FC	ASR	FC	ASR	FC
Anger	1	1	0.53*	0.61*	0.33*	0.27	0.12	0.21
Sadness	0.61*	0.54*	1	1	0.62*	0.55*	0.04	-0.16
Fear	0.55*	0.70*	0.71*	0.67*	1	1	-0.18	-0.10
Joy	-0.35*	0.06	-0.14	-0.15	-0.03	0.05	1	1

Table 4: Associations between discrete emotions in the internal (Affective Story Recall), and external (Film Clips), mood induction procedures.

3.1.5. A methodological comment

One possible criticism of these results is that the elicitation of multiple emotions in a single session generates contamination between emotional episodes, or produces an overall affective arousal reducing emotion selectivity. To investigate this issue we conducted a series of

investigations of possible order effects. These demonstrated that the magnitude of the *target emotion* was, no higher in initial, versus central, versus late presentations using film clips (Fear $F_{(3,36)} = 1.89$, $p=.15$; Sadness $F_{(3,36)} = 0.94$, $p=.43$; Joy $F_{(3,36)} = 0.76$, $p=.55$; Anger $F_{(3,36)} = 0.75$, $p=.53$) and affective story recall (Fear $F_{(3,36)} = 2.51$, $p=.07$; Sadness $F_{(3,36)} = 1.48$, $p=.24$; Joy $F_{(3,36)} = 0.88$, $p=.46$; Anger $F_{(3,34)} = 1.09$, $p=.37$). Moreover, the extent to which non-target emotions was elicited was also not significantly different across presentation order.

3.1.6. Discussion

As reviewed in the introduction, it has become clear that while emotion elicitation techniques are widely employed, several key issues remain under-investigated. Issues of effectiveness are probably the major concern: does a chosen MI procedure *elicit* the target emotion, and is one MI procedure more *effective* than another in eliciting a specific emotion? Given the central importance of these questions in the design for experimental work in the field, it is re-assuring to note that our results demonstrate that the affective story recall (internal MIP) and film clips (external MIP) were *equally* effective in selectively eliciting the target emotion intended (e.g. fear), to the exclusion of other emotions (e.g. sadness, anger, joy, see Figure 1).

One possible objection to the finding that the stimuli generate emotions which are selective to the target emotion is the issue of demand characteristics- that is that participants *report* that they have experience fear because they *have been told* to recall fearful life events or to watch fearful clips. Two points relate to this issue. Firstly, in the externally elicited procedure (film clips) we followed the widely employed guidelines designed specifically to avoid demand characteristics (Rottenberg, Ray & Gross, 2007). These generated, as expected, experiential evaluations which were higher for the target emotions, but also distributed to

non-target emotions (especially for negative-related target emotions). For the internally generated stimuli (affective story recall) there is, of course, a greater risk of demand characteristics, because participants are specifically requested to recall a single emotion (e.g. fear). Importantly, the reports of non-target emotions (e.g. sadness and anger) are also found for using this procedure – and non-target activation levels are just as easily elicited in the internally generated process as they are for externally generated stimuli. On this basis, demand characteristics do not seem to be a central factor in explaining the findings relating to emotion specificity.

A further way of addressing the 'effectiveness' issue comes from comparing the *intensity* of target emotions between procedures -by establishing which generates the stronger response in participants. The results of the present study show the interesting finding that only Joy appears to produce significantly higher levels of reported emotion when elicited by the internally generated procedure. This result does differ from that of Westerman et al. (1996), though their study did not compare the same participants in both internal and external procedures.

A second goal of the present study was to explore whether an internal (affective story recall) and external procedure (film clips) differed in the intensity of the emotions they generated (i.e. the average of *all* positive and negative emotions experienced while exposed to a fear stimulus). Notably, this was significantly higher for the *internal* procedure, for *each* stimulus. Thus, although the intensity of *target* emotions did not differ significantly when using an internal or external elicitation technique (with the exception of joy), the internal procedure tended to generate a greater level of *overall* emotional arousal.

Several possible explanations might account for this overall higher intensity of the internal procedure. A first possibility is that participants' scores in the film clips were more widely distributed than the ASR, lowering the average score of all emotions. However, this

seemed unlikely, considering that the standard deviation of the overall averaged scores was always greater in the internal procedure (see Table 3).

A second possibility considered is that internal mood induction procedures activated the associated non-target emotions with greater intensity than external procedures, because of its greater ecological validity. In other words, that when eliciting a negative emotion (e.g. fear) other negative non-target emotions (e.g. anger and sadness) may be activated more intensively. This claim is supported by previous studies examining *realistic emotion*, which suggested that negative emotion-producing situations are often complex and produce more than one emotional state (Ellsworth & Smith, 1988; Polivy, 1981; Scherer & Tannenbaum, 1986). However, the results of the present study did not support this explanation, or at least only supported it incompletely. When eliciting a negative emotion, the average of the associated non-target emotions was always higher in the internal procedure. However, this effect was not restricted to the associated non-target negative emotions. Thus, when fear was internally generated, joy (a non-target positive emotion) also appeared to be significantly higher, and when eliciting joy, negative non-target emotions (e.g. anger, sadness, fear) also were more intensively activated. In conclusion, it appears that the higher overall intensity of the internal procedure may not be exclusively explained by the higher intensity of valence-like emotions (negative blends) but to a higher intensity of *all* kind of emotions (negative blends and mixed blends).

It is possible to suggest that the overall higher intensity of the internal procedure may be related to the fact that recalling an emotional event is a more complex and dynamic process than observing a film clip- generating positive, negative and mixed blends of emotion. In fact, a qualitative analysis of the stories recalled by the participants (see Appendix 3) appears to supports this hypothesis. For instance, when asked to recall an 'angry' event, example A alternates between anger ('I absolute *hated* these girls who went to

do that to my sister') and sadness contents ('I *cry* a lot because she is going through so much and she is my baby sister and I'm supposed to protect her'). In this particular emotional event, it appears that different emotions seem to be simultaneously directed to different 'objects' (anger toward the bullying girls, and sadness about her sister).

The case of example B (Fear) and D (Joy) are clear instances of how negative and positive emotions may unfold sequentially over time, in an apparently interactive fashion. In subject B, the experience of fear ('I kept hearing noises upstairs and I just sort of *froze*') was followed by a more positive feeling, a mixture of embarrassment for being previously scared ('it was the cat playing around…feeling a bit stupid about myself') and relief ('and then I felt sort of calm'). In example D, the experience of joy ('getting a A+ was very, very *exciting* and it made me feel very *happy'*) is temporarily interrupted by the negative emotion of fear ('the Professor came to me and said "can we use your essay in the class as an example?... I was a bit *scared')*. It seems important to emphasize here how an emotion of different valence may emerge as a response of the subject to the initial emotional state (example B), or because an external event changes the initial trajectory of the emotional experience (example D).

In sum, it appears that a possible explanation of the higher overall intensity of the internal procedure might be related to the dynamic nature and complexity by which positive and negative emotions unfold when people recall emotional events from their life. This is consistent with the observation of higher frequency of positive blends, negative blends and mixed blends in real life scenarios (Ellsworth & Smith, 1988; Scherer & Tannenbaum, 1986; Scherer et al., 2004). However, it remains a matter of further research whether this complexity is inherent to emotional life itself, or is an artefact of the process of recollection.

The findings of this study also contribute to an on-going discussion regarding whether some mood induction procedures are better eliciting specific emotions. For example, one strand of literature suggests that anger may be difficult to elicit using film clips or

photographs (Harmon-Jones, Amodio & Zinner, 2007; Levenson, 2007; Rottenberg et al., 2007) - whereas personal and interactive procedures are more effective inducers (Harmon-Jones, Amodio & Zinner, 2007). However, these findings have been at their most clear with respect to physiological reactivity, and are less apparent in self-report (Lobbestael, Arntz & Wiers, 2008). In addition, there are a number of practical issues following from the use of interactive procedures - which usually require deception. Most notably, interactive procedures may well be unsuitable (or unethical) when exploring emotion in vulnerable groups, such as psychiatric or neurological populations. In consequence, although is necessary to acknowledge that some eliciting procedures may better activate of specific emotions (or some aspects thereof), the sorts of emotional induction procedures reported in the present study also have clear advantages of experimental control (one procedure for all emotions), and benefits as regards practical matters of implementation across various clinical populations.

In conclusion, the findings of this study appear to support the use of both internally and externally generated procedures in emotion elicitation research, by demonstrating that the recall of personal events (internal) is equally effective in selectively eliciting emotions when compared with well-standardized film clips (external). Furthermore, the findings of the present study suggest that affective story recall may be more suitable for experiments targeting positive emotions such as joy, or experiments aiming to produce higher levels of arousal.

Future research comparing the effectiveness of internal versus external MI procedures would also benefit by measuring other components of emotional response, such as behaviour and physiology. In addition, the consideration of age (including, for example, the greater sadness reactivity in older people, see Seider, Shiota, Whalen & Levenson, 2011) and gender

related issues (i.e. Brody & Hall, 2008) might reveal differences in patterns of response to specific emotional stimuli.

The use of internal procedures, such as the ASR, seems especially useful when exploring the dynamic aspect of emotional response. Because of the complexity of recalling a personal emotional event, emotional blends more readily can be observed. However, it remains a matter for future investigation whether these blends are better explained by sequential or simultaneous patterns of emotional experience (do participants experience two emotions simultaneously, although they appear to be sequential because of the linear nature of verbal narrative?), or if they are related to the complexities of the scenarios in which they unfold (as in example A, where two different emotions are directed towards two different objects in the same situation).

The scientific investigation of MI procedures appears to have favoured the use of externally generated stimuli (such as emotional pictures or film clips). This was a quite natural response to the desire for sound experimental control by the field, presumably with the expectation that a less controlled stimulus (recalling a personal event) might generate unpredictable and unreliable findings. The present study suggests that an internally generated MI procedure, such as the affective story recall, can produce effects that are as reliable as an external technique. Furthermore, such an approach opens the opportunity to investigate internally generated procedures in more detail, which by virtue of its verbal nature, provide a richer source of introspective data related to the subjective quality of emotional experience.

3.2. Internally and externally generated emotions in people with acquired brain injury: A brief report on right hemisphere damage.

3.2.1. Abstract

The study of emotional changes after brain injury has contributed enormously to the understanding of the neural basis of emotion. However, little attention has been placed on the methods used to elicit emotional responses in people with acquired brain damage. Of particular interest, for the topic of emotion elicitation, are subjects with unilateral right hemisphere [RH] lesions, whom have been described as presenting significant impairment in emotional processing and reactivity. In this article, an internal [affective story recall] and external [film clips] mood induction procedure [MIP] were used to trigger positive [joy] and negative [sadness] emotions, in a sample of 10 subjects with unilateral RH damage and 15 healthy matched controls. The participants' subjective emotional experience was registered by using a self-report questionnaire. As observed in previous studies, internal and external MIPs were equally effective selectively eliciting the target emotion. However, the internal procedure generated higher levels of emotional intensity when compared to the external. There were no significant group differences in relation to the level of intensity elicited by both MIPs, thus suggesting that participants with RH lesions were equally able to experience positive and negative affect. The results are discussed specifically in relation to the role of the RH in the capacity to experience negative emotions.

3.2.2. Introduction

Research on emotional changes after acquired brain injury has a long history (Adolphs, 2007; Borod, 2000; Gainotti, 1972, 2001), contributing to growing understanding of the neural basis of several emotional processes, such as emotional perception (e.g. Adolphs, Damasio, Tranel, Cooper, & Damasio, 2000; Borod, Bloom, Brickman, Nakhutina, & Curko, 2010; Tsuchida & Fellows, 2012), expression (e.g. Borod et al., 2010; Kazandjian, Borod, & Brickman, 2007; Nakhutina, Borod, & Zgaljardic, 2006) and experience (e.g. Anderson & Phelps, 2002; Feinstein, Adolphs, Damasio, & Tranel, 2010; Feinstein, Duff, & Tranel, 2010; Feinstein, 2013; Gillihan et al., 2010; Hornak et al., 2003).

Typically, studies on emotion use some type of mood induction procedure [MIP] to trigger the intended *target* emotion. There are a wide range of MIP, such as self-referential statements (e.g. Kenealy, 1986; Velten, 1968), autobiographical recall (e.g. Brewer & Doughtie, 1980; Schaefer & Philippot, 2005; Turnbull, Evans, & Owen, 2004), imagery (e.g. Tranel, Bechara, Damasio, & Damasio, 1998; Wright & Mischel, 1982) or film clips (e.g. Gross & Levenson, 1995; Rottenberg, Ray, & Gross, 2007; Schaefer, Nils, Sanchez, & Philippot, 2010). However, there is little research comparing the effectiveness of each method (Gerrards-Hesse, Spies, & Hesse, 1994; Isen & Gorgoglione, 1983; Westermann, Stahl, & Hesse, 1996). Recently, it has been reported that *internal* MIPs [autobiographical recall] generate higher levels of overall affect compared to *external* MIPs [film clips] (Salas, Radovic, & Turnbull, 2011).Because the successful triggering of target emotions is a prerequisite for measuring other more complex emotional processes, such as emotional comprehension or regulation (Rosen & Levenson, 2009), the comparative effectiveness of different forms of MIP is, itself, an important topic of research.

Notably, the correct selection of an MIP may well be critical when experimental subjects present with cognitive impairments that compromise the effective engagement with the

stimuli, especially after acquired brain injury (Levenson, 2007). For example, patients might struggle recalling details of personal events [autobiographical recall], or grasping the plot on a film [film clips] (Levenson et al., 2008). Unfortunately, there has been no systematic research effort to address this issue. Thus, it seems timely to explore which MIPs are best suited to elicit emotional states in neurological patients.

Among the variety of patients with acquired brain damage, subjects with unilateral lesions to the right hemisphere (RH) are of particular interest for the topic of emotion elicitation. It has been widely reported that, compared to subject with left unilateral lesions, they present a wide range of perceptive and expressive emotional deficits (for a review see Borod et al., 2010), as well as physiological hypo-reactivity to emotional stimuli (Heilman, Schwartz, & Watson, 1978; Morrow, Vrtunski, Kim, & Boller, 1981). Furthermore, it has been suggested that RH lesions compromise specifically the processing of negative, or withdrawal, emotions (for a review Gainotti, 2000), what have been called the valence hypothesis (Craig, 2005; Davidson, 1992a, 1992b, 2001). Unfortunately the evidence addressing this problem is small and present important methodological limitations. For example, most of the studies have focused on perceptual and expressive impairments, neglecting whether RH damage compromises or not the capacity to *experience* negative emotions.

Additionally, the studies that have addressed changes in emotional intensity have used rating by naïve judges as a method, thus not considering the patient's report of his own experience (Borod et al., 1996; Montreys & Borod, 1998) To our knowledge there is only one case study that has experimentally addressed this problem, using an internal MIP [affective story recall] to explore the capacity of a patient with RH damage to experience negative emotions (Turnbull et al., 2004). The authors found that the patient was able to experience similar levels of negative emotions than controls, thus challenging the valence hypothesis.

Nevertheless, a limitation of this study is that results are also based on the rating of naïve judges, thus not considering the patients subjective emotional experience.

The present study is the first to offer some insight on this problem, by comparing the effectiveness of two different MIPs in participants with unilateral RH damage and a matched sample of healthy controls. Following previous work on emotion elicitation (Salas et al., 2011), internal [affective story recall] and external [film clip] MIPs were used to elicit positive [amusement] and negative [sadness] emotions. The affective experience of participants was registered using a self-report questionnaire.

This study attempted to extend the findings of Salas et al. (2011), which were based on a student sample, to the key populations of patients with RH damage and normal elderly controls. In addition, this study also attempted to extend the findings of Turnbull and colleagues (2004), this time using both internal and external MIPs, and self-reports, to test whether participants with RH were able to experience negative emotions [sadness]. Based on the results of Salas et al. (2011) the following predictions were considered: 1) *Selectivity*: That internal and external MIPs will selectively trigger the target emotion but not the non-target emotion, this in both healthy controls and neurological groups; 2) *Intensity*: That internal MIPs will generate significantly higher levels of self-reported emotion compared with external MIPs in both groups; 3) *Right Hemisphere:* that patients with RH lesions will report less levels of negative emotional intensity than controls when using both MIPs.

3.2.3 Methods

3.2.3.1. Participants

The participants of this study were 10 subjects with right hemisphere damage (RH, Male = 4, Female = 6) and 15 healthy controls (HC, Male = 5, Female = 10). Both groups were matched in age (RH: *M* = 61.9, *SD* = 11.9; HC: *M* = 62.80, *SD* = 4.12) and education (ABI:

M = 13.63, *SD* = 1.36; HC: *M* = 13.93, *SD* = 1.75). The average time since injury in the ABI group was 55.8 months (*SD* = 34.84; *MIN* = 13, *MAX* = 114). Participants were referred by neurologists from Bangor University and the North Wales Brain Injury Service. Exclusion criteria for the neurological group were having a non-focal lesion, duration of less than 6 months since the brain injury, and moderate to severe language impairment. The details of patient's lesion location are described in Table 1.

Age/Sex	*Months since injury*	*Location*	*Aetiology*
57 F	84	Right Prefrontal	MCA Stroke
50 M	20	Right Prefrontal	MCA ACA Stroke
73 F	88	Right Prefrontal	MCA Stroke
45 M	70	Right Prefrontal	ACoA SAH
74 M	20	Right ventro-lateral prefrontal cortex, basal ganglia	MCA Stroke
65 M	65	Right frontal and TPJ	MCA Stroke
46 F	114	Right Prefrontal	MCA Stroke
63 F	60	Right Prefrontal and TPJ	MCA Stroke
78 F	13	Right Prefrontal	MCA Stroke
68 F	24	Right fronto-parietal	MCA stroke

ACoA = anterior communicating artery aneurism; SAH = subarachnoid haemorrhage, MCA = middle cerebral artery. ACA = Anterior cerebral artery. TPJ = Tempo Parietal Junction

Table 1. Clinical details of participants with acquired brain injury.

3.2.3.2. Procedure

The assessment took place mainly at the University of Bangor. In cases where participants with brain injury were not able to travel to the venue, researchers tested them at home. Participants were seen twice in order to collect data for the present study, and a larger one on emotion regulation. As a main introduction of the research, they were told that they would be asked to recall personal events in relation to certain emotions. In addition, they would have to watch some movies in the computer, and report on how they made them feel. For a detailed

description of the procedure adapted for this study see Salas, Radovic and Turnbull (2011) [see section 3.1].

During the first session the purpose of the experiment was explained to participants and their relatives and consent was obtained [see Appendix 2]. Participants completed a set of measures on overall cognitive functions and the Film Clip task. The Affective Story Recall was administered during the second session, along with other neuropsychological tasks that are not described here.

3.2.3.3 Mood Induction Procedures

3.2.3.3.1. *Internal Mood Induction Procedure: Film Clips*

In this task participants are asked to watch a series of film clips and report their emotional experience while watching the clips. For that purpose, participants were placed in front of a 15'' laptop screen. Headphones were provided to avoid any possible distracting noise. The following instruction was offered: '*I will ask you to watch a couple of short film clips. Please pay attention and watch them carefully'*. After each film clip a self-report questionnaire was administered. Before each 'emotional' video a neutral video was presented and the following instruction was given: *'Now I would like you to watch this video and try to relax and clear your mind of any thoughts'.* The 'emotional' clips [amusement-sadness] were counterbalanced across participants of each group to avoid order effects.

In relation to the neutral and emotional clips used, all of them have been previously validated regarding their capacity to elicit specific and discrete emotions (Gross & Levenson, 1995; Rottenberg et al., 2007). The neutral clips, *Sticks* (Gross & Levenson, 1995), were 60s in length and showed abstract shapes and colours. The amusement clip, *Bill Cosby Himself* (Cosby, 1983) showed a stand-up comedy performance by Bill Cosby (121 s). The sadness

clip, *The Champ* (Zeffirelli, 1979), showed a young boy facing the sudden death of his father after a boxing match (171 s).

3.2.3.3.2. *External Mood Induction Procedure: Affective Story Recall*

The Affective Story Recall task (Turnbull et al., 2004) was used as an internal MIP for both amusement and sadness.. In this task participants are asked to recall personal events from their lives related to specific emotions. In the same way that with the film clips, two neutral recall conditions (going shopping and fixing a meal) are used before each emotional recall as baselines. Each recall was prompted with the following phrase: "Try to recall an event in your life that has caused you to feel . . . (e.g., amusement). Try to be very detailed about the way you feel." Each participant had a maximum of 3 minutes to describe the event, although they could extend this if they felt they have not finished yet the story after the 3 min, or even finish before if they felt they have nothing else to say. A minimum of time was not set in order to avoid that task instructions could activate negative emotions by adding pressure and anxiety to the recollection. The same self-report questionnaire administered after each clip was also administered after each recollection.

3.2.3.4. Measures

In order to capture the emotional experience of participants, after each clip and affective story, a self-report measure was adapted from the PANAS-X (Watson & Clark, 1994). The PANAS-X is a 60-item self-report that assesses specific emotional states. It has a general dimension of positive and negative affect, but also includes specific scales for discrete emotions. Participants are asked to rate each of the 60 emotional words (e.g., cheerful, hostile, shaky) indicating the extent to which they felt each emotion according to a 5-point

scale (very slightly or not at all, a little, moderately, quite a bit, and extremely). For the purpose of this study, a total of 20 emotional words were selected from the discrete emotion scales, with 5 emotional words for each of the four basic emotions [***Joy***: cheerful, delighted, happy, joyful, energetic; ***Sad***: downhearted, sad, blue, lonely, alone; ***Anger***: disgusted, angry, loathed, irritable, hostile; ***Fear***: shaky, afraid, nervous, scared, frightened].

3.2.3.5. Score Data Analysis

The average score of each PANAS' subscales [Fear, Sadness, Joy, and Anger] was calculated, this for each eliciting stimulus [Neutral 1, Sadness, Neutral 2, Joy] on both tasks [ASR and FC]. To test the *Selectivity and Intensity* hypothesis, the PANAS' scores for each stimulus were compared using a mixed-anova test. More specifically, to answer the *Selectivity* hypothesis, the analysis was conducted separately for each stimulus and tasks, with the four subscales of the PANAS treated as the within-subject variable and controls v/s patients as the between-subject variable. The differences between the target emotion score and the other emotions were calculated using a simple contrast, with the target emotion as reference. In relation to the *Intensity* hypothesis, separated analyses for each stimulus were conducted, using the target emotion in both tasks [e.g. sadness or joy] as the within-subject variable and controls v/s patients as the between-subject variable. In both analysis, when the sphericity assumption was not respected, Greenhouse-Geisser correction was used (for Greenhouse-Geisser's between 0.9 and 0.7), or multivariate Pillai's Trace statistic was reported (for Greenhouse-Geisser's less than 0.7). Finally, the exploration of the interaction term in the mixed models allowed to test the third hypothesis (*Right Hemisphere*), and explore whether the comparisons in the within model differed between groups.

3.2.4. Results

The present study tested three hypotheses: (a) that internal and external MIPs will elicit the target emotion with grater intensity than the non-target emotions, this in both groups *(Selectivity)*; b) that target emotions elicited by the internal MIP will show greater intensity than the ones generated by the external MIP, in both groups *(Intensity)*; c) that the neurological group would present lower levels of negative emotional intensity compared to the healthy control group when exposed to internal or external stimuli *(Right Hemisphere).*

3.2.4.1. Selectivity

In relation to the *Selectivity* hypothesis, both MIPs elicited effectively the two target emotions (e.g. sadness) with higher levels of intensity than the other three emotions considered in the PANAS (e.g. joy / anger / fear) [See Fig. 1]. This result demonstrates that both internal and external MIPs are effective inducing an emotion selectively, this for people with and without brain injury [see Table 2].

Mood Induction Procedure	Target Emotion	Non-Target Emotion	Paired Differences	df	F	p	$\eta\rho^2$	r
Internal	*Sad*	Joy	9.91	1,19	89.35	<.001	0.83	0.91
(Affective Story Recall)		Anger	8.00	1,19	60.10	<.001	0.76	0.87
		Fear	5.81	1,19	36.44	<.001	0.66	0.81
	Joy	Sad	12.65	1,21	154.80	<.001	0.88	0.94
		Anger	13.17	1,21	202.65	<.001	0.91	0.95
		Fear	12.08	1,21	155.10	<.001	0.88	0.94
External	*Sad*	Joy	5.83	1,22	25.64	<.001	0.54	0.73
(Film Clips)		Anger	4.92	1,22	20.48	<.001	0.48	0.69
		Fear	4.37	1,22	17.76	<.001	0.45	0.67
	Joy	Sad	8.17	1,22	41.74	<.001	0.66	0.81
		Anger	8.3	1,22	54.03	<.001	0.71	0.84
		Fear	8.21	1,22	45.70	<.001	0.68	0.82

Table 2. PANAS score differences between target and non-target emotions for the internal (ASR) and external (FC) MIP.

Differences in the average intensity between target and non-target emotions, for the two emotional stimulus, were observed in the internal [Sadness: $F(3, 57) = 34.34$, $p < .001$, $\eta\rho^2 =$

.64[12], r = .80; Joy: v = .92, $F(3, 19) = 74.36$, $p < .001$, $\eta\rho^2 = .92$, $r = .96$] and external [Sadness: $F(2.44, 53.67) = 14.71$, $p < .001$, $\eta\rho^2 = .40$, $r = .63$; Joy: v = .75, $F(3, 20) = 19.45$, $p < .001$, $\eta\rho^2 = .75$, $r = .87$] MIP. All the planned comparisons between target and other emotions were significant in both MIPs, as seen in table 2. No differences across groups were found [Interaction *emotion*group*; Sadness Internal: $F(3, 57) = .19$, $p = .91$, $\eta\rho^2 = .01$, $r = .10$; Joy Internal: v = .12, $F(3, 19) = .84$, $p = .49$, $\eta\rho^2 = .12$, $r = .34$; Sadness external: $F(2.44, 53.67) = 1.13$, $p = .34$, $\eta\rho^2 = .05$, $r = .22$; Joy external: Joy: v = .14, $F(3, 20) = 1.07$, $p = .38$, $\eta\rho^2 = .14$, $r = .37$]. Thus the comparison between the target emotion and the other three emotions was statistically significant for each stimulus, in both tasks, showing the same pattern in healthy controls and neurological group.

3.2.4..2. Intensity

In relation to the *Intensity* hypothesis, the internal MIP elicited higher levels of intensity in the target emotion when compared to the external MIP, for both sadness and joy stimulus. No significant differences were found between groups. This result suggests that internal MIPs are more effective than external MIPs in triggering intense emotional experience [see Table 3].

Target Emotion	Internal (ASR)		External (FC)		Paired Differences					
	Mean	SD	Mean	SD	ASR-FC	df	F	p	$\eta\rho^2$	r
Sadness in SAD	15.50	3.90	12.40	4.30	3.10	1,18	22.53	<.001	0.56	0.75
Joy in JOY	18.05	4.53	13.18	5.09	4.87	1,20	11.1	<.01	0.36	0.60

Table 3. Comparison of the target emotion's level of intensity when using an internal (ASR) or external (FC) MIP. The internal MIP generates significantly higher levels of intensity.

The average intensity of the target emotion in the internal MIP was higher than the external MIP for both sad [$F(1, 18) = 22.53$, $p < .001$, $\eta\rho^2 = .56$, $r = .75$] and joy [$F(1, 20) =$

[12] $\eta\rho^2$ stands for partial eta square. To interpret $\eta\rho^2$ it was transformed to r, using the formula r = square root ($\eta\rho^2$). Then, standards guides given by Cohen (1992) could be used (r< .10= small; r< .30= medium; r< .50= large).

11.10, $p = .003$, $\eta\rho^2 = .36$, $r = .60$] stimulus. Thus the comparison between internally and externally generated target emotions was statistically significant for each stimulus, with no differences between participants with and without brain damage [Sadness: $F(1, 18) = .99$, $p = .33$, $\eta\rho^2 = .05$, $r = .22$; Joy: $F(1, 20) = 2.08$, $p = .17$, $\eta\rho^2 = .09$, $r = .30$].

3.2.4.3. Right Hemisphere

In relation to the *Right Hemisphere* hypothesis, participants with unilateral RH damage did not differ from healthy controls in level of intensity reached by positive and negative target emotions, this using internal and external MIPs. As described above, no significant interactions were observed between group and levels of intensity, for both positive and negative emotions. In conclusion, the data did not support the hypothesis suggesting that RH patients would have lower levels of emotional experience in response to negative stimulus. Results for all three hypotheses are described in Figure 1.

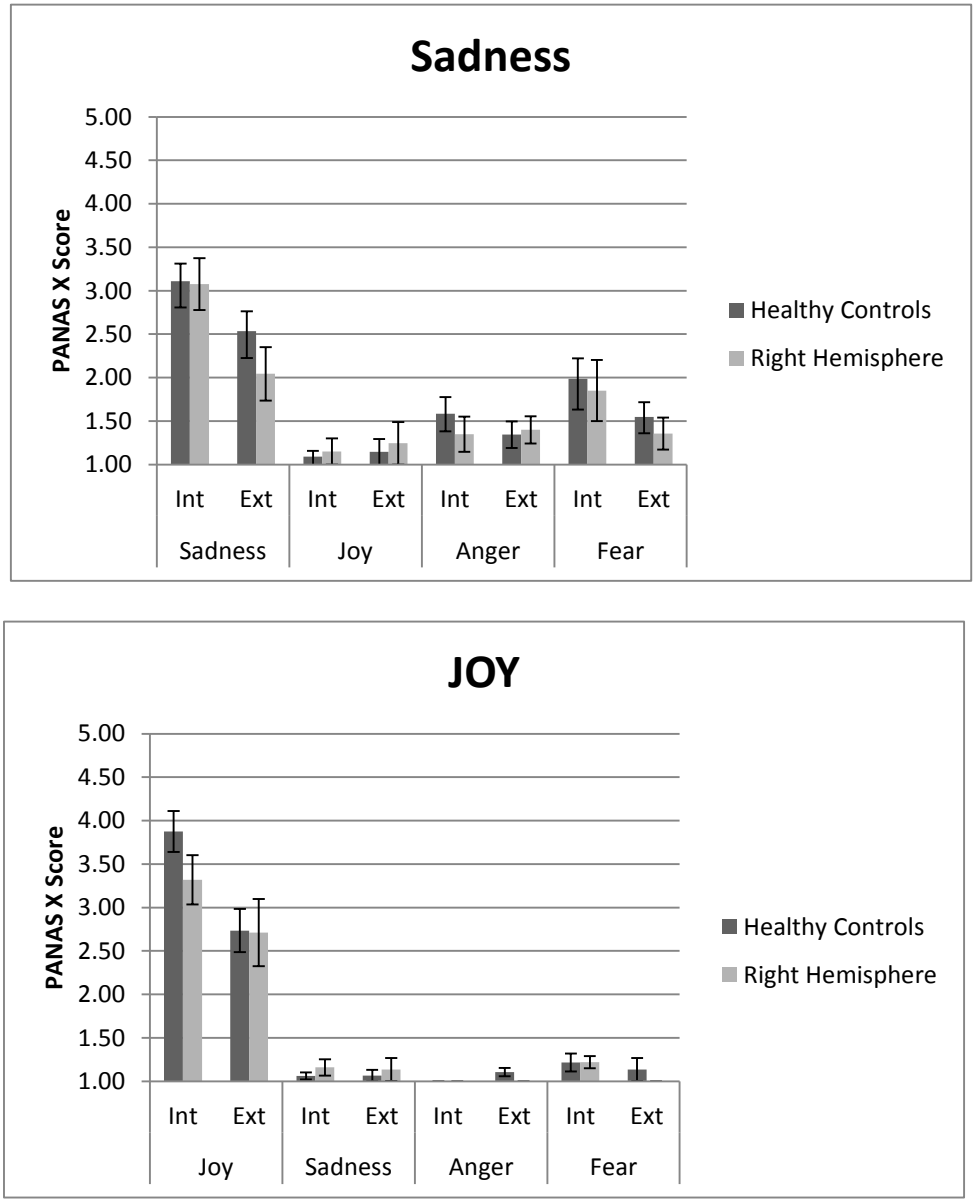

Fig. 1. PANAS score for internal (ASR) and external (FC) Sad and Joy stimuli. Both right Hemisphere participants and healthy controls are represented. In each case the internal and external procedure induce selectively the target emotion compared to non-target emotions. However, the internal procedure generates higher levels of experienced emotion. Participants with right hemisphere damage do not differ from controls in both measures.

3.2.5. Discussion

The goal of this study was twofold. Firstly, it explored the effectiveness of internal and external MIPs in an older adult sample of people with RH damage and healthy controls. Secondly, it tested whether participants with RH lesions were able to experience negative emotions using an internal and external MIPs.

A first finding of this study is that the levels of intensity were higher for the internally generated material. This is a replication of the results reported by Salas and colleagues in a student sample (2011), however it extends such results by suggesting that a similar pattern is also reported in elderly people and also in subjects with acquired brain injury. In addition, it was also found that emotional levels of intensity, measured by self-report, were not significantly different between participants with unilateral RH damage and healthy controls, for both positive [joy] and negative [sadness] emotions.

The results from this study contribute to a growing literature on the elicitation of emotion (Coan & Allen, 2007). When this results are interpreted in relation to previous studies comparing the efficacy of internal and external MIPs (Salas et al., 2011), they appear to suggest that the higher levels of emotion generated by the internal MIP are independent of age, for this phenomenon is observed in both young and elderly adults populations. This is an interesting finding for the literature on emotional intensity, which has tended to focus on the structure of emotional intensity (e.g. Sonnemans & Frijda, 1994; Verduyn, Van Mechelen, Tuerlinckx, Meers, & Van Coillie, 2009), and the individual determinants on intensity levels (e.g. Lynch, Robins, Morse, & Krause, 2001; Neumann, 2001; Sonnemans & Frijda, 1995), with less consideration of the impact of the MIPs used (for a review see Salas et al., 2011). This study offers important evidence supporting the view that emotion can be more intensely triggered when using personally relevant material.

The data obtained in this study is also of relevance to the elicitation and assessment of emotion in people with acquired brain damage (Levenson, 2007; Levenson et al., 2008). It shows that, in the same way than healthy controls, people with right hemisphere damage generate higher levels of positive and negative emotion when using internal MIPs. This finding suggests that the recollection of personal affective memories is also a powerful form of eliciting emotional states in this population, and that such recollection does not appear to be compromised by the cognitive impairments commonly associated to brain damage.

In terms of the RH debate on emotional experience, this data suggests that the capacity to experience positive and negative emotions, when measured by self-report, is preserved in patients with RH unilateral lesions. This finding supports the results reported by Turnbull and colleagues (2004), who described that negative emotional experience was not disrupted in a patients with right hemisphere damage, thus challenging the valence hypothesis (Craig, 2005; Davidson, 2001, 1992a, 1992b). Furthermore, it agrees with a recent literature proposing that the capacity to experience emotions is largely preserved after cortical damage and cortical atrophy (Merker, 2007), for such ability depends on rather deep subcortical structures (Damasio et al., 2000; Panksepp, 1998, 2011).

This study has two main limitations. The first one is the small sample used, which force us to consider these results as preliminary and requiring further confirmation with a larger population. The second limitation is rather technical, in that this study only focused on the subjective aspect of the emotional experience, without considering physiological or behavioural measures. This is relevant for future investigations, for it has been described that the three components of the emotional response [physiology, behaviour and experience] are not always associated (Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005). In fact, our findings challenge previous data from behavioural [facial] (e.g. Borod, Bloom, Brickman, Nakhutina, & Curko, 2002) and physiological studies (Heilman et al., 1978; Morrow et al.,

1981) on emotion after RH damage. Future studies on this topic should consider this componential view of the emotional response, and incorporate the simultaneous assessment of experiential, behavioural and physiological elements.

For decades emotional changes after brain injury have contributed to our understanding of the neural basis of emotional processes (Adolphs, 2007; Borod, 2000; Damasio, 1994; Feinstein, 2013; Gainotti, 2001; Robinson, 2006; Rosen & Levenson, 2009). However, little attention has been placed on the methods used to trigger emotional responses (Levenson, 2007; Levenson et al., 2008). This study has contributed to the field by showing that internal and external forms of elicitation are equally effective triggering selectively emotional states, although the internal procedure generates higher levels of intensity. More importantly, it suggests that these methods are also effective triggering negative emotional states in patients with RH unilateral damage, a population that has been traditionally described as impaired in the capacity to experience negative emotion.

Chapter 4: Group Design Articles

4.1. Emotion Regulation after Acquired Brain Injury: Response Modulation after Right Prefrontal Damage

4.1.1. Introduction

There is a growing literature reporting emotion regulation [ER] problems after brain damage, as well as commenting on its impact to emotional adjustment and social functioning (Abreu et al., 2009; Bechara, 2004; Beer & Lombardo, 2007; Lewis, Lamm, Segalowitz, Stieben, & Zelazo, 2006; McDonald et al., 2010; Obonsawin et al., 2007; Salas, 2012; Tate, 1999). Evidence from different sources appears to suggest that damage to the right hemisphere, and particularly to the right prefrontal cortex [rPFC], generates changes in the capacity to modulate emotion. It has been noted, for example, that individuals with rPFC damage present more severe emotional deficits, such as disinhibition of sympathetic centres (Hilz et al., 2006), emotion-based decision making impairment (Manes et al., 2002; Tranel, Bechara, & Denburg, 2002), delusional pathology (Feinberg, Venneri, Simone, Fan, & Northoff, 2010; Feinberg, 2011) and neuropsychiatric disorders like mania and alexithymia (Cummings, 1997, for a review). The mechanisms by which ER is disrupted after rPFC lesions are yet unknown, however, it has been proposed that this cortical area has a special role integrating cognition and affect (Stuss & Alexander, 1999; Stuss & Alexander, 2000).

There is a large body of literature suggesting that response inhibition, often regarded as the process of withholding unwanted, prepotent and reflective actions (Aron et al., 2004; Dillon and Pizzagalli, 2007), is a key ability to ER or self-regulation (e.g. Barkley, 1997, 2001; Hofmann, Schmeichel, & Baddeley, 2012; Quirk, 2007; Thayer & Lane, 2000). Interestingly, several sources of evidence have linked response inhibition to the rPFC. For example, neuroimaging findings (Aron, Robbins, & Poldrack, 2004; Aron, 2007; Chambers,

Garavan, & Bellgrove, 2009; Dillon & Pizzagalli, 2007; Forstmann et al., 2008; Levy & Wagner, 2011; Rubia, Smith, Brammer, & Taylor, 2003) and transcranial magnetic stimulation studies (Chambers et al., 2006, 2007) have suggested that response inhibition is mediated by a right hemisphere network, with a critical role for the inferior frontal gyrus [rIFG]. Lesion studies exploring the relationship between rPFC lesions and inhibition impairment appear to compliment neuroimaging findings. It has been reported that damage to the rIFG is associated with an increased reaction time in stop-signal inhibition tasks (Aron, Fletcher, Bullmore, Sahakian, & Robbins, 2003) and also to deficits in suppressing inappropriate responses on switching tasks (Aron, Monsell, Sahakian, & Robbins, 2004).

An important limitation of this literature on response inhibition is that it has mostly used *motor* inhibition tasks, and not *emotional* inhibition tasks. To our knowledge there is only one single neuroimaging study that has used an emotional inhibition paradigm, or interference paradigm, in exploring the neural correlates of inhibiting facial emotional expressions of a neurologically intact population (Lee, Dolan, & Critchley, 2008). During the task, participants were asked to look at pictures of people displaying a variety of emotions, and to voluntarily manipulate their facial [emotional] expressions to the pictures. Two conditions were used: to *inhibit* the expression of facial displays, and to *simulate* an emotion of opposite valence [e.g. frown when watching someone smiling]. Similarly to the above mentioned studies on motor inhibition, it was found that, among other right hemisphere areas [insula, superior temporal sulcus], the right inferior frontal gyrus [BA 47] was activated during the inhibition of emotional expression. More importantly, the activation of BA 47 was positively correlated with the self-reported use of a well-known ER strategy: suppression, or the ability to inhibit a behavioural display while affectively aroused (Gross & Levenson, 1997; Gross & John, 2003).

To our knowledge, the study of Lee, Dolan and Critchley (2008), is the only study that has considered a well-known ER strategy, that of suppression (Gross & Thompson, 2007), to explore the neural correlates of the inhibition of emotional behaviour. This limitation is, however, not exclusive of inhibition studies, but can also be found in the literature on ER changes after rPFC lesions. It is surprising that, despite the rapid growth of the field of ER in the last decade (Gross & Thompson, 2007), neuropsychology and lesion studies have not yet incorporated the conceptualization of ER (Beer & Lombardo, 2007; Salas, Gross, & Turnbull, submitted).

The Process Model of ER (Gross & Thompson, 2007) describes a set of strategies that people tend to use in modulating their emotional experience. One of these strategies, suppression, is extremely interesting in the exploration of the relationship between emotion modulation impairment after rPFC damage and inhibition deficits. Suppression has been described as a response modulation strategy, for its main goal is to influence emotion-response tendencies when they have been already elicited (Gross, 1998; Gross & Thompson, 2007). This process is commonly achieved by inhibiting the behavioral emotional display of an already triggered emotional response (Gross & Levenson, 1997). Available studies on the neural basis of suppression are consistent with reports on inhibitory control, suggesting also the activation of rPFC areas (e.g. Goldin, McRae, Ramel, & Gross, 2008; Lévesque et al., 2003). Nevertheless, no study has explored yet whether suppression ability is compromised in patients with rPFC lesions.

This is the first study that brings together these separate fields of research, by exploring whether individuals with rPFC lesions, compared to neurologically healthy controls, are impaired in voluntarily manipulating the facial expression of emotional behavior, particularly in relation to intense positive emotions [amusement]. For this purpose, 10 individuals with rPFC lesions and 15 matched healthy controls [HC] were tested on a

response modulation task, where they had to watch emotional film clips and manipulate their facial emotional expressions according to three instructions: watch spontaneously, hide and amplify. Self-report measures were used to assess the effectiveness of the emotional induction procedure and facial behavior analysis (*FACS*; Ekman, Friesen, & Hager, 2002) was used to measure facial markers of positive emotions.

It was hypothesized that individuals with rPFC lesions would: 1) present similar levels of spontaneous experienced positive affect than HC [*Emotion reactivity Hypothesis*]; 2) present a decreased range of response modulation compared to HC [*Response modulation range Hypothesis*] and 3) show higher levels of facial behavior when instructed to inhibit [*Inhibition Hypothesis*]. It was also hypothesized: 4) that performance in the inhibition task would be associated to measures of inhibition [*Cognitive Control Hypothesis*]; and finally that 5) that inhibitory impairment would be associated with damage to motor inhibition areas previously described in the literature [*Lesion Hypothesis*][13].

4.1.2. Methods

4.1.2.1. Participants

Healthy controls and participants with acquired brain injury were referred by neurologists from the Bangor University School of Psychology. The main inclusion criterion for the neurological group was to have a focal brain lesion that involved the rPFC. The lesion could involve exclusively the rPFC or extend to the posterior areas of the RH [rPFC+right posterior]. Several exclusion criteria were considered, such as time since injury [no less than 6 months] and language ability [no moderate or severe language impairment]. Clinical details and aetiology of the neurological sample are presented in Table 1.

[13] The results of this last hypothesis are not presented in this paper, for the lesion analysis is currently in progress

The overall sample involved a total of 25 participants, including individuals with rPFC lesions [*rPFC*, n = 10, female = 6, male = 4] and healthy controls [*HC*, n = 15, female = 9, male = 6]. Both groups were matched in age [**rPFC**, *M* = 61.78, *SD* = 12.64; **HC**, *M* = 62.80, SD = 4.12] and years of education [**rPFC**, *M* = 14, *SD* = 1.51; **HC**, *M* = 13.93, SD = 1.75]. The average number of months since the injury in the neurological group was 61.80 [SD = 40.37].

Age/Sex	*Aetiology*	*Months since onset*	*Location*
57F	MCA stroke	84	Right Prefrontal, insula
50M	MCA and ACA stroke	20	Right Prefrontal
73F	MCA stroke	97	Right Prefrontal, Frontal Eye Field
45M	ACoA SAH	70	Right Prefrontal
74M	MCA Stroke	20	Right ventro-lateral prefrontal cortex, basal ganglia
65M	MCA Stroke	65	Right Prefrontal, insula, Right TPJ
46F	MCA Stroke	114	Right Prefrontal, insula
66F	MCA Stroke	120	Right PFC, insula, middle and superior temporal gyrus, TPJ
78F	MCA stroke	13	Right Prefrontal insula
68F	MCA stroke	24	Right Prefrontal and parietal

Table 1. Clinical details and aetiology in the neurological group

4.1.2.2. Procedure

Eligible participants were seen twice. Assessment across two sessions was useful to avoid the impact of fatigue on the neurological group. During the first session the goal of the experiment was explained to participants and consent was obtained [see Appendix 2]. Measures of overall cognition function were also collected. In the second session participants completed a response modulation task and measures of executive function were obtained. At the end of this session participants were debriefed.

4.1.2.3. Instruments

Overall Cognitive Assessment: The *Mini Mental State Examination* (Rovner & Folstein, 1987), *Telephone search and telephone search dual task* (*TEA*, Robertson, Ward, Ridgeway, & Nimmo-Smith, 1994), *Token Test* (De Renzi & Faglioni, 1978), *Logic Memory* (Weschler, 1987), *Rey-Osterrieth Figure* (Stern, Singer, Duke, & Singer, 1994) and the *Frontal Assessment Battery* (Dubois, Slachevsky, Litvan, & Pillon, 2000) were used in order to obtain an overall profile of cognitive function.

Executive Functions Assessment: A set of neuropsychological tasks was used to obtain a profile of executive function. Digits Forward and Backward (Weschler, 1981), Verbal Fluency (Delis, Kaplan, & Kramer, 2001) and Similarities (Weschler, 1981). Measures of inhibition included conflicting instructions (Stuss & Benson, 1986) inhibitory control (Drewe, 1975) and environmental autonomy (Lhermitte, Pillon, & Serdaru, 1986).

Emotional Symptomatology and Suppression usage: In order to assess the presence of symptomatology, the Hospital Anxiety and Depression scale (HADS, Zigmond & Snaith, 1983), a self-report questionnaire, was employed. The HADS has been shown to be sensitive tool in assessing depression and anxiety symptoms in acquired brain injury population (Dawkins, Cloherty, Gracey & Evans, 2006). To calculate the use of reappraisal in daily life, the Emotion Regulation Questionnaire (Gross and John, 2003) was applied.

Emotional Experience Self-Report Emotional experience during the Response Modulation task [see below] was assessed using a self-report questionnaire adapted from the PANAS-X (Watson & Clark, 1994). Following a previous study (Salas et al., 2011), 12 emotional words were selected from the discrete emotion scales of the PANAS-X. A 5-point likert scale [very slightly or not at all, a little, moderately, quite a bit, and extremely] was used to rate the each word. Four emotional words were selected for the target emotion: *amusement* [cheerful,

delighted, joyful, happy, and energetic]. Six words related to negative affect were also considered: sad, lonely, downhearted, alone, blue, scared and angry.

Facial Imitation task: In order to determine participants' capacity to voluntarily generate facial expressions, a facial imitation task was adapted from Simmons, Ellgring and Smith (2003). Participants were asked to imitate facial movements of the researcher, which correspond to Action Units described in the Facial Action Coding System (Ekman et al., 2002). Each Action Unit was accompanied by a visual prompt from the examiner and a verbal instruction, in order to facilitate its understanding: AU 4 (frown), AU 9 (wrinkle your nose), AU 10 (rise your upper lip), AU 14 (make dimples), AU 20 (stretch your lips downward), AU 24 (press your lips together), AU 26 (drop your jaw), AU 1 + 2 (raise your eyebrows), AU 12 + 6 (do a big smile and wrinkle your eyes), AU 5 (make your eyes look bigger by raising your eyelids), AU 15 (pull the corners of your mouth down), AU 17 (move your chin boss up). If participants were not able to imitate the Action Unit immediately, a mirror was used in order to compare their facial expression with the researcher's.

*Response modulation task:*This task is an adaptation from a previous study on response modulation in dementia (Henry, Rendell, Scicluna, Jackson, & Phillips, 2009), in which subjects are asked to watch short video clips and manipulate their facial expression according to three different instructions: watch spontaneously, suppress or amplify. The spontaneous condition was always performed first. Then, inhibition and amplification conditions were alternated for each participant, this to avoid order effects. Based on the available evidence on emotion elicitation using film clips (Gross & Levenson, 1995; Hewig, Hagemann, Seifert, Naumann, & Bartussek, 2005; Rottenberg, Ray, & Gross, 2007), three validated clips were selected to induce amusement (*When Harry met Sally* [155s], *Robin Williams Live* [205s], *Whose line is it, Anyway?* [211s]). On the same premises, three short clips of 60 seconds each

were generated from *Alaska's Wild Denali* (Rottenberg et al., 2007) and used as neutral stimuli[14].

Participants sat in front of a 16" laptop in a quiet room. Headphones were used to avoid possible distractions. A video-recording camera was placed two meters in front of the subjects. At the beginning of the task participants were told that they would watch a series of clips from movies and they would have to follow some instructions during each clip. Then participants watched the amusement-inducing clips. Neutral clips were viewed before each emotional clip. A self-report questionnaire of experienced emotion, adapted from the PANAS-X (Watson & Clark, 1994), was administered immediately after viewing emotional clip [see Fig 1].

Fig 1. Response Modulation Task. Three amusement inducing clips were presented to participants under three different conditions [spontaneous, hide/amplify, amplify/hide]. Before each emotional clip a neutral clip was shown, and after each emotional clip a questionnaire to capture emotional experience during the clip was administered.

[14] The original experiment elicited not only joy but also sadness, using the same procedure described above. Such data is not reported here because the behavioural markers of sadness [Action Units] that have been reported previously in the literature were not found consistently for the neurological group or the healthy control group. Such failure to elicit the behavioural aspect of the emotion impedes any conclusions regarding the voluntary manipulation of such responses.

The instructions for each manipulation were also adapted from Henry et al. (2009). Before each neutral clip the following instruction was offered: *"now try to relax and clean your mind of any thoughts while you watch the following clip"*. In the spontaneous condition participants received the following instruction: *"please watch the following clip spontaneously, as if you were watching TV at home"*. In the suppress condition participants were told: *"this time I will ask you to hide any feelings that you might have while watching the following clip. As if later someone looking at the recording of this moment would not have a clue regarding how you were feeling. In other words, use a poker face"*. Finally, in the amplification condition participants were asked: *"this time I will ask you to amplify, to express as much as you can any feelings that you might have while watching the following clip. As if later someone looking at the recording would clearly understand how you were feeling*". After the instruction was given participants were asked to repeat it in order to confirm that they have understood the command. The task was not initiated until participants were able to describe what they were supposed to do. After each emotional clip participants were asked to recall the instruction given.

4.1.3. Data Analysis

4.1.3.1. Data reduction

In order to measure facial expressive behavior, the Facial Action Coding System, (*FACS*; Ekman, Friesen & Hager, 2002) was used to code the appearance and frequency of facial changes while watching the emotional clips. Based on previous research (Ambadar, Cohn, & Ian, 2009; Coan & Gottman, 2007; Paul Ekman, 2003; Rottenberg, Kasch, Gross, & Gotlib, 2002; Soussignan, 2002) action unit [AU] 12+6 was selected as a proxy for intense positive affect [see Fig 2]. Several studies have associated AU 12+6, also called Duchenne smile to

the felt experience of positive affect and enjoyment (Ekman, Davidson, & Friesen, 1990; Ekman & Friesen, 1982; Frank, Ekman, & Friesen, 1993; Soussignan, 2002).

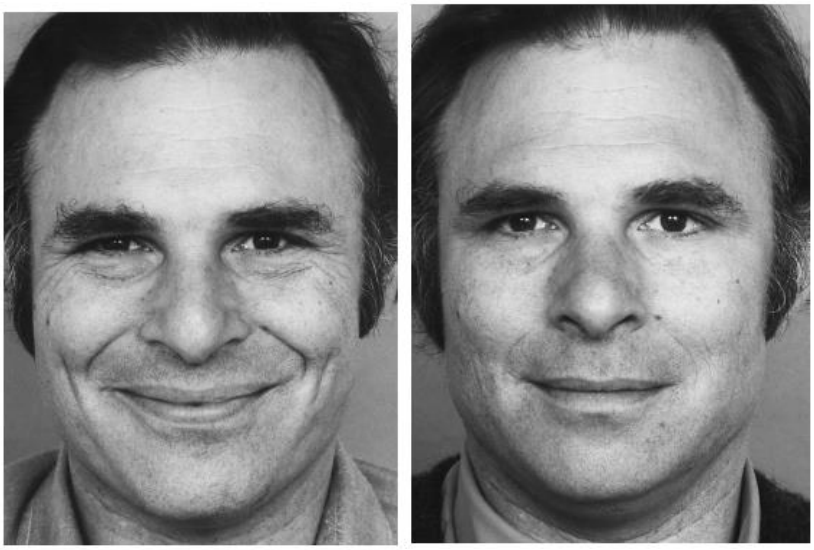

Fig 2. The Duchenne Smile. The Duchenne smile [left] involves activation of the zygomatic major [AU 12, oblique rising of the mouth corners] and the presence of orbicularis oculi activation [AU 6, crow's wrinkles in the eye region]. AU 12 alone [right], on the contrary, does not include AU 6.

The coding process involved two FACS certified coders who were blind to the participant's group, and to the video's condition [spontaneous, hide, amplify]. Coders watched each video and looked for the appearance of AU 12+6. There was a high agreement between coders in detecting the occurrence of AU 12+6 [$r_s = .80$].

4.1.3.2. Research hypotheses data analysis

Hypothesis 1 [*Emotion Reactivity*]: In order to respond Hypothesis 1, that individuals with rPFC lesions would present similar levels of experienced positive affect, the average scores of positive affect in each conditions were compared between groups. Because normality and independence of variance assumptions were not met, a non-parametric test for differences between two groups was used [Mann-Withney U].

Hypothesis 2 [*Response modulation range*]: To compare patients and controls in terms of their capacity to voluntarily manipulate positive emotion, a modulation range was calculated under the proposition that good regulators would be capable of decreasing the frequency of facial activity when instructed to suppress, and increasing it when instructed to amplify. In other words, good regulators would display a larger difference between suppression and amplification tasks. Following this logic, a *12+6 range score* was calculated by subtracting the number of AU 12+6 in the suppression condition to the number of AU 12+6 in the amplifying condition. To assess the normality distribution of this variable, histogram exploration and Kolmogorov-Smirnov test were performed for the entire sample and for each group separately. These analyses suggested that the distributions were relatively similar to a normal distribution [**Sample**: $D(25) = 1.10$, p = .18; **rPFC**: $D(10) = .70$, $p = .71$; **HC**: $D(15) = .80$, $p = .54$]. The 12+6 range score was then compared using independent sample *t*-tests reporting one-tailed probability values. When violations of homogeneity of variance assumptions were found, adjusted values were reported.

Hypothesis 3 [*Inhibition hypothesis*]: Because AU 12+6 reflects the experience and expression of *intense* positive emotion, no AU 12+6 was expected to be generated in the suppress condition. As a first step in the analysis of this variable, frequencies of appearance of AU 12+6 in the suppress condition were explored, both in neurological and healthy control groups. Then, all participants that had scores deviating from 0 were compared to the healthy control group using a modified *t*-test for small samples (Crawford & Howell, 1998). This *t*-test is a modification to the independent sample *t*-test which can be used to compare a single specimen with a small sample (less than 50), offering an estimate of the rarity or abnormality of the individual score. Neuropsychological studies of different sorts have used this approach

when addressing research questions with small samples (e.g. Bowles et al., 2007; Maguire, Nannery, & Spiers, 2006; Schiltz et al., 2006).

Hypothesis 4 [*Cognitive Inhibition*]: A non-parametric correlation test [Spearman, r_s] was used to assess the associations between neuropsychological measures of inhibition [conflicting instructions, response inhibition and environmental autonomy] and performance in the 'suppress' condition [number of AU 12+6].

4.1.4. Results

4.1.4.1. Cognitive and Emotional Assessment.

The assessment of emotional symptoms suggests that individuals with rPFC and healthy controls had scores below the cut-off point (> or = 8, Olssøn, Mykletun, & Dahl, 2005) in both the anxiety [rPFC: M = 6.70; SD = 4.73; HC: M = 4.70; SD = 3.10] and depression [rPFC: M = 6.40; SD = 4.62; HC: M = 2.67; SD = 1.79] subscales of the HADS. Nevertheless, the rPFC presented significantly higher scores in the depression subscale [$F(2,22) = 2.84$, $p = .017$] but not in the anxiety subscale [$F(2,22) = 1.25$, $p = .11$].

In addition, the neurological group did not differ from the healthy control group in the self-reported use of suppression, when measured by the ERQ [$F(2, 22) = .11$, $p = .45$]. A detailed description of the average scores of each group on the cognitive and emotional assessment can be found in table 2.

	Task	RH Group		HC Group	
		M	*SD*	*M*	*SD*
Overall Cognition	Minimental State Examination	28.11	1.45	29.00	1.25
	Sustained Attention (TEA)	7.88	1.64	11.00	1.57
	Divided Attention (TEA)	10.00	4.89	11.00	1.79
	Comprehensive Language (Token Test)	30.40	1.43	31.13	1.36
	Memory (WMS-R) Coding	12.50	3.20	13.67	3.43
	Memory (WMS-R) Free recall	14.30	3.65	16.33	3.77
	Memory (WMS-R) Recognition	13.22	1.39	13.86	1.52
	Executive Functions (FAB Total)	14.90	1.91	16.29	2.23
Cognitive Control	Working Memory (Digits, WAIS)	8.90	2.61	10.77	2.22
	Inhibition, Sensitivity to Interference	2.60	0.52	2.92	0.27
	Inhibition, Inhibitory control	2.40	0.96	2.38	1.04
	Inhibition, Environmental autonomy	2.90	0.31	3.00	0.00
	Verbal Fluency (DKEF-S) Letter	7.82	2.23	10.71	3.27
	Verbal Fluency (DKEF-S) Category	7.75	1.91	11.07	2.13
	Abstraction (Similarities, WAIS)	9.63	3.29	11.86	3.13
Emotional Functioning	Emotional Symptoms (HADS) Anxiety	6.70	4.73	4.70	3.10
	Emotional Symptoms (HADS) Depression	6.40	4.62	2.67	1.79
	Emotion Regulation (ERQ) Suppression	17.33	5.90	17.07	5.20

Table 2. Cognitive and Emotional performance of neurological groups and healthy controls

4.1.4.2. Voluntary generation of facial expressions

The rPFC group was equally able than the healthy control group to voluntarily generate facial expressions. Of particular interest for this study, all subjects in the neurological group obtained scores between 1 and 2 when generating AU 12+6[15]. The detail of both groups in the generation of a wider set of AU can be observed in Appendix 4.

4.1.4.3. Emotional Reactivity.

[15] Each AU was scored using a scale from 1 to 6 [1 = perfect intensity, long duration, no other AU; 2 = perfect intensity, short duration or another AU; 3 = prefer intensity, short duration and other AU; 4 = low intensity, short duration and other AU; 5 = movement does not correspond to AU; 6 = no movement].

Individuals with rPFC lesions did not differ from HC in the self-reported experience of positive affect during the three conditions. Average scores of positive affect across the three conditions are described in Table 3. Using a non-parametric comparison of two independent samples, it was observed that the average of positive experience affect was not significantly different between groups in all three conditions [**Suppress**: $U = 63$, $Z = -.68$, $p = .52$, r = -.14; *Mean Ranking*: rPFC = 11.8, HC= 13.8; **Spontaneous**: $U = 45$, $Z = -1.67$, $p = .10$, r = .33; *Mean Ranking*: rPFC = 10, HC= 15; **Amplify**: $U = 51$, $Z = -1.31$, $p = .21$, r = .26; *Mean Ranking*: rPFC = 14.57, HC= 10.65]. In consequence, the Emotional Reactivity Hypothesis is confirmed, thus suggesting that individuals with rPFC lesions experienced similar levels of positive emotions than controls.

Condition	rPFC			HC		
	M	*SE*	*Mean Rank*	*M*	*SE*	*Mean Rank*
Suppress	2.14	0.35	11.80	2.53	0.33	13.80
Spontaneous	2.36	0.44	10.00	3.28	0.23	15.00
Amplify	2.60	0.47	14.57	3.28	0.23	10.65

Table 3. PANAS scores of positive affect. Averages of self-reported positive affect, and mean ranks across the three conditions are presented in this table.

4.1.4.4. Response Modulation Range

Participants with rPFC damage were less able to voluntarily modulate the facial expression of positive affect. The comparison between the two groups showed that the HC group presented a larger response modulation range [$M = 13.8$, $SE = 3.53$] compared to rPFC group [$M = 3.7$, $SE = 1.2$]. This difference was statistically significant [$t(21,03) = 2.49$, $p = .01$, $r = .48$]. In consequence, the Modulation Range Hypothesis is confirmed, suggesting that individuals with rPFC lesions are less able to inhibit and amplify their facial expressions according to contextual demands.

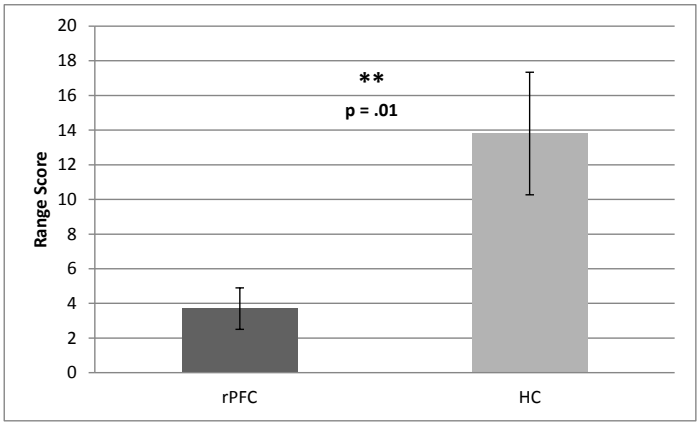

Fig 3. Comparison of Range Score between groups. The range score [12+6 suppress – 12+6 Amplify] was significantly smaller in subjects with rPFC lesions than controls.

4.1.4.5. Inhibition of positive affect. Healthy controls were highly able to suppress the expression of intense positive affect. Individuals with rPFC lesions, on the contrary, presented a mixed performance, with some successfully inhibiting while others presenting an abnormally high frequency of AU 12+6. Only 2 out of 15 [13.3%] healthy controls were not able to inhibit the expression of AU 12+6 in the suppress condition. These participants generated 1 and 2 AU 12+6 respectively. 4 out of 10 [40%] individuals with rPFC were not able to successfully inhibit the expression of AU 12+6. 3 out of the 4 participants[16] that failed inhibiting AU 12+6 had a higher score than the worst control [2 AU 12+6]. A t-test for small samples, (Crawford & Howell, 1998), suggested that the score of each of these four individuals with rPFC damage was significantly different than controls [for details see Table 4]. In consequence the Inhibition Hypothesis is only partially supported, suggesting that not all subjects with rPFC present with emotional inhibition impairment, but that an important proportion of the group does exhibit abnormal performance[17].

[16] These are the identification codes for the four participants that obtained abnormal performance on the suppression condition, and the number of AU 12+6 generated by each of them: ***45M***, 2 AU 12+6; ***46F***, 4 AU 12+6; **66F**, 5 AU 12+6; **65M**, 6 AU 12+6. The description of their lesion can be found in Table 1.

[17] This hypothesis is being further explored using lesion analysis techniques, correlating performance in the suppress task with damage to specific areas of the rPFC.

N° AU 12+6 Suppress	HC		rPFC				
	Frequency	*Percent*	*Frequency*	*Percent*	*T*	*p value*	*% population below case's score*
0	13	86.7	6	60			
1	1	6.7	0	0			
2	1	6.7	1	10	3.12	.003	99.61
3	0	0	0	0			
4	0	0	1	10	6.57	.001	99.99
5	0	0	1	10	8.29	.001	100
6	0	0	1	10	10.02	0.001	100

Table 4. Frequency of AU 12+6 in the suppress condition. The frequency of AU 12+6 on the suppression condition is compared here between healthy controls and individuals with rPFC lesions.

4.1.4.6. Suppression task and measures of cognitive inhibition.

The ability to suppress the emotional expression of positive affect was associated with only one measure of inhibition: inhibitory control. Using non-parametric correlations [Spearman, r_s], an inverse relationship was found between inhibitory control and suppression ability [r_s = -.36, p = .01]. No associations were found between the suppression condition and sensitivity to interference or environmental autonomy. These data appears to support the Cognitive Inhibition Hypothesis, suggesting that the capacity to withhold a prepotent response [inhibitory control] is involved in the ability to suppress the expression of emotional facial displays.

This data is interesting in view that suppression usage, as measured by the Emotion Regulation Questionnaire subscale, is positively associated to inhibitory control [r_s = .43, p =.02]. In other words, it suggests that both, behavior [suppression task] and frequency of use [suppression subscale ERQ] are related to response inhibition.

4.1.4.7 Results Summary

In sum, data from this study appears to partially support the idea that the rPFC is associated to ER, particularly to the use of one ER strategy: suppression. Individuals with rPFC lesions are less able to voluntarily manipulate their emotional facial expressions according to context demands. In other words, compared to controls, they struggle inhibiting and amplifying the expression of intense positive affect. Nevertheless, this impairment is not present in all

individuals with rPFC lesions. In fact, while some of them have abnormal performances, others appear to fall within the normal range. Data from the PANAS and the imitation task suggest that this impairment cannot be explained by an inability to trigger, and experience positive emotions, or a difficulty voluntarily generating positive facial displays [AU 12+6].

Finally, these results also offer supporting evidence to the relationship between an ER strategy [suppression] and inhibitory control, for behavior in the suppression task and suppression frequency of use are both associated to the capacity to withhold prepotent responses.

4.1.5 Discussion

There is an important body of literature that suggests that emotion dysregulation is frequent after rPFC lesions. However, one of the main limitations of this line of research has been the lack of an adequate theoretical framework for ER. This study is the first to explore ER impairment after rPFC using a well-known model of ER, the Process Model (Gross & Thompson, 2007). Furthermore, this study is unique in that it tests whether response modulation, or the capacity to voluntarily manipulate [suppress and amplify] the expression of emotional experience, is compromised after rPFC damage.

Results from this study are consistent with previous literature on emotion reactivity after brain injury, suggesting that the experience of emotion [in this case positive emotion] is largely preserved after cortical damage [for a detailed discussion on this point see section 3.2] (e.g. Feinstein, 2013; Merker, 2007; Solms & Panksepp, 2012). Furthermore, findings from this study appear to support previous claims on the role of the right hemisphere, and particularly the rPFC cortex, in emotion regulation (Borod, 2000; Stuss & Alexander, 1999; Stuss & Alexander, 2000; Turnbull, Evans, & Owen, 2004).

A novel finding of this study is that individuals with rPFC lesions are less able than controls to voluntarily manipulate the facial emotional expressions. It could be argued that such impairment does not reflect a regulatory defect, but instead, the inability to produce facial expressions. There are two reasons against this interpretation. The first one is that, in this study, individuals with rPFC were equally able than controls *posing* facial expressions [see imitation task results]. The second argument is that the literature on *posed* facial expression impairment after right hemisphere is not conclusive. For example, from the nine studies review by Borod et al. (2002) only four reported impairment of posed expression in right hemisphere patients.

Another possible explanation to this finding is that individuals with rPFC present an impairment producing *spontaneous* facial expressions. In fact, the same review from Borod et al. (2002) reported that eight out of thirteen studies described an impairment spontaneously generating facial expressions after right hemisphere damage. This interpretation of the data seems unlikely when the performance of individuals with rPFC lesions in this study is considered. Data from the spontaneous condition here, where participants were not asked to manipulate their facial expressions, is here informative. It was observed that, even though the rPFC group produced less AU 12+6 than controls [rPFC: $M = 3.60$, $SD = 5.37$; $HC = 7.40$, $SD = 9.2$], this difference was not significant with a medium effect size [$T(2, 23) = -1.75$, $p = .12$, $d = .50$]. This result suggests that the spontaneous facial expression of emotions is not drastically reduced in patients with rPFC, and that there is a wide variability even between controls.

This study offers novel data regarding the relationship between suppression, inhibitory control and rPFC damage. Firstly, it suggests that suppression impairment after rPFC lesions is not a homogenous deficit, with some subjects performing equally as controls, and other presenting remarkably abnormal performance. A possible explanation of this

finding is that the extension of the damage, and the involvement of key cortical structures, may play a critical role on generating a deficit on suppression. For example, as reviewed in the introduction, it has been suggested that the right inferior frontal gyrus, or BA 47, is particularly important in implementing motor inhibitory control. The question of which areas of the rPFC are related to the performance on the suppression condition of this experiment is still a work in progress. However, it is interesting to note that three out of the four subjects with abnormal performance on this task present large fronto-posterior lesions with insula involvement.

In the last couple of years, a handful of studies have explored the relationship between cognitive control and the suppression of emotional experience. So far, the only cognitive control process that has been found to be associated to the suppression of emotional experience is verbal fluency (Goodkind, Gyurak, McCarthy, Miller, & Levenson, 2010; Gyurak et al., 2009). It is intriguing that associations between suppression and inhibition have not been found, especially considering that suppression involves the process of inhibiting the behavioral emotional display of an already triggered emotional response (Gross & Levenson, 1997). This study is the first one to offer supporting evidence to this theoretical assumption, by reporting inverse associations between the capacity to withhold prepotent responses [response inhibition] with the ability to suppress or hide the expression of feelings.

It is interesting that the studies addressing the role of cognitive control in suppression have used a Stroop task as an inhibition task (Gyurak et al., 2009; Gyurak, Goodkind, Kramer, Bruce, & Levenson, 2012). In this task participants are presented with color words [e.g. 'red'] that are either printed in congruent ink colors [e.g. the word red printed in red ink], or incongruent ink colors [e.g. the word red printed in blue ink]. Then, participants are asked to read the words and not the colors. The inhibitory effort demanded by this task is that verbal command [read the words and not the color] conflicts with sensory information [the

ink color of the word], so subjects must withhold the tendency to say the color [which is more automatic] in order to read the word. In this study we tested three tasks that assess different aspects of inhibition. One of them, referred in this study as *sensitivity to interference* task, follows the same logic of the Stroop task, for it requires participants to obey verbal commands and refrain from what they see [e.g. when I tap once, you tap twice]. This task did not present significant associations to the suppression task, thus replicating the above mentioned studies. The task that did exhibit significant relationship was slightly different, and is more related to go-no go paradigms. In this paradigm, subjects must inhibit a response that was previously given [e.g. not tapping when the examiner taps twice]. It would be interesting to explore in the future what makes the response inhibition task more sensitive to suppression impairment than the sensitivity to interference task. Perhaps the use of several measures of inhibition in studies interested in suppression would allow understanding this point further.

The results presented in this study are of relevance for neuropsychological rehabilitation, for they suggest that some individuals with rPFC are particularly impaired hiding and amplifying their feelings according to contextual demands. These deficits may have a great impact on patients' social life, compromising the interpersonal regulation of emotions (Niven et al., 2009) and the attainment of intimate relationships (Bowen et al., 2010). For example, when experiencing joy at other's people misfortunate, a phenomenon also known as Schadenfreude (Smith et al., 996; van Dijk, Ouwerkerk, Wesseling, & van Koningsbruggen, 2011), people may need to inhibit their initial expression of joy, in order to show concern, or sympathy. In this case, inhibition of facial expression would allow suppressing of the prepotent feeling, and thus tuning to the negative emotions experienced by the other. In a similar way, the inability to amplify positive emotions when witnessing someone else's accomplishment, also known as Synhedonia (Royzman & Rozin, 2006), may also have interpersonal detrimental effects. In this case, the amplification of joy, would allow

connecting to others, thus triggering a spiral of positive affect and increasing mutual well-being (Gable, Reis, Impett, & Asher, 2004). In the future it would be interesting to describe in detail how these patterns of suppression impairment may impact patients' quality of life.

4.2. Reappraisal generation after acquired brain damage: the role of laterality and cognitive control.

4.2.1. Introduction

Emotion dysregulation has been long recognized as a common impairment after focal and diffuse brain damage, compromising emotional adjustment and social functioning (Abreu et al., 2009; Bechara, 2004; McDonald et al., 2010; Obonsawin et al., 2007; Tate, 1999). However, little is known about the mechanisms that underlie such impairment. The process model of emotion regulation (Gross, 1998; Gross & Thompson, 2007) suggests the existence of a set of five mechanisms, called strategies, that people commonly use to modulate how they feel. Recently, it has been proposed that these strategies may be selectively impaired in groups of patients with damage to discrete brain areas (Beer & Lombardo, 2007).

Reappraisal is an emotion regulation [ER] strategy of special interest to understand emotion dysregulation after brain injury. It refers to the capacity to modify the meaning of a situation in order to manipulate its emotional impact (Gross, 1998; Gross & Thompson, 2007), this by re-interpreting the event in less negative, or more positive, terms (McRae, Ciesielski, & Gross, 2011; Ochsner & Gross, 2007). In view that brain damage, and particularly frontal lesions, impair different components of discursive thinking (Goldstein & Scherer, 1941; Goldstein, 1936; Gomez Beldarrain, Garcia-Monco, Astigarraga, Gonzalez, & Grafman, 2005; Luria, 1966), it is likely that reappraisal generation may be compromised as well.

From a neuropsychological point of view, it has been suggested that reappraisal depends on capacities like *set shifting* [to inhibit the current negative appraisal and generate a new one] and *language* [to generate a narrative or history to tell oneself] (Mcrae, Jacobs, Ray, John, & Gross, 2011). These abilities are frequently compromised after left prefrontal

lesions (Alexander, Stuss, Picton, Shallice, & Gillingham, 2007; Coelho, Lê, Mozeiko, Krueger, & Grafman, 2012; Geva et al., 2011; Luria, 1959; Luria, 1966; Mecklinger, Von Cramon, Springer, & Matthes-von Cramon, 1999; Morin, 2009; Robinson, Shallice, Bozzali, & Cipolotti, 2010).

Neuroimaging studies on reappraisal appear to support the relevance of the left hemisphere. It has been reported that reappraisal is closely associated with the activation of cognitive control and language areas, particularly in the left PFC (Goldin, McRae, Ramel, & Gross, 2008; Green & Malhi, 2006; Kalisch, 2009; Kim, Cornwell, & Kim, 2011; Ochsner & Gross, 2005; Ochsner et al., 2004; Ochsner, Bunge, Gross, & Gabrieli, 2002; Vanderhasselt, Baeken, Van Schuerbeek, Luypaert, & De Raedt, 2012). To our knowledge there is only one case study that has explored how damage to the left hemisphere [a fronto-parietal lesion], and impairment to cognitive control and language areas, is related to reappraisal generation difficulties (Salas et al., 2013). However, no group study has tested whether unilateral lesions to the left hemisphere have a larger impact on reappraisal generation compared to right hemisphere lesions.

Finally, a couple of studies have begun to unpack the cognitive control processes associated with reappraisal in non-brain injured subjects. It has been suggested that working memory, response inhibition, abstract reasoning and verbal fluency may be necessary capacities to reappraise (Mcrae et al., 2011). Nevertheless, evidence has only supported a relationship between working memory and reappraisal ability (Mcrae et al., 2011; Schmeichel, Volokhov, & Demaree, 2008). The study of people with brain damage may contribute considerably to this line of research, by exploring whether impairment in a specific neuropsychological capacity [e.g. verbal fluency] is related or not to reappraisal generation ability.

This is the first study to experimentally test whether lesion laterality has an effect on reappraisal generation. In addition, this study is also the first to explore the relationship between cognitive control impairment and the capacity to generate reappraisals. For this purpose, participants with unilateral lesions to the left and right hemisphere, and matched healthy controls, were tested on a reappraisal generation task. In order to obtain a profile of neuropsychological impairment, a set of cognitive tasks was administered. Additionally, measures of emotional symptomatology and emotion regulation were collected.

Based on the available literature, this article explored two hypothesis: (1) that participants with left hemisphere [LH] lesions will present a more marked impairment generating reappraisal compared to subjects with right hemisphere lesions and controls [*LH Reappraisal Hypothesis*]; (2) that cognitive control abilities, such as, response inhibition, working memory, verbal fluency and abstraction will be negatively associated to reappraisal difficulty and positively associated to reappraisal productivity [*Cognitive Control Hypothesis*].

4.2.2. Methods

4.2.2.1. Participants

Healthy controls and participants with unilateral lesions and healthy controls were referred by neurologists from the Bangor University School of Psychology. The main inclusion criterion for the neurological group was to have a focal brain lesion. Several exclusion criteria were considered, such as time since injury [no less than 6 months] and language ability [no moderate or severe language impairment].

The overall sample involved a total of 30 participants, including individuals with left hemisphere damage [*LH*, $n = 8$, female = 4, male = 4], right hemisphere damage [*RH*, $n = 8$, female = 5, male = 3] and healthy controls [*HC*, $n = 14$, female = 9, male = 5]. The three

groups were matched in age [LH: *M* = 62.83, *SD* = 9.98; RH: *M* = 57.43, *SD* = 13.34; HC: *M* = 62.86, *SD* = 4.28] and education [LH: *M* = 12.83, *SD* = 1.3; RH: *M* = 14.14, *SD* = 1.3; HC: *M* = 14, *SD* = 1.8]. One subject from the LH group died before completing the assessment. Both neurological groups were matched in relation to the average of months since the injury [LH: *M* = 69.16, *SD* = 49.19; RH: *M* = 63.22, *SD* = 44.35]. For details on lesion location and aetiology see Table 1.

Lesion Laterality	*Age/Sex*	*Aetiology*	*Months since onset*	*Location*
Left Hemisphere	52F	MCA stroke	104	Left Prefrontal
	76F	MCA stroke	25	Left Prefrontal, Insula
	64F	MCA stroke	129	Left temporo-parietal
	76M	MCA stroke	65	Left temporo-parietal
	49M	Herpes encephalitis	24	Left hippocampus, amygdala, insula
	59M	MCA stroke	48	Left temporo-parietal
	57M	MCA stroke	126	Left temporo-parietal
	72M	MCA stroke	72	left fronto-parietal
Right Hemisphere	57F	MCA stroke	84	Right Prefrontal, insula
	50M	MCA and ACA stroke	20	Right Prefrontal
	45M	ACoA SAH	70	Right Prefrontal
	74M	MCA Stroke	20	Right ventro-lateral prefrontal cortex, basal ganglia
	46F	MCA stroke	114	Right Prefrontal, insula
	66F	MCA stroke	120	Right parietal
	78F	MCA stroke	13	Right Prefrontal insula
	68F	MCA stroke	24	Right Prefrontal and parietal

ACoA = anterior communicating artery aneurism; SAH = subarachnoid haemorrhage, MCA = middle cerebral artery.ACA = Anterior cerebral artery.

Table 1. Clinical details of Left Hemisphere and Right Hemisphere groups

4.2.2.2. Procedure

Healthy controls and participants with brain injury were tested at Bangor University. In cases where participants with acquired brain injury had mobility problems or could not travel, they were seen at home. Eligible participants were seen twice. Assessment across two sessions

was useful to avoid the impact of fatigue on the neurological group. During the first session the general goal of the research was explained and consent was obtained [see Appendix 2]. Measures of overall cognition were also collected and participants completed the reappraisal task. In the second session the response modulation task was carried out and measures of executive function were obtained. Finally participants were debriefed upon completion.

4.2.2.3. Instruments

Overall Cognitive Assessment: The *Mini Mental State Examination* (Rovner & Folstein, 1987), *Telephone Search* [*TEA*, Robertson, Ward, Ridgeway, & Nimmo-Smith, 1994], *Token Test* (De Renzi & Faglioni, 1978), *Logic Memory* (Weschler, 1987), *Rey-Osterrieth Figure* (Stern et al., 1994) and the *Frontal Assessment Battery* (Dubois et al., 2000) were used in order to obtain an overall profile of cognitive function.

Cognitive Control Assessment: A set of neuropsychological tasks was used to obtain a profile of several cognitive control capacities: working memory [*Digits Forward and Backward*, Weschler, 1981], abstraction [*Similarities*, Weschler, 1981], verbal fluency [*D-KEFS*, Delis, Kaplan, & Kramer, 2001] and inhibition [*conflicting instructions*, Stuss & Benson, 1986; *inhibitory control*, Drewe, 1975; *environmental autonomy*, Lhermitte, Pillon, & Serdaru, 1986].

Emotional Functioning Assessment: In order to assess the presence of symptomatology, the Hospital Anxiety and Depression scale (HADS, Zigmond & Snaith, 1983), a self-report questionnaire, was employed. The HADS has been shown to be a sensitive tool to in assessing depression and anxiety symptoms in the acquired brain injury population (Dawkins, Cloherty, Gracey & Evans, 2006). To assess the use of reappraisal in daily life, the *Emotion*

Regulation Questionnaire (Gross and John, 2003) was applied. This instrument has not been used in acquired brain injury population before.

4.2.2.4. Reappraisal Generation Task

This task is adapted from several studies on reappraisal ability, and is described in detail in Salas and colleagues (2013) [see section 5.2]. Thirteen pictures[18] were selected from the International Affective Picture System (*IAPS*; Lang, Bradley, & Cuthbert, 2008), depicting negative events of different sorts. These pictures were selected to cover the wide range of possible negative scenarios that people commonly face (death, natural disasters, accidents, illness, violence, etc.) [See Fig 2]. The pictures were displayed in a 14' computer screen.

At the beginning of the task participants were trained to generate reappraisals, using three practice IAPS pictures. The task was introduced as follows: "Sometimes people try to feel better by looking on the bright side of things. You will watch pictures of negative events and will be asked to think aloud about the positive side of these situations. Try to be fast and say as many positive sides you can think of".

In order to avoid memory bias for the neurological group, on the screen, above each picture, the task instruction was summarized: "Think aloud about the positive side of this situation. Try to be quick". Participants were informed that their answers would be timed, and recorded verbatim. They were also informed that the aim of the task was to produce as many positive reinterpretations as possible. If they were not able to generate a correct reappraisal for the first picture, several reappraisal examples were offered (e.g. Car Crash [9903]: 'when looking at this picture some people say that help is on the way' or 'is not as bad as it looks'). The same procedure was followed with the second and third trial picture. Both, the

[18] Pictures' theme and IAPS number: Riot (2691), Shoplifting (2745), Sick baby (3350), Burnt building (9471), Funeral (2799), Tornado (5971), Pollution (9341), Flood (9926), Car crash (9903), Graveyard (9220). The picture set had an overall negative valence (M = 3.01; SD = 0.8) according to the IAPS database

neurological group and the non-brain injured group were able to offer adequate reappraisals by the end of the trial run.

Fig 1. IAPS pictures used in the Reappraisal Generation Task. From upper left corner [clockwise]: Sick Baby (3350); Funeral (2799); Car crash (9903); Pollution (9341).

4.2.3. Data Analysis

In order to respond *Hypothesis 1* [that participants with left hemisphere lesions will present a more marked impairment generating reappraisals compared to subjects with right hemisphere lesions and controls], two variables were generated, following a similar analysis to that used by Salas et al. (2011). *Reappraisal difficulty* was obtained averaging the number of seconds that each subject required to offer the first reappraisal in each picture. *Reappraisal productivity* was measured by averaging the number of spontaneous reappraisal [with no prompting] generated during each picture. The two variables were compared between groups.

Because normality and independence of variance assumptions were not met, a non-parametric test for differences between 3 or more groups (Kruskal-Wallis) was used. If the overall model showed significant differences, two planned comparisons were tested using Mann-Whitney test [Healthy controls v/s Brain injury patients (Left + Right Hemisphere); Left Hemisphere v/s Right Hemisphere].

Hypothesis 2 [that cognitive control abilities would be negatively associated to Reappraisal Difficulty, and positively related to Reappraisal Productivity] was addressed using correlational methods. As a first step, bivariate correlations were used to explore independent associations between Reappraisal Difficulty and Reappraisal Productivity with cognitive control abilities. As a second step, a multiple linear regression model was tested. From the 7 initial tasks considered to assess the four cognitive processes, three were dropped. Two of them, related to inhibition [sensitivity to interference and environmental autonomy], did not show enough variability [most of the participants performed with the highest score]. The two fluidity tasks [Letter Fluency and Category Fluency] showed a medium correlation between them [$r = .5$, $p = .01$]. In consequence, category fluidity was discarded in order to reduce the number of parameters and avoid possible multicollinearity. The decision to preserve letter fluency was based on evidence suggesting that, compared to category fluency, it is more strongly associated to cognitive control abilities (Henry & Crawford, 2004).

4.2.4. Results

4.2.4.1. Cognitive and Emotional functioning

The assessment of emotional symptoms suggests that all three groups had subclinical levels of anxiety [LH: $M = 3.67$; RH: $M = 5$; HC: $M = 4.64$] and depression [LH: $M = 4.33$; RH: $M = 4.75$; HC: $M = 2.71$]. In addition, none of the neurological groups differed from healthy

controls in the self-reported use of reappraisal [$F(2, 22) = 2.38$, $p = .11$]. A detailed description of the average scores of each group on the cognitive and emotional assessment can be found in table 2.

	Task	LH Group		RH Group		HC Group	
		M	*SD*	*M*	*SD*	*M*	*SD*
Overall Cognition	Minimental State Examination	25.67	2.88	27.83	1.60	29.17	1.34
	Sustained Attention (TEA)	7.67	1.52	7.83	1.83	11.00	1.78
	Divided Attention (TEA)	9.00	3.60	11.16	5.11	11.22	1.73
	Comprehensive Language (Token Test)	26.00	9.53	31.17	1.17	31.33	0.78
	Memory (WMS-R) Coding	6.67	2.50	13.00	3.84	14.58	2.97
	Memory (WMS-R) Free recall	8.67	3.05	14.00	4.33	16.83	3.51
	Memory (WMS-R) Recognition	13.67	0.57	13.33	1.36	13.75	1.60
	Executive Functions (FAB Total)	13.33	4.04	15.50	0.23	17.08	0.99
Cognitive Control	Working Memory (Digits, WAIS)	10.00	4.58	9.17	2.56	10.67	2.31
	Inhibition, Sensitivity to Interference	2.83	0.41	2.63	0.51	2.91	0.30
	Inhibition, Inhibitory control	2.00	1.92	2.50	0.92	2.58	0.79
	Inhibition, Environmental autonomy	3.00	0.00	2.83	0.40	3.00	0.00
	Verbal Fluency (DKEF-S) Letter	7.67	5.13	7.50	2.43	10.71	3.27
	Verbal Fluency (DKEF-S) Category	4.33	0.57	8.00	2.09	10.73	2.19
	Abstraction (Similarities, WAIS)	6.67	2.08	10.00	3.69	11.64	3.50
Emotional Functioning	Emotional Symptoms (HADS) Anxiety	3.33	0.57	6.17	4.04	4.27	3.52
	Emotional Symptoms (HADS) Depression	4.00	6.92	6.00	5.40	2.55	1.86
	Emotion Regulation (ERQ) Reappraisal	23.67	14.50	33.00	4.19	32.00	5.07

Table 2. Cognitive and Emotional performance of neurological groups and healthy controls

4.2.4.2. Reappraisal difficulty and productivity

Reappraisal Difficulty: the average amount of seconds used to generate a first reappraisal did not differ between the LH and RH groups. However, significant differences were found between patients with brain injury in general [LH+RH], and HC. A Kruskal-Wallis non-parametric test was used to compare the amount of seconds used by each group. It was observed that the number of seconds was significantly different between groups [$H(2) = 10.77$, $p = .002$; *Mean Ranking*: LH = 21.71, RH = 18.25, HC = 9.79]. According to planned comparisons, it was found that such difference was only significant, and had a large effect size, between the HC group and the Brain Injury group (LH+RH) [$U = 32$, $Z = -3.19$, $p <$

.001, r = .59], but not between LH and RH groups [U = 25, Z = -.24, p = .43, r = -.04]. In conclusion, data does not support the *LH Reappraisal Hypothesis* as patients with LH and RH damage were equally slower in generating reappraisals.

Reappraisal Productivity: the average number of reappraisals generated in each picture did not differ between groups, suggesting that individuals with LH and RH lesions, and healthy controls, are equally productive when time is not considered. A Kruskal-Wallis non-parametric test was used to compare the total reappraisals generated by each group. It was observed that the number of reappraisals was not significantly different between groups [H(2) = 3.51, p = .175; *Mean Ranking*: LH = 14.07, RH = 10.88, HC = 17.82]. A detailed description of the groups' performance can be found in Table 3. A graphic summary of the results can also be found in Fig 2.

	LH Group		RH Group		BI Group		HC	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Reappraisal Difficulty	43.3	25.88	33.58	17.6	38.13**	21.62	16.7	7.07
Reappraisal Productivity	1.82	0.59	1.61	0.95	1.71	0.78	2.22	0.79

** = p < .001

Table 3. Descriptive statistics of reappraisal generation and productivity, for both neurological groups and healthy controls.

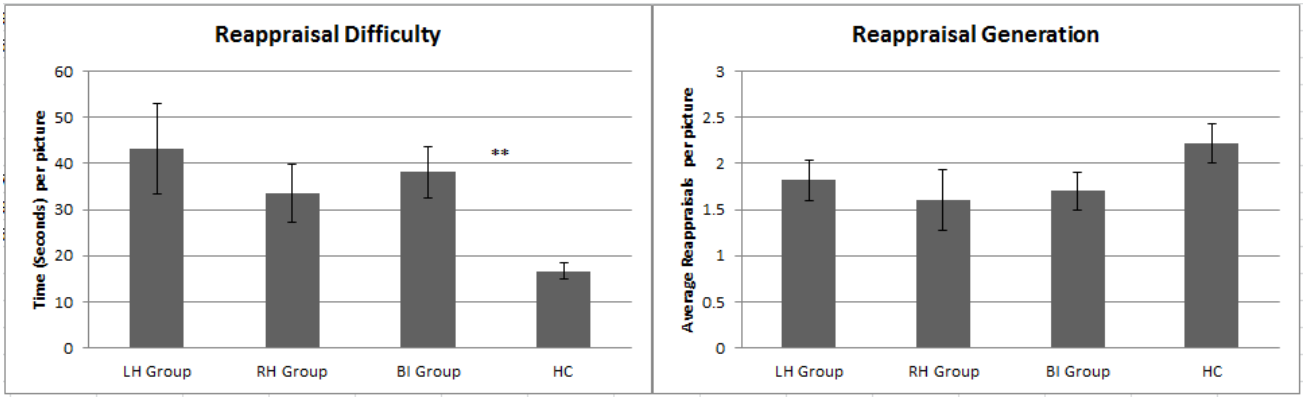

Fig 2. Laterality and Reappraisal Generation. The figures describe differences between neurological groups and controls in reappraisal *difficulty* and *generation*. Patients with LH and RH lesions are equally slow generating the first reappraisal [left side figure]. However, they do not differ from controls in the average number of reappraisal produced in each picture [right side figure]. ** = p < .001

4.1.4.3. Cognitive Control Hypothesis

Reappraisal Difficulty. From the four cognitive control processes considered, only two [inhibition and verbal fluency] were significantly associated with reappraisal difficulty. The final model tested used Reappraisal Difficulty as a dependent variable and Working Memory, Abstraction, Verbal Fluency and Inhibition as predictors. Multicollinearity, homoscedasticity and independency of errors assumptions were met. The model was significant [$F(4,24) = 7.70$, $p < .001$] and explained a 49% of the variance. The final model is presented in Table 4. From the four predictors, Inhibition and Verbal Fluency were the only two significantly associated to Reappraisal Difficulty. By looking at the standard coefficient it is possible to conclude that Inhibition showed the strongest predictive value. In relation to the other two independent variables, abstraction was marginally related and working memory showed no relationship.

Reappraisal Productivity. The same model was tested for Reappraisal productivity. Contrary to the previous model, this one did not explain significant amounts of variance [adjusted $R^2 = .022$, $F(4,24) = 1.16$, $p = .35$]

	Unstandardized Coefficients		Standardized Coefficients		
	B	Std. Error	Beta	*t*	Sig.
(Constant)	87.12	12.59		6.92	0.00
WORKING MEMORY (Digits)	0.26	0.93	0.04	0.28	0.78
ABSTRACTION (Similarities)	-1.64	0.87	-0.27	-1.88	0.07
VERBAL FLUENCY (Letter)	-2.28	0.9	-0.37	-2.53	0.02*
INHIBITION (Inhibitory Control)	-10.27	3.07	-0.47	-3.35	0.003*

a Dependent Variable: REAPPRAISAL DIFICULTY

Table 4. Multiple linear model for Reappraisal Difficulty. The table describes the percentage of variance explained by each cognitive control process. Inhibition and Verbal Fluency are the only two significant variables. Abstraction presents a marginal relationship with reappraisal difficulty.

4.2.4.4. Results summary

In sum, data from this study does not support the *LH Laterality Hypothesis*, thus challenging the assumption that LH lesions generate a more marked impairment in the quick generation of a first reappraisal [reappraisal difficulty] or the total number of reappraisals that can be produced [reappraisal productivity]. Nevertheless, the results of this study suggest that having a brain injury, with disregard of the laterality of the lesion, does have an impact on reappraisal difficulty. This pattern is not observed in relation to reappraisal productivity, where participants with brain injury reach similar levels than controls. In conclusion, participants with brain injury are slower, but equally productive than people with no brain injury.

The results of this study appear to support the *Cognitive Control Hypothesis*, suggesting that the velocity to generate a first reappraisal, which is compromised in brain-injured patients in general, is associated with two cognitive control abilities. Thus, inhibition and verbal fluency present both negative relationships to reappraisal difficulty. In relation to abstraction, it only exhibits a marginally significant negative association. Working memory does not appear to be associated to the amount of time required to reappraise. Interestingly, no cognitive control variable appears to be associated with reappraisal productivity.

4.2.5. Discussion

In the last decade there has been a growing body of literature exploring the neuroanatomical basis of reappraisal through the use of neuroimaging. These studies have suggested that reappraisal tasks activate a set of areas in the left hemisphere that are commonly associated with language and cognitive control. The main goal of this study was to investigate such a hypothesis in two ways. Firstly, to test whether participants with unilateral lesions to the LH were more impaired than participants with RH lesions, and neurologically healthy controls

with a reappraisal generation task. Secondly, to explore which cognitive control abilities are associated with reappraisal generation.

In relation to the LH reappraisal hypothesis, it was observed that participants with lesions to the LH and RH needed considerably more time than controls to generate a first reappraisal for each picture. In other words, it appears that RH damage generates similar levels of impairment than LH damage. However, both neurological groups, and healthy controls, were equally productive when time was not considered. As for the cognitive control hypothesis, a negative relationship was observed between inhibition, verbal fluency and reappraisal difficulty. In contrast, no cognitive control ability was associated to reappraisal productivity. The relevance of these findings is discussed in relation to the neuroanatomical basis of reappraisal, the relationship between cognitive control and reappraisal, and the implications of reappraisal for neuropsychological rehabilitation.

4.2.5.1. The neuroanatomical basis of reappraisal generation

This is the first study that uses a brain injury population to explore the neural basis of reappraisal. More specifically, this study has focused on reappraisal generation, or the capacity to *produce* positive reinterpretations of negative events. This is an important point to clarify, because most behavioral and neuroimaging studies tend to focus on reappraisal ability, or the capacity to modulate emotion through the use of reappraisal. Such emphasis on *ability* is sensible, considering that people with healthy brains probably have an intact capacity to *generate* reappraisals. However, a robust set of evidence on the neuropsychological consequences of brain injury suggests that the generation and manipulation of thoughts can be compromised by lesions to diverse brain areas (Goldstein & Scherer, 1941; Gomez Beldarrain et al., 2005; Luria, 1966; McGrath, 1991). If reappraisal

relies on the flexible use of thinking (Ochsner & Gross, 2004), then it is likely that people with brain injury may struggle using such strategy to modulate how they feel.

Data from this study suggests that lesions to the LH, which has been long related to language functions (Corballis, Badzakova-Trajkov, & Häberling, 2012; Frost et al., 1999), and verbally mediated cognitive control (Gruber & Goschke, 2004; Henry & Crawford, 2004; Stephan et al., 2003), do not impair reappraisal difficulty more markedly than lesions to the RH. This is interesting, since reappraisal has been mostly considered as a language- mediated ER strategy (Gross & Thompson, 2007; Mcrae et al., 2011). A possible explanation for this negative finding may well be that lesions to the RH compromise reappraisal generation via impairment of other non-language mechanisms, such as attention. If individuals with RH lesions present marked attentional difficulties (Anderson, Jacobs, & Harvey, 2005; Leclercq, 2002; Molenberghs et al., 2009; Robertson, Tegnér, Tham, Lo, & Nimmo-Smith, 1995; Shallice, Stuss, Alexander, Picton, & Derkzen, 2008; Wilkins, Shallice, & McCarthy, 1987), and reappraisal involves the manipulation of attentional focus (Ochsner et al., 2002), it is likely that such an impairment may also have an impact on reappraisal generation. However, this explanation remains speculative and requires scientific exploration.

4.2.5.2. Neuropsychological components of reappraisal generation

A different perspective to this problem is to consider the correlational data between reappraisal generation and cognitive control abilities. This approach might offer important information on the neuropsychological mechanisms that sustain reappraisal generation.

The available literature on cognitive control and reappraisal is scarce and focuses exclusively on reappraisal ability. So far, it has suggested that reappraisal ability is positively related to working memory (Mcrae et al., 2011; Schmeichel et al., 2008) and marginally

related to abstraction (Mcrae et al., 2011). Interestingly, no associations have been reported for abilities that reflect key theoretical aspects of reappraisal, such as inhibition [e.g. to detach from the negative emotional experience], or verbal ability [e.g. to generate alternative interpretations].

This study suggests that an inhibitory control task (Drewe, 1975), where participants must withhold a response that was given to the same stimulus before [e.g. tap once when the examiner taps twice], can significantly predict the amount of time that it would take to generate a first reappraisal. In other words, if an individual decreases his score by one point in the inhibition task (Dubois et al., 2000), his response time to generate a first reappraisal will increase by 10.27 seconds. This finding is the first to support the view that inhibition is a key ability in decreasing the salience of automatic negative appraisals (Mcrae et al., 2011), for when inhibition is impaired, reappraisal generation requires considerably more time. There is one case study that has reported this relationship in detail (Salas et al., 2013) [see Chapter 5], describing how inhibition impairment after a left prefrontal lesion produced a remarkable difficulty in spontaneously generating reappraisals. Finally, it is important to note that this data is consistent with models of executive function that propose behavioral inhibition as a requisite to any goal directed behavior (Barkley, 1997, 2001) [see Fig 3].

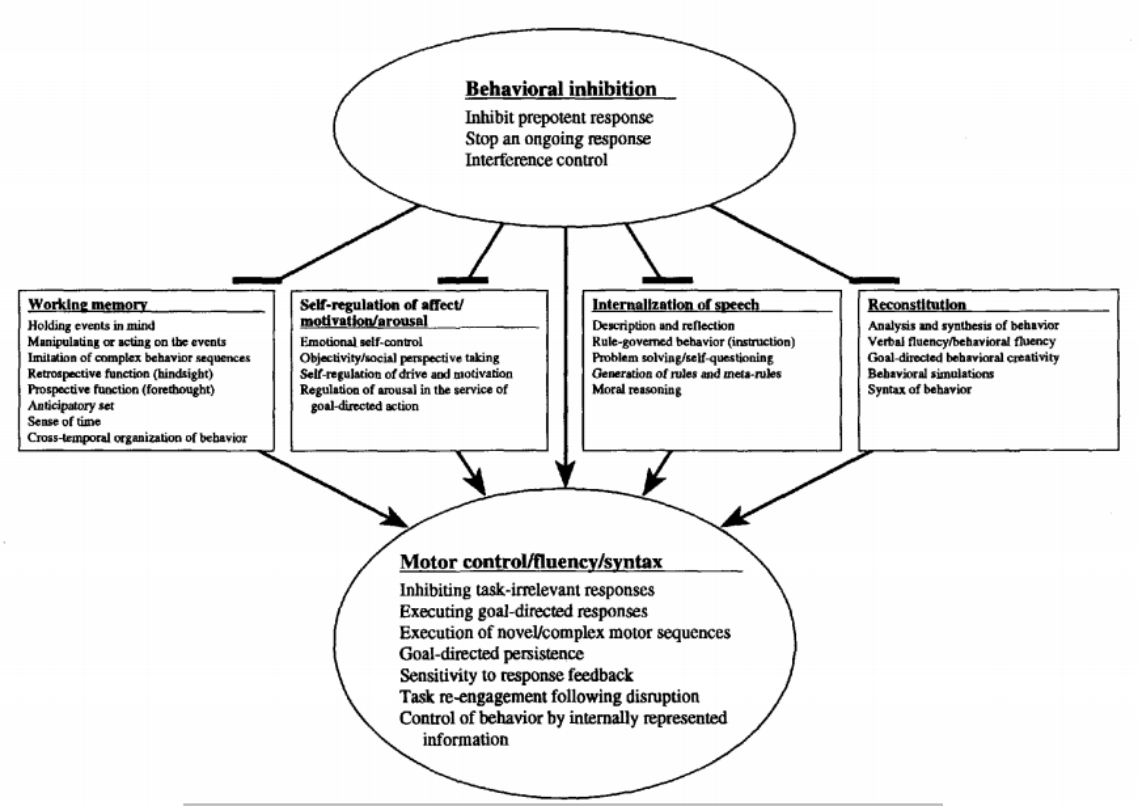

Fig 3. Behavioral Inhibition and Executive Functions. The figure describes the relationship between behavioral inhibition and related executive functions (from Barkley, 1997).

The results obtained in this experiment also suggest that performance on a verbal fluency task (Delis et al., 2001), in which participants are expected to generate as many words that begin with a letter in one minute, also predict the amount of time used to generate a first reappraisal [this, to a less extent than inhibition]. Verbal fluency is an interesting measure in understanding reappraisal, for it presents high associations with verbal ability (Crawford, Moore, & Cameron, 1992; Crawford, Obonsawin, & Bremner, 1993; Miller, 1984; Sollberger et al., 2010), a core component of reappraisal (Mcrae et al., 2011). In addition, verbal fluency also appears to recruit other processes that are key to reappraisal, such as the retrieval and recall of information, self- monitoring and inhibition (Henry & Crawford, 2004; Perret, 1974).

Based on these findings, and considering previous studies on reappraisal ability (Mcrae et al., 2011; Schmeichel et al., 2008), a two-stage reappraisal model is here proposed.

In a first stage [*reappraisal generation*] inhibition is required to disengage from the automatic negative appraisal. If inhibition is successful, alternative interpretations can be generated -this moderated by verbal fluency. In a second phase [*reappraisal ability*], working memory ability allows the retention of the previously generated interpretations. This, in order to reevaluate the meaning of the emotional stimulus, which still remains in the focus of attention (Kanske, Heissler, Schönfelder, Bongers, & Wessa, 2010), and engage with the most adequate interpretation (Mcrae et al., 2011). The successful engagement with a positive reinterpretation will down-regulate the initial levels of negative emotion.

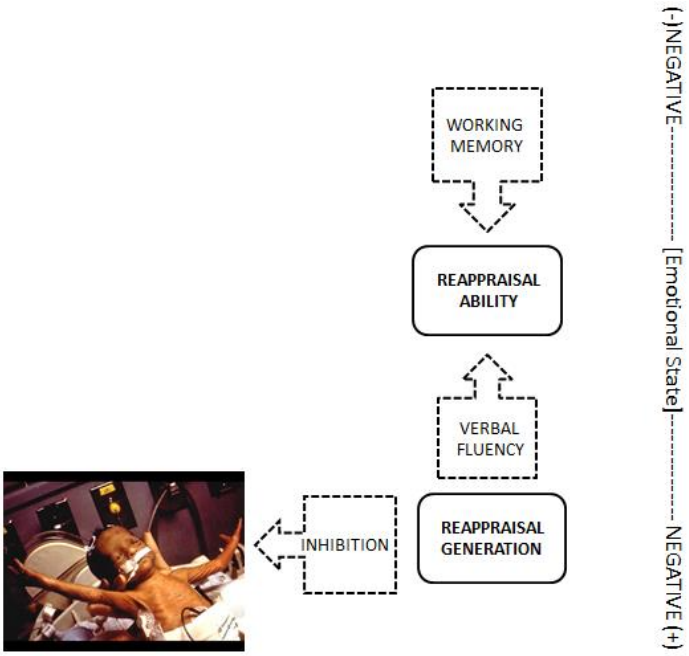

Fig. 4 A two-stage process of reappraisal. The figure describes a two phase model of reappraisal. On a first phase reappraisals are generated. On the second stage reappraisals are mentally manipulated, so they can compete with the negative stimulus and down-regulate emotion. Inhibition and verbal fluency are key abilities for reappraisal generation, while working memory is central for reappraisal ability.

4.2.5.3 Reappraisal generation impairment and neuropsychological rehabilitation

This study offers interesting information regarding how brain damage may compromise the use of emotion regulation strategies. It suggests that brain injury, without regard for lesion laterality, impairs the capacity to quickly generate positive reinterpretations of negative events. However, when longer periods of time are provided, patients are able to produce reappraisals as well as the controls did. These findings are relevant for two reasons. Firstly, they offer supporting evidence to the idea that brain-injured patients in general are vulnerable to experiencing emotion dysregulation (Salas, 2012), particularly when confronted with emotional situations where they need to react quickly. This is consistent with studies describing that focal and non-focal brain damage compromises the capacity to rapidly react to environmental demands (Gerritsen, Berg, Deelman, Visser-Keizer, & Meyboom-de Jong, 2003; Goldstein, 1995; Mathias & Wheaton, 2007; Winkens, Van Heugten, Fasotti, Duits, & Wade, 2006), as well as to reports suggesting that negative emotional events can compromise cognitive abilities (e.g. Demanet, Liefooghe, & Verbruggen, 2011).

Secondly, they suggest that a psychological capacity for reappraisal can be externally modulated, by manipulating environmental demands [time]. This is in line with evidence proposing that neuropsychological impairments are not stable, but can be modulated by physical or interpersonal context (Bowen, Yeates, & Palmer, 2010, for a review). For example, it has been reported that the use of prompts facilitates the process of disengagement from negative visual material, increasing dramatically the capacity to generate positive reinterpretations (Salas et al., 2013). This evidence appears to support the theoretical proposition that extrinsic forms of emotion regulation, such as affective and cognitive engagement (Niven et al., 2009), can be used to compensate for intrinsic emotion regulation difficulties [reappraisal] (Freed, 2002; Salas, 2012).

4.2.6. Conclusion

In recent years, scientists have become increasingly interested in the neural basis, and neuropsychological foundations, of emotion regulation. This study has contributed to such literature by exploring the role of each hemisphere, and the relevance of several cognitive control abilities, in reappraisal generation. In addition, and perhaps most importantly, this study has addressed these issues using a well-known ER paradigm on a sample of individuals with acquired brain injury. Such an approach opens important possibilities to ER research, complementing behavioral studies with healthy controls and neuroimaging studies.

Chapter 5: Single Case Studies

5.1. Concrete Behaviour and Reappraisal Deficits after a Left Frontal Stroke: A Case Study[19]

5.1.1. Abstract

Concrete behaviour, the inability to disengage from immediate experience in order to manipulate ideas and thoughts, has long been understood to be a common problem after frontal lobe lesions. However, there has been little consideration of the impact that concreteness may have on emotional functioning, specifically in the use of thinking to manipulate emotional responses. One widely-studied emotion regulation strategy is reappraisal, which depends on several frontal lobe related cognitive control processes. While there have been numerous neuroimaging findings on reappraisal, no study has used brain injured patients to investigate this issue. The present case study is the first to describe the capacity to generate reappraisals in a patient (Mrs M), whose behaviour became concrete after a left prefrontal stroke. Using a picture-based reappraisal paradigm, her performance was compared to non-concrete brain-lesioned patients, and neurologically-healthy controls. Although Mrs M showed relatively preserved overall cognitive function, she was completely unable to spontaneously generate reappraisals. In striking contrast, once external support was offered, in the form of prompts, her capacity to reappraise dramatically improved. The results are analyzed in terms of three neuropsychological capacities – all compromised in Mrs M– previously proposed as reappraisal components: response inhibition, abstraction and verbal fluency. A number of implications for rehabilitation are discussed, including how the use of prompting may facilitate reappraisal capacity.

[19] Salas, Gross, Rafal, Viñas-Guasch & Turnbull (2013). Concrete Behaviour and Reappraisal Deficits After A Left Frontal Stroke:A Case Study. *Neuropsychological Rehabilitation*, 37-41,10.1080/09602011.2013.784709

> "Blue feels betrayed by the man who was once like a father to him, and when he finishes the letter he feels empty (…) I'm on my own, he thinks, there is no one to turn to anymore. This is followed by several hours of despondency and self-pity, with Blue thinking once or twice that maybe he'd be better off dead (…) But eventually he works out his way out of the gloom (…) By the time supper rolls around, he has even began to look on the bright side. It might be a good thing after all, he says to himself. It might be better to stand alone than to depend on anyone else. Blue thinks about this for a while; he is no longer an apprentice. There is no master above him anymore"
> Paul Auster, *The New York Trilogy.*

5.1.2. Introduction

Concrete behaviour, or concreteness, is often defined as a difficulty disengaging from immediate experience in order to manipulate ideas and thoughts (Fuster, 2008; Goldstein, 1936a, 1936b; Goldstein & Scheerer, 1941; Mesulam 2000, 2002; Salas, Vaughan, Shanker & Turnbull, 2013). Concrete behaviour is common after lesions to the frontal lobe (Fuster, 2008; Luria, 1966; Mesulam, 2000, 2002). It has long been associated with diffuse lesions, as in traumatic brain injury (Judd, 1999; Klonoff, 2010; Ogden, 1996), as well as more localized lesions in the left hemisphere (Goldstein, 1942), particularly to its dorsolateral surface (Fuster, 2008; Scott and Schoenberg, 2011).

In recent decades, there has been only a modest amount of research relating to the idea of concreteness, though the concept is still commonly used amongst rehabilitation professionals (see Salas, Vaughan, Shanker & Turnbull, 2013 for review). Such clinical value appears to derive from the fact that concreteness, while it clearly overlaps with concepts of executive function [see Table 1], offers clinical insights *beyond* the cognitive domain, for example, in relation to patients' transformations in emotional and interpersonal life (Hanfmann, Rickers-Ovsiankina & Goldstein, 1944).

Symptomatic features in concrete patient's performance *(Goldstein & Scheerer, 1941)*
Concreteness is characterized by difficulty:
detaching our ego from the outer world or from inner experience
assuming a mental set wilfully and consciously
accounting for acts to oneself and *verbalizing* the account
shifting reflectively from one aspect of the situation to another
holding in mind simultaneously various aspects
grasping the essential of a given whole; *breaking up* a given whole into parts, *isolating* and *synthesizing* them
abstracting common properties reflectively; *forming* hierarchies and concepts
planning ahead ideationally; *assuming* an attitude towards the 'mere possible', thinking and performing symbolically

Table 1. Goldstein's description of common deficits observed in patients with acquired concreteness

In modern neuropsychology, however, discussions of concreteness still view it predominantly as a *thinking* problem, as in concrete thinking (Cicerone and Giacino, 1992; Judd, 1999; Ponsford, Sloan and Snow, 1995), without reference to the impact that concreteness may have on emotional life, and to emotional adjustment to brain injury (for a review see Salas, Vaughan, Shanker and Turnbull, 2013). Nevertheless, one particularly important implication of concrete behaviour may be its influence on emotion regulation (Gross, 1998), especially on one widely studied type of emotion regulation strategy, reappraisal, or the ability to change a situation's *meaning* in a way that alters its emotional impact (Gross and Thompson, 2007).

Reappraisal involves changing the way that we *think* about a situation, usually reframing the meaning of an aversive event in less negative or more positive terms (McRae, Ciesielki and Gross, 2011; Ochsner and Gross, 2007). To achieve this goal, several cognitive control processes are thought to be required, including: (1) keeping the automatic appraisal of a negative situation to mind (*working memory*), (2) decreasing the salience of that appraisal (*inhibition*), (3) generating alternative interpretations (*working memory manipulation* and *verbal ability*), (4) identifying and engaging the interpretation that is most appropriate (*set shifting*), (5) keeping the new appraisal in mind (*working memory maintenance*), and (6) keeping track of the success of regulation (*monitoring*) (McRae, Jacobs, Ray, John & Gross, 2011).

This study suggests the possibility that patients with *marked* concrete behaviour may experience difficulties generating reinterpretations of negative events in order to alter its emotional impact – with substantial consequences for the emotional lives of such patients and their families. Indeed, most of the processes thought to be implicated in reappraisal (McRae, Jacobs, Ray, John & Gross, 2011) are compromised in concrete patients [see Table 1], who often struggle to voluntarily *disengage* from immediate experience and to *manipulate* ideas

and thoughts (Beldarrain et al., 2005; Fuster, 2008; Goldstein, 1936a; 1936b, Goldstein & Scheerer, 1941; Mesulam, 2000; 2002). Furthermore, reappraisal appears to engage left prefrontal cortical regions (Ochsner & Gross, 2007) which, when lesioned, are often associated with concrete behaviour (Goldstein, 1942).

Surprisingly, despite exponential growth in the emotion regulation field (Gross, 2007), and growing understanding of emotion regulation problems after brain damage (Abreau et al., 2009; Cattran, Oddy, Wood and Moir, 2011; McDonald et al., 2010; Obonsawin et al., 2007; Rochat, Ammann, Anoni and Linden, 2009; Tate, 1993), there is an absence of brain injury studies using emotion regulation paradigms (Beer and Lombardo, 2007). This is unfortunate because the investigation of patients showing concrete behaviour could offer especial insight into the underlying neurological and neuropsychological basis of reappraisal. While there have been several neuroimaging findings on this topic (Goldin et al., 2008; Green and Mahli, 2006; Ochsner, Bunge, Gross, Gabrieli, 2002; Ochsner et al., 2004; Kalisch, 2009; Vanderhasselt et al., 2012), no study has investigated how damage to specific brain areas, and neuropsychological functions, might compromise the capacity to *generate* reappraisals. This is a promising avenue of research to complement the correlational findings offered by neuroimaging studies.

The present study describes one's patient capacity to generate reappraisals (Mrs M) after her behaviour became markedly concrete, following a stroke that involved the left dorsolateral PFC (dlPFC). Employing a set of neuropsychological tasks, her profile of concreteness is characterized, as well as her overall level of cognitive function. Using a picture-based reappraisal paradigm, Mrs M's performance is analysed in detail, and compared to the profile of patients with no concrete thinking and neurologically healthy controls. Three main issues are considered: a) *Reappraisal difficulty*: Whether Mrs M would present with larger reaction times when generating a reappraisal, compared to non-concrete

brain injured patients and controls; b) *Reappraisal generation*: Whether Mrs M would generate fewer reappraisals for each picture, compared to non-concrete brain injured patients and controls; c) *Reappraisal monitoring*: Whether Mrs M would require more external prompts to reappraise, compared to patients without concrete thinking and controls. Finally, in addition to the experimental assessment of reappraisal, information regarding Mrs M's awareness of her capacity to use reappraisal is reported.

Mrs M is a 72 year old right handed woman, married and mother of two sons. She finished secondary school but did not pursue any further education. She is Welsh-English speaking and at the time of the stroke was living with her husband. Thirty-five months prior to this study Mrs M suffered an ischemic stroke that involved the left middle cerebral artery territory, damaging parts of the left inferior and frontal gyri, BA 44 and 46 including Broca's area, the frontal operculum and anterior insula (see Fig. 1). She had no history of psychiatric problems. Medically, the only remarkable antecedent was a history of hypertension and elevated cholesterol.

She presented with loss of muscle tone to the right side of her face, having also had some weakness of the right leg, which had recovered within a few days. In the acute stages she was reported to have had some difficulty in expressive language, taking the form of a Broca-type aphasia. She is described in her language report as being able to offer information about herself, but needed prompting and cueing, with trouble naming objects and with occasional perseverations and misnaming. She could read single words, follow simple commands, and understand simple questions. Four days later her comprehension was substantially improved, and at two months later, she was reported to have had a mild expressive aphasia, which had in many respects recovered by the time of the study, though she still had some problems with correct articulation and repetition, and a mild word-finding difficulty. Her comprehension was intact, and she was able to communicate effectively. The

neurological examination did not reveal any other deficit apart from the facial weakness, and there were no features of apraxia. According to Mrs M's husband, the more important observable changes after the injury were difficulties in planning and decision making. He also reported a moderate increase in depression and anxiety.

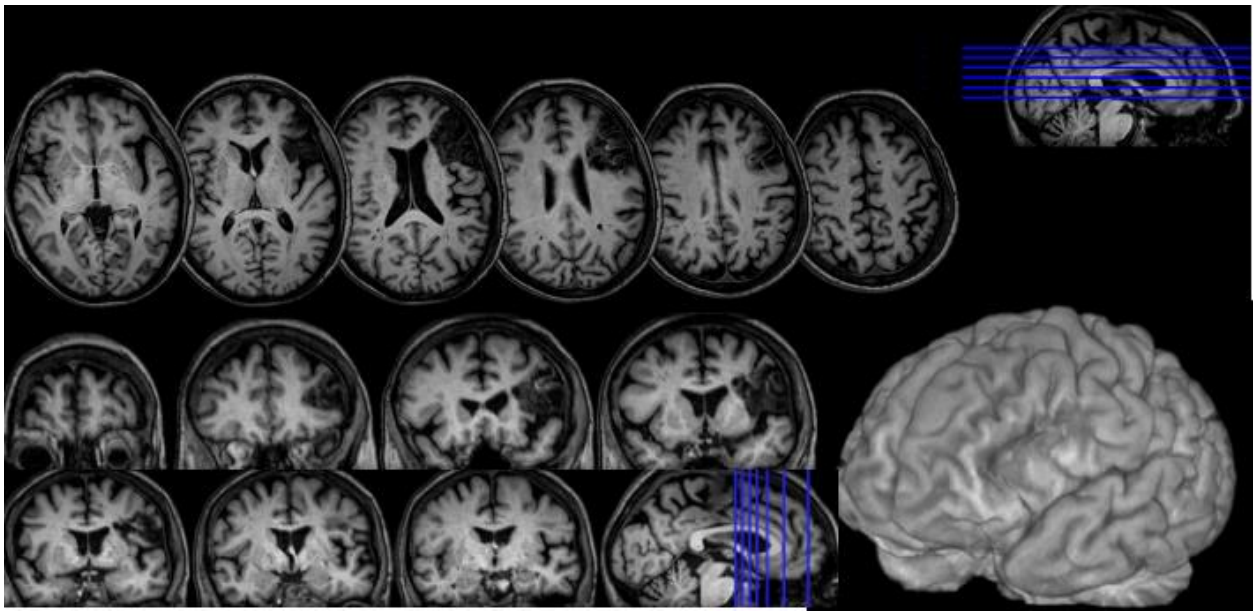

Figure 1. *Mrs M's lesion*. T-weighted MRI images obtained on a Philips Achieva 3T scanner with .7mm isometeric voxel resolution. Top: axial sections; Bottom left: Coronal sections; Bottom right: Volume rendered reconstruction viewed from the left front. The lesion involves the middle and inferior frontal gyri of the left hemisphere, including Broca's area, the frontal operculum and parts of the anterior insula.

5.1.3. Methods

5.1.3.1. Participants

Mrs M's performance on the cognitive assessment and reappraisal task was compared to a group of 12 brain injured patients and 12 healthy controls. Mrs M's age was not significantly different from the control group [$t(11) = 1.55$, $p = .07$, $M = 63.57$, $SD = 5.2$] or brain injured group [$t(11) = 0.76$, $p = .23$, $M = 63.27$, $SD = 10.99$]. Similarly, Mrs M's number of years of education was not significantly different from the control group [$t(11) = -1.07$, $p = .15$, $M = 14$, $SD = 1.79$] or the brain injury group [$t(11) = -1.68$, $p = .06$, $M = 14.27$, $SD = 1.34$]. Only one subject from the entire sample, a patient with brain injury, was left handed. Clinical details of Mrs M and the neurological control patients are shown in Table 2, including aetiology, duration of illness and lesion localisation.

5.1.3.2. Procedure

Healthy controls, and participants with acquired brain injury, were referred by neurologists from the Bangor University School of Psychology. Exclusion criteria for the neurological group were duration of less than 6 months since their brain injury, and moderate to severe language impairment. Eligible participants were seen twice, in order to collect data for the present study. Assessment across two sessions was useful to avoid the impact of fatigue on the neurological group. During the first session the main goal of the research was explained, and measures of overall cognitive and emotional function were collected. The second session started with the administration of the reappraisal task, after which participants completed several measures of concreteness.

Age/Sex	*Aetiology*	*Years since onset*	*Location*
72F	*MCA stroke*	*2*	*Left prefrontal, insula*
51M	MCA and ACA stroke	2	R prefrontal
74F	Falx meningioma resection	9	Bilateral medial frontal
46M	ACoA SAH	6	Right prefrontal
61F	Lobar haemorrhage from venous sinus thrombosis	14	Right prefrontal, small haemorrhage in the left frontal eye field
75M	Right MCA stroke	2	Right ventro-lateral prefrontal cortex, basal ganglia
63M	MCA stroke	3	Rigt prefrontal and orbitofrontal
65F	MCA stroke	9	Left prefrontal
66F	MCA stroke	11	Right parietal
78M	MCA stroke	5	Left parietal
49M	Herpes encephalitis	1	Left hippocampus, amygdala, insula
59F	MCA stroke	4	Right prefrontal, insula
72/M	MCA and ACA stroke	6	Left prefrontal and parietal

ACoA = anterior communicating artery aneurism; SAH = subarachnoid haemorrhage, MCA = middle cerebral artery. ACA = Anterior cerebral artery.

Table 2. Clinical details of Mrs M (in italics) and non-concrete brain injury control patients

5.1.3.3. Measures

Concrete Behaviour Assessment

Considering the set of cognitive abilities compromised by concrete behaviour [see Table 1], and the neuropsychological components of reappraisal proposed by McRae and colleagues (2011), five tasks were used as proxies for different aspects of concreteness that are of relevance to reappraisal:

a) *Inhibition*: A Conflicting Instructions (Stuss and Benson, 1986) and Inhibitory Control (Drewe, 1975) task were used to assess subjects' "detaching" capacity (Goldstein & Scheerer, 1941). In the *conflicting instructions* task participants must provide an opposite response to the examiner's alternating signals, following verbal command and withholding automatic responses based on visual input (e.g. tapping once when the examiner taps twice). In the *inhibitory control* task subjects must inhibit a response that was previously given to the same stimulus (e.g. no tapping when the examiner taps twice).

b) *Abstraction*: *Similarities* (*WAIS-R*; Wechsler, 1981) was used to assess subjects' ability to "abstract common properties reflectively and form hierarchic concepts" (Goldstein & Scheerer, 1941). In this task participants were asked to think in which way two items were alike (e.g. boat/car: "means of transport"), thus detaching from the most salient perceptual features (e.g. "both have motors"; "one has wheels the other does not").

c) *Verbal fluency*: *Verbal fluency* (*D-KEFS*; Delis, Kaplan and Kramer, 2001) was included to assesses individual's capacity to "assume a mental set" (Goldstein & Scheerer, 1941), by initiating a verbal response and retrieving specific information in accordance with a rule (e.g. say as many words you can think of that start with the letter 's', excluding people's or place's names). Of special relevance here, for

concrete thinking and reappraisal, is the subtask of category *switching*, which taps into the capacity to "shift reflectively from one aspect of a situation to another" (Goldstein & Scheerer, 1941). In this task subjects are asked to flexibly alternate between two different mental sets (furniture/fruits).

d) *Divided Attention*: *Telephone search* and *Telephone search dual task* (*TEA,* Robertson, Ward, Ridgeway and Nimmmo-Smith, 1994) were used to assess participants' ability to "hold in mind simultaneously various aspects of a situation" (Goldstein & Scheerer, 1941). In the first task subjects are asked to visually scan for target symbols in a telephone directory (e.g. □□, ++), while in the second one a simultaneous auditory task is added to the visual search (counting the number of tones played in a recording). A divided attention score is calculated by subtracting the amount of seconds used in the first task from the second one.

e) *Working Memory: Digits Forward and Backward* (*WAIS-R*, Wechsler, 1981) was used to assess participants' capacity to 'manipulate ideas and thoughts' (Goldstein, 1936a; 1936b, Goldstein & Scheerer, 1941). In the *digit forward* task participants listened to a series of single-digit numbers and repeat them in the same order. The number of digits in each series increased from two to nine. The *digit backward* task had the same structure, but the participant repeated the numbers in reverse order.

Overall Cognitive Assessment

The *Mini Mental State Examination* (Rovner and Folstein, 1987), *Token Test* (De Renzi and Faglioni, 1978), *Logic Memory* (*WMS-R*, Wechsler, 1987), *Rey-Osterrieth figure* (Stern et al., 1994) and *Frontal Assessment Battery* (Dubois, Slachevsky, Litvan and Pillon, 2000) were used in order to obtain an overall profile of cognitive function.

Emotional functioning

In order to assess the presence of symptomatology, the Hospital Anxiety and Depression scale (HADS, Zigmond & Snaith, 1983), a self-report questionnaire, was employed. The HADS has demonstrated to be a sensitive tool to assess depression and anxiety symptoms in acquired brain injury population (Dawkins, Cloherty, Gracey & Evans, 2006). The Iowa Scale of Personality Change, ISPC (Barrash et al., 1997; Barrash et al., 2011) was also applied. The ISPC is an informant questionnaire that offers a quantification of the magnitude of several affective, behavioural and social disturbances after brain damage, as well as the assessment of the extent to which there has been change from pre-morbid levels.

Reappraisal use frequency

To assess the use of reappraisal in daily life, the *Emotion Regulation Questionnaire* (Gross and John, 2003) was applied. This questionnaire differs from the reappraisal task used in this study in that captures people's *perception* of how frequently they use reappraisal to manage emotions, and not reappraisal *ability*.

Reappraisal Generation Task

The paradigm used in this experiment is adapted from several studies on reappraisal ability, where participants are exposed to negative emotional pictures, and asked to decrease their emotional response by reinterpreting the displayed situation in more positive terms (Kanske, Heissler, Schonfelder, Bongers & Wessa, 2010; Lieberman, Inakagi, Tabibnia & Crockett, 2011; McRae, Ciesielski & Gross, 2011; McRae, Jacobs, Ray, John & Gross, 2011; Ray, McRae, Ochsner & Gross, 2010; Urry, 2010). Thirteen pictures[20] were selected from the International Affective Picture System (IAPS, Lang, Bradley and Cuthbert, 2008), depicting negative events of different sorts. These pictures were chosen to cover the wide range of possible negative scenarios that people commonly face (death, natural disasters, accidents, illness, violence, etc.).

Before beginning the task, participants were trained to generate reappraisals, using three practice IAPS pictures. Instructions were simplified, in order to facilitate the patients' grasp of what reappraisal meant, using a model of previous research on reappraisal in children (Carthy, Horesh, Apter, Edge & Gross, 2010). For example, an image of a glass half full (or half empty) was used as a graphic metaphor to demonstrate the idea of reappraisal. The task was introduced as follows:

> *"Sometimes people try to feel better by looking on the bright side of things. You will watch pictures of negative events and will be asked to think aloud about the positive side of these situations. Try to be fast and say as many positive sides you can think of".*

[20] Pictures' theme and IAPS number: Riot (2691), Shoplifting (2745), Sick baby (3350), Burnt building (9471), Funeral (2799), Tornado (5971), Pollution (9341), Flood (9926), Car crash (9903), Graveyard (9220). The picture set had an overall negative valence ($M = 3.01$; $SD = 0.8$) according to the IAPS database.

The pictures were displayed in a 14' computer screen and participants were sat at no more than 40 cm from it. In order to avoid memory bias for the neurological group, above each picture the task instruction was summarized: "*Think aloud about the positive side of this situation. Try to be quick*". Participants were informed that their answers would be timed, and recorded verbatim. They were also informed that the aim of the task was to produce as many positive reinterpretations as possible. If they were not able to generate a correct reappraisal for the first picture, several reappraisal examples were offered (e.g. Car Crash [9903]: '*when looking at this picture some people say that help is on the way'* or '*is not as bad as it looks*'). The same procedure was followed with the second and third trial picture. Both, the neurological group and the non-brain injured group were able to offer adequate reappraisals by the end of the trial run.

Once the task started, each participant had a maximum of 120 seconds to offer the first reappraisal in each picture. In cases where no reappraisal was generated during the 120 seconds, the examiner prompted the participant by reading the instruction on the screen aloud: '*Mr X, could you please try to think now of a positive side of this situation'*. In cases when participants were *still* unable to generate a reappraisal, or commented that the only things they could think of were the negative elements of the picture, a more extended prompt was offered: *'Mr X, do not worry. This task is difficult for many people. The reason why it is hard is because you need to ignore the negative elements of the pictures, in order to think of a positive side. Could you try to do that one more time?'*.

5.1.4. Results

5.1.4.1. Concrete Behaviour Assessment

In relation to measures of concreteness, Mrs M's profile is characterized by marked difficulties in *inhibitory control* (3/6 max score), *abstraction* (*Similarities* scaled score[21] 5), *phonological fluency* (scaled score 2), *category fluency* (scaled score 4) and *set shifting* between categories (scaled score 4), *working memory* (*Digit Span* scaled score 6). However, unexpectedly, she presents an average performance in *divided attention* (scaled score 10). Mrs M is the *only* brain injured subject with a consistent low performance across concrete behaviour proxy measures (see Table 3). She has the lowest score in 4/7 tasks and the second worst score in 2/7 tasks. When the six proxy scores of concreteness are ranked and averaged, she is placed first. The two brain injured participants that follow her are positioned almost in third place, but none of them has low scores in abstraction (*similarities*), a cardinal feature of concrete behaviour according to Goldstein and Scheerer (1941). In sum, although some brain injured patients also present low performance on some of the proxy measures of concreteness, none of them is as consistently impaired as Mrs M.

5.1.4.2. Overall Cognitive Assessment

Mrs M presents a relatively preserved *overall* cognition, which is reflected by the mild level of cognitive impairment (24/30) detected in the *Mini Mental State Examination* (see Table 3 for comparison to control group and brain injury group). Her profile of cognitive deficits is characteristic of lesions to the left dorsolateral frontal cortex (Anderson and Tranel, 2002).

In language comprehension tasks (*Token Test*) she is below average (15/32), failing on items where the manipulation of three or more units of information is required. A related

[21] Scaled Scores are adjusted by age

difficulty can be observed in tasks where she is instructed to mentally manipulate information, such as the *100-7* (*MMSE*, score 3/5) or *digits backwards* (*WAIS-R*, maximum 2 items).

Her memory profile appears as characterized by impairment in coding and retrieving new information. In a *Logical Memory* task (*WMS-R*) her immediate recall was highly compromised

	Tasks	*Mrs M*	*BI (n = 12)*		*Controls (n = 12)*	
		score	*M*	*SD*	*M*	*SD*
Concreteness	Similarities	5	10.00*	2.71	11.86*	3.13
	Letter Fluency	2	8.33*	2.45	10.71*	3.26
	Category Fluency	4	7.56	2.46	11.07*	2.13
	Category Fluency Switching	4	7.44	2.60	12.43*	2.17
	Telephone Search	8	7.5	4.84	11.50*	1.95
	Telephone Search Dual Task	10	7.63	1.60	11.00	1.57
	Inhibition	3	4.20	1.32	5.50*	0.79
	Digit Span	6	9.18	2.13	10.77*	2.24
Overall Cognition	Mini Mental State Examination	24	26.64	0.69	29	0.38
	Token Test 20	15	30.73**	0.40	30.92**	0.41
	Ray-Osterrieth Copy	30	30.27	2.02	33.58	0.97
	Logical Memory Immediate Recall	4	12.27**	1.08	14.17**	1.05
	Logical Memory Delayed Recall	8	13.00*	1.31	16.42**	1.12
	Logical Memory Recognition	14	13.73	0.33	13.83	4.77
	Frontal Assessment Battery	9	14.64**	0.71	16.5**	0.57
	Reappraisal Score (ERQ)	38	29.36**	1.49	31.17**	1.62

BI: Brain Injury group. *P*-values for modified *t*-test comparing Mrs M to BI group and controls: * = $p < .05$; ** = $p < .001$

Table 3. Overall Cognitive Performance and Concrete Behaviour profile. Mrs M is the only brain injury subject who consistently presents the lowest scores in proxy tasks for concrete behaviour.

(4/15 units), but it improved considerably (12/15 units) after a second exposure. The free recall of information, after a delayed period of time, appears moderately compromised (8/15 units) improving significantly with cues (14/15 units). She did not present executive or constructive errors in the *Rey-Osterrieth Complex Figure* copy (30/36 units) and the delayed recall was within normal limits compared to controls (16/30 units).

Executive impairment was evident in the brief *Frontal Assessment Battery* (FAB). Although this is a bed side screening test for executive abilities, it has shown concurrent validity with more standardized executive function measures, such as the Stroop and Wisconsin Card Sorting Task (Cunha, 2010). In addition, significant correlations between the FAB scores and frontal lobe metabolism have also been reported (Guedj et al., 2008; Sarazin et al., 1998). Mrs M obtained a score of 9/18 in the FAB (12 is the cut off score for presence of frontal lobe deficits), where she failed on tasks of concept formation, verbal-fluency and inhibitory control. Her performance on tasks that assess motor programing, sensitivity to interference and environmental dependency was normal. These results are congruent with information obtained from the Iowa Scale of Personality Change (Barrash et al., 1997; Barrash et al., 2011), where the *executive dysfunction/decision making* subscale presented the highest average score of change ($\Delta = 1.67$), with a dramatic increase in lack of planning ($\Delta = 4$) and poor judgement ($\Delta = 4$) according to her husband.

5.1.4.3. Emotional functioning

Mrs M. does not refer significant levels of emotional distress according to the Hospital Anxiety and Depression Scale (Zigmond & Snaith, 1983). She reports sub-clinical levels of anxiety [score = 4], referring to occasions where she experiences frightened feelings, worrying thoughts, difficulties to relax and sudden feelings of panic. Mrs M does not report any sign of depression [score = 0]. Nevertheless, the Iowa Scales of Personality

Change (Barrash et al., 1997; Barrash et al., 2011) suggests the presence of post injury emotional disturbances. According to her husband, she has exhibited mild changes in the Distress Scale ($\Delta = 1.3$), with moderate increase in depression ($\Delta = 2$) and anxiety ($\Delta = 2$).

5.1.4.4. Reappraisal use frequency

In relation to the use of reappraisal in daily life, Mrs M reports using reappraisal more frequently than controls [$t(11) = 4.05$, $p < .01$] and non-concrete brain injured patients [$t(11) = 5.57$, $p < .001$]. This is extremely interesting in view of Mrs M's low performance on the reappraisal task, perhaps reflecting some type of dissociation between reappraisal awareness and reappraisal ability (see discussion section).

5.1.4.5. Reappraisal Performance: Quantitative Analyses

In order to answer our hypotheses regarding Mrs M's performance on the reappraisal task three variables were generated. *Reappraisal difficulty* was obtained averaging the number of seconds that each subject required to offer the first reappraisal in each picture. *Reappraisal generation* was measured as the average number of *spontaneous* and *prompted* reappraisal answers during the complete task. Finally *Reappraisal monitoring* was calculated by averaging the number of prompts that each subject needed throughout the complete task in order to reappraise. Results are summarized in Table 4.

	Mrs M	BI	Control	BI (n = 12)		Controls (n = 12)	
	Score	worst	worst	M	SD	M	SD
Reappraisal Difficulty	95.67	66.9	32.8	37.65*	20.64	16.69**	7.07
Reappraisal Generation (spontaneous)	0.0	0.4	0.9	1.69	1.05	2.17*	0.82
Reappraisal Generation (prompted)	0.8	0.8	1.0	1.87	0.92	2.29*	0.79
Reappraisal Monitoring	10	4	2	1.8**	1.61	0.57**	0.77

BI: Brain Injury group. *P*-values for modified *t*-test comparing Mrs M to BI group and controls * = $p < .05$; ** = $p < .001$

Table 4. Mrs M's Reappraisal task performance compared to Brain Injury and Control Group. Mrs M present the lowest scores in all four reappraisal measures.

Reappraisal difficulty: Mrs M's average time generating a reappraisal was substantial. At 95.67s, it was almost 30s slower than the worst patient (66.9s) and more than 50s slower than the worst control (32.8s). Using a modified *t*-test for small samples (Crawford and Howell, 1998)[22], it is clear that Mrs M required considerably more time to generate a reappraisal when compared to non-concrete patient [$t(11) = 2.67$, $p = 0.03$] and controls [$t(11) = 10.730$, $p < .001$].

Reappraisal Generation (spontaneous): Mrs M was not able to produce a single reappraisal on her own, and her performance was significantly lower compared to controls [$t(11) = -2.54$, $p = 0.03$] and marginally lower compared to other brain injured patients [$t(11) = -1.546$, $p = 0.08$]. However, Mrs M's score of 0 was below the worst non-concrete brain injured patient (0.4) and the worst control (0.9).

[22] This method is a modification to the independent sample *t*-test which can be used to compare a single specimen with a small sample (less than 50), offering an estimate of the rarity or abnormality of the individual score. Neuropsychological studies of different sort have used this approach when addressing research questions with small samples (see for example Bowles, Crupi, Mirsattari, Pigott, Parrent, Pruessner, Yonelinas and Kohler, 2007; Maguire, Nannery and Spiers, 2006; Schiltz, Sorger, Caldara, Ahmed, Mayer, Goebel, and Rossion, 2006)

Reappraisal Generation (prompted): Mrs M's performance improved considerably when prompted to reappraise, reaching an average of 0.8 reappraisals per picture. When considering the total of reappraisals per picture, including prompting, Mrs M was not the worst score among patients (three subjects obtained a score of 0.8), but still was below the lowest control (one subject obtained a score of 1.0).

Reappraisal Monitoring: Mrs M required the highest number of prompts during the task (10/10), differing significantly from controls [$t(11) = 11.766$, $p < .001$] and non-concrete brain injury patients [$t(11) = 4.893$, $p < .001$]. The worst non-concrete patient required 4/10, and the worst control 2/10.

In sum, Mrs M's reappraisal performance could be described as significantly *slower*, with more marked *generative* problems and requiring more external *monitoring*, when compared to other brain injured patients and controls. However, effectiveness improved to similar levels than non-concrete patients, when external compensation (in the form of prompts) was offered.

5.1.4.6. Reappraisal Performance: Qualitative Analyses

A qualitative analysis of Mrs M performance on the reappraisal task offers valuable additional information. We would like to comment further on this point given that neuropsychological assessment is not restricted to a patient's formal tests scores, but perhaps more importantly, to *how* the task is performed (Luria, 1966; Lezak, Howieson and Loring, 2004).

Mrs M's overall behaviour might be described as an 'over engagement' with the task (see Appendix 5 for a transcript of Mrs M's answers during the reappraisal task). Most of the

time she leaned forward on top of the desk, some 20 centimetres from the screen, looking somehow 'absorbed' by the pictures, a phenomenon not seen with other patients. This physical proximity to the stimulus was exhibited throughout the task, only altered during the final moments of her responses -when she was asked to offer reappraisals and replied "I don't know", while returning to an upright position (*Funeral* [9220]; *Flood* [9926]). The amount of time visually engaged with the slide was also greatly prolonged.

The most striking observation about Mrs M's behaviour was her emotional hyper-reactivity toward the pictures[23], in the form of frequent emotional *exclamations* ('Oh! -Smiles with surprise-What's the matter with that…what…what…what… oh! Oh dear -sad expression', *Sick Boy* [3350]; 'this is terrible!', *Flood* [9926]) and recurrent *facial emotional expressions* (*Sick Boy* [3350]; *Graveyard* [9220]; *Flood* [9926]) when the stimulus was presented.

Mrs M expressive language was informative but lacked fluidity and synthesis; sentences were short and often interrupted by pauses. The most common aphasic feature was a difficulty naming objects, which she compensated by substitution or circumlocution ('lots of *things* here' instead of *machines* or *cables*, *Sick Baby* [3350]; 'curly wind' instead of tornado, *Tornado* [5971]).

Mrs M's thinking process seemed highly guided by the perceptual features of the slides. Firstly, there was as an apparent *capture* of the thinking process by pictorial details ("All the tires though…tires there…all tires there"- pointing to tires placed in different parts of the picture, *Pollution* [9341]; "one, two, four factories…black smoke" –while touching the screen, *Factory* [9280]), with a failure to filter from amongst complex stimuli, and an inability to follow the more abstract instructions of reappraisal. This was exemplified by

[23] A similar description of emotional hyper-reactivity to pictures (neutral and emotive), in concrete patients, can also be found in Hanfmann, Rickers-Ovsiankina and Goldstein (1944).

awareness of unusual details, of a sort rarely mentioned by other subjects ('that is John or something -reading the name in one of the graves', *Graveyard* [9926]; 'there is a man standing on the right upper corner', *Pollution* [9341]). A second feature was the *intrusion* of stimulus-related thoughts after a prompt to reappraise has been offered ('doesn't he look old although he is tiny?', *Sick Baby* [3350]; 'although I don't know what this is…a hand – pointing to a small hand in the lower right corner', *Funeral* [9220]; "they are all standing...and he is standing there, so one, two, three…no really", *Pollution* [9341]). These types of responses were significantly less frequent in patients with brain injury and almost non-existent for controls. The mean number of intrusions per picture for Mrs M was 1.6, for the Brain Injury Group was 0.24 [$t(11) = 4.083$, $p < .001$] and 0.08 for controls [$t(11) = 13.90$, $p < .001$]. Taken together, these peculiarities of her thinking process suggest an inability to abstract relevant cues from the pictures and to inhibit perceptual elements that are irrelevant to the task.

5.1.5. Discussion

The last decade has seen dozens of neuroimaging studies offering insights into the neural basis of reappraisal. This case is the first description of reappraisal performance in a neurological patient, who appears to present with a specific impairment in disengaging from immediate experience in order to manipulate ideas and thoughts, when looking at emotional images.

The case of Mrs M is interesting because she presents with relatively preserved overall cognitive function, but exhibits a remarkable impairment of reappraisal. Compared to controls, and to other non-concrete brain injured patients, Mrs M appears to be completely unable to spontaneously generate reappraisals, despite having extended periods of time to

complete the task. In striking contrast, once external support is offered in the form of prompts, her capacity to reappraise is dramatically improved.

The prolonged periods of time for Mrs M's answers were not empty, as someone might expect of a patient with mild expressive aphasia. Instead, they were filled with remarks about observable elements of the picture, often repeated several times ('Oh dear…very sick boy…I don't know what you can say really…very sick', *Sick Baby* [3350]). These were accompanied by emotional comments (see Appendix 5), again often repeated in different ways ('Oh…it's sad…who are these? Does it matter who they are? It could be the relatives…that is John [pointing at a name in one of the graves] very sad', *Graveyard* [9220]). There were also often substantial latency periods between these comments and observations while Mrs M appeared to be gathering her thoughts.

An especially notable aspect of Mrs M's performance was that she often mentioned elements from the pictures that *were* used by controls as the raw material to construct appropriate reappraisal solutions. For example, in the *Sick Baby* [3350] picture, she specifically mentions the hand holding the baby ('very sick boy… although there is a hand there really… a hand there'). For controls (see Appendix 5), this pictorial element often elicited reappraisals where the baby was *being held* and *cared for* - in other words, where the hand represented something else (support/comfort), typically with an inferential or abstract positive meaning. However, despite noticing this useful element, Mrs M failed to capitalize on this material, perseverating on the perceptual elements and their emotional consequences ('a hand there… I don't know what you can say about that really…he is sick').

Finally, it seems necessary to comment on the remarkable discrepancy between Mrs M's poor performance reappraising and her self-assessment of such capacity. She rated herself high on the use of reappraisal (*Emotion Regulation Questionnaire*) as a strategy to feel 'less negative' or 'more positive' about a situation. This interesting discrepancy could be

interpreted as a deficit in self-awareness, a common problem in brain-injured patients, and especially in patients with concrete thinking, who may underestimate their physical, cognitive and social difficulties (Sherer, Boake, Levin, Silver, Ringholz and High, 1998; Prigatano, Altman and O'Brien, 1990; Sawchyn, Mateer and Suffield, 2005). A similar observation can be made to account for the discrepancy between her self-report of emotional difficulties (HADS) and the relative's perceptions of her emotional problems (Iowa Scale of Personality Change).

Two clear questions seem to arise from Mrs M's data: 1) if Mrs M is able to generate reappraisals once external aid is provided, how can we explain her selective deficit in performing the task by herself? In other words, which is the neuropsychological component that, once damaged, compromises her ability to spontaneously reappraise? 2) How Mrs M's performance, in the reappraisal task, might be related to her neuropsychological profile of concreteness? In other words, which neuropsychological components, associated to concrete behaviour, might account for the reappraisal impairment?

5.1.5.1. Concreteness and Disengagement

During the reappraisal task, Mrs M's main difficulty appears to be one of detaching or *disengaging* from the initial appraisal of a picture, in order to acquire a reflective stance to reinterpret such negative events. Once such detachment is accomplished, by means of external compensation in the form of prompts, Mrs M is able to reappraise at a similar level than other brain injured patients.

These observations appear to offer support to the idea that concrete behaviour impairs reappraisal because, in order to reappraise, a decrease in the emotional *salience* of the initial (automatic) response is required (McRae et al., 2011a). This is consistent with the classical definition of concreteness as an inability to disengage from immediate experience (Goldstein,

1942; Goldstein & Scheerer, 1941), which has been re-cast (by more contemporaneous authors) as the resurgence of a *default mode*, where inflexible stimulus-response linkages remain impervious to modification by context or experience (Mesulam, 2000; 2002), or as a form of *temporal concreteness*, where behaviour is anchored in the present, devoid of temporal perspective, and principally dominated by present needs and stimuli (Fuster, 2008).

The specific neuropsychological features, related to Mrs M's lesion site (BA 44, 46, frontal operculum), appear to support the disengagement hypothesis. Her lesion encompasses the left dlPFC, an area commonly reported as underpinning cognitive control (Petrides, 2005; Ridderinkhof, van den Wildenberg, Segalowitz and Carter, 2004) and thought to exert inhibitory effects on the posterior brain regions involved in perception (Anderson and Tranel, 2002). It has also long been reported that patients with damage to this area may exhibit a dependency on immediately-present environmental cues (Lhermitte, Pillon and Serdaru, 1986). In the case of Mrs M, this could be interpreted as the emotional capture by the picture's contents, and the intrusion of task-irrelevant perceptual elements.

5.1.5.2. Concreteness and Reappraisal Generation

We have reported that once Mrs M is prompted, her reappraisal ability improves substantially [see Appendix 5 for examples]. In other words, it seems that once the process of inhibiting the emotional appraisal is facilitated, she is able to generate positive reinterpretations of the negative scenes, achieving similar levels of performance to that of other non-concrete patients. However, the average number of reappraisals offered by Mrs M and non-concrete patients after prompting is *still* significantly lower than that of controls, suggesting a more *general effect* of frontal lesions on the *generative* component of reappraisal, which seems crucial for the production of multiple alternative interpretations. This is consistent with evidence suggesting that frontal lobe lesions, in general, compromise

spontaneous counterfactual thinking (Baldarrain, Garcia-Monco, Astigarraga, Gonzalez & Grafman, 2005). Thus, the ability to *detach* from initial emotional appraisals appears to be a necessary (but not sufficient) component in the complex multi-step process of reappraisal (Ochsner and Gross, 2008), and is *selectively* compromised by concreteness. However, the subsequent *generation* of alternative interpretations, although heavily dependent on effective disengagement, appears to be compromised not by concreteness in particular, but by frontal lesions in general. This claim appears to receive support from executive function models that place response inhibition (here disengagement), or the ability to delay prepotent responses, as a pre-requisite for other executive functions, such as word generation (Barkley, 1997, 2001).

5.1.5.3. Alternative Accounts

While we have argued that Mrs M's deficits in reappraisal are a consequence of a failure to disengage from perceptual elements (and its emotional consequences) we would like to briefly consider some alternative explanations for her performance, and discuss why we think they do not serve as sufficient explanations. Mrs M's deficit is clearly not a failure to identify the relevant pictorial elements in each image. She is invariably accurate in recognising objects in a scene (e.g. baby, medical equipment, hand etc.), identifying the overall theme of each picture (e.g., 'sick baby', *Sick Baby* [3350]; 'someone has died', *Funeral* [9220]) and specifying its emotional consequences (e.g., 'they are all very sad that he has died' *Funeral* [9220]).

Language impairment cannot *fully* account for her performance. Her failure in reappraisal is not the consequence of inability understanding task instructions, because her performance is so much improved after prompting. A skeptical reader might also wonder whether Mrs M's impairment generating spontaneous reappraisals is not just the consequence of an *expressive* language disorder. This seems unlikely, and cannot explain two facts.

Firstly, although lacking fluidity, Mrs M is able to offer informative accounts about the emotional pictures. In other words, her expressive language seems preserved enough to describe the slides and its emotional consequences (see Appendix 5). Secondly, her response time improves dramatically when prompted (*spontaneous M* = 95.67s, *SD* = 26.28; *prompted M* = 23.59s, *SD* = 20.17). When cued she is as quick (*Mrs M: M* = 23.59s, *SD* = 20.17; *BI group*: *M* = 37.65s, *SD* = 20.64) as other brain injured patients – so that it is unlikely that the spontaneous reappraisal deficit can be exclusively the result of expressive aphasia which improves after prompting. Nevertheless, it is necessary to acknowledge that Mrs M does present difficulties in executive function-related language ability, verbal fluency, a task that is highly dependent on cognitive control mechanisms such as self-monitoring, initiation and strategic retrieval (Henry and Crawford, 2004). The involvement of a more executive aspect of language in Mrs M's reappraising deficit appears to be supported by evidence that relate her lesion (BA 44, 46), to deficits in other executive language capacities, such as *selecting* among multiple competing propositions for language generation (Robinson, Shallice, Bozzali & Cipolotti, 2012), updating of task representations (Derrfuss, Brass, Neumann & von Cramon, 2005), inner speech (Geva et al., 2011; Jones, 2009) and conceptual self-referential thinking (Morin & Hamper, 2012).

In the domain of executive function Mrs M's failure is also not a consequence of attentional impairment, or of the ability to sustain attention. She is within normal limits also in measures of such capacities (*Telephone Search* and *Telephone Search dual task*), and she is also able to stay focused for up to two minutes, on each picture. However, as described above, several other executive functions that require disengagement from immediate experience *are* compromised by concrete behaviour (Barkley, 1997, 2001), most notably *abstraction*, *verbal fluency* (categorical and lexical) and *set shifting*. Apparently, impairment in one or two of these tasks makes it difficult to generate reappraisals -but not impossible,

whereas failing in most or all of them –which might reflect a more marked profile of concreteness, does appears to severely compromise the capacity to reappraise at all. This data is in line with clinical observations suggesting that concreteness is not a homogeneous deficit (Goldstein & Scheerer, 1941, Prigatano, 1989) and, therefore, only patients with more marked concrete behaviour will present more difficulties generating reappraisals.

5.1.5.4. Concreteness Profile and Reappraisal Components

Mrs M's profile of concreteness is characterized by marked impairment in three neuropsychological domains, all of which have been also proposed as relevant for the process of reappraisal (McRae et al., 2011a): i) response inhibition, ii) abstraction and iii) verbal fluency.

i) Response inhibition (*disengagement*) has been suggested as having an important role in emotion regulation, by enabling the suppression of the more automatic aspects of emotional responding (Barkley, 1997, 2001; Gyurak et al., 2012). This claim seems supported not only by Mrs M's impairment in disengaging from initial appraisals during the task, but also by her performance on response inhibition tasks. In a similar way to other patients with dlPFC lesions described in the literature (Anderson and Tranel, 2002), Mrs M presents with difficulties inhibiting responses associated with previously learned tasks (*Motor Inhibition*) and also inhibiting the most salient attributes of objects, in order to generate a superordinate category (*Similarities*).

To our knowledge, only one study (McRae et al., 2011a) has directly addressed the relationship between response inhibition and reappraisal, concluding that reappraisal *ability*, as measured by a decrease in negative affect, was not related to response inhibition (on a Stroop-like task). A possible explanation for the discrepancy with the data in the present case may lie in the different use of experimental paradigms. Most studies (which use

neurologically-normal participants) have taken the reappraisal *generation* aspect of the process for granted, and have investigated only reappraisal *ability*. However, an important feature of the focus of the present study, on neurological patients, derives from the fact that impairments in idea generation are a common feature of frontal lobe lesions and concrete behaviour (Goldstein & Scheerer, 1941; Jones-Gotman and Milner, 1977; Baldo, Shimamura, Delis, Kramer and Kaplan, 2001). The present study suggests that response inhibition (disengagement) is an initial, and necessary, step in the generation of alternative versions of reality (Barkley, 2001). If this ability is compromised, negative emotional thoughts tend to persist. These observations in Mrs M are supported by findings (in psychiatric populations) that relate depressive rumination, or the persistence of negative thoughts, to difficulties inhibiting negative information (Joorman, 2005; Withmer and Banich, 2007).

ii) Abstract reasoning, though little explored, has also been suggested as a component in reappraisal (McRae et al., 2011a). Abstraction is typically viewed as allowing humans to grasp the essentials of a given situation, or to break up a given whole into part in order to isolate and to synthesize them (Goldstein & Scheerer, 1941). The loss of an abstract attitude is a cardinal feature in patients with concrete behaviour, who often struggle with abstraction tasks because their thinking is captured by the most salient features of immediately present objects and mental representations (Goldstein & Scheerer, 1941, Salas, Vaughan, Shanker & Turnbull, 2013). For example, in the similarities task, when asked in which way socks and shoes are alike, Mrs M answers: "for your feet… warmth". Or when asked in which way a table and a chair are alike she replies: "good for eating" Both answers are concrete in the sense that, although they *do* establish a proper relationship between objects; the link is based on *physical/sensory* and *use* properties (*socks and shoes*: sensory/warmth; *table/chair*: use/eating). Reappraisal is, in contrast, an abstract exercise, in the sense that it demands that one inhibits what is seen or felt in the present moment ("Oh! What's the matter with that…

what… what… oh! Oh dear! Very sick boy", *Sick Baby* [3350]), in order to emphasize abstract *ideas* or *beliefs* that can change the emotional trajectory of a situation ("He will receive good treatment", "he is lucky to be alive", *Sick Baby* [3350]).

It is interesting to note that McRae and colleagues (2011a) did not find significant associations between abstract reasoning and reappraisal ability in their study. Their task, however, differed from ours in the sense that responses were 'closed' – with offering a limited set of choices. In order to better investigate abstraction, tasks should ideally be open-ended, or take the form of an 'ill-structured' situation. These are the properties of the real life scenarios where reappraisal unfolds, and map onto the ecological validity criterion that is so central for measures of executive function (Burgess et al., 2006; Goel, 2010).

iii) Verbal fluency has also been suggested as a relevant component for emotion regulation, by enabling the rapid generation of alternative interpretations, in accordance with a predetermined criterion (Gyurak et al., 2012; McRae et al., 2011a). Although no study has directly explored the relationship between verbal fluency and reappraisal, verbal fluency appears to be related to other emotion regulation strategies, such as response modulation, for basic (Gyurak et al., 2012) and primitive emotional responses (Gyurak et al., 2009).

As with other brain injured patients with left dlPFC lesions (Baldo, Shimamura, Delis, Kramer and Kaplan, 2001; Costafreda et al., 2006; Jones-Gotman and Milner, 1977; Robinson, Shallice, Bozzali & Cipolotti, 2012), Mrs M presents with impairment on verbal fluency tasks. Compared to healthy population standards, her capacity to generate sets of ideas (her phonological and semantic fluency), and to switch between them (her switching category fluency), is highly compromised. Nevertheless, when compared to non-concrete brain injured subjects Mrs M's performance only differs significantly on phonological (letter) fluency. This is especially interesting because phonological fluency, by comparison with semantic (category) fluency, appears to rely more heavily on the integrity of executive

functions (Henry & Crawford, 2004). Furthermore, and of interest to the discussion on disengagement and concreteness, it has been suggested that phonological fluency relies on an inhibitory component, which is required to suppress the habitual use of words in relation to their meaning (Perret, 1974). Taken together, it is likely that this impairment in phonological fluency might contribute to the low number of reappraisals she is able to generate spontaneously, and once prompted (on average less than one per picture), and this might have important implications for the effectiveness of this strategy. A difficulty generating alternative interpretations may seriously narrow the scope of possibilities from which a reappraisal can be selected, thus compromising the flexibility needed to test and modify a chosen reappraisal when it is not effective.

Finally, we note that there are two reappraisal components, often suggested in the literature, which did not appear selectively compromised in Mrs M's neuropsychological profile. The first is working memory, which is thought to enable the storage and manipulation of perceptual and contextual information (Gyurak et al., 2011), and which has been significantly associated to reappraisal ability (McRae et al., 2011a; Schmeichel, Volokhov and Demaree, 2008). Although Mrs M presents with some difficulties in mentally manipulating information (*100-7*), her level of impairment does not differ significantly from that of other brain injured patients (*Digit Span)*. In consequence, it seems unlikely that a working memory deficit (as measured by the digit span task used in this study) can account for her selective reappraisal impairment. The second component that did not appear compromised by Mrs M's lesion was divided attention. This ability would be required to bring to mind alternative thoughts or images that contradict the initial appraisal (Sheppes and Gross, 2011), thus demanding a splitting of attentional resources between competing representations. Unexpectedly, Mrs M's performance on the *Telephone search dual task* was at, or above, the levels achieved by the non-concrete patient group, and within the range of

controls (Table 3). A possible explanation for this apparent preservation is that the dual task used in this study might be more associated with *sustained* attention than attentional *switching* (Robertson, Ward, Ridgeway and Nimmo-Smith, 1996), a capacity that is preserved in left dlPFC patients, but often impaired in subjects with damage to the superior medial or right lateral prefrontal cortex (Wilkins, Shallice, McCarthy, 1987; Shallice, Stuss, Alexander, Picton and Derkzen, 2008).

5.1.5.5. Implications for Rehabilitation

The results presented in this case study expand our understanding of how brain injury in general, and concreteness in particular, may disrupt emotional life. By narrowing experience to the immediacy of the present moment, concreteness appears to limit not only a patient's ability to process appropriate information, but also their capacity to modulate how they feel, by means of detaching from experience and reflecting upon it.

The implications of this type of impairment for rehabilitation are diverse. Firstly, Mrs M's performance suggests that reappraisal improves greatly when offered external scaffolding, through prompts (which facilitate disengaging from the initial appraisal). Several explanations may account for this improvement. For example, it is possible that prompts function as an 'external' form of speech (the *social speech* of Vygotsky, 1986[1934]) that allows Mrs M to compensate for her difficulty of internally guiding her behaviour through *inner speech* (e.g. '*stop describing what you see and think about a positive side of this*'). This interpretation of the data would be in line with Luria and Vygotsky's early work on the regulatory role of speech (Luria, 1959; 1960; 1966; Vygotsky 1986[1934]), as well as with more modern developmental research, suggesting that the externalization of speech in children facilitates the detachment from the immediate environment (Muller, Jacques, Brocki & Zelazo, 2009).

Another possibility, from the perspective of restoration of function, is that prompting might work as an 'orientation of thought' (Luria, 1963), allowing Mrs M to go beyond passively describing what she sees [e.g. a sick baby], allowing her to temporally restore *active* thinking, or the capacity to create an inner plan of thinking. Even though these explanations remain tentative, and require further research, both stress the fact that reappraisal, in the same way as other psychological processes (Bowen, Yeates, Palmer, 2011), can be influenced externally and interpersonally for the purposes of rehabilitation. Furthermore, it also suggests the need to consider *extrinsic* mechanisms of emotion regulation when compensating for *intrinsic* regulatory failures (Nive, Totterdell and Holman, 2009; Rime, 2007; Salas, 2012).

There is a large literature suggesting that the externalization of speech, also known as *private* speech, has a mediating role in self-regulation, in both healthy and clinical populations (for a review see Winsler, 2009). The therapeutic possibilities of this idea with brain injured patients have also been explored, for example, in relation to the use that children with TBI make of 'thinking aloud' in classroom environments (Rees & Skidmore, 2011), or the use of self-advocacy video tapes during the process of identity reconstruction (Ylvisaker & Feeney, 2000). It is in this context that the use of prompting to facilitate reappraisal is relevant as a therapeutic tool. Furthermore, it is even possible to suggest that prompting may be used by therapists in different forms, all of them aiming to scaffold reappraisal capacity. For example, therapists may use prompts to [1] change the patient's focus of attention when he/she is stuck on a specific negative appraisal (*e.g. let's not talk about what you lost, let's talk about what you have now*); [2] offer content that can be used as raw material for new reappraisals (e.g. *what if I ask you now to think about your kids? Let's talk about your kids*); [3] help patients to adopt a different point of view from which to reappraise (e.g. *what would your wife think in this same situation?*); [4] or synthetize

reappraisals from more diverse associations *(e.g. you talked about being at home… you also mention the kids… watching movies… am I right if I say that not working anymore has changed your lifestyle for the better?)*. Relatives and caregivers can thus also be trained in how to manage patients' regulatory difficulties. For example, they may learn to detect when patients are stuck on a negative emotion or thought, as well as to learn the steps that facilitate the transition out of such states. In some exceptional cases, patients themselves may even learn to recognize when they feel stuck, and look to significant others as a source of external regulation (Salas, in press).

5.1.6. Limitations and Future Directions

This case study also reveals some limitations that might be addressed in the future. We note that the observed difficulties in reappraising are likely to be amplified in real life (rather than laboratory) situations, where personally-meaningful events appear to generate higher levels of arousal (Salas, Radovic and Turnbull, 2011), compromising even further the necessary cognitive control abilities required to reappraise (Demanet, Liefooghe and Verbruggen, 2011; Oei et al., 2011). This is an extremely interesting possibility for future research, and taps into a developing trend in affective research, which is the study of emotion in more naturalistic contexts (Scherer & Tannembaum, 1986; Scherer, Wranik, Sangsue, Tran & Scherer, 2004). Even though this article does not address such matters directly, it seems possible in the future to partially address this issue in experimental settings, for example, by exploring the relationship between a picture's valence and the reappraisal's level of difficulty.

The analysis of Mrs M's performance suggests new avenues of research on a problem only partially addressed by the neuroimaging literature on reappraisal: that of lateralization. Neuroimaging studies have long reported left hemisphere activation when reappraising, thus

suggesting a shared neural substrate with language and cognitive control abilities. However, the present study bears on the question of how the *right* hemisphere might contribute to the process of reappraisal. Lesion studies have suggested that cognitive control involves a number of distinct processes, which they may be localized across *both* hemispheres (Alexander, Stuss, Picton, Shallice and Gillingham, 2007). For example, while patients with left lateral damage show difficulties in task setting, or disengaging from a previous task to initiate a new one (as in the case of Mrs M), subjects with right lateral lesions exhibit a rather different profile of monitoring impairment (Stuss, 2011). The study of patients with such monitoring impairments might offer valuable information regarding how damage to this specific cognitive control component can differentially compromise reappraisal ability.

We have primarily addressed the impact of concreteness on a specific emotion regulation strategy, but this does not consider the impact of concreteness in emotion *generation;* which often relies on abstract cognitions (McRae et al., 2011b; Ochsner et al., 2009). If we acknowledge that emotion generation and regulation are two interactive processes (Gross and Barret, 2011), the impact of concreteness on how emotions are triggered is clearly important and needs to be addressed in future studies. In addition, a further limitation of this study is its focus on the *generative* component of reappraisal, not considering reappraisal *ability* or efficacy. It remains to be explored how left dorsolateral lesions may impact the down-regulation of negative affect. Finally, in order to further understand the relationship between reappraisal generation/effectiveness and underlying neuropsychological components, a multiple single-case group study would be required, to confirm the associations between cognitive deficits (e.g. response inhibition) and reappraisal performance suggested by this case study.

The growing number of neuroimaging studies on reappraisal underscores the scientific and clinical relevance that this emotion regulation strategy has acquired in recent years. We

have proposed that the study of brain injured patients may offer a complementary source of information, describing a patient with a selective impairment in reappraisal generation. In addition, we note that, even by studying the profile of deficit in a single patient, it is possible to identify new rehabilitation approaches, of substantial clinical importance, which tackle problems that lie on the border of cognition and affect – for it is increasingly clear that the most disabling difficulties that brain injured patients have to face are not cognitive, but are emotional and interpersonal.

5.2. Opening to a new dimension: Changes in Emotion Reactivity and Emotion Regulation in a Case of Executive Impairment after Left Fronto-Parietal Damage[24].

5.2.1. Abstract

Dysexecutive impairment is a common problem after brain injury, particularly after damage to the frontal lobes. There is a large literature describing the cognitive deficits associated to executive impairment, however, little is known about its impact on emotional functioning. This case study describes changes in a 72 years old man [Professor F], who became markedly dysexecutive after a left fronto-parietal stroke. Professor F's case is remarkable in that, despite exhibiting typical executive impairments [e.g. set shifting, verbal fluency], abstraction and working memory capacities were spared. Such preservation of insight-related capacities allowed him to offer a detailed account of his emotional changes. A mixed method approach was adopted, using quantitative [self-report and informant-report questionnaires, experimental tasks] and qualitative tools [in depth interview] to explore modifications in several well-known emotional processes [e.g. emotion recognition, emotional reactivity, emotional understanding, emotion regulation]. The results suggest that Professor F's two main emotional changes were in the domain of emotional reactivity and emotion regulation. Modifications in emotional reactivity were observed as an increase in the experience of both positive and negative emotions. As for emotion regulation, he reported important difficulties down-regulating negative emotions [particularly sadness]. Professor F related both changes to difficulties in his thinking process, especially to a difficulty generating and manipulating

[24] Salas, C.E., Radovic, D., Yuen, K.S.L., & Turnbull, O.H. (in preparation). Opening to a new dimension: Changes in Emotion Reactivity and Emotion Regulation in a Case of Executive Impairment after Left Fronto-Parietal Damage.

thoughts during moments of negative arousal. These results are discussed in relation to the literature on executive function and emotion regulation, both intrinsic and extrinsic. The relevance of these findings for the debate on the neural basis of emotional reactivity and emotion regulation is also addressed.

And the question is,
was I more alive
then than I am now?
I happily have to disagree;
I laugh more often now,
I cry more often now,
I am more me

Peter, Bjorn and John, *Object of my affection*

5.2.2. Introduction

Executive impairment is a common problem after frontal lobe lesions, often compromising a wide range of necessary capacities to resume productive and social life (Crepeau & Scherzer, 1993), such as initiation, problem solving, self-monitoring and behavioural regulation (Burgess & Robertson, 2002; Evans, 2005; Fuster, 2008; Wilson & Evans, 2008). Even though executive impairment is commonly associated with *cognitive* difficulties, there is a small literature suggesting that *emotional* deficits, and particularly emotion *regulation* deficits, may also co-occur (Burguess, Alderman, Wilson, Evans & Emslie, 1996; Eslinger, Flaherty-Craig & Chakara, 2013). For example, some reports have described the presence of shallow levels of emotion itself (Baddeley & Wilson, 1988; Burgess & Robertson, 2002), while others have referred to difficulties in controlling emotion (Fuster, 2008; Godefroy et al., 2010, Tang et al., 2009; Yeates et al., 2008). Unfortunately there has not been a systematic exploration of this problem in order to interpret such apparently contradictory reports.

Addressing the topic of emotional changes after brain injury has been a complex task for researchers, presenting important limitations. For instance, emotional change has been poorly operationalized, with dissimilar conceptualizations used by authors, defining emotional change as a modification in: emotional expression (Baird et al., 2006; Brahman et al., 2009; Hornak, Rolls & Wade, 1996; Hornak et al., 2003), intensity and frequency of discrete emotional experience (Hornak et al., 2003; Rolls, Hornak, Wade & McGrath, 1994), transient and dispositional emotion (Gillihan et al., 2010), public behaviour and interpersonal

relationships (Brahman et al., 2009; Hornak et al., 2003) or mood and personality (Cohen et al., 2001; Robinson, 2006). Nevertheless, there is a recent literature addressing this gap, by suggesting a set of key emotional processes to be explored when assessing emotional change after brain injury (Levenson et al., 2008; Rosen & Levenson, 2009).

Another limitation has been the under-reporting of the patient's subjective experience of affective changes, including the way that patients perceive these changes, and how they impact on their lives. Even though some studies have tried to capture this using semi-structured questionnaires that ask about emotional changes (e.g. Rolls, Hornak, Wade & McGrath, 1994; Hornak et al., 2003), such data have never been reported in detail. This class of evidence could inform, in a bottom-up fashion, our understanding of emotional change, for example in subjects with executive impairment (Turnbull & Solms, 2004). Furthermore, a better description of emotional changes, from the *inside*, could also contribute to the understanding of related problems in neuropsychological rehabilitation, such as *identity* change (Carrol & Coetzer, 2011; Coetzer, 2008; Gelech & Desjardins, 2011; Salas, 2009; Segal, 2010; Ylvisaker & Feeney, 2000).

Why might researchers have avoided the *in depth* exploration of emotional changes in dysexecutive patients? A possible answer is that this population often presents with a wide set of cognitive deficits [disorganized thinking, cognitive rigidity, concreteness, or impaired working memory], deficits that may compromise the capacity to reflect upon, and report, subjective experience (Eslinger, Flaherty-Craig & Chakara, 2013; Salas, Vaughan, Shankers & Turnbull, 2013; Ylvisaker, Szekeres & Feeney, 1998). Nevertheless, these methodological concerns can be moderated by a number of arguments. First, there is an emerging literature addressing the technical challenges imposed by interviewing people with cognitive (Paterson & Scott-Findlay, 2002) and language difficulties after brain injury (Carlsson, Paterson, Scott-Findlay, Enfhors & Ehrenberg, 2007; Lloyd, Gatherer & Kalsy, 2006). Secondly, it is clear

that the so-called dysexecutive syndrome has heterogeneous presentations (Stuss & Alexander, 2007), and the degree of cognitive impairment can vary widely (Evans, 2005). Some dysexecutive patients may *preserve* substantial capacity to reflect upon experience, and offer a detailed account. In our opinion, the study of such *high-functioning* dysexecutive patients opens important opportunities to understand the nature and extent of emotional changes in dysexecutive patients.

There is a long history of subjective reports of patients with neuropsychological impairment. However, this literature is almost exclusively about patients with posterior lesions, who have intact executive function (e.g. Kapur, 1996; Luria, 1972). This article explores the emotional change in a man [Professor F] who acquired dysexecutive symptoms after a left fronto-parietal lesion, caused by an ischemic stroke. What is unique about this case is Professor F's extraordinary premorbid capacity and the remarkable preservation of the ability to abstract, which offers a rare window into the subjective experience of dysexecutive patients. In order to address some of the limitations of previous studies on emotional change, this article considers a wide range of emotional key processes (Levenson, 2007; Levenson et al., 2008). In addition, by using a case study methodology, different data collection methods are combined (Yin, 1994): with reports of in-depth interviews, self-report questionnaires, relative-report questionnaires and a range of experimental tasks. It has been suggested that such convergence of methods, often called *triangulation* (Denzin, 1978), allows a more complete and contextual portrait of the phenomenon at study (Jick, 1979).

5.2.3. Methods

5.2.3.1. Case Description

Professor F is a 72 year old right-handed male, married and father of three. At the time of his brain injury, 6 years ago [2006], he was actively working in academia as a lecturer [in a social science discipline] and as the dean of his department. His teaching career had extended for almost forty years, with an active research agenda and numerous publications [mostly essays and qualitative papers] that can be traced up to the year of the accident. He was taken to the emergency service several hours after the stroke, which occurred at night while sleeping. At that point, he was confused, also presenting severe expressive aphasia and right hemiparesis. According to medical records, after being pharmacologically treated [details about this procedure were not clear on his medical records], within hours there was a positive recovery of language deficits and hemiparesis. There are no records of radiological examinations at the time of the injury.

Once Professor F became stable, he was immediately transferred to a sub-acute inpatient rehabilitation unit, where he stayed hospitalized for a period of two months. Records of the initial neurological and neuropsychological assessment suggest good understanding of this immediate environment and relatively preserved communication –with some difficulty finding words- and high levels of anxiety. At three months of evolution he was discharged from the sub-acute unit and moved back to his house. At that point [2006] he started attending an outpatient rehabilitation clinic.

When interviewed at arrival, he –and his wife- reported the following problems: difficulties managing time, fatigue and high levels of anxiety. When queried about the possible causes of his anxiety, Professor F commented that his main concern whether he would ever recover his intellectual capacities, for he had noticed changes in the way his mind now worked. He found himself forgetful, unable to focus and with moments where his mind

went blank. He also commented on difficulties following conversations, a problem that made him feel lost and frustrated.

The outpatient rehabilitation phase lasted 15 months [2006-2007] and Professor F attended the centre twice a week. During this period the main goals of treatment were: 1) to improve cognitive function, specifically his tolerance to mental effort, fluidity and mental flexibility; 2) to incorporate the use of compensation strategies to manage the impact of executive problems in everyday life [calendars, diary]; 3) to facilitate Professor F's return to academic activities; 4) to emotionally support the increased awareness of changes generated by the exposure to his former working routine; 5) to educate and support family members [especially Professor F's wife] in the process of adjustment. By the end of this phase [2007], and despite his executive disorder, Professor F was working two mornings a week at the University.

Even though the outpatient rehabilitation phase was over, Professor F continued privately with the support of a clinical neuropsychologist, an occupational therapist and a physiotherapist, this one more sporadically. Such work has been sustained until present time [2008-2013].

A radiological examination, five years after the vascular event [2011], suggested a watershed stroke at the junction of the middle and anterior cerebral artery territories, in the left frontal and parietal lobes. The damage involved the medial part of the dorsal prefrontal cortex and extended into the intra-parietal cortex [see figure 1].

Interaction with Professor F suggests that he is a relatively independent individual. At the time of writing [2013], he has many areas of elementary cognitive impairment, but works hard to compensate for them. He is able to use the telephone, but needs to take notes to remember conversations. He remembers appointments only if they are registered in his diary, particularly new ones. He can shop independently for small purchases. He collaborates

proactively with household chores. He is able to travel independently by subway or taxi. However, he only feels confident when travelling a familiar route. In cases where he needs to arrive to a new place [as it was the case when he started attending to the session with his psychologist], he needs to be accompanied by a family member initially. However, after only a couple of closely monitored trips, he is able to start doing the route alone. His capacity to move independently has also been limited by his right leg, which did not recover completely, generating fatigue and balance problems. He was also able to manage money but needs help with banking and major purchases. In fact, all the household finances were taken over by Professor F's wife after the accident.

Today, Professor F still works twice a week at the University. However, his duties have changed in import ways. He does not teach anymore, neither is he in charge of any administrative duties. His capacity to read highly theoretical books, a common task in his pre-stroke everyday work, is rather limited, especially for texts that present new and unfamiliar ideas. His actual work has mostly focused on tutoring undergraduate and postgraduate students, who are working on areas related to his past field of expertise. In addition, he had begun marking student theses, a task that was slowly mastered [it took almost a year], thanks to the development of a system that organizes and register information. This system consists of making small notes and comments on each paragraph. Then, after the complete text has been read and commented, Professor F goes over it a couple of times, but now just reviewing his comments. Based on this second reading, new and more inclusive comments are generated, allowing him to synthetize his impressions and elaborate a report.

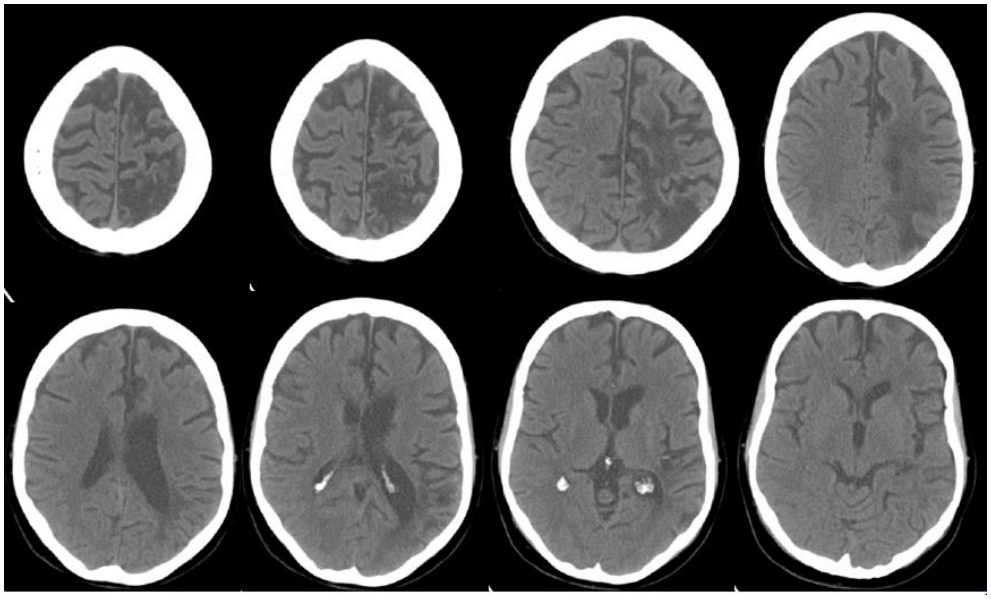

Fig 1. Professor F's Lesion. Coronal sections of Computerised Tomography. The lesion involves the medial part of the dorsal prefrontal cortex and extended into the intra-parietal cortex

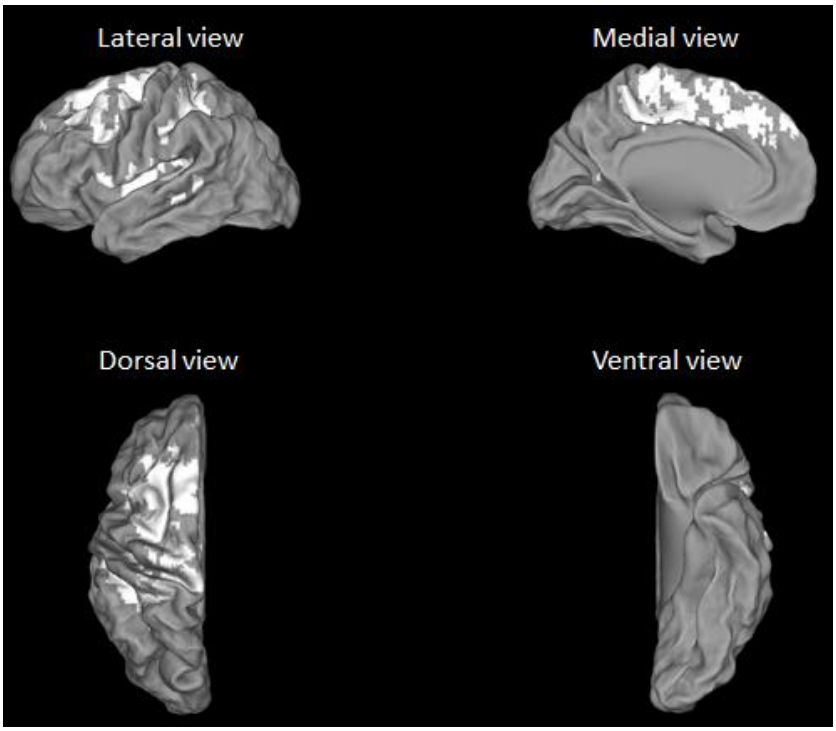

Fig 2. 3D reconstruction of Professor F's lesion. The image shows how the damage mostly compromised the dorsolateral and dorso-medial left PFC.

During his spare time, when he does not attend to university, he has started writing. He has completed short autobiographical stories, and published two historical books about the institutions where he has worked. Even though this level of productivity is impressive, Professor F has to invest high levels of effort to write and re-write such documents. The most prominent difficulties he faces when approaching these tasks are generating and organizing ideas, as well as multitasking. A common problem is that he does not know how to start writing, often reporting that he is not able to come up with ideas, or that he struggle manipulating ideas in his mind or coming up with a scheme [or plan] of what to write. However, writing down his thoughts appears to facilitate the idea generation process, and focusing for months exclusively on the same project appear to help him to keep information fresh and at hand. The support of the occupational therapist has been crucial here, especially during periods where the writing process becomes 'stuck' or needs to be planned.

Professor F's social life has shrunk, now clustering mostly around his nuclear family. Although such changes in social life are common with age, Professor F's changes have been specifically influenced by several factors. In first place, because of his leg problem, he gets tired when walking long distances, thus limiting his mobility range. Secondly, he reports struggling in social situations, which are experienced by him as exhausting, and difficult to follow. Thirdly, his capacity to plan social activities appears to be reduced. He has complained that he does not spontaneously think about 'fun activities' to do with his wife. He justifies this by suggesting that it may be related to his mind focusing completely on his writing during his spared time.

5.2.3.2. Quantitative Data Collection

5.2.3.2.1. Procedure

The neuropsychological and emotional assessment was carried out in 2012, at Professor F's house in three separate sessions (2 hours each). In the first session the rationale of the study was explained to him and his wife, and consent was obtained. The neurological tasks and questionnaires were spaced throughout the sessions in order to reduce the impact of fatigue on performance. Professor F's questionnaires were administered under close supervision from one of the researchers (CS) in order to check that the participant understood each question and the logic of the corresponding scale. Relative-report questionnaires were also introduced in detail to Professor F's wife, explaining the logic of each instrument, so she could answer them on her own. In the last session the main researcher and Professor F's wife revised the questionnaires checking for items that might not have been clear.

5.2.3.2.2. Instruments

5.2.3.2.2.1. Neuropsychological Assessment

A battery of neuropsychological tasks was used to obtain a profile of Professor F's overall cognitive, especially focusing on his executive capacities: Speed Processing Index (*WAIS-R*; Weschler, 1981), Free and Cued Selective Recall Reminding Test (*FCSRT*, Grober & Buschke, 1987),Verbal Fluency (*D-KEFS*; Delis, Kaplan, & Kramer, 2001), Wisconsin Card Sorting Test (*WCST*, Heaton, Chelune, Talley, Kay, & Curtiss, 1993), Similarities (*WAIS-R*) and Matrix Reasoning (*WAIS-R*). In order to further understand Professor F's profile of dysexecutive impairment, the Dysexecutive Questionnaire (*DEX*, Wilson, Alderman, Burguess, Emslic, & Evans, 1996) was applied. The DEX is a 20-item questionnaire that explores a range of problems commonly associated with the Dysexecutive Syndrome. The DEX comes in two versions, one to be completed by the patient and another by the relative.

5.2.3.2.2.2. Emotion and Personality Assessment

Emotion and personality were assessed by considering the outcome of experimental tasks, self-report questionnaires and relative-report questionnaires. In order to capture the complexity of emotional processes, the following key emotional components were assessed: emotion recognition, emotional expressiveness, emotional comprehension, emotion regulation, emotional symptomatology and personality change.

a) *Emotion recognition* is a basic aspect of emotional and interpersonal life. Professor F's emotion recognition ability was assessed using the Facially Expressed Emotion Labelling Test software (*FEEL*, Kesler, Bayerl, Deighton & Trauce, 2002). The FEEL measures the perception of facially expressed emotions and has been used in psychiatric (Kessler, Schwarze, Filipic, Traue, & von Wietersheim, 2006) and brain injury (Brau, Traue, Frisch, Widder, Deighton & Kessler, 2005) populations. During the task a slideshow of portrait images is presented, each for a period of 300ms, expressing six possible basic emotions: happiness, sadness, disgust, anger, fear and surprise. After the image is presented participants are asked to choose the corresponding emotion, from six available options (words) displayed on the screen. For each basic emotion seven portraits are presented during the task.

b) *Emotional Expressiveness* refers to the behavioural changes that typically accompany emotion, or the form in which an individual manifest emotional impulses behaviourally (Gross & John, 1997). This capacity is often assessed using the Berkeley Expressivity Questionnaire (*BEQ*, Gross & John, 1997). The BEQ is a 16 questions self-report, where participants have to rate how much they agree with each statement using a 7 point-likert scale. It is composed of three subscales that measure

different facets of emotional expressiveness: Negative Expressivity, Positive Expressivity and Impulse Strength.

c) *Emotional Understanding* refers to a more complex aspect of emotional life. Professor F's ability to understand emotions was assessed using the Toronto Alexithymia Scale 20 (*TAS-20*, Bagby, Parker & Taylor, 1994). This self-report questionnaire has three subscales that capture different aspects of emotion understanding. The *Difficulties Identifying Emotions* (DIE) subscale refers to problems distinguishing between emotions and bodily sensations and also between different emotions. The *Difficulty Describing Emotions* (DDE) subscale assesses the inability to verbally express emotions. Finally, the *External Oriented Thinking* (EOT) scale refers to a concrete non-introspective cognitive style.

d) *Emotional Symptoms*. Emotional symptoms, such as anxiety and depression are common after brain injury (Robinson, 2006). The presence of emotional symptoms was assessed using a self-rating questionnaire, the Hospital Anxiety and Depression Rating Scale (*HADS*; Olssøn, Mykletun, & Dahl, 2005). The HADS has been widely used in subjects with brain injury (Dawkins, Cloherty, Gracey, & Evans, 2006). The scale consists of 14 items that assess different manifestations of anxiety [HADS-A subscale] and depression [HADS-D subscale]. In each item, participants are expected to select an answer from four possible choices that reflect the frequency of such symptoms in a time period of a week.

e) *Emotion Regulation* refers to the set of processes that individuals use to influence *which* emotions they have , *when* they have them and *how* they experience and

express those emotions (Gross, 1998). Emotion regulation has been considered as a key emotional process (Rosen & Levenson, 2009), that is often compromised after brain injury (Abreau et al., 2009; Beer & Lombardo, 2007; Salas, 2012). In order to explore Professor's F regulatory capacities, the Emotion Regulation Questionnaire (*ERQ*, Gross & John, 2003), was used to assess the frequency of usage of two emotion regulation strategies: suppression and re-appraisal. Suppression has been defined as the conscious inhibition of one's own emotional expressive behaviour while affectively aroused (Gross & Levenson, 1997), while re-appraisal has been described as the ability to change a situation's meaning in a way that alters it emotional impact (Gross & Thompson, 2007).

f) *Personality Change* following acquired brain injury has been described as an alteration or discontinuity in personhood post injury (Yeates, Gracey & McGrath, 2008), often reported after frontal lobe lesions (Chow, 2000 for a review). The Iowa Scale of Personality Change (*ISPC*, Barrash, Anderson, Hathaway-Nepple, Jones & Tranel, 1993) is an informant questionnaire that assesses disturbances in several personality dimensions, such as decision making, social behaviour, irascibility, motivation and distress (Barrash, Asp, Markon, Manzel, Anderson & Tranel, 2011). It is composed of 30 items, each of them targeting one type of personality disturbance. For each item the informant rates the presence of the behaviour after (*Now* Score) and before (*Before* Score) the injury. A change score (Δ) is generated by subtracting the *Now* score from the *Before* Score. Thus, a positive Δ score implies an increase in a determined disturbance, while a negative Δ score suggests a decrease in such behaviour.

5.2.3.3. Qualitative data Collection

5.2.3.3.1. Procedure

In order to capture Professor's F subjective experience of his emotional changes, a series of three semi-structured interviews were carried in 2012, using a thematic analysis approach (Braun & Clarke, 2006). The interview schedule intentionally focused on the impact that the brain injury had on emotional life. For example, initial questions asked Professor F to report on any changes that he had noticed on individual and interpersonal aspects of emotional life. Following this, Professor F was asked to reflect more specifically on possible changes on his capacity to experience, understand and regulate emotions. Each of these interviews lasted 120 minutes approximate and was digitally audio-recorded for analytical purposes. See Appendix 6 for the interview protocol.

5.2.3.3.2. Technical adaptations to address dysexecutive impairment during the interview process

In order to manage the impact of Professor F's dysexecutive impairment on the interviews, a series of strategies were developed. First, as suggested by several researchers, interviews were spaced in time to avoid fatigue and overstimulation (Carlsson et al., 2007; Patterson & Scott-Findlay, 2002). In addition, during the course of each session, Professor F's level of fatigue was constantly monitored and short breaks were suggested (Lewis & Porter, 2004).

Different compensatory strategies were used to manage the impact of dysexecutive impairment on the interview process. Firstly, as a form of compensating for Professor F's difficulty *recalling* information [see Table 1 below], direct interview questions were formulated. These questions were intended to collaboratively find concrete situations that would facilitate the recall process (Patterson & Scott-Findlay, 2002). Once such 'typical situations' where flagged [e.g. a social situation where he felt he could not manage his

emotions] the interviewer actively facilitated the re-construction process, managing typical dysexecutive phenomena such as distraction and perseveration. Such facilitation was accomplished mainly by helping Professor F *fixing* the focus of attention on specific contents [e.g. 'let's stay for a while on how you experienced your mind at that time?'], and also by *shifting* the focus of attention to other related contents in order to enrich the narrative [e.g. 'let's try to think now how you tried to recover for those intense feelings?'].

Compensatory strategies were also used to manage Professor's F difficulty in moving from *detailed* to *general* levels of thinking, and *vice versa*. This is a common difficulty in subjects with dysexecutive impairment, who tend to get stuck into the details of a situation (Eslinger, Flaherty-Craig, & Chakara, 2013), thus compromising the transit from offering examples to abstracting meaning from them. The interviewer actively formulated questions that facilitated the transition from *concrete to abstract* levels of thinking [e.g. 'So you tell me about this situation where you did not know what to do… Were you trying to tell me this story as an example of how hard is for you to manage how you feel?'], and from *abstract to concrete* levels of thinking [e.g. 'You say that when there are too many stimuli you don't know what to do…Can you tell me how your mind feels, or how your body feels, in those moments?'].

5.2.3.3.3. Qualitative Data Analysis

All data were collected prior to analysis and interviews were transcribed *verbatim*. The qualitative data analysis software NVIVO™ (QSR International Pty Ltd., 2002) was used to retrieve and code data, as well as to develop theory building. A thematic analysis, which allows the identification and reporting of patterns within qualitative data, was conducted in accordance to the procedure described by Braun and Clark (2006). Three main features characterize the type of thematic analysis used in this research. First, it adopted an

essentialist/realist approach, which holds that language reflects, and enables us to articulate, meaning and experience. Second, it was *theoretically driven*, offering a detailed description of certain aspects of the data which are relevant for the research question [in this case emotional change], and not the entire data set. Third, it followed a *semantic approach*, which implies that themes are identified within the *explicit* meanings of the data, and that data handling involves a progression from descriptive to interpretative levels.

Data analysis followed general guidelines established by the Grounded Theory approach. Firstly, data was fragmented into codes, which were organized in more general concepts [categories]. Secondly, data was reorganized, by generating connections between categories (Strauss & Corbin, 1998). More specifically, and as a first step, the interview was read and re-read separately by two coders [CS and DR], in order to identify preliminary themes. Then the interview was broken down into codes, which were organized into categories and subcategories. This analysis was made using the constant comparison approach, in which researchers go back and forth between newly uncovered and pre-existing data, thus constantly modifying the emerging categories (Glaser & Strauss, 1967). Comparisons were made between objects [codes] and theory [categories], as suggested by Strauss and Corbin (1998). Themes where then refined, and their names revised according to the different codes extracts and the entire data set. The reorganization of the data was made by relating categories in consideration of their properties and dimensions, as well as looking on how they linked together. Finally, in order to report the results of the analysis, vivid and compelling extracts were selected.

5.2.4. Results

5.2.4.1. Neuropsychological Assessment

Data from neuropsychological tasks, self-report and relative-report questionnaires suggest that, 7 years after the stroke, Professor F presents with moderate dysexecutive deficit. The profile of such impairment [see Table 1 for details] may be characterized as: (1) a decrease in speed of processing, requiring long periods of time to correctly deal with complex information [*Speed Processing Index*, *WAIS III*]; (2) Verbal fluency problems, especially when using categories as a guide [*D-KEFS*]; (3) increased sensitivity to interference [*failure to maintain set errors*, *WCST*] and cognitive inflexibility [*perseverative errors*, *WCST*]; (5) preserved capacity to store new information, but severely impaired free recall [*Free and Cued Selective Recall Reminding Test, FCSRT*, Grober & Buschke, 1987]. It is interesting to note that, in contrast with the impairment in several domains of executive function, Professor F's *abstract* capacities appear to be relatively spared [*Similarities* and *Matrix Reasoning*, WAIS]. Professor F's range of executive impairments can be better appreciated by considering his self-report on the DEX. He describes himself as having *frequent* difficulties planning ahead, assessing the magnitude of problems, avoiding distractions, making decisions, and frequently acting without thinking. When compared to a sample of 78 patients with diverse neurological disorders (Wilson et al., 1996), Professor F reports similar levels of dysexecutive problems in everyday life [DEX, $t(77) = 0.32$, $p = .37$, $M = 27.21$, $SD = 14.48$]. Notably, he acknowledges more dysexecutive problems than his wife on the DEX [Professor F's score = 32; Professor F wife's score = 22].

Table 1
Summary of Professor F's neuropsychological assessment

Neuropsychological Processes	Task	Score	Interpretation
Speed Processing	Processing Speed Index (WAIS)	88	mildly impaired
	Digit Symbol-Coding (WAIS)	7ss	mildly impaired
	Symbol Search (WAIS)	9ss	mildly impaired
Language	Letter fluency (D-KEFS)	9ss	mildly impaired
	Category fluency (D-KEFS)	4ss	impaired
Working Memory	Working Memory Index (WAIS)	111	normal
	Arithmetic (WAIS)	10ss	normal
	Digit Span (WAIS)	11ss	normal
	Letter-number sequencing (WAIS)	14ss	above average
Memory	Free Recall FCSRT	11	impaired
	Total Recall FCSRT (free and cued recall)	26	impaired
	Recognition FCSRT	15	normal
Category Generation and Set Shifting	Categories Completed (WCST)	4	impaired
	Perseverative errors (WCST)	24	impaired
	Failures to maintain set (WCST)	8	impaired
	Category switching fluency (D-KEFS)	3ss	impaired
Abstraction	Similarities (WAIS)	10 ss	normal
	Matrix Reasoning (WAIS)	12ss	above average

ss = scaled scores;WAIS = Wechsler Adult Inteligence Scale; D-KEFS = Delis Kaplan Executive Function System; FCSRT = Free and cued selective Recall Reminding Test; WCST = Wisconsin Card Sorting Test

5.2.5.2. Emotion and Personality Assessment

a) *Emotion Recognition* [FEEL test]: Professor F's capacity to recognize accurately, and label, basic emotions is preserved for all emotions except disgust. When compared to a sample of 15 age matched controls he does not present significant differences in the total score [$t(14) = -0.19$, $p = .18$, $M = 33$, $SD = 5.5$]. More specifically, Professor F's performance is in normal range for anger [$t(14) = 0.13$, $p = .44$, $M = 3.6$, $SD = 2.8$], sadness [$t(14) = -0.32$, $p = .37$, $M = 5.6$, $SD = 1.8$], surprise [$t(14) = -0.48$, $p = .31$, $M = 5.8$, $SD = 1.6$], happiness [$t(14) = 0.48$, $p = .31$, $M = 6.7$, $SD = 0.6$] or fear [$t(14) = -0.53$, $p = .30$, $M = 5.1$, $SD = 2.0$]. Nevertheless, Professor F appears significantly impaired in the recognition of disgust [$t(14) = -3.55$, $p < .001$, $M = 6.3$, $SD = 0.9$] .

b) *Emotional Expressivity* [BEQ]: Professor F capacity to express negative and positive emotions, as well as the intensity of his emotional reactions, appears to be in normal range. When compared to a sample of 19 non brain-injured subjects of matched age (Gross et al., 1997), no significant differences were found in terms of negative expressivity [$t(18) = 0.73$, $p = .23$, $M = 3.69$, $SD = .85$], positive expressivity [$t(18) = 0.57$, $p = .28$, $M = 5.18$, $SD = .96$] or impulse strength [$t(18) = 0.18$, $p = .43$, $M = 4.5$, $SD = .93$].

c) *Emotional Understanding* [TAS-20]: Professor F capacity to identify and describe emotions appears to be within normal range. Interestingly, he seems to use a rather abstract cognitive style when relating to emotions. When compared a sample of 58 non brain-injured older (Henry et al., 2006), Professor F's scores do not differ significantly in terms of identifying emotions [$t(57) = 0,67$, $p = .25$, $M = 16.1$, $SD = 5.77$] or describing emotions [$t(57) = 0.51$, $p = .30$, $M = 13.4$, $SD = 3.10$]. However, his score on the concrete cognitive style subscale is significantly lower [$t(57) = -3.93$, $p < .001$, $M = 22.9$, $SD = 3.02$].

d) *Emotion Regulation* [ERQ]: Professor F's usage of reappraisal [the capacity to generate positive interpretations of negative events] and suppression [the ability to withhold the expression of emotional reactions] appear to be within normal range. When compared to a sample of 20 non brain-injured older adults (Winecoff et al., 2011), no differences were found in the frequency of use of suppression [$t(19) = 0.26$, $p = .40$, $M = 14.6$, $SD = 5.2$], but marginal differences were observed in relation to reappraisal [$t(19) = -1.39$, $p = .09$, $M = 32.4$, $SD = 4.6$].

e) *Emotional Symptoms* [HADS]: Professor F reported no clinical signs of depression or anxiety. His scores for the Anxiety Scale (score = 4) and Depression Scale (score = 1) were not higher than the proposed cut-off score [< 8] (Olssøn et al., 2005).

f) *Personality change* [ISPC]: According to Professor F's wife, there was a *moderate* change in the Distress Scale ($\Delta = 3$), with high scores of change in the items anxiety ($\Delta = 4$) and vulnerability to pressure ($\Delta = 4$). The Executive Dysfunction/Decision Making Scale presented *mild* levels of change ($\Delta = 1.4$), with lack of initiative ($\Delta = 3$) and indecisiveness ($\Delta = 3$) as the items with highest score. Finally, the Hypo-emotionality Scale also presented *mild* levels of change ($\Delta = 1.3$), with social withdrawal ($\Delta = 3$) and apathy ($\Delta = 2$) as the items with the highest score. Interestingly, blunted affect presented a mild decrease ($\Delta = -1$), suggesting an augmentation of emotional experience. See Table 2 for further details.

Table 2

Summary of Professor's F emotional and personality assessment. Professor's F scores are compared to age matched control samples.

Emotional Components		Professor F	Healthy Controls		
		Score	*M*	*SD*	*p*
Emotion Recognition (FEEL test)	Anger	4	3.6	2.8	.44
	Sadness	5	5.6	1.8	.37
	Disgust	3	6.3	0.9	.001**
	Surprise	5	5.8	1.6	.31
	Happiness	7	6.7	0.6	.31
	Fear	4	5.1	2.0	.30
Emotional Expressiveness (BEQ)	Negative Emotions	4.33	3.69	0.9	.23
	Positive Emotions	5.75	5.18	1.0	.28
	Impulse Strength	4.66	4.5	0.9	.43
Emotional Understanding (TAS-20)	Identifying Emotions	23	16.1	5.8	.25
	Describing Emotions	15	13.4	3.1	.30
	External Oriented Thinking	11	22.9	3.0	.001**
Emotion Regulation (ERQ)	Reappraisal	26	32.4	4.6	.09
	Supression	16	14.6	5.2	.40
Emotional Symptoms (HADS)	Anxiety	4			
	Depression	1			
Personality Change (ISPC)	Distress	Δ = 3.0			
	Executive Dysfuntion/ Decision Making	Δ = 1.4			
	Hypoemotionality	Δ = 1.3			
	Disturbed Social Behavior	Δ = 0.5			
	Obsessive	Δ = 0.0			
	Irascibility	Δ = -0.2			
	Self-centered	Δ = -0.5			
	Self-serving	Δ = -0.5			

Δ = difference between the post injury and preinjury scores.

5.2.4.3 Summary of neuropsychological and emotional findings

In sum, Professor F's neuropsychological profile appears characterized by slow information processing [Speed Processing Index, WAIS III], difficulties with free recall of verbal information [FCRST], difficulties in mental generation [Verbal Fluency, D-KEFS], distractibility [WCST] and mental inflexibility [WCST]. Such profile of impairment is consistent with the executive difficulties that Professor F reports on the DEX (e.g. planning ahead, avoiding distractions, making decisions and acting without thinking) and his wife's report of change on the Executive Dysfunction /Decision Making Scale [ISPC].

It is interesting, however, that not all executive-related abilities appear compromised in Professor F's profile. Both, abstraction [Similarities and Matrix WAIS] and working memory [Working Memory Index, WAIS] appear within the normal range. The possible implications of this discrepancy between damaged and spared capacities for ER would be addressed in the discussion section.

As for the emotional assessment, Professor F appears within normal range in most of the measures of key emotional processes, such as emotion recognition for basic emotions [FEEL], emotional expressivity [BEQ], emotional understanding [TAS-20], emotion regulation [ERQ] and depressive/anxious symptomatology [HADS]. Nevertheless, he does present some abnormalities. For example, he has a low performance in the recognition of disgust [FEEL], and a markedly higher performance in the Concrete Cognitive Style subscale (TAS-20), suggesting a clearly abstract approach to thinking about emotions. This last point is interesting in relation to the preserved abstraction reported by formal tests. There is also a trend suggesting a less frequent use of reappraisal compared to controls [ERQ]. It is important to note that, even though Professor F does not report significant emotional difficulties [HADS], his wife mentions several. For example, she reports that the most marked change in Professor F's personality is in the Distress Scale [ISPC], particularly with

an increase in anxiety and vulnerability to pressure. She also mentions an increase in social withdrawal and a decrease of blunted affect (i.e. increased emotional reactivity).

5.2.4.4 In-depth interview results

A first point that needs to be mentioned, before describing the main themes of the interview, is the impact of Professor F's executive impairment on the interview process. He is able to understand and address questions correctly; however, his train of thought is often fragmented and constantly interrupted by related, but peripheral, contents. This generates a difficulty getting to the point, requiring longer periods of time and external support to answer. This profile appears to be consistent with previous studies that have described patients with left PFC lesions' discourse as vague, disorganized and repetitive (Michael P Alexander, 2006; Coelho et al., 2012).

5.2.4.4.1. Descriptive analysis of themes

The analysis identified four themes related to emotional change, which are presented individually. The theme *Opening to a new dimension* describes how Professor F attributes meaning to acquiring a brain injury, by stressing changes in the emotional domain. *Becoming dysexecutive* addresses his experience of having a disorganized mind, by means of describing his main cognitive difficulties. *Emotional Changes* describes modifications in basic emotional processes, such as emotional reactivity and emotional understanding. Finally, *Emotion Dysregulation and Regulation* draws on Professor F's difficulties regulating negative emotional states and on the intrapersonal and interpersonal mechanisms he has discovered to overcome such problems.

Theme one: 'Opening to the emotional dimension in me'

According to Professor F the main change that he perceives is the emergence of a new 'dimension', from which he now relates to himself and to the world. This aspect is referred by him as the *emotional* dimension:

> [A1] The impact of the accident in me has been multiple. First is this thing of being opened to a new world. This is related to what I just said, I thank the accident because deep down I start to perceive in myself, to perceive from reality, things that I did not perceive before. I mean, ways of relating, of being with myself, ways that I did not have before. This has been the strongest change…to be now beginning to grasp the emotional thing, the emotional dimension in me.

The predominance of the emotional dimension implies a personality change, a re-arrangement of the way in which cognition and emotion used to interact. To him, emotions are not shadowed by reason any more, and being rational appears as something from the past:

> [A2] I have always had this [emotional] dimension, but I do not know why in hell…I do not know…that dimension…or that aspect of my life … was silenced. Emotions were hidden by rationality … with the accident, this intellectual dimension, the rationality, this serious way of being that I had…that was lost. I do not have to be serious to anyone, because I can't…because to be serious would be to read a thesis, to properly write a thesis, intellectually speaking […] with the accident… there are two things, one is that I can't do it anymore, and the other one is that I do not want to do it anymore.

The re-arrangement of the rational and emotional dimensions has not only transformed the way he relates to himself, but also how he relates to others. According to him, others notice this change, and he notices that he relates to them differently:

[A3] The change that others see in me is that…that the Professor F, who was responsible and serious…like a brain to them…he is not that one anymore. I had a reputation of being a serious guy. Now at the university I relate to people very differently, I'm friendlier, more waggish, supporting them, caring for them…not about what they have to do or study…that really makes me happy…I see that when I get to the University in the morning and I talk to them, as an equal. I wouldn't have done that before.

Theme two: 'Becoming Dysexecutive'

Along the years, Professor F has gained awareness of the cognitive difficulties left by the brain injury, mostly by realizing its impact on his professional life. When questioned about it, he refers to mental slowness and mental inflexibility as the main problems:

[B1] I used to articulate theory, to make stories…that is what I used to like. Well…and that yet…I don't feel like playing that game anymore…because I do not have the capacity to do it, the required speed, creativity…many things are slower now…I know I am creative, but I'm creative in other ways, in things that were unknown to me before.

In relation to problems of mental flexibility he comments:

[B2] Every situation…work…to figure out what to do in life…is a muddle. Situations where change is required… I do perceive those changes, but I do not manage to know how to react to them. Technological, political and cultural changes….they belong to completely different levels, but the sum of those levels is what confuses me.

Professor F acknowledges that cognitive deficits compromise thinking and introspection, particularly in situations where he needs to think about something on the spot:

[B3] Something really unpleasant is not being able to argue. In other words, not being able to think. To really try to think…realizing that what other people are saying is

> nonsense… that I had arguments to reply to that, but that I was not able to. That is really unpleasant… wanting to think but not being capable of doing so.

It is interesting to note that the inability to think is often associated with the experience of his mind going blank, with no contents. In such moments of 'silence', as he likes to call them, active thinking is replaced by contemplation:

> [B4] I have these spaces… that I often call spaces of silence…quiet spaces, silent ones. And my mind often goes blank in those moments, without any explicit content…without any reflexive content…but with truths and emotional certainties. This often occurs during praying…with my affection towards my wife, my kids. What I try to do then is to remain silent and contemplate. If you ask me contemplate what? I do not know. It is some form of empty contemplation, with no images, but certainly it has reality.

The subjective experience of Professor F during these moments of silence is that of heightened bodily sensations and peace:

> [B5] In those moments of silence I do not look at anything, but I feel a great serenity. They are moments of pure sensation. For example, often when my wife works and the news is on…I often disconnect myself from the news, because I get tired and bored. But I stay there quietly. Then I am just feeling. I have learnt to sense that at those moments I can pay attention to what I feel. And because before I did not know how to do that…now I like it. I like it and I have become somehow addicted to those spaces, where I can just stay like that. In the car… for instance… when we travel with my wife to the north…sometimes there is music playing in the car…but I stay in that space … which was occupied before by reflection…and now is simply being there.

Theme three: 'Emotional Changes'

One of the most remarkable changes reported by Professor F is an increase in emotional *reactivity* for positive and negative emotions. According to him, this is related to a higher variation of emotional intensities, which boosts his capacity to be emotionally moved by events.

> [C1] Happy feelings …the accident did not reduce them, but on the contrary, it augmented them. The capacity to feel moved, the capacity to cry […] now I experience these things where I am happier, or sadder, or that I have variations on intensity. That did not exist before. Because now I feel things, and this is part of my life. The accident made me more sensitive to that.

The subjective experience of increased emotional reactivity appears to depend on which type of emotion is heightened. In the case of positive emotions, this seems to generate feelings of wellbeing and satisfaction, as well as to facilitate the capacity to empathize with other's people happiness:

> [C2] I am certainly happier than before. That is what my friends tell me, everybody does. Since the accident…more satisfied…I do not know about what…I do not know if it is related to something…it is just that I live more happily. Sometimes I have these low moments, but in general the experience is that I feel happy […] yesterday I realized I was feeling happy because my son accomplished something at work. There I noted something different. I used to think 'well, this is not my businesses'. But to feel 'how great that he did good'…well I noticed there something unusual, like surprising myself. I did not have that register before.

The increased intensity of negative emotions, on the contrary, appears to have a different effect. Professor F describes that in moments where he experiences such emotions he feels bewildered, with no control and somehow trapped:

[C3] For example, this happens when I feel nervous. It is a very ugly sensation that one of being suddenly perplexed…of feeling really uneasy about a situation that you can't control, something you can't react to. It is a feeling of instability, of danger […] I have the feeling that this intensity…that I feel this intensity with more intensity now. And that is why…especially in relation to sadness…the intensity pulls me to be sadder than I am.

Professor F reports that he has no difficulties naming the emotions he feels [emotional labelling]. However, he recognizes that understanding why he feels that way [emotional understanding], particularly when aroused by negative emotions, is highly taxing:

[C4] For example, there are times when I have been sad and I do not know why. When you are there is like… like a little oppression…like something obscure…like an oppressive feeling and darkness, because I enter in something dark without knowing why. Then is where the confusion appears, because at that point I start looking for reasons.

During moments of high negative arousal, where thinking is compromised, Professor F describes to experience something like a mental block. This experienced is a vicious cycle where negative emotions, and unpleasant bodily sensations generated by the disorganized mind render Professor F powerless:

[C5] The mental block is something in the head. There are two things that I feel in the head, sadness and tiredness. I feel that like a stitch in the top of my head. Then it is a real struggle to think. The mental block is like a little stitch, a physical sensation. For example that time at the meeting…the first thing that I felt was an increase in blood pressure. My face got really red…I noticed that I was feeling hot and that the other guy was talking…and I also started feeling a headache…which I rarely do…then a stomach discomfort. It is so unpleasant not to be able to think. It was like a fight with myself '*How come you are not able to say something…you…Professor F!'* Because at that

> point I thought I knew the truth…that I was the authority… So it was an awful mix of physical sensations…of powerlessness. That is the word…to feel powerless…in every way you could imagine.

Theme four: 'Emotion Dysregulation and Regulation'

The mental block might be considered an episode of emotion dysregulation, where impaired cognitive resources do not allow the self-modulation of negative affect. Professor F exemplifies this when describing his inability to voluntarily shift away from sad states, which he calls the 'sticky sadness':

> [D1] Having difficulties walking is something that saddens me. But then I have this experience that… well…I know is ok to feel sad, but this is not just feeling sad, this is like a sticky sadness, which makes me feel sadder than I should. That is really, it is like when you step in the mud…so you need to make an effort to lift your foot, and in order to lift your foot you have to clean it too. I can't get out of the mud by myself […] it's not a matter of intensity here, but the sensation of a weight, of mud, of something that grabs your foot. I'm afraid of the mud, because it is so sticky that even my capacity to imagine collapses.

Professor F comments that it is only thanks to external help that he manages to come out of such persistent negative states. It is his wife who helps him to generate an external dialogue, which allows him to consider alternative ways of thinking about his feelings:

> [D2] When I'm in the sticky sadness it's very difficult to change the way I think. There, my wife is of great help. She is the one that pulls me out of such states. Because when I'm feeling sticky it's written all over my face. So she starts asking questions: *What is wrong? why do you feel like this?* She does it until I'm able to come up with an answer. And she always asks me for the other side…she says: *We have moved forward on this…you are walking well enough…if we need to do more exercise we will do it*. It is as

> if she helps me to get out of that place, by putting another image in my mind…like those old machines where you pass slides [slide projectors]. She removes the bad slide and puts in a good one. She removes one and says… *now look at this one…instead of using those specs use this ones*. So then I start to see that new one. If you ask me if I can change the slide by myself…I don't think so.

According to Professor F, it is the help provided by his wife, in the form of questions, which facilitates the generation of an external dialogue, thus compensating for his inability to internally think about what he feels:

> [D3] To ask me questions, questions that unfold me…that allows me to reflect upon me, to see myself. It is like putting it out there so you can see it as an image. My wife asks me: *Why are you sad? What happened to you?* She asks me questions, and those questions help me to put what I'm feeling out and think about it. It is a form of putting my head in order. There is something cognitive there…the fact that there is another possible way of arranging the same pieces. And by re-arranging them you can breathe. I can't do that by myself.

Such conversations can be later remembered by Professor F, thus activating internal thoughts:

> [D4] My head works with the memory of these conversations. It is weird. For example yesterday you spoke with my wife and then I talked to her about it…so we discussed how we could help each other more. With that memory new things appeared in my mind. What I was telling you before, about work…that appeared when I was in the shower today…in relation to what my wife had been talking with you. I do not know how that link was made, but when I woke up it was there, so I kept thinking about it

Even though Professor F has important difficulties modulating how he feels in a moment of dysregulation, he reports that he has developed strategies to avoid such situations.

These typically consist of detecting the preliminary signs of a possible 'mental blockage', so that current behaviour can be stopped. He comments on this point:

> [D5] There was a second meeting where I really managed the situation. I said to myself *here, F, you shut your mouth! Stay out of this!* I even managed to have a good time, by talking and listening to what other people were saying. But there was another meeting where there was really a huge discussion, and even though I knew I had to stay away from it, I got carried away. But I managed to notice when things were starting to go wrong…The turning point was when I felt my mouth dry…so I realized I was entering into something bad…something turbid…so I realized I had to disconnect myself.

Another strategy that he has developed to deal with the mental block, especially during interpersonal conflict, is writing. He describes different uses of writing. The first refers to the writing of his thoughts after experiencing the mental block, as a form of resuming a discussion or argument. The second refers to the organization of his thinking beforehand, so that a situation that may potentially generate an episode of mental block can be contained:

> [D5] I remember that I had this big argument with my sons. During the argument I felt confused and couldn't think. So after that I wrote my thoughts about how I felt at that moment and why I felt so bad. And then I read what I wrote to them. So writing was a good strategy because the problem was that I couldn't argue properly. […] Now, for the community meeting I also write my thoughts, when we have to talk about ourselves in front of the group. I write my ideas all the time. That helps me…because if I have wanted to say them from the top of my head…as once I tried to…good bye! That does not work.

5.2.4.4.2. Thematic map of analysis

As reported above, two main themes related to emotional change are described by Professor F during the interview. The first one is *emotional reactivity* [see Fig. 2], which refers to an

increase in the intensity of experienced positive and negative emotions. This augmentation of felt experience is associated by Professor F to a decrease in thinking abilities, particularly his capacity to generate ideas. Professor F exemplifies this with the metaphor of having a blank mind, or mind without contents. Interestingly, the increased reactivity for positive and negative emotions appears to trigger quite different psychological processes and self-states. Positive emotions are experienced as an increase in somatic sensations, which becomes the focus of contemplation. The self-state generated by this dynamic is one of wellbeing. The increase of negative emotions, on the contrary, generates a mental block, which further increases negative arousal and dysregulation. The self-state generated by this dynamic is one of powerlessness.

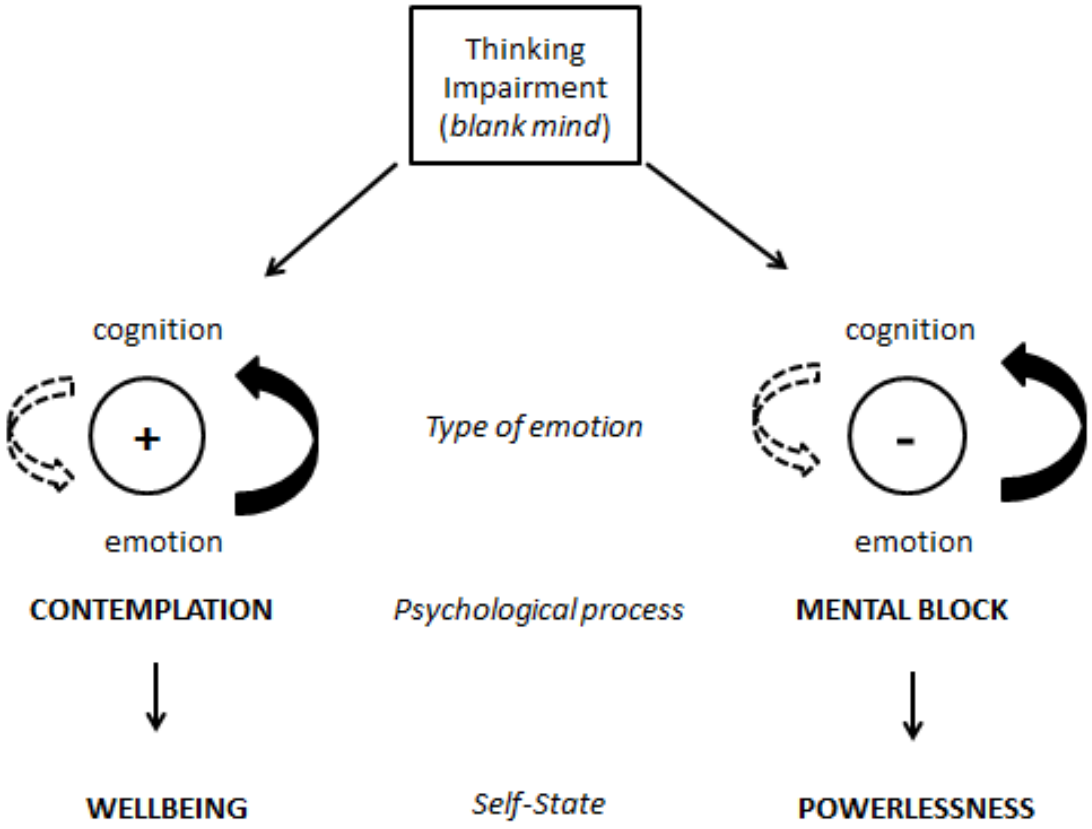

Fig 2. *Emotional Reactivity thematic map*. The figure describes the differential impact of thinking impairment [blank mind] on the experience of positive [left] and negative [right] emotions. In both cases, thinking impairment generates an imbalance between cognition and emotion. Nevertheless, it triggers quite different psychological processes, and creates radically opposing self-states.

The second main theme described by Professor F is *emotion dysregulation and regulation* [see Fig 3]. It could be considered that this theme is an amplification of the dynamics generated by the mental block [dysregulation]. However, it extends beyond that by also describing how such states can be modulated. The basic idea here is that the interaction of executive impairment and increased emotional reactivity generates a difficulty to use thinking as a tool to modulate negative emotional states. Such inability to use one's own mind to think about one's own feelings increases even further the negative arousal, thus compromising even more the already debilitated executive capacities. In consequence Professor F becomes stuck in the negative emotion, a situation that he describes as the sticky sadness. Nevertheless, it appears that in such moments of emotional perseveration emotions can still be regulated, but from outside. This seems to be accomplished by his wife's scaffolding of executive abilities, which promotes reflective capacity and mental flexibility.

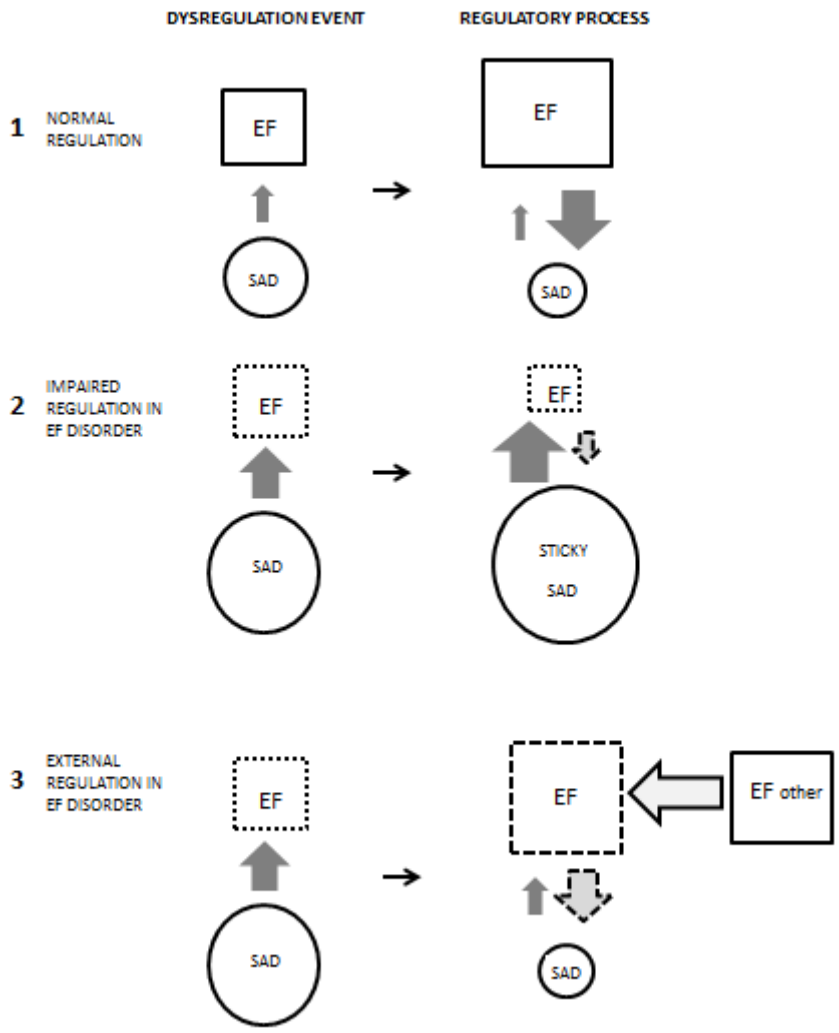

Fig 3. *Emotion Dysregulation and Regulation thematic map*. The figure describes the processes by which emotion is dysregulated and regulated, using Professor F's example of the 'sticky sadness'. [1] In persons with no brain injury [left] the experience of an intense negative emotion impacts executive functions (EF), which demands a further strengthening of executive control, thus down-regulating the negative state [right]. [2] In patients with executive impairment, the baseline situation is unbalanced by default: EFs are less efficient and emotional reactivity is higher [left]. In consequence, there is a larger impact of negative emotions on EF, thus compromising even further its regulatory capacity. The negative emotion, in this case sadness, is therefore experienced with greater intensity and persistence: a 'sticky sadness' [right]. [3] The emotional state of patients with executive impairment can, nevertheless, be modulated externally, by relying on the EF of another. In the case of Professor F, such scaffolding process attains the down-regulation of sadness.

5.2.5. Discussion

For many decades neuropsychologists have tried to elucidate the mechanisms that underlie emotional change after brain injury. Such a task has not been simple, for a range of important theoretical and technical reasons (as discussed in the Introduction). While there have been some case reports of non-dysexecutive patients describing their subjective experience of change, this study is the first one to portray such transformations in a patient with executive impairment, a population that has been often considered as challenging because of deficits in reflective capacity. However, Professor F is remarkable in that, partly because of his excellent premorbid abilities, and also because of the preservation of some executive capacities related to insight [abstraction and working memory], he was able to offer an extraordinarily detailed description of his experience of change. The novelty of this account is also technical, in that it uses a mixed methodology to evaluate a range of well-known emotional processes.

5.2.5.1. What type of dysexecutive patient is Professor F?

A first point that needs to be clarified is that Professor F's profile of dysexecutive impairment is more in the domain of *thinking* than in the domain of *behavioural* disinhibition. The three major difficulties reported by him during interview were *mental slowness*, *mental inflexibility* and *lack of fluidity* [*B1, B2, B3*][25]. This is consistent with his neuropsychological assessment, which suggests his main deficits to be: slow information processing [WAIS], mental inflexibility [WCST] and difficulties in word generation [D-KEFS]. In addition, Professor F's description of everyday problems [DEX] emphasized deficits of executive *cognition* (e.g. planning ahead, avoiding distractions, acting without thinking) but not behavioural regulation. Professor F's wife offers further supporting evidence on this issue. She reports (in

[25] The codes refer to the interview's paragraph from where information has been taken.

the Iowa Scale of Personality Change) modifications on the Executive Dysfunction/ Decision Making dimension ($\Delta = 1.4$), but almost no change on the Disturbed Social Behaviour dimension ($\Delta = 0.5$). The location of Professor F's lesion is consistent with this distinction, the damage involves the lateral and superior medial surfaces of the left prefrontal cortex, both areas commonly associated with *cognitive* impairment rather than *behavioural* impairment (Anderson & Tranel, 2002; Eslinger et al., 2013; Tim Shallice, Stuss, Picton, Alexander, & Gillingham, 2008; Stuss & Alexander, 2007).

An interesting feature of Professor F's profile of executive impairment is his relatively preserved capacity for abstraction, with scores in the average or above average range [Similarities and Matrix Reasoning, WAIS]. It is possible that this preservation of abstraction ability, along with his excellent premorbid intellectual capacity, are the elements that allow him to offer such an insightful account of his emotional changes. It is also interesting to note here that, compared with people without brain injury, Professor F has a significantly lower score on the External Oriented Thinking scale [TAS-20], suggesting a highly introspective or abstract cognitive style. From a neuroanatomical perspective, a likely explanation is that Professor F's lesion did not compromise rostral areas of the PFC [e.g. BA 10], known to play a key role in abstract forms of cognitive control (Badre, Hoffman, Cooney, & D'Esposito, 2009; Christoff, Keramatian, Gordon, Smith, & Mädler, 2009; Petrides, 2005).

5.2.5.2. Increased subjective emotional reactivity: The balance between feeling and thinking.

A main emotional change reported by Professor F is the increase in emotional reactivity for positive and negative emotions. In his interview Professor F suggested that the accident augmented the intensity of 'happy' and 'sad' feelings [*C1, C2, C3*]. This phenomenon is also reported by his wife in the Iowa Scale of Personality Change, where she mentions a decrease

of blunted affect (Δ = -1). An interesting point here is that, to Professor F, such change is closely related to the 'inability to think' or the 'blank mind' [*B5*]. In other words, Professor F's proposal is that his inability to generate thoughts –in relation to a felt emotion- modifies the intensity level with which such emotion is experienced. This observation is especially interesting because is consistent with the idea that emotional changes after dlPFC lesions are *secondary* to the cognitive deficits generated by such damage (Anderson & Tranel, 2002). Professor F's subjective account may offer some preliminary evidence to support such claim. One should always be cautious about how to interpret subjective reports; however, Professor F's description of increased reactivity is provocative because it may allow us to observe the interaction between emotion and cognition. It suggests that the somatic aspect of an emotional response is influenced by the mental representations originated from such visceral states. In other words, when the representational machinery is ineffective [the blank mind], somatic states become less constrained and acquire a heightened quality. This is perhaps what Professor F refers to when reporting that his emotions are no longer 'silenced' or 'hidden' by rationality [*A2*], or when describing that in moments with no thinking [moments of 'silence'] there is 'pure' sensation [*B5*]. This interpretation of the data is consistent with our increasing awareness of a dynamic balance between cognitive control areas [PFC] and emotion generation areas [subcortically] (Knight, Staines, Swick, & Chao, 1999; Solms & Panksepp, 2012; Thayer & Lane, 2000), a balance that can be altered after damage to neocortical structures [see fig 1]. Another source of evidence that appears to support Professor F's description comes from studies exploring the impact of thinking-about-feelings [or affect labelling] in emotional reactivity. Recent work suggest that labelling has a dampening effect on both positive and negative emotions (Lieberman, Inagaki, Tabibnia, & Crockett, 2011), presumably by inhibiting the activity of subcortical structures in charge of emotion

generation (Creswell, Way, Eisenberger, & Lieberman, 2007; Hariri, Bookheimer, & Mazziotta, 2000; Lieberman et al., 2011; Payer, Baicy, Lieberman, & London, 2012).

Professor F's enhanced capacity to experience emotions taps directly into one of the most relevant issues in affective neuroscience today, which is the debate about the role of the neocortex in emotional processes. For decades, neuropsychologists have studied patients with focal brain lesions to shed light on this matter, investigating whether damage to selective cortical areas compromises the capacity to *experience* discrete emotions. Typical examples of this approach have been studies on hemispheric laterality and the processing of positive and negative emotions (Borod et al., 2010; Demaree, Everhart, Youngstrom, & Harrison, 2005; Shenal, Harrison, & Demaree, 2003), as well the recognition of disgust and the insula (Calder, Keane, Manes, Antoun, & Young, 2000). This opinion has also been supported by a range of physiological studies (Craig, 2010; Craig, 2008; Craig, 2009; Davidson & Irwin, 1999; Davidson, 1992a, 1992b, 1998). The main idea of this approach is that damage to the neocortex impairs the *processing* of emotion, a view that has been often interpreted as compromising the *experience* of emotions.

This approach to the problem has recently been challenged by case studies reporting that emotional experience is largely *preserved* after cortical damage (Turnbull, Evans, & Owen, 2004, Damasio, Damasio, & Tranel, 2012; Feinstein et al., 2010), and also after cortical atrophy (Merker, 2007). Indeed, even patients with bilateral lesions to subcortical structures [amygdala] traditionally assumed to participate in emotion generation [fear] still appear to experience such emotions in specific contexts [survival threat] (Feinstein et al., 2013). Instead, it has been argued that emotion is generated not by cortical areas, but rather by *deep* subcortical structures, with the neocortex playing its principal role in emotion regulation (Damasio et al., 2000; Holstege et al., 2003; Ochsner & Gross, 2007; Panksepp, 1998, 2011).

If Professor F's emotional change cannot be characterized as an inability to *experience* an emotion, it remains to be answered how such change might be defined. An alternative approach is to suggest that cortical injuries do not compromise the capacity to *experience* emotions, but they may alter the *dynamics* of such experience. Emotions dynamics have typically defined as the response parameters that modify the quality [the magnitude and temporal features] of emotional behaviour: such as intensity, range, lability, latency, rise time, recovery or persistence (Thompson, 1991). In the case of Professor F, for example, it appears that his executive impairment produces –indirectly mediated by cognition- a change the *intensity* [increased in magnitude] and *recovery* time [e.g. sticky sadness]. The perspective of emotion dynamics offers the possibility to disentangle the role of different cortical areas, offering a window into the ways in which the dynamic landscape of the emotional response may change.

5.2.6.3. Emotion dysregulation, reappraisal impairment and neurologically induced rumination

Another emotional change reported by Professor F is his difficulty in self-regulating negative emotions, particularly sadness, by the use of thinking. He describes that the increased intensity of negative emotions impacts his capacity to react to them [*C3*]. In such moments, Professor F comments, he experiences a 'mental block' where 'it is a real struggle to think' [*C5*]. This mental state should be viewed not merely as a cognitive block, given that it is [in his account] also a mixture of felt emotion, unpleasant physical sensations and fatigue [*C5*]. When in this state he remains able to recognize what he is feeling [*D1, I know it is ok to feel sad*]. However, his ability to understand the cause of this feeling seems beyond his grasp [*C4*]. The final consequence of this dysregulatory process is that the initial emotion is experienced more intensely and persistently [*D1, this is like a sticky sadness, which makes me*

feel sadder than I should], together with the fact that he is unable to get out of such states without external assistance [*D1*, *I'm afraid of the mud, because it is so sticky that even my capacity to imagine collapses*].

The idea that thinking helps people to modulate how they feel has history (Freud, 1946; Freud, 1964[1910]; Janet, 1909). During the last decade there has been a substantial research effort on the topic. Studies have particularly focused on how people regulate their emotions, especially by changing the way they think about negative events, a capacity often described as cognitive change or reappraisal (Gross & Thompson, 2007; McRae, Ochsner, & Gross, 2011; Ochsner & Gross, 2004). It is interesting to consider Professor F's regulatory impairment from this perspective, in other words, to view his 'inability to think' as a difficulty generating, and shifting to, more positive reinterpretations. This explanation seems supported by the fact that Professor F's two main executive deficits [fluency and set shifting] have been described as key components of reappraisal ability (Mcrae et al., 2011). Furthermore, Professor F's score on the reappraisal scale of the ERQ (Gross & John, 2003) is relatively low [$p = .09$], suggesting that he uses cognitive change less frequently than people with no brain injury. Consistent with this, Professor F's lesion encompasses the high order language and cognitive control areas in the left PFC that are commonly recruited during neuroimaging studies on reappraisal (Goldin et al., 2008a; Green & Malhi, 2006; Kalisch, 2009; Kim et al., 2011; Ochsner & Gross, 2007; Ochsner & Gross, 2005; Ochsner et al., 2004, 2002; Vanderhasselt et al., 2012).

Professor F's self-regulatory impairment can be further understood if we consider his emotional experience during the 'mental block' as a neurologically-induced ruminative process. A phenomenon reminiscent of the rumination of some psychiatric patients, Professor F remains focused on the negative emotions in a *repetitive* and *passive* way, staying somehow *fixed* on the feelings states, and unable to do anything about them (Nolen-

Hoeksema, 1991). Professor F and such ruminative patients are alike, in that both present with an inflexible cognitive style that interferes with active problem solving (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Thus, both are unable to disengage from affective states (Genet, Malooly, & Siemer, 2012; Joormann & Gotlib, 2008; Watkins, 2008), which leads to the prolonged activation of negative content in working memory (Genet et al., 2012) and the exacerbation of negative affect (Joormann & Siemer, 2011).

There is, however, an important distinction between such ruminative patients and Professor F. Psychiatric patients exhibiting rumination are often rather productive in generating repetitive [negative] thoughts (Genet et al., 2012), while Professor F generates considerably less contents [the blank mind]. This distinction is interesting in the view that ruminative thoughts in psychiatric patients tend to dwell on the causes and consequences of current feelings (Nolen-Hoeksema et al., 2008), a mental state that Professor F reports as highly taxing to generate [*C4, I enter into something dark without knowing why, then is when the confusion appears*]. A possible result of his not being able to think about causes and consequences, when in such a state, may well be the impossibility of attributing meaning to negative experience [*C4, I don't know why I feel sad*], thus adding further to his confusion and distress.

5.2.5.4. Emotion dysregulation and emotional symptomatology

There is substantial evidence suggesting that emotion dysregulation lies at the core of a range of psychopathological symptoms (for reviews see Kring & Sloan, 2010; Kring, 2010; Werner & Gross, 2010). In consequence, it is interesting to consider whether or not Professor F's regulatory difficulties are related to the presence of emotional symptomatology. Data from his emotional assessment [HADS] suggests that he does not present with clinically significant levels of anxiety [score = 4] or depression [score = 1]. However, in relation to anxiety, he

does reports some symptoms, such as feeling tense 'from time to time', experiencing worrying thoughts 'a lot of the time' and having 'mild difficulties' in relaxing. As for depression, he merely reports that he does not enjoy experiences 'quite as much' as he used to do.

Professor F's self-report is especially interesting when contrasted with his wife's assessment of his personality change [ISPC]. She *does* suggest an increase in symptomatology. From her perspective, the personality dimension with the highest score was a moderate change on *Emotional Distress* ($\Delta = 3$), which measures the presence of negative emotionality (Barrash et al., 2011). Interestingly, and following a similar trend to the anxiety symptoms reported by him on the HADS, Professor F's wife rated him with *moderate* levels of change in anxiety related items [*Anxiety* ($\Delta = 4$), *Vulnerability to Pressure* ($\Delta = 4$)], and *mild* levels of change in depression related items [*Depression* ($\Delta = 2$)]. This are of course self-report measures, should be treated with caution. However, it seems clear that there appears to be some inconsistency between the reports of Professor F and his wife on measures of emotional symptomatology.

The increase in anxiety, reported by Professor F's wife, is consistent with his own description of the emotional experience associated to his executive impairment. His inability to think, *the mental block*, is often accompanied by substantial levels of anxiety, which makes him vulnerable in cognitively demanding situations [*C3, When I feel nervous…is an ugly sensation that one of being suddenly perplexed…of feeling really uneasy about a situation that you can't control, something you can't react to*]. This suggests that the impaired executive function is the *cause* of the change in emotional state. Of course the direction of causality may be reversed, with anxiety serving as a cause of the mental block. Notably, however, when the executive impairment can be resolved by compensation [by the action of an external agent] this immediately results in a reduction in anxiety [see section below on

intrinsic and extrinsic emotion regulation strategies]. This description is also in line with evidence suggesting a regulatory role of executive functions in the production and management of anxiety (Olssøn et al., 2005). In addition, it is interesting to note that Professor F's anxious reaction is quite similar to the long recognized 'catastrophic' reaction, as an emotional response of anxiety and confusion experienced by people with brain injury, when they fail to use their cognitive resources to respond to environmental demands (Hendrawan, Yamakawa, Kimura, Murakami, & Ohira, 2012; Vytal, Cornwell, Arkin, & Grillon, 2012).

According to both, Professor F and his wife, his depressive symptoms are not as disruptive as anxiety symptoms. However, the emotion that Professor F reports as most difficult to regulate is sadness [*D1* 'sticky sadness']. It is interesting that while Professor F struggles to self-regulate sad events, such episodes do not escalate to a fully fledge depression. Why this might be? Firstly, Professor F's premorbid personality may operate as a protective factor, somehow buffering the impact of emotion dysregulation after brain injury. This seems probable, given that well established that pre-morbid personality is a key element in socio-emotional adjustment after brain injury (Aben, 2002; Greenop, Almeida, Hankey, Van Bockxmeer, & Lautenschlager, 2009; Storor & Byrne, 2006).

Secondly, as it will be discussed below, Professor F's dysregulatory episodes are often externally modulated by his wife, thus limiting their time length and impact. This observation is consistent with a range of evidence suggesting that the availability of a supporting environment is crucial in facilitating emotional adjustment (Hammond et al., 2012; Sady et al., 2010; Schönberger, Ponsford, Olver, & Ponsford, 2010)

Thirdly, Professor F's emotional changes are not restricted to the increase of negative emotions, and its dysregulatory impact, but they also extend to the amplification of positive emotions [*C1*]. This is interesting in view of a literature suggesting that the experience of

positive emotions buffers, and even undoes, the impact of negative affect (Fredrickson & Levenson, 1998; Fredrickson, Mancuso, Branigan, & Tugade, 2000). In other words, Professor F's increased reactivity to positive emotions may act as a regulatory influence.

Finally, it is important to consider that the frequency of emotion dysregulation episodes also depends on the success of coping strategies. Professor F offer several examples of using avoidant coping strategies to control the possible impact of challenging situations [*D5, I say to myself here, F, you shut your mouth! Stay out of this!*]. This is consistent with evidence suggesting that avoidant coping is a common strategy among brain injured patients (Krpan, Levine, Stuss, & Dawson, 2007; Krpan, Stuss, & Anderson, 2011a, 2011b). Notably, such strategies, despite reducing the range of interaction with the environment, appear to enhance the patient's capacity to react to it (Goldstein, 1995; Hanfmann, Rickers-Ovsiankina, & Goldstein, 1944; Krpan et al., 2011b; Riley, Brennan, & Powell, 2004; Salas, 2012).

5.2.6.5. Intrinsic and extrinsic ER strategies: The use of social speech and antecedent-focused ER strategies.

Professor F is a remarkable example of how both intrinsic and extrinsic forms of emotion regulation are *functionally* related (Gross & Thompson, 2007). A clear example of this is that, when intrinsic ER is not possible, Professor F defaults to interpersonal regulatory strategies [*D2*]. These might be classified as taking the form of affective and cognitive engagement (Niven, Totterdell, & Holman, 2009). Niven and colleagues (2009) define affective engagement as the efforts directed to try to improve the way the 'target individual' [Professor F] feels about a situation. Two examples of these strategies might be Professor F's wife's approach of active listening [*D2, So she starts asking questions? Why do you feel like this?*], and her positive attitude emphasising his achievements [*D2, we have moved forward on this…you are walking well enough*]. Cognitive engagement, on the other hand, is usually

defined as the efforts to change the way the 'target individual' person thinks about a situation (Niven et al., 2009). This can be observed in Professor F's wife's attempts to generate positive reinterpretations of negative events [*D2, And she always asks me for the other side*].

From a neuropsychological point of view it is interesting to consider the relationship between Professor F's executive deficits and his wife's regulatory interventions. A better insight into how these processes are linked might shed some light on the mechanisms by which intrinsic regulation failure is compensated by extrinsic regulation.

According to Professor F the first step in the extrinsic regulatory process is when his wife starts asking questions to understand what is going on [*D2, What is wrong? Why do you feel like this? She does it until I'm able to come up with an answer*]. Such basic questioning allows him to generate a rudimentary schema, from which more complex meaning can be built [*I am feeling X because of Y*]. The questions offered by his wife facilitate the *generation* of ideas, thus re-activating a reflective process that was frozen because of executive impairment [*D3, To ask me questions, questions that unfold me…that allow me to reflect upon me, to see myself*]. The use of such questions as a form of 'breaking down the stimuli', or to clarify a narrative, has been described before by Luria, when discussing the restoration of active thinking after brain injury (Luria, 1963[1948] p. 213). An alternative, and probably complementary, way of understanding this point is that questioning allows some form of *detachment* from immediate experience [*D3, questions that allow me to see myself*]. There is some evidence supporting this interpretation. For example it has been shown that questioning [in the form of prompting] dramatically improves the performance of concrete patients who struggle to detach from emotional stimuli (Salas, Gross, Rafal, Vinas-Guasch, & Turnbull, 2013). It has been also suggested, in neuroimaging studies, that the self-referential talk generated by questioning, activates the same brain areas that are lesioned areas in Professor F (Geva et al., 2011; Jones, 2009; Morin & Hamper, 2012).

Another particularity of the extrinsic regulatory process in which Professor F and his wife engage is that it occurs *externally* in the space *between* the two mind/brains [*D3, It is like putting it out there so you can see it as an image. She asks me questions, and those questions help me to put what I'm feeling out and think about it*]. Professor F needs to say aloud what he thinks, or to hear it from his wife, as a way of perceiving [hearing] himself. This is a well- established neuropsychological concept. The externalization of internal speech was extensively described by Luria, principally as an strategy that facilitates the regulation of behaviour in patients with left PFC lesions (Luria, 1959, 1966 pp. 250-265). Recently, it has been suggested that externalizing voiced thoughts, or thinking aloud, may function as a cognitive tool that engages with internal dialogue, thus facilitating problem solving (Rees & Skidmore, 2011). In sum, it can be argued that a core part of the extrinsic regulatory process used by Professor F is based on the use of early evolutionary forms of thinking, which are both social and inter-mental (Leont'ev, 1979; Luria, 1963[1948]).

Professor F's dialogue with his wife also appears to compensate for executive impairment by *organizing* his thinking, and facilitating the mental *manipulation* of information [*D3, It is a form of putting my head in order. There is something cognitive there…the fact that there is another possible way of arranging the same pieces*]. Such a new arrangement suggests the inhibition of current negative appraisals, and the change of attentional focus towards more positive mental representations [*D3, It is as if she helps me to get out of that place, by putting another image in my mind. She removes the bad slide and puts in a good one*]. In consequence, it can be argued that Professor F's wife scaffolds two basic executive processes that are compromised in him: set shifting and fluency. These observations are consistent with a range of clinical and experimental data suggesting that cognitive impairment can be modulated by interpersonal and contextual influence (Bowen, Yeates, & Palmer, 2010; Freed, 2002; Salas, 2008, 2009, 2012; Ylvisaker & Feeney, 1998).

A point that remains to be discussed is the long term influence of Professor F's wife interventions. In other words, do these new mental structures, which are conjointly constructed, persist in time? Or more simply put, does this extrinsic regulation process become intrinsic? Professor F does report some form of internalization, described by him as remembering conversations [*D4, My head works with the memory of these conversations. It is weird. For example yesterday you spoke with my wife and then I talked to her about it…so we discussed how we could help each other more. With that memory new things appeared in my mind*]. However, such recollection does not appear to be voluntarily guided, because mental representations appear rather unexpectedly [*D4, what I was telling you before, about work…that appeared when I was in the shower today…in relation to what my wife had been talking with you. I do not know how that link was made, but when I woke up it was there, so I kept thinking about it*]. This phenomenon may be related to his difficulties freely retrieving information. In sum, it appears that Professor F does internalize, at some level, the episodes of extrinsic regulation, which he can access to, more or less voluntarily. However, the question of how such schemas are triggered still remains to be answered. It is possible that such encoded mental schemas may be more easily activated by emotionally relevant stimuli, as it has been suggested by Ylvisaker and Feeney (2000) when describing the advantages of implicational coding after brain injury.

One last observation needs to be made regarding Professor F's intrinsic regulatory capacities. Even though he struggles to manage dysregulation events on the spot, he has developed a series of alternative strategies. As mentioned before, he has learnt to deal with situations that typically trigger catastrophic reactions by completely avoiding them, or by changing his behaviour when it is leading to dysregulation *[D5, There was a second meeting where I really managed the situation. I said to myself here, F, you shut your mouth! Stay out of this!].* This is interesting in that avoiding situations resemble an ER strategy known as

situation selection, while changing behaviour to alter the emotional trajectory of a situation is often defined as *situation modification* (Gross & Thompson, 2007; Werner & Gross, 2010). It is possible that, because he is not able to use cognitive resources optimally during moments of negative arousal, Professor F has enhanced the use of *antecedent-focused* ER strategies, which are implemented before the emotional response is triggered (Gross & Muñoz, 1995).

Another important strategy developed by Professor F to cope with dysregulation is writing. He describes to use writing as a form of organizing his ideas before attending to a situation where he has to express himself, thus preventing the collapse of his executive abilities by having to think on the spot *[D5, Now, for the community meeting I also write my thoughts…That helps me…because if I have wanted to say them from the top of my head…as once I tried to…good bye! That does not work].* He also uses writing as a form of organizing his thoughts after a dysregulation episode, as a way of re-gaining, *a posteriori*, some form of control of the situation. This is particularly important when a core aspect of his identity is threatened [*D5, I remember that I had this big argument with my sons. During the argument I felt confused and couldn't think. So after that I wrote my thoughts about how I felt at that moment and why I felt so bad. And then I read what I wrote to them*]. The mechanisms that underlie the efficacy of using writing as a strategy to restore thinking remain to be explained. However, it has been described before that writing may compensate for impairments in the 'flow of thought', by allowing patients to fragment the narrative they have in their heads and re-arrange their components through visual control (Luria, 1963[1948], pp. 217-218).

5.2.6. Conclusions

Case studies are a well-established methodology in neurology (see *Neurocase* Journal), neuropsychology (Andrewes, 2001; Funnell, 2000; Humphreys, 1999; Ogden, 1996; Parkin, 1997) and neuropsychological rehabilitation (Wilson, 1999) . However, few case studies have considered the topic of emotional change after brain injury (Damasio, 1994; Kaplan & Solms, 2000). This methodology is particularly suited to address such area of research, for it allows the simultaneous consideration of subjective *and* objective aspects of complex psychological processes (Turnbull & Solms, 2004). The articulation of these two dimensions has been a recurrent technical issue in the field (Mauss et al., 2005).

This paper has tried to show the potential of the case study approach to explore emotional changes in a man [Professor F], who became dysexecutive after a left fronto-parietal lesion. A remarkable feature of Professor F was that, despite exhibiting important set shifting and fluency problems, his abstract ability remained largely preserved, thus allowing him to offer a detailed account of his emotional changes. The use of a mixed methodology, which combines qualitative [subjective] and quantitative [objective] sources of information, appears to be particularly promising in the study of *high functioning* dysexecutive patients like Professor F.

The data gathered by this study suggests that dysexecutive impairment not only compromises cognitive capacities, but may also have an impact on several emotional processes, such as emotion reactivity and emotion regulation. These findings are consistent with a literature proposing that executive functions have a role in emotional reactivity (Ochsner et al., 2009), and are central to emotion regulation (Barkley, 2001; Hofmann et al., 2012). The results offered by this study need, however, to be considered with caution, for they are based in data obtained from one single subject. It will be necessary to gather data

from other patients, or group of patients, with similar lesions and neuropsychological profiles, in order to determine whether the same pattern of emotional change is observed.

It has been widely suggested that self-regulatory deficits are a core feature of the dysexecutive syndrome (Eslinger et al., 2013; Wilson et al., 1996). However, there is little evidence on the mechanisms that underlie such difficulties. Professor F's account is unique in that it describes how impairment to specific neuropsychological processes [e.g. verbal fluency], compromise the use of some emotion regulation strategies [e.g. reappraisal] but not others [e.g. situation selection]. Thus, this article contributes to a small, but emergent, literature exploring the neuropsychological basis of emotion regulation processes (Beer & Lombardo, 2007; Salas et al., 2013; Salas, Gross, & Turnbull, submitted).

In the last decade, the field of neuropsychological rehabilitation has become more interested in socio-emotional functioning and participation problems after brain injury (Bowen et al., 2009; Mateer, Sira, & O`Connell, 2005; McGrath, 2004; Wilson, 1997, 2008; Wilson, Gracey, Evans, & Bateman, 2010). This 'relational turn' in neuropsychological rehabilitation (Bowen et al., 2010) has generated some interest on the socio-emotional, and interpersonal consequences, of having a dysexecutive syndrome (Godfrey & Shum, 2000; Rochat, Ammann, Annoni, & Linden, 2009; Yeates et al., 2008). Professor F's case contributes to such a literature in several ways. Firstly, it offers evidence to support the idea that emotional change after dorsolateral PFC damage is closely related to the underlying cognitive impairments generated by such lesions (Anderson & Tranel, 2002). Secondly, it offers preliminary data to understand the mechanisms by which intrapersonal emotion dysregulation is compensated through the use of another person's executive capacities [extrinsic regulation]. Even though this is a well-recognized clinical phenomenon, the use of other people's minds to compensate for cognitive and emotional impairment has received little attention (Freed, 2002; Salas, 2008, 2012; Yeates, 2009). This case study emphasizes

the need to further explore how the 'inter-mental space' (Leont'ev, 1979; Luria, 1963[1948]) is used by brain injury survivors to regain cognitive reintegration and affective balance. Such an approach is in line with a growing neuroscientific interest in how cognitive and affective processes emerge from brain to brain coupling (Hasson, Ghazanfar, Galantucci, Garrod, & Keysers, 2012; Konvalinka & Roepstorff, 2012).

Chapter 6: Discussion

The main goal of this thesis was to bridge the theoretical and experimental gaps between the fields of emotion regulation [ER] and neuropsychology, particularly neuropsychological rehabilitation. The relevance of such an enterprise is justified by the surprising lack of dialogue between these two fields, despite the exponential scientific growth of ER research in the last decade, and the well-recognized presence of ER problems after brain injury. Because the intersection of these two disciplines is so complex [as outlined in the introduction], this thesis has adopted a multilevel approach, considering several experimental designs [group and single case studies] and methodologies [quantitative and qualitative].

Despite the variety of topics treated in this thesis, there is one common thread that crosses almost every chapter, which is a *clinical interest* in understanding how brain injury modifies people's capacity to regulate emotion. Such a perspective is heavily influenced by the fact that the author of this thesis is not at core an experimental psychologist, but a clinical neuropsychologist and psychotherapist. In consequence, this background infuses these articles, and can be observed in the constant interest to capture the subjective experience of emotional changes, the impact of such changes on relationships, and the acknowledgement that experimental settings are limited in recreating the complexities of emotional life. This clinical interest can also be perceived in seeking to identify the therapeutic value of the new acquired knowledge: what is the relevance of this new finding for neuropsychological rehabilitation? How does this change the way that rehabilitation professionals understand patients' problems? How does this data modify the way we offer treatment to patients?

In this closing section, the main findings of this thesis will be discussed. Firstly, the contribution of this work to the existing literature on ER and brain injury will be addressed.

Then, novel findings will be described in more detail. Finally, the limitations of these studies will be identified, as well as suggestions for future research on the topic.

6.1. A reappraisal of what is known.

Since the early case description of Phineas Gage, changes in emotion regulation have been associated with brain injury (Harlow, 1848, 1868; Macmillan, 2000). However, only recently, a handful of articles has offered supporting scientific evidence for this relationship (e.g. McDonald, Hunt, Henry, Dimoska, & Bornhofen, 2010; Obonsawin et al., 2007; Rochat, Ammann, Annoni, & Linden, 2009; Tate, 1999). An important limitation of this emergent literature has been the absence of a comprehensive theoretical framework to explain such impairments. This thesis has addressed this gap in two general ways. Firstly, it brings together the fields of ER and neuropsychology, by using a well-known theoretical framework [Gross's Process Model, Gross & Thompson, 2007], and also by importing widely used ER paradigms to study brain injured patients. Secondly, and most importantly, data from this thesis supports the clinical assumption that individuals with acquired brain injury are particularly vulnerable to experience episodes of emotion dysregulation (Salas, 2012). For example, people with acquired brain injury are slower to re-interpret negative events, and appear to struggle inhibiting the expression of strong emotions [see below Novel Findings].

In addition, data from this thesis also support neuroimaging and behavioural studies proposing that ER depends on a set of cognitive control processes, commonly regarded as relating to frontal lobe functioning (Ochsner & Gross, 2007; Ochsner & Gross, 2005; Ochsner et al., 2004; Quirk & Beer, 2006; Zelazo & Cunningham, 2007). This thesis offers the first set of data to confirm a long assumed relationship between inhibition, verbal ability [fluency] and

reappraisal generation [see chapter 4.2], as well as between inhibition and suppression [see chapter 4.1]. Thus, evidence from patients with brain damage appears to complement behavioural studies on neurologically intact individuals (Gyurak et al., 2009; Gyurak, Goodkind, Kramer, Bruce, & Levenson, 2012; Mcrae, Jacobs, Ray, John, & Gross, 2011; Schmeichel & Demaree, 2010), by allowing the exploration of how specific deficits [e.g. inhibition] are associated with the impairment of ER strategies. These findings suggest that, despite recent criticisms to the value of lesion methods in a neuroimaging era (Rorden & Karnath, 2004, for a review), the study of patients with acquired brain damage is an important complement to neuroimaging and behavioural approaches. Such a convergence of methods is a central tenant of modern cognitive neuroscience (Gonsalves & Cohen, 2010).

Finally, this thesis also offers substantial support for the commonly accepted idea that emotion dysregulation in people with brain injury can be externally modulated, by manipulating context demands, or by offering cognitive and emotional support (Bowen, Yeates, & Palmer, 2010; Freed, 2002; Goldstein, 1995). The case studies described in chapter 5 are notable examples of this, describing in detail how intrinsic and extrinsic forms of ER are functionally related (Gross & Thompson, 2007; Niven, Totterdell, & Holman, 2009). This data is consistent with authors that have stressed the necessity of including an attachment viewpoint on brain injury rehabilitation (Salas, 2012; Wilson et al., 2010), as well as considering the value of the working alliance between patients and teams (Klonoff, 2011; Lustig, Strauser, Weems, Donnell, & Smith, 2003; Schönberger, Humle, & Teasdale, 2006).

6.2. Novel Findings.

This thesis has contributed to the literature on ER and brain injury in several domains. In this section, novel findings, and their implications, will be discussed.

6.2.1. Mood induction and ER. Emotion induction is a key methodological problem for any researcher interested in ER (Coan & Allen, 2007). The reason is that in order to regulate emotion, an emotional response needs to be adequately triggered. This issue is especially relevant when experimental subjects can be impaired in triggering emotional reactions based on emotional stimuli, the phenomenon commonly referred to as 'emotion reactivity' problems. The first methodological article of this thesis (Salas, Radovic, & Turnbull, 2011, section 3.1.) compared the effectiveness of an internal [autobiographical recall] and an external [movie clips] mood induction procedure, across four basic emotions [fear, joy, anger, sadness], on a student population sample. It was found that both procedures were equally effective triggering the target emotions. However, the internal one generated considerably higher levels of felt emotion. This is the first study to systematically compare two commonly used mood induction procedures across four basic emotions, offering valuable information a sound methodological base for the design of emotional assessment protocols.

In a related experiment [section 3.2] the same two mood induction procedures where used to elicit a positive [joy] and negative [sadness] emotion, on a sample of people with unilateral right hemisphere [RH] lesions and healthy adult controls. Interestingly, similar results were found; the internal procedure generated higher levels of emotional intensity, in both emotions. Furthermore, this finding was observed in the healthy controls and also in the group with RH lesions. Taken together with the results reported above, these two articles contribute to the literature on emotion elicitation, by concluding that the higher intensity

generated by the internal mood induction procedure is independent of age [with the same phenomenon is observed in young and older samples].

6.2.2. Emotional Reactivity. As commented above, emotional reactivity is a key issue when studying ER after brain injury. This thesis contributes to the literature with two novel findings on this topic. Firstly, and related to the emotion elicitation experiment described in the previous section, it suggests that individuals with unilateral RH lesions present a preserved capacity to experience positive and negative emotions [when measured by self-report]. These data extend previous findings by Turnbull et al. (2004), which described the case of a man with a large right hemisphere lesion who was able to experience positive and negative emotions. These data are specially interesting because it challenges behavioural and physiological reports suggesting that individuals with RH damage are hypo-reactive to emotional stimuli (e.g. Heilman, Schwartz, & Watson, 1978; Morrow, Vrtunski, Kim, & Boller, 1981). In addition, this finding contributes with important information to the debate on the role of the cortex in the generation of emotion (Panksepp, 1998, 2011), by supporting the view that emotional experience is largely preserved after cortical damage (Damasio, Damasio, & Tranel, 2012; Feinstein et al., 2010) and even cortical atrophy (Merker, 2007).

A second contribution of this thesis relates to the role of the dorsolateral prefrontal cortex in emotional reactivity. It has been suggested that emotional changes after dorsolateral lesions are secondary to the cognitive deficits generated by damage to such areas (Anderson & Tranel, 2002). However, until today, such a claim had never been supported by robust evidence. The case study presented in section 5.2 (Salas, Radovic, Yuen, & Turnbull, in preparation) offers great insight on this matter, for it describes how impairment in thought generation is related to an increase in emotional reactivity for positive and negative emotions. This finding appears to suggest that damage to the dorsolateral prefrontal cortex does not

compromise emotion generation *in itself*, but instead causes a change in emotion regulation, by heightening emotional experience and compromising the capacity to think about it.

6.2.3. Brain Injury and Emotion Regulation: Evidence to support the impact of brain injury on ER abilities is offered extensively through this thesis [see chapter 4 and 5]. These findings are particularly relevant because they are the first to interpret ER changes after brain injury using a well-established model of ER, the Process Model (Gross & Thompson, 2007). More specifically, this thesis offers important information on how brain injury modifies two widely studied ER strategies: reappraisal and suppression. Reappraisal is an interesting ER strategy to explore after brain injury, because it refers to the use of thinking to change the interpretation of a negative event, and thinking impairment is common after diffuse (e.g. Goldstein & Scherer, 1941) and focal brain damage (e.g. Luria, 1966). Data from this thesis suggests that people with brain damage, independent of the laterality of their lesion, are considerably slower than healthy controls in the generation of reappraisals. However, when time constraints are removed [when they are allowed as much time as they need], they can generate as many reappraisals as healthy controls. This is an interesting finding, because it not only points to brain injured patients' difficulty using thinking to quickly deal with emotional situations, but also recognizes the preservation of these abilities when environmental demands are modulated [time]. This point has substantial implications for rehabilitation and will be addressed in more detail later.

A second novel finding is the presence of changes in the capacity to inhibit the expression of intense positive emotions in patients with right PFC lesions. This is interesting in view of a growing literature suggesting that damage to the rPFC generates marked emotional disturbances (Stuss & Alexander, 1999; Stuss & Alexander, 2000), and that such brain area is related to the inhibition of motor and emotional responses (Dillon & Pizzagalli,

2007). Results suggest that not all patients with right frontal lesions present deficits inhibiting emotional expressions. However, a considerable number of subjects with damage to such area are particularly unable to conceal the expression of strong positive emotions. On a related note, this thesis offers supporting evidence to the idea that the suppression of emotional expressions is associated to measures of response inhibition.

In addition, it is also reported here that individuals with rPFC lesions, compared with people with no brain damage, present a more limited range of voluntary manipulation [amplification and inhibition] of facial emotional expressions. This is also a novel contribution to the literature of RH damage and emotional expression impairment, which has mainly tended to focus on expressive and perceptive deficits (Borod et al., 2010, for a review), without addressing the voluntary manipulation of facial emotional expressions.

6.2.4. Emotion Regulation Theory. The study of ER changes after brain injury not only offers information on how brain damage alters peoples' capacity to modulate their feelings, but also contributes to a better conceptualization of ER and its mechanisms. This thesis addresses this point in two ways. Firstly, it suggests the involvement of several psychological processes that have not been previously considered as relevant for ER [see section 2.2], such as time-traveling [for situation modification] and inner speech [for reappraisal]. These two processes can be selectively impaired after lesions to specific brain areas [hippocampus and left inferior frontal gyrus, respectively]. In the future it would be interesting to explore whether these processes are related to ER performance on neurologically intact individuals.

A second contribution in this regard is the generation of a two stage model of reappraisal, based on the correlational results from section 4.2. This model suggests that in the first stage, [reappraisal generation] automatic negative appraisals are inhibited and reappraisals are generated. In the second stage [reappraisal ability] the generated reappraisals

are kept in mind so the subject can engage with the most appropriate reinterpretation, while the negative stimuli remain within the focus of attention. The novelty of this model is that it incorporates reappraisal generation, an often neglected aspect of reappraisal in studies with non-brain injured subjects, which tend to focus exclusively on reappraisal ability.

6.2.5. Neuropsychological rehabilitation and adaptation of psychotherapeutic tools. A main goal of this thesis has been to stress the importance of emotion regulation [intrinsic and extrinsic] for neuropsychological rehabilitation [see section 2.1]. Such interest is not purely theoretical, but most importantly practical. By understanding the ways in which ER ability can change after brain injury, it is possible to generate better diagnostic formulations and to modify our therapeutic tools. The modification of therapeutic tools is a recent concern in neuropsychological rehabilitation, especially when using psychological interventions to deal with emotional adjustment after the injury (Klonoff, 2010; Weatherhead et al., 2013).This thesis makes several contributions on this point. Firstly, it offers a theoretical model of understanding how concreteness impacts on emotional life, and describes several technical modifications to address such changes in a psychotherapeutic setting [section 2.3]. Secondly, it proposes compensatory strategies to address reappraisal impairment, such as the use of prompting [section 5.1], questioning [section 5.2] or the modification of time constraints [section 4.2]. The usefulness of these strategies to overcome cognitive difficulties is not new in neuropsychological rehabilitation (e.g. Luria, 1963). Nevertheless, this is the first time that scientific evidence is gathered to support its relevance when compensating for emotion regulatory failures.

6.3. Limitations

As one might expect, this thesis has limitations. The majority are related to the fact that bringing together two large fields of knowledge [emotion regulation and neuropsychology] imposes multiple theoretical and methodological challenges. In this particular case, there are few precedents to answer these challenges. In addition, these difficulties are amplified when the complexities of assessing people with brain injury are considered.

Sample size, which is a main limitation of the group studies in chapter 4, stands out as the most obvious and important. A large sample not only guarantees more statistical power, but also allows more fine grained neuroanatomical hypotheses to be established, this by clustering patients in smaller groups with similar lesion sites and profiles of cognitive impairment.

A limitation of working with a small sample is a decrease in the capacity to detect an effect [associations or differences]. In other words, a small sample may lead to a type II error. In the paper on response modulation and right PFC damage [4.1], for example, no significant differences were found between the neurological group and the healthy control group on the average of AU 12+6 [Duchenne smiles] produced during the spontaneous condition. This suggests that the behavioural reactivity of both groups was similar. However, this difference presented a medium effect size [d = .50], thus requiring this result to be considered with caution.

A small sample size can also compromise the use of statistical analyses that attempt to capture multiple associations between variables. For example, in the paper on reappraisal and laterality [4.2.], where the contribution of several cognitive control abilities to reappraisal generation was explored, the ideal statistical procedure to use was a multiple regression analysis. However, it has been suggested that in order to test a regression model with a high level of power, a minimum of 10 participants per group is needed (Field, 2009). Therefore,

the available sample of the study (n = 30) was considerably smaller than the number of participants required to build a model with four predictors (n = 40). Nevertheless, considering that a main goal of the paper was to explore associations between cognitive control abilities and reappraisal generation, it was decided to use a multiple regression model in order to control for the shared variance. This is particularly important when the predictors are theoretically related [cognitive control], as was the case here. However, the results of this model need to be considered with caution, as the use of a small sample may artificially increase the explained variance. In addition, some predictors that show small and non-significant associations [working memory and abstraction] might become significant if using a larger sample.

Finally, and as suggested above, an important problem when working with small samples of patients with focal lesions is that the capacity to test fine grained neuroanatomical hypotheses is limited. This is particularly relevant when exploring complex psychological processes [e.g. reappraisal] that depend on several neuropsychological components which are associated to diverse brain areas. For example, in the case of reappraisal, it has been suggested that this strategy heavily relies on language abilities. It is widely known that posterior areas of the LH [temporal and parietal lobes] are associated with more basic aspects of language [e.g. comprehension], while more anterior areas of the LH [PFC] are related to executive language [e.g. expression and mental manipulation]. In consequence, when exploring the contribution of language areas to reappraisal, a model should consider, at least, these two basic groups of patients [anterior and posterior lesions]. This is an important limitation of the study on reappraisal and laterality [4.2], where due to the small sample size all patients with unilateral lesions to the LH were considered as one group, with the common factor of impaired language abilities. Even though this is partially correct [patients with LH lesions were more impaired in language abilities than individuals with RH damage], such

clustering does not reflect the different forms of language impairment that may emerge after lesions to anterior and posterior areas of the LH. Therefore, conclusions regarding the relevance of those areas for reappraisal cannot be established. This might be particularly important considering that, as reviewed in chapter 2, neuroimaging studies on reappraisal have predominantly shown activation of the left PFC. It is possible, and remains to be tested, whether anterior lesions to the LH, involving the PFC, may result in more marked disruptions of reappraisal than lesions to posterior areas [e.g. temporal lobe].

The understanding of how several cognitive processes are related to ER strategies was a main goal of the group studies. In recent years there has been a growing interest in this relationship, with a handful of behavioural studies exploring such associations on non-neurological populations. Surprisingly, no relationships have been found between key theoretical processes [e.g. verbal ability] and ER strategies [e.g. reappraisal]. As discussed in chapter 2.2, a possible explanation may well be that people without a brain injury perform at the upper limit of what neuropsychological instruments report, with low variance [ceiling effect]. This may also be related to the fact that tasks that assess neuropsychological abilities are not designed to capture variations in normal population, but are more sensitive to detect marked abnormalities in performance. Following this logic, it was decided in the article on reappraisal and laterality [4.2.] to apply the multiple regression model to the whole sample [patients + healthy controls], as a way of increasing the power of the analysis, but also as a means of increasing variability on the neuropsychological performance. This approach raises questions about the applicability of the model to these two different populations separately, a question that could not be tested because of the small sample size.

Beyond issues of sample size, the group studies also present limitations in terms of the methods employed to measure emotional response. They used reaction time measures, self-report and facial behaviour. However, they do not consider physiological measures. This is a

second limitation – particularly on the response modulation study [section 4.1]. The technical demands of collecting behavioural and subjective data were already substantial, especially in a neurological patient population. Because this was a preliminary investigation, which implied studying the same phenomenon from different points of view, it was not possible to implement all these measures simultaneously. Nevertheless, this point is relevant and needs to be addressed in the future. The emotional response has been typically described as composed of several components [behavioural, subjective and physiological], which may not always cohere (Mauss et al., 2005). This constitutes an important limitation, particularly when some of the results presented here: that patients with RH preserve emotional experience, when measured by subjective self-reports, challenge previous findings reporting that patients with RH damage present a behavioural and physiological hypo-reactivity. It is possible that, in the same way as healthy controls, the components of the emotional response in patients with brain injury may not cohere. It is also possible, that such non-coherence may be abnormally exacerbated. Unfortunately such a discussion cannot be settled without testing all three components at the same time.

6.4. Future Directions

Perhaps the main goal of a research project of this sort like this is not to offer definitive findings on how brain injury compromises ER, but to open new avenues of enquiry. In this section several possible directions of future research are identified.

6.4.1. ER and non-focal brain damage. An interesting question is whether individuals with diffuse brain lesions present difficulties implementing specific ER strategies, and if these difficulties are similar to those exhibited by patients with focal brain damage. It has been frequently reported that emotion dysregulation is a core feature of traumatic brain injury, TBI

(e.g. Tate, 1999), however no studies have yet used ER tasks based on the Process Model to investigate this. Naturally, a limitation of using subjects with diffuse lesion [e.g. TBI] is that neuroanatomical conclusions are more difficult to establish. However, an advantage is that TBI presents with a well-known profile of executive impairment, offering an impressive opportunity to explore how cognitive control is associated to ER strategies, a topic of substantial clinical importance.

6.4.2. ER and circumscribed cognitive impairment. The study of patients with restricted cognitive impairment is a very promising line of enquiry. It would allow the exploration of how such deficits impact one or several ER strategies. For example, in patients with amnesia [e.g. after hippocampal damage] it would be of great interest to understand the psychological basis of situation selection. In another line of enquiry, individuals with deficits of expressive speech, and perhaps also inner speech [after damage to the left inferior frontal gyrus] would allow offer a better understanding of how important internal thinking is for reappraisal. A larger list of these possible 'target' patients can be found in section 2.2.

6.4.3. Single case studies on emotion dysregulation after RH damage. Single case studies are a key approach to understand ER changes after brain injury. They offer insight into the subjective experience of such change, and also of how the individuals [and their environment] has spontaneously adapted [or not] to it. The two case studies presented in this thesis (Salas et al., in preparation; Salas et al., 2013) are of individuals with left fronto-parietal lesions, offering important insight into the role of language and verbal cognitive control for reappraisal. It would be interesting to explore in depth cases of subjects with say [right fronto-parietal lesions], in order to compare the profiles of dysregulation that may arise. It has been suggested, for example, that left lateral lesions compromise a task-switching aspect of cognitive control, while right lateral lesions impair monitoring (Shallice, Stuss,

Picton, Alexander, & Gillingham, 2008a, 2008b). Our case studies appear to support such a claim, and offer additional evidence of how left lateral lesions compromise the use of specific ER strategies. It would be interesting to see the effect that monitoring impairment may have on the implementation of ER strategies.

6.4.4. Brain injury and reappraisal ability. This thesis has explored one particular aspect of reappraisal, which is reappraisal generation. The rationale behind such an approach is that the generation and manipulation of ideas is commonly compromised after brain injury. It remains to be studied how well patients with brain injury are able to use the generated appraisals to down-regulate negative states. It is possible that a different set of cognitive control abilities than those recruited in the generative phase are required at this point of the process.

6.4.5. Use of extrinsic emotion regulation strategies. An interesting, but anecdotal finding of this thesis is that extrinsic forms of ER appear to be able to compensate for intrinsic regulatory failures. The case of Professor F (Salas et al., in preparation) is a notable example of how these two processes are connected, and more importantly of how such compensation might emerges spontaneously. This finding is of substantial relevance when developing interventions that can facilitate patient and family emotional adjustment. In consequence, it appears necessary to explore more systematically the way in which relatives address intrinsic regulatory failures, thus mapping patterns of interaction that facilitate and hinder emotional adjustment. A better understanding of these interpersonal mechanisms may offer valuable information to educate and train family members.

6.4.6. Implementing and testing interventions to improve ER. This thesis has offered important guidelines in how to scaffold patient's intrinsic regulatory abilities. For example, it

has been suggested that the use of prompting and questioning may enhance patients' capacity to reappraise. It would be interesting to explore whether these strategies can be applied, and are equally effective in therapeutic and family settings, and if there are family dynamics that facilitate or impede their use.

6.5. Closing words

Clinical neuropsychologists are constantly dealing with the behavioural and interpersonal difficulties caused by brain damage. However, they do not always label such difficulties as ER problems, or have a clear theoretical framework to think about them and to justify why they do what they do. This is an important limitation, for the lack of an appropriate theoretical model reduces the capacity to share information [with colleagues, families and funding agencies] and test whether what we do is effective or not.

It is strange how this task has not been done before, particularly considering that ER problems have been reported since the foundation of our discipline. A possible explanation to this situation is that, only recently, we have become aware of the limitations of a cognitive 'restorative' approach, and realized the relevance of addressing emotional adjustment issues. However, there seems to be another reason, perhaps more influential. In order to understand ER, we need to comprehend the mechanisms by which emotion and cognition influence each other. And today, such knowledge is still limited. Nevertheless, clinical neuropsychologists are in a privileged position to offer insight into this matter. We have the skills to assess cognitive functions, and also the ability to explore subjective, and interpersonal, emotional experience. We are trained to test patients in rigorous experimental settings, but at the same time are skilled in capturing how deficits impact everyday life. This thesis is an example of the many possibilities that a *clinical* and *neuropsychological* perspective opens for the study of ER.

Appendixes

Appendix 1. Ethics permission letter, information sheet and consent form for undergraduate student study.

Date: Tue, 02 Nov 2010 11:50:07 +0000 [02/11/10 11:50:07 BST]
From: Everil McQuarrie <e.mcquarrie@bangor.ac.uk>
To: Oliver Turnbull <pss40d@bangor.ac.uk>, Salas,Christian Eduardo <pspa35@bangor.ac.uk>
Subject: Ethics proposal 1658

Dear Colleagues

* Emotion regulation in patients with Acquired Brain Injury Ethics 1658
*

Your research proposal referred to above has been reviewed by the School of Psychology Research Ethics Committee and they are satisfied:

(i)That the research proposed accords with the relevant ethical guidelines.

(ii)That the research proposed is appropriate for sponsorship by BangorUniversity.* *

* Approval is granted subject to you submitting Welsh translations of your information/consent and debrief forms to me. *

If you wish to make any non-trivial modifications to the research project please inform the committee in writing before proceeding.Please also inform the committee as soon as possible if research participants experience any unanticipated harm as a result of participating in your research.

You should now forward the application to NRES and to the appropriate Local Research Ethics Committee (LREC). * If you need a signature on the form regarding research sponsorship by the University, and/or a letter confirming this sponsorship, please send the final version of your NRES form to me and I will make arrangements for this.*

* *

The NHS Research Ethics Committee expect one of the investigators to make an oral presentation in support of the proposal at their meeting.You will be contacted by their committee with details as to the date and place of the meeting at which your proposal will be considered.

You may not proceed with the research project until you are notified of the approval of the Local Research Ethics Committee and have R&D approval from the relevant NHS Trusts.

The approval for this project is given on the understanding that you will complete a review form on the project when requested; to this end I would be grateful if you could complete the form below and return it to me.

Yours sincerely

Everil

* UWB-SPONSORED RESEARCH PROJECTS*

**

MONITORING ARRANGEMENTS FORM

Principal Investigator: _____

Project Title: _____

Because Bangor University has agreed to act as research sponsor for the research project named above, we are required to ensure that arrangements are in place to monitor the progress of the project. Please read through the information below, tick the box that applies to this project, and return to the ethics coordinator.

o* This research is funded by an external agency that requires regular progress reports. *

In this case, please copy all such progress reports to the ethics coordinator for review.

o* This is student research under your supervision.*

It is the responsibility of the supervisor to monitor the progress of research conducted by students and to report any significant changes or issues arising to the ethics coordinator.

o* Progress reports are not required for this research by the external funder, or this is non-funded research conducted by you as a staff member.*

The ethics coordinator will contact you at regular intervals for a short progress report.

--

/Everil McQuarrie,
Research and PhD Administrator,
Room 113,
School of Psychology
Brigantia Building,
Penrallt Road,
Bangor
LL57 2AS

Tel: 01248 383671/

*/THE LAST DATE FOR PAPER ETHICS SUBMISSIONS IS/**/30th September 2010/*

*/THE LINK TO THE ELECTRONIC FORM IS
/**/https://intranet.psychology.bangor.ac.uk/ethics//*

Ysgol Seicoleg

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Bangor University

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Information sheet

Researchers

Mr. Christian Salas, Dr. Oliver Turnbull.

1. **Study title**

Emotional Persistence: transitions between basic emotions.

2. **Invitation paragraph**

You are being invited to take part in a research study. Before you decide it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with others if you wish. Ask us if there is anything that is not clear, or if you would like more information. Take time to decide whether or not you wish to take part.

Thank you for reading this.

3. **What is the purpose of the study?**

The aim of the study is to look at how people experience different emotions in time. Some people tend to be more affected by certain emotions than others, and can experience emotions for longer or shorter periods of time. These differences between people are important when trying to understand how people manage their emotions.

4. **Why have I been chosen?**

For the purpose of this study we need to recruit university students, who are English native speakers.

5. **Do I have to take part?**

Participation is entirely voluntary. If you change your mind about taking part in the study you can withdraw at any point during the session, and at any time in the two weeks following the

session. You can withdraw by contacting me by email, providing me with your participant code. If you decide on withdrawal your data will be destroyed and will not be used in the study. There are no consequences to deciding that you no longer wish to participate in the study.

6. **What will happen to me if I take part?**
If you decide to take part in the study, you will take part in a 1-hour session. During that session you will have to complete two tasks. In the tasks you will be asked to watch short movie clips as well as telling personal episodes associated to certain emotions. In both tasks you will have to report systematically how you feel. The second task will be video taped for later analysis.

7. **What are the possible disadvantages and risks of taking part?**
Because this study intends to explore how emotions work, you might experience different feelings during the assessment and afterwards. Making people feel emotions is the only way to investigate them. Many people usually do not experience any distress after being exposed to a film clip, or after being asked to recall a personal event. However, there is a chance that you keep experiencing some emotions after the session, or that negative personal memories keep coming into your mind. If this is the case we ask you to get in touch with the principal researcher, so we can give you advice where you might seek help, for example referring you to the University Counselling Office.

8. **What are the possible benefits of taking part?**
As an undergraduate student, by taking part in this study you will gain an insight into how a psychology research project is conducted, and what is like to be a participant. This may be helpful to your own undergraduate project and how to design it. You will also receive credit for your participation.

9. **Will my taking part in this study be kept confidential?**
Yes. The raw data extracted from the sessions will be handled under strict confidentiality regulations: your name will be changed to a letter-digit code, the data registered (including videos) will be stored in a safe place, and only the named researchers will have access to it. To analyze the data some other researchers might have to access to it. Before they do it they will have to sign an agreement of confidentiality.

10. **What will happen to the results of the research study?**
The results of the study will be written up as part of my PhD thesis. If the results are novel they might also be presented at academic conferences or written up for publications in specialized journals. In all these cases there would not be any personal reference to the participants, and we will not display video material one it has been coded.

11. Who **is organising and funding the research?**
The research project has been organised by Christian Salas, who is a PhD student at the School of Psychology, Bangor University. This project is not externally funded.

12. **Who has reviewed the study?**
The project has been approved by the Ethics Committee of the School of Psychology, Bangor University and North Wales Research Ethics Committee.

13. **Contact details for** further information

Mr. Christian Salas
School of Psychology,
Adeilad Brigantia, Of. 261
Penrallt Road
Gwynedd LL57 2AS
pspa35@bangor.ac.uk
Tel: (01248) 388895

Dr. Oliver Turnbull (Supervisor)
Tel: (01248) 388261
o.turnbull@bangor.ac.uk

17. **What if something goes wrong?**

If you have any complaints about how this study is conducted please address these to either of the persons below:

Dr Paul Downing
Deputy Head of School
School of Psychology
University of Wales, Bangor
Brigantia Building
Bangor, ll57 2AS

Ysgol Seicoleg

Prifysgol Cymru, Bangor

Adeilad Brigantia, Ffordd Penrallt
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e-mail: psychology@bangor.ac.uk

Consent Form

Emotional Persistence II. Transitions between Basic Emotions.

Participant reference code_____

- I have read and understood the attached information sheet and, by signing below, I consent to participate in this study.
- I understand that I have the right to withdraw from the study without giving a reason at any time during the study itself.
- I understand that I also have the right to change my mind about participating in the study for a short period after the study has concluded (two weeks).

Participant signature:_____

Print name:_____ Sex: _____

Participant psu: _____Age:_____

If you want to receive information about the results of this research write here your e-mail address. It will be used for this exclusive purpose:

_____

Researcher signature:_____

Print name:_____

Appendix 2. NHS Ethics committee approval letter, information sheets and consent forms for studies with acquired brain injury population.

NHS

Pwyllgor Moeseg Ymchwil Gogledd Cymru (Y Orllewin)
North Wales Research Ethics Committee (West)

Betsi Cadwaladr University Health Board
Ysbyty Gwynedd
Clinical Academic Office
Bangor, Gwynedd
LL57 2PW

Telephone/ Facsimile: 01248 - 384 877
Email: Rossela.Roberts@wales.nhs.uk

PRIVATE & CONFIDENTIAL
Mr Christian Salas
School of Psychology
Bangor University
Brigantia Building, Penrallt Rd,
Bangor, Gwynedd
LL57 2AS

7 December 2010

Dear Mr Salas,

Full title of study: **Emotion Regulation in People with Aquired Brain Injury**
REC reference number: **10/WNo01/61**
Protocol number: **1 dated 05/11/2010**

Thank you for your letter of 03 December 2010, responding to the Committee's request for further information on the above research and submitting revised documentation.

The further information has been considered on behalf of the Committee by the Chairman.

Confirmation of ethical opinion

On behalf of the Committee, I am pleased to confirm a favourable ethical opinion for the above research on the basis described in the application form, protocol and supporting documentation as revised, subject to the conditions specified below.

Ethical review of research sites

The favourable opinion applies to all NHS sites taking part in the study, subject to management permission being obtained from the NHS/HSC R&D office prior to the start of the study (see "Conditions of the favourable opinion" below).

Conditions of the favourable opinion

The favourable opinion is subject to the following conditions being met prior to the start of the study.

Management permission or approval must be obtained from each host organisation prior to the start of the study at the site concerned.

Management permission ("R&D approval") should be sought from all NHS organisation(s) involved in the study in accordance with NHS research governance arrangements. Guidance on applying for NHS permission for research is available in the Integrated Research Application System (IRAS) or at http://www.rdforum.nhs.uk

Where a NHS organisation's role in the study is limited to identifying and referring potential participants to research sites ("participant identification centre"), guidance should be sought from the R&D office on the information it requires to give permission for this activity.

For non-NHS sites, site management permission should be obtained in accordance with the procedures of the relevant host organisation.

Sponsors are not required to notify the Committee of approvals from host organisations. It is the responsibility of the sponsor to ensure that all the conditions are complied with before the start of the study or its initiation at a particular site (as applicable).

Approved documents

The final list of documents reviewed and approved by the Committee is as follows:

Document	*Version*	*Date*
Covering Letter		05 November 2010
REC application (Submission code: 59182/163057/1/589)		08 November 2010
Response to Request for Further Information	-	03 December 2010
Protocol	1	05 November 2010
Participant Information Sheet	2	29 November 2010
Participant Information Sheet: Family	2	29 November 2010
Participant Consent Form	2	29 November 2010
Participant Consent Form: Family	2	29 November 2010
GP/Consultant Information Sheets	1	05 November 2010
Questionnaire: Berkeley Expressivity Questionnaire		
Questionnaire: Social Emotional Questionnaire		
Questionnaire: Emotion Regulation Questionnaire		
Questionnaire: Difficulties in Emotional Scale		
Questionnaire: Hospital Anxiety & Depression Scale		
Questionnaire: Toronto Alexythimia Scale		
Questionnaire: Iowa Scale of Personality Change		
Evidence Supervisor CV of insurance or indemnity	UMAL	01 August 2010
Letter from Sponsor		04 November 2010
Investigator CV (Mr CE Salas)		05 November 2010
Supervisor CV (Prof O Turnbull)		05 November 2010

Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees (July 2001) and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

After ethical review

Now that you have completed the application process please visit the National Research Ethics Service website > After Review

You are invited to give your view of the service that you have received from the National Research Ethics Service and the application procedure. If you wish to make your views known please use the feedback form available on the website.

The attached document *"After ethical review – guidance for researchers"* gives detailed guidance on reporting requirements for studies with a favourable opinion, including:

- Notifying substantial amendments
- Adding new sites and investigators
- Progress and safety reports
- Notifying the end of the study

The NRES website also provides guidance on these topics, which is updated in the light of changes in reporting requirements or procedures.

We would also like to inform you that we consult regularly with stakeholders to improve our service. If you would like to join our Reference Group please email referencegroup@nres.npsa.nhs.uk.

10/WNo01/61	**Please quote this number on all correspondence**

With the Committee's best wishes for the success of this project

Yours sincerely

Mr David Owen
Chairman

Email: rossela.roberts@wales.nhs.uk

Enclosures: "After ethical review – guidance for researchers"

Copy to:
Sponsor: Professor Oliver Turnbull
School of Psychology
Bangor University
Brigantia Building, Penrallt Rd,
Bangor, Gwynedd
LL57 2AS

R&D office for lead NHS Site Clinical Academic Office
BCUHB, Ysbyty Gwynedd
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Prifysgol Cymru, Bangor

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Tel:(01248) 382211 - Fax:(01248) 382599

Participant Identification Number:
Date: 29/11/10
Version: 2

Information sheet

Researchers
Dr Oliver Turnbull, Mr Christian Salas

1. **Study title**
Emotion Regulation in People with Acquired Brain Injury.

2. **Invitation paragraph**
You are being invited to take part in a research study. Before you decide to take part in the study it is important for you to understand why the research is being done and what it will involve. Please take the time to read the following information carefully and discuss it with others if you wish. Please don't hesitate ask us if there is anything that is not clear or if you would like further information. Take time to decide whether or not you wish to take part.

Thank you for reading this.

3. **What is the purpose of the study?**
The aim of this study is to look at how people regulate their emotions after a brain injury. Some people that have acquired a brain injury may experience changes in their capacity to feel or control positive or negative emotions. We are planning to look at how lesions to different parts of the brain may impair different aspects of this capacity to regulate emotions, and how this may impact people's ability to cope with the injury and return to social life.

4. **Why have I been chosen?**
You have been invited to take part in this study because you have had a brain injury and because the borders of this injury are well defined, so we know which areas of your brain were affected. Neurologists and neuropsychologist from Bangor University and Staff from the North Wales Brain Injury Service (NWBIS) think that you would be able to take part in this study and the tasks this involves.

5. **Do I have to take part?**
It is up to you to decide whether or not to take part. If you do decide to take part you will be given this information sheet to keep and be asked to sign a consent form. If you decide

to take part you are still free to withdraw at any time and without giving a reason. A decision to withdraw at any time, or a decision not to take part, will not affect the standard of care you receive.

6. **What will happen to me if I take part?**

If you decide to take part in the study, you will need to attend two sessions of approximately 2 hours each. In the first session an initial assessment will be carried out, where we will ask you to complete some questionnaires about how you are feeling at the present moment and how you manage your emotions (with help from the researcher if you need it). We will also briefly assess your concentration, language, memory and capacity to organize. We will also need a relative of yours (wife, husband, parent or son/daughter) to complete two questionnaires about you.

Approximately one or two weeks after your initial assessment, the second session will be carried out. In this session you will be asked to perform different tasks, for example, watch short clips from movies and report how they make you feel, or remember an event in your life when you felt a specific emotion (for example joy or sadness).

During all these tasks we will need to video-record you. This is because we are interested in studying emotions and facial expression are one of the most important sources of information about how people feel. Although we know it might feel uncomfortable, we hope you agree with this, because this information is very important to understanding the emotional changes that occur after a brain injury.

The assessments will take place either in the North Wales Brain Injury Service in Colwyn Bay, in another health service venue that the Brain Injury Service uses for patient appointments (such as your GP surgery or a community hospital) or at the university in Bangor. If it is difficult for you to travel to one of these places, it may also be possible for you to be seen at your home. You will be offered money for travel expenses (£ 6 for each hour you spend travelling) and each assessment session you attend (£ 10), in recognition of the time and energy that participation in this project will ask of you.

7. **What do I have to do?**

Other than the need to attend to two assessment sessions, the research study will not affect your lifestyle or normal routine in any other way.

8. **What are the possible disadvantages and risks of taking part?**

The risks of this study are the same as watching a movie on TV or having a conversation with another person about you. We have run this experiment before and rarely found it to be distressing for participants.

9. **What are the possible benefits of taking part?**

We hope that taking part in the study will be helpful in two ways. First, in case that you are interested, after the study we will give you brief feedback letter which may be used in your rehabilitation process, or simply as a way of knowing yourself better. Second, we believe that the emotional changes that a person has after a brain injury are very important in how he/she may cope with the brain injury and return to their work and family life. Because there is such

scarcity of information on this, the results of this study may help in the future to design interventions that would help people to cope better with brain injury.

10. **What happens when the research study stops?**
The information obtained will be studied in detail and the main conclusions of the study will be shared with the professionals to generate new ways of helping people with brain injury. In case you want it, a brief feedback letter will be offered to you too.

11. **Involvement of my Family Doctor**
We will write to your family doctor to let them know that you have decided to take part in this study.

12. **Will my taking part in this study be kept confidential?**
All information that is collected about you during the course of the research will be kept strictly confidential and you will not be identified in any report or publication. This agreement of confidentiality only will be broken in cases where your well-being and safety are put at risk. If something like this happens, the researcher will report the situation to the professionals in charge of your care (rehabilitation team or consultant neurologist).

13. **What will happen to the results of the research study?**
When the study is finished, you will receive a letter telling you about the results. Our findings will also contribute to an educational qualification, copies of which will be given to the Library of the University of Wales, Bangor. The results will be published in scientific journals and may be presented at conferences.

14. **Who is organising and funding the research?**
The research project has been organised through the School of Psychology, Bangor University.

15. **Who has reviewed the study?**
The project has been approved by the Ethics Committee of the School of Psychology, Bangor University and North Wales Research Ethics Committee.

16. **Contact details for further information**
Mr. Christian Salas
Chief Investigator
School of Psychology
Bangor University
Bangor, Gwynedd LL57 2AS, UK.

Available during the week
Tel: 01248 38 8895

17. **What if something goes wrong?**
If you have any complaints about how this study is conducted please address these to either of the persons below:

Dr Valerie Morrison	Dr. Rosella Roberts

Deputy Head of School School of Psychology University of Wales, Bangor Brigantia Building Bangor, ll57 2AS Tel: +44 (0) 1248 383624 Fax: +44 (0) 1248 38 2599	Clinical Governance Officer Betsi Cadwaladr University Health Board Ysbyty Gwynedd Bangor, Gwynedd LL57 2PW Tel: +44 (01248) 384877

1 for patient; 1 for researcher; 1 to be kept with hospital notes

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Tel:(01248) 382211 - Fax:(01248) 382599

PARTICIPANT CONSENT FORM

Study title: Emotion Regulation after acquired brain injury.

Name of Researchers:
Mr Christian Salas, Dr Oliver Turnbull.

Name of Researchers who will obtain consent: Mr Christian Salas

Please initial box

1. I confirm that I have read and understand the information sheet dated ..... (version .....) for the above study and have had the opportunity to ask questions. ☐

2. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. ☐

3. I understand that relevant sections of my medical notes may be looked at by Mr Christian Salas. I give permission for this researcher to have access to my records. ☐

4 I understand that all collected information during this research will be kept strictly confidential. However if there is a situation where my well-being or safety is compromised, I understand that the investigators will have to report the situation to the professionals on charge of my care. ☐

xv

5. I agree to my family doctor being informed of my participation in the study. ☐

6. I agree that the tasks in the study can be video-recorded. ☐

7. I agree that the video recordings can be used for educational purposes. ☐

8. I agree to a close relative to participate in the study by answering two questionnaires ☐

9. I agree to take part in the above study. ☐

_____	_____	
Name of Patient	Date	Signature
_____	_____	
Name of Person taking consent (if different from researcher)	Date	Signature
_____	_____	
Researcher	Date	Signature

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Tel:(01248) 382211 - Fax:(01248) 382599

Participant Identification Number:
Date: 29/11/10
Version: 2

Information sheet family members

Researchers
Dr Oliver Turnbull, Mr Christian Salas

1. **Study title**
Emotion Regulation in People with Acquired Brain Injury.

2. **Invitation paragraph**
You and your relative have been invited to take part in a research study. Before you decide it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with others if you wish. Ask us if there is anything that is not clear or if you would like more information. Take time to decide whether or not you wish to take part.

Thank you for reading this.

3. **What is the purpose of the study?**
The aim of this study is to look at the capacity of people to regulate emotions after a brain injury. Some people that have acquired a brain injury may experience changes in their capacity to feel or control positive or negative emotions. We are planning to look at how damage to the brain may impair different aspects of this capacity to experience and regulate emotions, and how this may affect people's ability to cope with the injury and return to social life. Because the capacity to regulate emotions is usually at work when we relate to friends and family, and because as a relative you also have knowledge of the patient from before the injury, your information is extremely relevant for this research.

4. **Why have I been chosen?**
You have been invited to take part in this study because you have a close relationship with the patient (partner, parent, sibling, etc.) and are able to offer information on the patient's level of social functioning. Neurologists and neuropsychologist from Bangor University and Staff from the North Wales Brain Injury Service (NWBIS) think that your relative is suitable to take part in this study and the tasks this imply.

5. **Do I have to take part?**

It is up to you to decide whether or not to take part. If you do decide to take part you will be given this information sheet to keep and be asked to sign a consent form. If you decide to take part you are still free to withdraw at any time and without giving a reason. A decision to withdraw at any time, or a decision not to take part, will not affect the standard of care your relative receive.

6. **What will happen to me if I take part?**

If you decide to take part in the study, you will need to answer two questionnaires, which will take no more than an hour. If you accompany your relative while he or she is being tested you might use that time to fill in the questionnaires. If you are not attending we might send you the questionnaires by post.

8. **What are the possible disadvantages and risks of taking part?**

To participate in this study has no risks for you and the information you will provide will be kept confidential.

9. **What are the possible benefits of taking part?**

Taking part on this study will be useful in several ways. First, in case that you and your relative want it, after the study, we will give you a feedback which may be used in your relative's rehabilitation process. Second, we believe that the emotional changes that a person has after a brain injury are very important in how he/she may cope with the brain injury, return to their work and resume family life. Because there is such scarcity of information on this, the results of this study may help in the future to design interventions that would help people to cope better with brain injury.

10. **What happens when the research study stops?**

The information obtained will be studied in detail and the main conclusions of the study will be shared with the professionals to generate new ways of helping people with brain injury.

11. **Will my taking part in this study be kept confidential?**

All information that is collected about you or your relative during the course of the research will be kept strictly confidential. You or your relative will not be identified in any report or publication. This agreement of confidentiality only will be broken in cases where the well-being and safety of the patient is put at risk. If something like this happens, the researcher will report the situation to the professionals in charge of the patient (rehabilitation team or consultant neurologist).

12. **What will happen to the results of the research study?**

When the study is finished, you will receive a letter telling you about the results. Our findings will also contribute to an educational qualification, copies of which will be given to the

Library of the University of Wales, Bangor. The results will be published in scientific journals and may be presented at conferences.

13. **Who is organising and funding the research?**
The research project has been organised through the School of Psychology, Bangor University.

14. **Who has reviewed the study?**
The project has been approved by the Ethics Committee of the School of Psychology, Bangor University and North Wales Research Ethics Committee.

15. **Contact details for further information**
Mr. Christian Salas
Chief Investigator
School of Psychology
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Bangor, Gwynedd LL57 2AS, UK.

Available during the week
Tel: 01248 38 8895

16. **What if something goes wrong?**

If you have any complaints about how this study is conducted please address these to either of the persons below:

Dr Richard Bental Deputy Head of School School of Psychology University of Wales, Bangor Brigantia Building Bangor, ll57 2AS Tel: +44 (0) 1248 383624 Fax: +44 (0) 1248 38 2599	Dr. Rosella Roberts Clinical Governance Officer Betsi Cadwaladr University Health Board Ysbyty Gwynedd Bangor, Gwynedd LL57 2PW Tel: +44 (01248) 384877

1 for relative; 1 for researcher; 1 to be kept with hospital notes

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CONSENT FORM FAMILY MEMBERS

Study title: Emotion Regulation after acquired brain injury.

Name of Researchers:
Mr Christian Salas, Dr Oliver Turnbull.

Name of Researchers who will obtain consent: Mr Christian Salas

Please initial box

1. I confirm that I have read and understand the information sheet dated ..... (version .....) for the above study and have had the opportunity to ask questions. ☐

2. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my relatives' medical care or legal rights being affected. ☐

3. I understand that all collected information during this research will be kept strictly confidential. The only exception is a situation where my relative's well-being or safety is compromised. I understand that in such circumstances the investigators will have to report the situation to the professionals on charge of my relative's care. ☐

3. I agree to take part in the above study. ☐

_____	_____	
Name of Relative	Date	Signature
_____	_____	
Name of Person taking consent (if different from researcher)	Date	Signature
_____	_____	
Researcher	Date	Signature

1 for relative; 1 for researcher; 1 to be kept with hospital notes

Appendix 3. Vignettes from the internally generated mood induction procedure (Affective Story Recall). In the right column the self-report scores of the internal (ASR) and external (FC) procedures are compared.

Participant	Target Emotion		ASR and FC Scores
Example A Female, 19	Anger	The most *anger* probably would be weeks ago… one of my younger sister is going through so much. It is the youngest sister, so you can imagine all the things she goes through. She had acne, so doctors prescribed tablets, and as a side effect they cause *depression*. She has been always really *happy*, one of those persons you actually *love*. She went on this tablets and her *mood dropped* completely. She is going through so much… a few weeks ago she started to get bullied by these girls at school. And is not so much the bullying, but are these little things like leaving her out, making calls…because her boyfriend is very popular…is one of those boys every girl want to be with. And my sister is with her… and there were a lot of comments on why is she with him… you know… he can do better. For a 15-year-old girl who is going through so much already… in a way, I really *hated* those girls that hurt my sister. My mother told me that my sister was quite *upset* because I was not around. And we are so far away… there is nothing I can do to help her and that… it *kills me*. I *cry* a lot because she is going through so much and she is my baby sister and I'm supposed to protect her. And these girls were so cruel. She came from school *crying*, she missed school… and she can't miss school this year. And I absolute *hated* these girls who went to do that to my sister. And they just seem not to care… and that is the worst thing, when someone do something to someone and really hurt her... and not realize the effect that this has in my sister. I mean maybe they see my sister *crying*, but they don't know the effect of this on my family or me. Just so much *rage* the fact that people can be so cruel and don't realize how they can affect other people. Yeah… this is probably the worst time.	Anger ASR A = 5.0 S = 4.3 F = 4.0 J =1.0 Anger FC A = 2.7 S = 2.0 F = 1.0 J =1.0
Example B Female 19	Fear	It was the first time I was in the house alone for a few months. I was back from reading week and my mom and dad were away. It was late at night… I know you should tell yourself that is a good neighbourhood and that nothing is gonna happen, but I kept hearing noises upstairs and I just sort of *froze*. And then my dog, she is quite sensitive to things like that, started barking and growling. So I was there *frozen* in my seat not knowing what to do just having my phone ready… So I turned the volume down of the television thinking 'what if someone can hear me?'. But everything that was in my head at that moment was ilogical. And I knew that there is no possible way that someone have got upstairs. But I got myself into this *fear* like state that made me think there is someone upstairs and is going to come down and murder me… you know… something stupid. I was very *scared* and even started to think how to get out of the house crawling down to the front door. But finally it was something very ridiculus, it was the cat playing around. But at that time I was just lying in bed, because I was in this state… so wound up… and then I felt sort of calm…feeling bit stupid about myself. I think I was very, very *scared* of being alone at home..	Fear ASR F = 3.7 A = 1.3 S = 2.3 J = 2.0 Fear FC F = 2.7 A = 1.0 S = 1.7 J = 2.
Example C Female 18	Sadness	One of the *sadest* memories I have is one when I was very young. It is one of the first memories I have, so it makes it	Sad ASR S = 5.0

		even worst. Is my mom and my dad fighting in the kitchen and I was hidden behind the washing machine, looking. I can't remember what they are saying, I can't rememeber any sound at all. All I can remember is the *anger* in their faces. My mom is leaning against the sink and she is screaming at my dad and my dad is screaming back and I couldn't handle it anymore, so I ran out of the kitchen and my younger brother was in the front room crying and he could hear everything as well. That moment when I saw him *crying* I thought well I can't *cry,* he needs to be looked after. They carried on screaming so eventually I took all my brothers upstairs and we went to bed together. And then after that… it was a year or so after that… and I remember that was a tuesday morning and my mom came and said you are not going to school. I hated school…she started getting me dress with my normal clothes and I ran down the stairs and bang my head into the hand rail of the stair and knocked my tooth out… so I just thought that I was gonna be grounded… but she sat me down and I knew there was something wrong… and she took me into the front room and told me that my dad had killed himself. So yeah… that was a pretty crap day. And I don't remember her saying anything afterwards. I remember she sat there *crying*, and my grandma came through the door and she huged me and she huged my mom. Then I went upstairs to my room and didn't speak to anyone for three months.	A = 5.0 F = 4.7 J = 1.0 Sad FC S = 3.0 A = 1.3 F =1.0 J =1.0
Example D Female, 18	Joy	I wrote an essay, my first essay ever and I got an A+ on it. So that was very, very *exciting* and it made me feel very *happy* and also I didn't expect it. So I remember calling my parents and telling them how *happy* I was and the feeling of how proud they were of me made me even *happier*. Well, and at the department the professor came to me and said "can we use your essay in the class as an example?". And I was like… of course… wow… that made me very *happy*. I was a bit *scared*, but mostly happy because it just meant that they thought that my work was good enough to be presented as an example… or … I don't know it made me very happy because it was one of the first things that I have done in the university and I didn't expect an A+.	Joy ASR J =3.7 A = 1.0 S = 1.0 F = 2.7 Joy FC J = 2.3 A = 1.0 S = 1.7 F = 1.0

Appendix 4. Facial expression generation. rPFC and HC mean scores for the voluntary generation of a large set of AUs.

AU	rPFC		HC	
	M	*SD*	*M*	*SD*
12+6	1.13	0.35	1.29	0.46
4	1.63	0.91	1.21	0.47
9	1.88	0.64	1.86	0.36
10	2.75	1.58	2.29	0.99
14	2.25	1.28	1.64	0.63
20	1.63	0.74	1.93	1.07
24	1.50	0.75	1.29	0.47
26	1.13	0.35	1.07	0.27
1+2	1.50	0.53	1.57	5.14
5	2.38	0.91	2.00	0.78
15	3.50	1.41	3.36	1.39
17	2.25	1.48	1.50	0.65

Appendix 5. Reappraisal answers of Mrs M, non-concrete brain-injured patients and controls.

IAPS Picture		Mrs M	Non-concrete brain-injured patients	Controls
Sick Baby (3350)	Free Reappraisal	*Mrs M*: Oh! (Smiles with surprise). What's the matter with that… what… what… what… oh! What, what, what am I supposed to... Oh dear! (6.53s) Very sick boy (sad expression/stretches down the corners of the lips) (3.39s) I can't think what else to say there really (2.53s)… Very sick boy (8.53s) Although there is a hand there isn't it really? *Examiner*: Yes. *Mrs M*: Hand there (16.6s)... I don't know what you can say about that really (frowning)… He is sick. *(62s Total)*	*P10 (Female, Bilateral medial frontal lesion)*: Oh gosh! (6s) Poor baby. He is very, very ill. He is very, very ill. Because, I think, he has been starved... Starving situation isn't it? (20s) Well, let's think that the baby would get better if people contributed towards giving money to help the situation. Examiner: Like charity? *P10*: Yes, yes. *Examiner*: Ok...anything else? *P10:* Oh! (10s) I'm very slow at thinking things like this, sorry. I can't think in anything else. *(88s Total)*	*C16 (Female)*: Well… Some case, but somebody is there… There is a hand behind the child, supporting him. He is getting medical treatment…for the malnutrition, he is getting treatment… There are monitors attached to him, so somebody is caring, which is good because lots of children in that situation don't have that. Mmmm! That he is being taken care of is good for the family... Good that he is there, you know, knowing that things are being done... And good for the child if he survives... So he has a chance. *Examiner*: Anything else? *C16:* Not really *(82s Total)*
	Prompting	*Examiner*: Can you think about the positive side of this situation? *Mrs M*: (3.46s) at least she is somewhere where they do him good (emphasis added/ raises eyebrows and the upper eyelid). Examiner: Where they will do him good? *Mrs M*: Yes… lots of things here… here (pointing to medical equipment in the picture)… Oh it is a shame! *Examiner*: Anything else you want to add? *Mrs M*: (8s) (smiles) Doesn't he look old, although he is tiny? *Examiner*: Yes, that is true Mrs M: I don't know.		
Funeral (9220)	Free Reappraisal	*Mrs M*: (5s) That is someone that has died (4s). And they are all very… very… sad that he has died (frowning). Looks like it's all family… I should think… Don't you think? *Examiner*: Probably. *Mrs M*: (14s) yes (9s). I don't know (she moves away from the screen while frowning). Very sad (she sighs and frowns). *(57s Total)*	*P34 (Female, Right dorsolateral frontal and insula lesion)*: (11s) They are mourning him (35s) At least they have somebody with them to look after them (26s) The women in these countries seem to get very excitable (15s). *Examiner*: You mean as a cultural difference? *P34*: Yes (20s). *Examiner*: Anything else? *P34:* (4s) He is comforting her... He's got hold of her wrist. And she must be comforting that one, because she is touching her. *(140s Total)*	*C17 (Female)*: Looks as if the gentleman has died, and I'm going back to what I said about the old lady in the bed (she refers to a picture used during the training period)... His organs can be used, again, who knows? One of these people around the coffin could be in need of a transplant one day, or even now, so he can be used. I have never put down death as being the end of it, and there is always another kind of life afterwards... But I think he can be used if he has agreed, and also his family. *Examiner*: Anything else? *C17:* The grief will become less with time. We all have to do that, because life goes on... And we will remember him with nicer thoughts in the head than actually seeing him as he is in a coffin now. *(74s Total)*

	Prompting	*Examiner*: Can you think of a positive side of this situation? *Mrs M*: (she frowns) (9s) No, I can't really (raises both eyebrows). *Examiner*: Difficult to think in a positive aspect? Mrs M: Yes. (8s) Although it looks like that is the wife. And that is the… (8s)… Although I don't know what this is (points to a hand that appears from the left lower corner)… A hand… (9s)… Yes (keeps staring at the picture without saying anything).		
Car Crash (9903)	Free Reappraisal (65s)	*Mrs M:* Wow! That is a big a (3s), big car there (she smiles and then bites the lower lip). (16s) I should think there is a lot of people there that didn't get out (she moves very close to the screen, and frowns while contemplating the picture, then she moves back to a straight position and stretches her lips horizontally, generating dimples in her face). (20s) Lots of people helping people there. (13s) No, I don't know. *(61s Total)*	*P24 (Female, left dorsolateral frontal and Frontal Eye Field lesion):* (5s) A car… Overturned… Somebody is hurt… Somebody is on the stretcher…(9s) it is like it was a chain thing, because all you see are these things here but maybe they had to jack up everything (5s) Violent cutting out... Person is on a stretcher and... Hopefully he is alive. *Examiner:* Anything else? *P24:* No. *(54s Total)*	*C15 (Female)*: (6s) The positive thing there is that help has actually arrived hasn't it? Just hope that the people are rescued … Gosh! There is not much left of that car… You just hope they got out in time… But at least there is help, the fire brigade is there... The policeman... Yes so they've got the best around them... Haven't they? *Examiner:* Anything else? *C15:* No. *(45s Total)*
	Prompting	*Examiner*: Can you think of a positive side of this situation? Mrs M: (4s) He went too fast, he was going too fast, I would think (raises her eyebrows). It tripped over, fell over like that (makes the gesture with her hand of something hitting the table) So therefore lots of people got (frowning). There is nothing else there, is it? Not really (She moves her head trying to see the picture from different angles). (6s) *Examiner*: So what would be a positive side of this situation? *Mrs M*: they are looking after the people there... Whether they do anything... At least they are helping aren't they?		
Graveyard (9926)	Free Reappraisal	*Mrs M*: Oh! (opens mouth and frowns)… It's sad (10s)… (Looks at the picture from different angles) Yes (11s). Who are these (frowning and pointing to the people in the slide)? Does it matter who they are? *Examiner*: We don't know who they are. *Mrs M*: Could be their relatives (4s) More like relatives than anything… (4s) Yes, looks like relatives, really. (10s) That's John (pointing at a name in the grave stone contemplated by the couple) *Examiner*: I think it says Jake… (11s) *Mrs M:* Jake… Very sad (sad expression/stretches the corners of the mouth down). Looks like if… (6s) Very sad… (10s) I don't know (moves away from the screen while frowning). *(93s Total)*	*P13 (Female, Bilateral dorsolateral frontal lesion):* (5s) At least they have got each other. They have lost… I think is their child (5s)…Mmmm. At least they've got each other, so they can comfort each other (10s) They are all the same names… Wilcox, Wilcox, Wilcox and Wilcox (referring to the names on the graves). (20s) Oh dear! *Examiner:* Anything else? *P13*: No. *(55s Total)*	*C3 (Female):* Obviously this couple is mourning a lost person. I should imagine it's a child, probably,. But it looks as if they love each other, and can console each other. The child, the person had a really nice burial, and there is a lovely head stone and flowers. And the hope is that they will get over it... But otherwise they look... They are sad... but they look as if they are together, and in a good relationship. That's it I think. *(40s Total)*

Prompting	*Examiner:* Can you think about the positive side? *Mrs M:* (19s) They've got each other (her frowning disappears, and she smiles while looking at the examiner). And they are sorry that this happened (moves her head from side to side while showing a sad expression, by frowning, and at the same time raising the inner corners of the eyebrows).

Appendix 6. Semi-structured Interview Protocol.

1. Has your brain injury had an impact on your emotional life?
    a. Individually
    b. Interpersonally
2. Has your brain injury had an impact on the way you experience your emotions?
    a. Positive or negative emotions
    b. Intensity and frequency of emotions
3. Has your brain injury had an impact on the way you understand your emotions?
    a. Understand what you feel
    b. Capacity to name what you feel
    c. Understand why you feel in a certain way
4. Has your brain injury had any impact on the way you regulate the emotions you feel?
    a. Capacity to distract yourself from negative thoughts or feelings
    b. Capacity to focus on a positive thought or feeling when you are feeling negative
    c. Capacity to supress or disconnect from negative thoughts or feelings
    d. Capacity to find a positive side on negative event as a way of feeling better

References

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