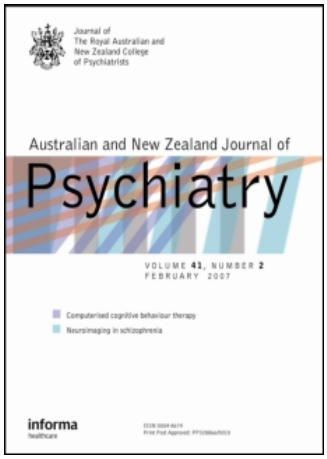

Australian and New Zealand Journal of Psychiatry

Publication details, including instructions for authors and subscription information:
http://www.informaworld.com/smpp/title~content=t768481832

Traumatic memories: bridging the gap between functional neuroimaging and psychotherapy

Julio F. P. Peres [a]; Alexander McFarlane [b]; Antonia G. Nasello [c]; Kathryn A. Moores [d]

[a] Neuroscience and Behavior, Institute of Psychology, University of São Paulo, São Paulo, Brazil
[b] Centre of Military and Veterans' Health, University of Adelaide, Adelaide, South Australia, Australia
[c] Department of Physiological Science, Medical School of Santa Casa of São Paulo, Brazil
[d] Cognitive Neuroscience Laboratory, School of Psychology, Flinders University, South Australia, Australia

Online Publication Date: 01 June 2008

To cite this Article: Peres, Julio F. P., McFarlane, Alexander, Nasello, Antonia G. and Moores, Kathryn A. (2008) 'Traumatic memories: bridging the gap between functional neuroimaging and psychotherapy', Australian and New Zealand Journal of Psychiatry, 42:6, 478 — 488

To link to this article: DOI: 10.1080/00048670802050561
URL: http://dx.doi.org/10.1080/00048670802050561

Traumatic memories: bridging the gap between functional neuroimaging and psychotherapy

Julio F. P. Peres, Alexander McFarlane, Antonia G. Nasello, Kathryn A. Moores

Objective: Neuroimaging studies have highlighted important issues related to structural and functional brain changes found in sufferers of psychological trauma that may influence their ability to synthesize, categorize, and integrate traumatic memories.
Methods: Literature review and critical analysis and synthesis.
Results: Traumatic memories are diagnostic symptoms of post-traumatic stress disorder (PTSD), and the dual representation theory posits separate memory systems subserving vivid re-experiencing (non-hippocampally dependent) versus declarative autobiographical memories of trauma (hippocampally dependent). But the psychopathological signs of trauma are not static over time, nor is the expression of traumatic memories. Multiple memory systems are activated simultaneously and in parallel on various occasions. Neural circuitry interaction is a crucial aspect in the development of a psychotherapeutic approach that may favour an integrative translation of the sensory fragments of the traumatic memory into a declarative memory system.
Conclusion: The relationship between neuroimaging findings and psychological approaches is discussed for greater efficacy in the treatment of psychologically traumatized patients.
Key words: multiple memory systems, neuroimaging, neuroscience, psychotherapy, traumatic memory.

Australian and New Zealand Journal of Psychiatry 2008; 42:478–488

The aim of this article was to synthesize evidence from neuroimaging and psychotheraphy research in relation to post-traumatic stress disorder (PTSD), so that integration might bring greater efficacy to the treatment of patients suffering from the re-experiencing of traumatic memories. The reorganization and reconstruction of past traumatic experiences into more adaptive memories is used as the model guiding the therapeutic process for trauma survivors.

A single traumatic event can be processed in very different ways by individuals who experienced the same traumatic episode [1]. Jones *et al*. demonstrated that the psychopathological signs of trauma are not static over time, nor is the form of the expression of traumatic memories [2]. This fluidity is a consequence of the sensitization [3–5] that is driven by reminders of the traumatic event and the vulnerability of memory to being modified with repeated recall [6].

Julio F.P. Peres, Research Associate in Neuroscience and Behavior

Neuroscience and Behavior, Institute of Psychology, University of São Paulo, Rua Maestro Cardim 887, São Paulo 01323-001, Brazil. Email: julioperes@yahoo.com

Alexander McFarlane, Professor of Psychiatry

Centre of Military and Veterans' Health, University of Adelaide, Adelaide, South Australia, Australia

Antonia G. Nasello, Professor of Physiology

Department of Physiological Science, Medical School of Santa Casa of São Paulo, Brazil

Kathryn A. Moores, Research Associate in Psychology

Cognitive Neuroscience Laboratory, School of Psychology, Flinders University, South Australia, Australia

Received 9 December 2007; accepted 18 February 2008.

The sensitization of neural pathways involved in this reactivity [7] is central to understanding the neurobiology of PTSD [8].

One of the main psychological sequelae of traumatic experiences is conditioning of specific fears [9]. In addition to PTSD, traumatic memories can significantly influence simple phobia [10], major depression [11], and somatoform disorders [1,12], among other disorders. Comorbidities occur frequently, and typically include major depression, substance abuse, panic and anxiety disorders, obsessive–compulsive disorders, and phobic disorders [13]. Traumatic memories have also been observed in many people who manifest dysfunctional behaviour patterns, but do not meet diagnostic criteria for psychiatric disorders. For example, the prevalence of partial PTSD in the general population is estimated to be approximately 30% [14].

Neuroimaging studies of sufferers of traumatic memories

Single-photon emission computed tomography (SPECT), positron emission tomography (PET), and functional magnetic resonance imaging (fMRI) techniques have provided information about the dynamics of brain activity in sufferers of traumatic memories [15]. The neural substrates underlying traumatic memories have been induced with personalized narrative trauma scripts, images, sounds and virtual reality equipment. The diversity of findings and the heterogeneity of symptomatology among people suffering psychological traumas suggest that it may not be possible to identify one specific neural circuit underlying PTSD. Nevertheless, neuroimaging studies of symptom provocation have identified some consistent patterns, including reduced left hemisphere activity, and hypoperfusion in the anterior cingulate (AC), dorsolateral prefrontal cortex (PFC), hippocampus, and Broca's area. Other areas have shown consistently increased activation, including the parahippocampal gyrus, posterior cingulate, and amygdale. Less consistent findings include bilateral reduction in activation of the thalamus and fusiform gyrus, and increase in activation of the right insula and cerebellum [15–17].

Rauch was the first to use PET and personalized script-driven imagery to temporarily provoke symptoms in individuals with PTSD [18]. The study found increased perfusion in limbic and paralimbic structures of the right hemisphere, including the orbitofrontal cortex, insular cortex, anterior temporal pole, and middle temporal cortex. Broca's area in the left inferior frontal cortex showed significantly decreased blood flow during provocation of traumatic memories, although not all studies have replicated this finding [19].

There is consensus that limbic and paralimbic regions are involved in the expression of emotional memories [3–5,7]. More specifically, activation of the amygdala and anterior paralimbic structures is implicated in the processing of negative emotions such as fear. Studies of war veterans with PTSD, during visualization of combat images showed increased activation of the anterior ventral cingulate gyrus and right amygdala, and reduced activity in Broca's area [20,21]. A SPECT study by Liberzon *et al.* using combat veterans with and without PTSD and healthy controls, found left amygdala activation in response to combat sounds in PTSD patients only, but no amygdala activity in response to neutral sounds [22]. Similarly, Rauch *et al.* found that people with PTSD had greater activation of the right amygdala when shown frightening faces, compared to the controls [23].

PET has been used to study veterans to measure patterns of neural activity associated with traumatic images and sounds. Decrease in activity of the left PFC and AC cortex of those individuals has also been demonstrated [24]. In another study with war veterans, fMRI was used to measure changes in activation of the left AC cortex in response to a cognitive activation paradigm (counting Stroop), where subjects connected the number of combat-related, negative, and neutral words [25]. Individuals with PTSD had decreased activity in the left PFC and AC when compared to the control group. In contrast, other SPECT studies of war veterans, did not find differential activity in these regions in response to trauma-related stimuli in PTSD, but found increased activity in the middle PFC that was not correlated with symptoms [20,26]. Two PET studies of women victims of childhood sexual abuse utilized directed scripts of neutral images and events related to the trauma [16,27]. The scripts evoked memories of abuse experiences in all women, and resulted in increased bilateral activation of the posterior cingulate and motor cortex, but there was no differential activity in the middle PFC or AC in women with PTSD relative to controls. Liberzon *et al.* and Zubieta *et al.* conducted SPECT using similar methodology, involving war veterans with PTSD, publishing findings on PFC activity, which appeared to modulate the response to fear [22,26]. In contrast to the typical findings, a regional increase in blood flow (not decrease) was

found in PFC in individuals with PTSD. This discrepancy may relate to the selection of this region in a region-of-interest analysis, which was derived from a separate control group or a confounding variable of a dissociative subtype of PTSD. Lanius *et al*., using a script-driven symptom provocation paradigm, have observed greater activity in the right posterior cingulate, right parietal lobe, and right occipital lobe, in PTSD and less activity in the left hemisphere [28]. These findings support the suggestion of the inherently non-verbal nature of traumatic memory recall in PTSD subjects, compared to a more verbal pattern of traumatic memory recall in subjects without PTSD.

Despite some inconsistencies, there are reproducible neuroimaging findings in studies of traumatic memories. Functional neuroimaging has indicated greater activation of the right amygdala and anterior paralimbic regions, structures that are known to be involved in processing negative emotions, deactivation of the Broca's area and other non-limbic cortical regions, and decreased activity of the left PFC and cingulate cortex in response to trauma-related stimuli in individuals with PTSD [15,29].

There is also evidence to suggest that the failure of the medial PFC/AC network to regulate amygdala activity may extend beyond the situations of threat reminders associated with traumatic memory [24], because these circuits are also implicated in the processing of facial expression and affect [30], and have been found to be abnormal in PTSD [23,31]. The exact nature of the disruption of the networks in PTSD is unclear, but such findings suggest that there is an abnormality in the networks involved in this processing of affective states. The Felmingham *et al*. study was of particular interest, and demonstrated less ability to differentiate between fearful and resting facial expression in PTSD [31], possibly reflective of the emotional numbing symptom in PTSD, which has disruptive social implications.

Williams *et al*. explored the time course of activations associated with processing of fearful faces in PTSD and found that while traumatic emotions had a primary impact on the medial prefrontal systems, there was also a breakdown of the laterality of AC responses, which intensified with repeated exposure [32]. The lack of coupling of the amygdala and AC in the PTSD subjects may account for the disruption of spatiotemporal activity observed in this disorder.

Another body of research has examined the processing of non-trauma-related stimuli in PTSD [33–35]. This line of research is of particular interest because it explores the question as to whether there are differences between PTSD subjects and controls in their ability to manage their day-to-day environment [34]. PTSD patients demonstrated reduced activity in the left dorsolateral and inferior parietal cortex, indicative of decreased recruitment of these key areas involved in verbal working memory updating. Event-related potential (ERP) data from these same subjects [35] showed an abnormal pattern of cortical source activity during this updating process in PTSD, with a strong reduction in left frontoparietal activity, systems involved in attention, working memory and interactions with medial temporal areas during episodic memory. The abnormalities that have been identified raise the question as to whether the difficulties that individuals with PTSD have in dealing with traumatic reminders may, in part, reflect a more pervasive abnormality of information processing [19]. There is extensive work demonstrating ERP abnormalities in PTSD [36].

One of the challenges in interpreting these data is in understanding the extent to which such changes are indicative of primary pathology in the processing of traumatic memories or whether they are part of compensatory changes that would represent partial resilience to trauma exposure. Britton *et al*. found decreased activation in the amygdala to neutral memories in individuals with PTSD and increased activation to traumatic reminders in both PTSD and trauma-exposed individuals who did not develop PTSD [37]. In general, the pattern of activation for PTSD patients was midway between those for combat-exposed and non-traumatized controls, indicating that they may have partial or less effective regulation of amygdala activation than combat-exposed controls. PTSD patients also showed a failure of activation in the AC and diminished medial PFC activity in response to traumatic memories. These findings emphasize that the interaction between neural circuits, rather than activity of specific neuroanatomical regions, is central to understanding the neurobiology of PTSD. The AC is of importance in the monitoring of emotional experience [38] and the greater intensity of negative emotions in PTSD may represent a failure of this region to exert appropriate top-down inhibition [37].

One interesting study investigated the temporal dynamics of amygdala activity in PTSD, and found increased early amygdala responses, which in the left hemisphere correlated with symptom severity [39]. PTSD patients also failed to show the normal pattern of habituation to threat-related words (unrelated to trauma), and instead showed a pattern suggestive of sensitization. In summary, this pattern of reactivity

and increasing responsiveness to threat stimuli in PTSD provides valuable neurobiological insights into the difficulty that patients have in modulating their reactivity. Chung *et al.* in a SPECT study on patients in a resting condition, found increased blood flow in limbic regions and decreased perfusion in the superior frontal gyrus and parietal and temporal regions in PTSD, further suggesting general dysregulation of regions involved in memory and emotion in PTSD [40].

A novel fMRI study explored the processing of social cognitions associated with empathy judgments in PTSD [41]. Participants were scanned before and after modified cognitive behavioural therapy, with healthy people showing increased activation in the left middle temporal gyrus, associated with empathy judgments, and posterior cingulate gyrus activation, associated with forgivability judgments. In patients, activity in regions activated by empathy and forgivability judgments increased as PTSD symptoms resolved, suggesting networks that might underpin the symptoms of social withdrawal and emotional numbing.

Limitations

Individuals with PTSD typically present with various comorbidities, including substance abuse, depression and insomnia [11–13], among others. Different symptoms or emotions may accompany specific neural interactions during retrieval of traumatic memories [42]. For example, dissociative experiences may be markers of potential psychoneurophysiological differences in PTSD [43]. There are also important interindividual variations in the processing of life events and basic emotions, which are likely to account for many of the inconsistencies in previous work [44]. Thus, symptomatological heterogeneity and the peculiarities of traumatic memories are complex and difficult factors to control in neuroimaging studies. Further, the heterogeneous nature of trauma associated with PTSD may also cause difficulties in inducing reproducible responses in patients, as well as comparable activations in control subjects. It is now clear that the division of PTSD into more specific subtypes is necessary in future diagnostic manuals to better categorize patterns of symptomatology and the respective neural substrates involved.

Another important consideration is recency of the memories being studied, which may be important for neuroimaging studies involving war veterans or survivors of childhood sexual abuse. We know that memory expression may be modified over time, causing changes in the neural substrates involved [15].

Comprehending neuroimaging findings

As advances are made in interpreting the meaning of neuroimaging findings, this work may lead to important refinements of therapeutic interventions for the treatment of traumatized patients [45]. Clinical studies suggest that abnormalities in interpretation, synthesis, and integration of emotionally salient episodes play a crucial role in experiences being received as traumatic [46].

Decreased hippocampal volume, often associated with PTSD, may have etiological significance for dissociation and errors in interpretation of information related to threats [47]. Moreover, reduction or blockage of hippocampal integrative function can fragment the various aspects of the memory of the traumatic experience into body sensations, smells, and sounds that seem strange and separate from other life experiences [46]. It has been proposed previously that impaired hippocampal function may contribute to the fragmentation of experience in patients with PTSD [12].

People with PTSD exposed to personalized narratives of their trauma demonstrate a different pattern of activation, highlighting networks that are more associated with affective processing and less associated with linguistic representation. It appears that disruption of activity in the left frontal region is of particular importance in PTSD [21,46] and the propensity to engage right hemisphere networks. It has been suggested that the left hemisphere sequentially organizes information and is responsible for problem solving and categorization operations [15,46], which may explain why traumatic memories are experienced as 'belonging to the present', because brain regions necessary for sequencing and categorizing experiences are not adequately activated.

Individuals with PTSD were examined with SPECT before and after treatment involving eye movement desensitization and reprocessing [48]. After treatment there was increased activity in the AC and left frontal lobe, perhaps influencing neuronal activity in the areas implicated in PTSD, particularly the left hemisphere. The Farrow *et al.* study further indicated that after treatment there was greater activation of left hemisphere pathways associated with empathetic responses, with concomitant symptomatic improvement in PTSD [41].

During exposure to traumatic narratives, several studies have also shown a decline in activation in Broca's area of the left inferior frontal gyrus. Shin *et al*. verified that only individuals with PTSD exhibited a failure of activation in Broca's area and the AC [20]. Other studies have also identified significantly decreased activity in Broca's area, and are perhaps linked to the difficulty that PTSD individuals have in assimilating the traumatic event into a narrative structure [15].

The PFC and AC have been shown to be deactivated during retrieval of traumatic memories in patients with PTSD. These structures may inhibit responses to emotional stimuli [7,8]. In addition, dysfunction of dorsolateral PFC may mediate problems with language, cognition and integration of verbal expression with emotions. Decreased PFC activity may extinguish response to the symptoms of PTSD, attenuating the negative feedback of amygdala activity [16,17,49]. Interestingly, studies that have examined the temporal dynamics of these neural networks suggest that one of the key factors in PTSD is a progressive sensitization and increasing responsivity to non-specific threat stimuli, even in a brief period of time [39].

The failure to adaptively process threat suggests that in PTSD, there is a propensity for increasing strength of affective responses with time, which disrupts the modulation of affect. Such neuroimaging findings highlight the experience of patients and underscore the disruptions of processing of the external world. Further evidence for a pervasive problem of information processing of non-trauma-related stimuli in PTSD suggests that treatment needs to address this aspect of the phenomenology of the disorder [34–36]. The sense of being confused and aroused by the external world goes beyond specific reminders of the trauma.

Although neuroimaging studies of PTSD are still in an embryonic stage, disruption of hippocampal function, deactivation of Broca's area, the left hemisphere, and PFC are consistently implicated in the pathophysiology of PTSD, expressed as a difficulty in synthesizing, categorizing, and integrating the traumatic memory [45]. The subtle impact of the processing of facial expression may affect the sense of engagement and empathy in the therapeutic setting. These abnormalities occur against the background of a more pervasive disruption of information processing in PTSD of stimuli unrelated to the trauma. These limitations should be considered as an important factor challenging the capacity of these patients to engage in the therapeutic process.

Trauma and memory systems

'Trauma', in its Greek etymological root, means lesion caused by an external agent. The term 'psychic trauma' was first coined by Freud during his studies on the aetiology of neurosis, in which he stated that psychic traumatism is characterized by excessive excitement related to an individual's tolerance and capacity to integrate and psychically elaborate this stimulus [50]. But characterization of an event as traumatic does not depend only on the stressor stimulus, and there is no single human response to the same traumatic events or a 'universal reaction to trauma' [2]. The search to understand idiosyncratic responses to trauma has turned to the contribution of personality factors [51], with the way that people process the stressor event appearing to be a critical factor in determining whether an event will be encoded as traumatic or not [52].

There are several complex memory systems involved, including declarative memory [53]. Emotional memories interact with the neural substrates of declarative memory [54]. Clinical observations clearly demonstrate that unpleasant emotional memories (charged with sadness, disgust, fear, or rage) can lead to maladaptive changes, such as distortions of perception, assessment, and judgment [55]. Although such distortions may not characterize a traumatic event, unpleasant emotional memories can remain vivid over time, and serve as references for expression of avoidance behaviours. In contrast, an event of greater emotional impact that is perceived as traumatic can lead to abnormal memory phenomena that are typical of PTSD, including the extreme imprinting of the experience, fragmentation of memories for the event, partial forgetfulness, or even amnesia [12,46]. Studies of adults with a history of sexual abuse during childhood present with a consistent picture of dissociative amnesia, occurring more often in victims of interpersonal violence during childhood than in combat soldiers and accident victims [56]. Amnesias for emotional and cognitive content appear to be related to the age at which the trauma occurred, as well as the constancy of the stressor event, with younger age and prolonged duration of traumatic stressor associated with greater probability of significant amnesia [57]. Thus, terrifying experiences can either totally resist integration, or can be etched in an 'indelible' manner in a person's memory and, under many circumstances, traumatized individuals report a combination of these two phenomena [46]. For example, in studies of post-traumatic nightmares, some individuals reported that they repeatedly experienced the same traumatic scenes

without change over a 15 year period. It is curious to note that few patients describe their perceptions as exact representations of sensations experienced at the time of the trauma [58]. The permeability of traumatic memories to cultural influences, and changes of their expression over time has been demonstrated [2].

Van der Kolk investigated the differences in recovering memories of traumatic experiences from recovering memories of significant but non-traumatic events [46]. Non-traumatic memory recall was associated with narratives and was without strong sensorial manifestation. In contrast, 78% of individuals who were questioned about traumatic memories from both childhood and adult traumas, initially reported not having any memory of the event and were unable to give an account of what happened. Regardless of the age at which the trauma occurred, all individuals stated that they initially remembered the trauma in the form of sensorial flashbacks, such as visual, olfactory, affective, or auditory impressions, with the awareness and capacity to describe what actually happened developing over time. That study demonstrated that the key distinction between the recovery of the traumatic and emotional events was the relative absence of any narrative expression of the traumatic memory.

Functional neuroimaging studies suggest that explicit retrieval is preferentially associated with increased activity in prefrontal and medial temporal regions [59], and the phenomenological awareness that accompanies episodic memories may arise within the hippocampal–frontal memory system. This information has to be bound together to be retrievable as a conscious memory, and the hippocampus is critical to this binding function [60]. Studies point to an important distinction between hippocampally dependent and non-hippocampally dependent forms of memory that are affected differently by extreme stress [61]. One form, termed 'verbally accessible memory' (VAM), supports ordinary autobiographical memories that can be modified and interact with other autobiographical knowledge, so that the trauma is represented within a personal context consisting of past, present, and future. These traumatic memories are influenced by information that the individual has encoded before, during, and after the traumatic event, and that received sufficient conscious processing to be transferred to long-term memory in a form that can be explicitly retrieved and verbally communicated. Another form, termed 'situationally accessible memory' (SAM), contains information that has been obtained from lower-level perceptual processing of the traumatic scene (e.g. visuospatial information that has received little conscious processing) [62] and from the person's bodily (e.g. autonomic, motor) responses. This form of memory is consistent with the phenomenon of trauma-related flashbacks that are a characteristic of severely traumatized people. Because SAMs do not involve verbal representations, these memories are difficult to communicate and may not therefore interact with other autobiographical knowledge. During periods of intense emotion, reduction of hippocampally dependent processing of information and formation of SAMs may result in increased probability of amygdala reactivity to trauma reminders and the person experiencing a sense of current threat. A longitudinal study of the reliability of memories for trauma and other emotional experiences, demonstrated that traumatic memory imagery tended to persist with no apparent decrement, whereas emotional memories were subject to considerable distortion over time [63]. The findings converge on the non-hippocampally dependent nature of traumatic memories, and suggest a tendency of these memories to resist change with the passage of time. Nevertheless, it is clear that that at any time multiple memory systems are activated simultaneously and in parallel, and findings suggest that these systems may interact [64–66]. One treatment study, using an exposure and cognitive restructuring process, suggests an interaction between SAM and VAM systems [49].

Clinical observation indicates that the narrative organization of mnemonic content will assist its permeability to change. If an event, once charged with emotions, can be integrated into an individual's autobiographical memory, it tends not to be available anymore as a separate and immutable entity. The memory becomes modified by associated experiences, emotional context and a state of consciousness during the recall process [53]. Breuer and Freud asserted that bringing early traumatic material to consciousness would allow 'abreaction' and quick remission of symptoms, with psychotherapeutic approaches favouring the retrieving the mis-stored memory and integrating this memory with narratives. This reworking consisted of building cross-links between the traumatic memory and other memories and thoughts, believed to reintegrate the isolated traumatic memory into 'normal' memory systems [50].

Implications of neuroimaging findings in psychotherapeutic treatment: the challenge of integration

Questions concerning the neurobiological effects of psychotherapeutic interventions are now given con-

siderable importance within the field of psychiatry and psychology. Neuroimaging studies have provided evidence for changes in cerebral dynamics after pharmacotherapy or psychotherapy [45,67]. PET, ERP and fMRI studies have provided substantial evidence that the cognitive and behavioural changes that occurr within a psychotherapeutic context can cause alterations in the regional cerebral metabolism of patients with obsessive–compulsive disorders [68], major depression [69], as well as in patients with social phobia [70] and specific phobia [71]. The findings suggest that the psychotherapeutic interventions have the potential to modify dysfunctional neural circuits associated with the disorders studied [72].

Psychological treatments are presently considered the first-line intervention of choice for sufferers of traumatic memories with PTSD [73]. According to the Expert Consensus Guideline Series for treatment of PTSD [74], exposure-based therapy was indicated as a psychological treatment of choice for flashbacks, intrusive thoughts, trauma-related fears, and avoidance. All of the multicomponent treatments that include cognitive interventions have exposure as one of their key elements [48,75]. In fact, revisiting traumatic memories can bring therapeutic benefits, as long as a well-structured process of restructuring of the emotional content is used [76].

Ehlers *et al*. evaluated the quality and content of memories of individuals who had been through different traumatic experiences [77]. The authors emphasized the importance of identifying the moment of greatest emotional salience, so that associations and patterns of arousal established at that moment could be reprocessed. Conscious attention to unfolding events is likely to result in richer VAM representations and, theoretically, sustained attention to flashbacks may promote information transfer between these systems, leading more rapidly to amygdala inhibition [61]. Therefore it is reasonable to postulate that well-designed exposure and cognitive restructuring psychotherapies may enable the critical translation of the fragmented sensory elements of the traumatic memories into a more integrated, narrative representation of the memory [53]. In this respect, psychotherapy should facilitate a new framing of the traumatic experience by reviving and strengthening memories of successful coping and self-effectiveness prior to the trauma. These memories, their respective emotional valences and states of consciousness, may be recognized and interconnected with the memory of the trauma during a restructuring session. We found that each time a patient narrated a traumatic episode, the narrative could be structured with new cognitive and emotional elements extracted from reinforced memories of successful coping. Therefore, the reinterpretation and reconstruction of traumatic memories may lead to changes in neural networks involved, and relieve symptoms [49].

Increasingly, psychological interventions have focused on exposure-based therapies for cognitive restructuring of past events [42,78], with the essential component involving repeated exposure to memories of the traumatic stressor. Is should be noted, however, that confrontation with traumatic memories through debriefing has not been effective in treating individuals with PTSD [79]. Thus, confrontation of the memories does not appear to be sufficient to provide a therapeutic effect, but also requires the restructuring and integration of memories. A point worth noting is that Breuer and Freud found pre-psychoanalytic cathartic treatment alone generally ineffective, and the latter turned to a more narrative type of approach in transference-based therapy [50]. Moreover, we believe it is critical for narrative to involve the search for constructive lessons. Thus, psychotherapy will sensitize the traumatized individual's resilient traits by propitiating access to this repertoire from their pre-trauma life history. Good examples of successful coping by individuals who drew lessons from their traumatic experiences and so developed their resilience may also provide models for trauma victims when developing new types of cognitive processing.

Memory reconstruction

There is consensus that emotionally charged memories are not static, but rather are interpretations, new reconstituted versions of the original event [80]. Loftus and Polage observed the imprecise nature of remembering by examining the phenomenon of false memories [57]. It has also been demonstrated that responses to traumas are guided by emotional beliefs, independently of the precision of the information [52,81]. Thus, neuroscience findings provide crucial insight for psychotherapy, highlighting that emotionally charged memories are peculiar representations of an event, distant from the original episode, but salient in their significance for the individual.

We postulate that the re-interpretation and reconstruction of traumatic memories can be used with exposure and cognitive restructuring psychotherapies, to alleviate some of the distressing symptoms of PTSD, by changing the nature of the representa-

tions of the traumatic event. It is therefore crucial to consider that the most important modulators of the acquisition, formation, and evocation of traumatic memories are the emotions involved and the individual's conscious access to the memories [82,83]. The retrieval of traumatic memories, whether spontaneous or provoked, occurs in an altered state of consciousness. Vermetten and Bremner reviewed remarkable similarities in neuroimaging studies of traumatic recall and hypnotic processes [84]. The same brain structures – thalamus, hippocampus, amygdala, medial PFC, and AC cortex – were involved in both research lines. We propose that therapeutic interventions focusing on emotions and the conscious processing of these events will modulate the memory for these events, effectively changing the interactions between underlying neural networks. It is argued that this shift of consciousness, will result in changes in the perception of the same event [85]. Retrieval and interpretation of the original altered states of consciousness also permit the transformation of 'early' traumatic memory into 'later' explicit memory [86]. In accordance, Breuer suggested that hypnosis might be useful to access and modulate that altered state of consciousness and remobilize memory systems for the purpose of cross-linking them with narrative memory functions [50]. Other work supports the idea that the use of altered states of consciousness can be an effective tool in the formation of new patterns of perception involving thought, feelings, and behaviour [87,88]. By re-experiencing the trauma in different states of consciousness and, consequently, acquiring different perceptions of the same traumatic event, the individual may efficiently transfer information from the non-hippocampally dependent memory store to the hippocampally based memory system [49,53]. In many cases the trauma per se must be accessed before mourning can proceed. In this respect, Pierre Janet's hypnotherapy and its approach based on a dissociation model has been used satisfactorily for cases in which traumatic grief occurs when psychological trauma obstructs mourning [89].

Psychotherapy can be informed by the neuroimaging literature in relation to the difficulties that PTSD patients have with processing both trauma-related and trauma-neutral information. The challenge is to be able to grasp the experience of an individual whose registration of the environment is fundamentally different from normal perception, and explore the attribution of meaning. Neuroimaging research highlights that a large component of a patient's cognitive and affective experience has changed their capacity to create meaning and manage their perceptions of those experiences, particularly with regard to interpretations of their current environment. The challenge of the psychotherapy is to draw the patient out of this world by facilitating changes in perception and meaning.

When a traumatic memory can be reconstructed and reintegrated in this way, it loses intensity and evolves from a traumatic memory into an emotional one. Psychotherapy can facilitate the search for a narrative and integrated translation of the traumatic event, so the experience can be understood and conveyed in communicable language. We argue that psychotherapeutic interventions involving exposure and cognitive restructuring, and accommodating the altered states of consciousness during traumatic memory retrieval, will make an important contribution to the treatment of PTSD.

Conclusions

Psychotherapy appears efficacious in enabling sufferers of psychological trauma to cope better with the memories of their traumatic experience, with the reconstruction of the traumatic memories [49]. Emotionally charged memories are subjective representations of an event, often distorted and distant from the original episode, but salient in their significance to the individual. Although there is a marked degree of inter-individual variability in the processing of memory of life events and basic emotions [44], we postulate that the re-interpretation and reconstruction of traumatic memories will be efficacious in relieving PTSD symptomatology. This process will influence the neural networks subserving these experiences, leading to the formation of new memories that are less fragmented and available for narrative expression, an idea that is consistent with neuroimaging and clinical observations [66]. The modulation of neural circuitry, involving PFC, hippocampus and Broca's area, is a crucial aspect in the development of a psychotherapeutic approach that favours the search for narrative and integrative translations of the sensory fragmented traumatic memory [49,53].

Although insights from neuroimaging research have been linked to psychotherapy, this synthesis is embryonic. Future multicentre studies addressing specific types of traumatic memories, and the age at which they were formed, should be encouraged. Construction of coherent bridges between psychotherapy and neuroimaging must continue, in

order that the two complementary and interdependent bodies of work can bring greater efficacy to the treatment of psychologically traumatized patients [45]. The growing understanding of the neurobiology of emotionally charged memories and their modulation may inform treatment of the victims of psychological trauma.

Acknowledgements

Professor McFarlane and Dr Moore were supported by the National Health and Medical Research Council (NH&MRC) of Australia (Program grant number: 300403) held by R. Bryant., A. McFarlane, D. Silove, R. Clark and M. Creamer. Dr Peres was supported by Fudacao Espirita Americo Bairral–Brazil, and Bial Foundation–Portugal.

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