EASY, EASY

with

PTSD

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Preface

Despite our attempts toward objectivity, I am cognizant that subjectivity is everywhere. Even quantum physics provides us with experiments showing how the experimenter influences physical realities. We negotiate in a world of relative truths, knowing that our understandings are approximations at best of the reality at hand. Subjectivity is present in research, and thus in clinical research also. Throughout this book, researchers' and clinicians' biases are highlighted. In a matter of fairness, an overview of my own conscious biases is provided below. Having been trained as psychotherapist and psychotherapy researcher for many years, I hope to provide a useful comprehension of PTSD and its treatment in this book.

I came to psychotherapy by reading a book, *Dibs: In Search of Self* by Virginia Axline (1964). This transcription of self-psychotherapy with a 5 year old boy opened a completely new wonderful world to me, the world of psychotherapy. It was a revelation because I hereby witnessed the most beautiful human relationship I had ever seen. Not surprisingly, my main research focus later became the alliance, within the larger context of evaluating the efficacy of psychological treatments.

I have profound beliefs that human relationships are transformative, for better and for worst, and psychotherapy unfolds with techniques embedded within the greater background of a human relationship. I have been trained in behavioral, cognitive, humanistic, and psychodynamic approaches, and I know these techniques intimately because I have follow behavioral, cognitive, humanistic and dynamic psychotherapy. I have been trained the use of relaxation, meditation, mental imagery, hypnosis, EMDR, etc. I have used trauma-focused techniques parsimoniously, when significant trauma-related information was inaccessible at the usual waking state of consciousness. I learned that preliminary conditions need to be in place before employing such potentially destabilizing techniques, conditions such as a well-established therapeutic alliance and a demonstrated capacity for affect-modulation.

Given the diversity of nature, I consider that it is always best to remember that there are "different strokes for different folks" and that each therapeutic model has its rightful place. I believe that the more models and methods we know, the more we are likely to choose the most pertinent interventions to adequately and efficaciously assist our patients. I have studied major theoretical approaches, including humanistic, psychodynamic, cognitive, behavioral, and neurobiological models. I have learned behavioral, cognitive, humanistic, dynamic, and other models of psychotherapy.

I hope this book will answer practitioners' needs in their sincere efforts to treat PTSD in a comprehensive and informed fashion. This book is likely to validate the already-present competencies of most practitioners, while orienting them toward the specific needs of individuals with PTSD. Particular techniques can be learned through weekend trainings, but an overall capacity to treat PTSD resides in a long-term learning process.

The field of PTSD has become more and more narrowly focused on standardized therapeutic methods, mostly with cognitive-behavioral models. This emphasis on cognitive-behavioral methods is not due to their particular efficacy, but rather to selective governmental funding provided only for studies concerning cognitive-behavioral methods. Mental health is more and more oriented toward an emphasis on pharmacotherapy and rapid psychological techniques. These therapies can be helpful, but they are inherently limited, like any other. All therapeutic models and methods have a unique and necessary contribution to treating PTSD in a required adequate, comprehensive, and multidimensional model of PTSD.

Trauma-focused therapies have been heavily promoted in the last two decades. In response to this emphasis and bias, I realized the need to say to academicians, researchers, funding agencies, and some psychotherapists "Easy, easy with PTSD".

Only a minority of patients presenting with PTSD can undergo trauma-focused interventions which produce iatrogenic effects and trigger therapy dropout. Clinical trials do not reflect clinical practice and patients' needs and fragilities. In this book, my intention is to remind all of us, with ample empirical and clinical examples, of our basic desire to care for individuals battered by traumatic events and PTSD rather than to focus on standardized 'only partly' empirically-supported treatments for PTSD. For most clinicians, the neurobiological knowledge presented in this book, along with clinical considerations, will mostly be validating of their already careful and caring clinical practice with individuals suffering from PTSD.

In 1991, I founded a clinic specialized in evaluating and treating PTSD, named TRAUMA*TYS.* As PTSD specialists, we employ trauma-focused techniques at times, but only techniques providing full control to patients throughout the whole exercise, and only in a timely fashion. Trauma-focused techniques are always used within a comprehensive therapy and prerequisite conditions are well established. Otherwise, with regret, we found trauma-focused techniques to be ineffective at best or unnecessarily destabilizing. From our clinical experience and errors, we have become cautious in employing trauma-focused techniques. We also welcome people who have been destabilized by the use of a trauma-focused technique by another practitioner. These patients have abandoned the prior therapy without informing with the clinician of the reasons for their dropping out. As one person said, "The therapist was too keen on her technique for me to go back there. She would not listen and would try to convince me to try it again."

Key ingredients of a successful psychotherapy for PTSD appear to be diversified competencies in the psychotherapist, toward PTSD and personality disorders, as well as the psychotherapist's flexibility in using various techniques in a timely manner. Many PTSD specialists have, indeed, integrated therapeutic considerations beyond the scope of their original model. For example, among others, Smyth (1994), a cognitive-behavioral therapist, has integrated core elements of Horowitz's brief dynamic model for treating PTSD.

As with any psychotherapy, a dance is involved in treating PTSD: a dance between persons, a dance between psyches, a dance between mind and body, a dance between avoidance and

approach, a dance between conscious and unconscious. Given that PTSD involves the whole person, all dimensions need to be considered in a comprehensive psychotherapy for PTSD. In this book, we hope to illustrate the dance in which the clinical reader can partake. Now, let's appraise the effectiveness of such a dance, the dance of an integrative psychotherapy for PTSD.

To illustrate the background of the assertions provided in this book, let's take a look at the effectiveness found at TRAUMA*TYS.* Before let's remember that trauma-focused techniques are effective for about 50% of participants in controlled clinical trials comprising of only PTSD with comorbidity and after not including the data of those who had abandoned treatment, about 30% of participants (Schottenbauer, Glass, Arnkoff, Tendick, & Gray, 2008).

At a specialized clinic for treating PTSD, TRAUMA*TYS*, where an integrative treatment for PTSD is employed, an independent study was conducted Brunet (2004), using 100 randomly selected files. Pre- and post-therapy PTSD diagnoses were available in the file using the Structured Clinical Interview for DSM-IV-PTSD (SCID). A remission PTSD rate of 96% was observed; full PTSD remission was found in 48% of patients and partial PTSD remission was observed in another 48%. The psychotherapy provided is integrative and specialized for treating PTSD (Gaston, 1995), based on the dynamic model of Horowitz (1986) for PTSD, with the additions of neurological, behavioral, cognitive elements, along with Masterson (1989) model for treating personality disorders. Patients were not pre-selected and usually present with severe PTSD, several co-morbid disorders (anxious and depressive) and a personality disorder. Psychotherapy lasts as long as needed, with an average of 9 months (Brunet, 2004), ranging from few months to few years. Psychotherapy terminates beyond the resorption of PTSD symptoms after therapeutic gains have been well established.

With patients treated at TRAUMA*TYS*, a neurobiological study was conducted. Results showed that PTSD changes were significantly correlated with activity changes in key brain areas involved in PTSD arousal and arousal-modulation: amygdala, hippocampus, and anterior cingulate cortex (Dickie et al., 2011). Using the Clinician's Administered Interview for PTSD (CAPS), the PTSD rate of remission was found to be 65% after 6 to 9 months psychotherapy, while many patients pursue their therapy (Dickie et al., 2011). The sample comprised subjects with severe PTSD, co-morbidity, functional limitations, and personality disorders.

In this book, neurobiological findings are highlighted to illustrate how trauma-focused techniques for PTSD can destabilize patients and even create long-standing damages. This book does not consist of a scholarly review of the field. The purpose is to provide practitioners with a sufficient knowledge of the neurobiology of PTSD so they can appreciate the particular needs of their patients presenting with PTSD, heightened arousal, co-morbid disorders, functional deficits, and personality disorders. My emphasis is on the need for adjusting PTSD psychotherapy to patients' needs and capacities.

Introduction

Posttraumatic stress disorder or PTSD is one of those rare disorders in the Diagnostic and Statistical Manual for Mental Disorders, DSM, elaborated from a theoretical perspective instead of solely phenomenological observations. In the DSM-5 (APA, 2013), the dynamic interplay between reexperiencing and avoiding is still acknowledged, along with the phenomena of classical conditioning, hyperarousal, and dysphoric cognitions and moods.

PTSD represents a wonderful window to explore and come to better understand the human psyche. Treating PTSD patients is most rewarding and most challenging at times. The art and science of psychotherapy is well accomplished when therapeutic models are interwoven with sound empirical findings, in a way that a unique understanding of each individual can be created.

The occurrence of a traumatic event can be a crisis in a person's life. PTSD is always a crisis in one's life. When PTSD is set in place, psychological phenomena are amplified. All dimensions of a human being are under attack, which can be both a challenge and an opportunity. Patients often come in psychotherapy highly distressed, confused, symptomatic, dysfunctional, suffering, and attempting to continue as if nothing happened. Symptoms of reexperiencing and avoidance can be readily observed and reported. One's psychological defenses and coping mechanisms are not as subtle and mature as previously. Well-adapted individuals often regress to surprising levels of dysfunction, relying often on mechanisms such projective identification and acting out. Yet, individuals presenting with PTSD can undergo a true metamorphosis, leaving psychotherapy stronger than they have ever been. They are forced to work on themselves and care for themselves, to acquire a greater capacity to modulate affects, more differentiated representations of self and others, and a more flexible psychological structure overall. Along with PTSD remission, the brain areas involved in arousal, arousal-modulation, non-emotional memory and long-term memory can end up showing more adapted levels of activity.

Beyond symptomatic alleviation, practitioners wish to help patients get back in the flow of life. Psychotherapy of PTSD is truly successful when patients say, "I don't know why, but I am now happy that this traumatic event happened to me. I am different, stronger and yet more sensitive." Patients can accomplish a successful accommodation of their psychological structure, by including traumatic experiences and their psychological wounds in their overall psyche. For practitioners, treating PTSD can also be a blessing because we are brought to witness with wonder those precious human capacities of adapting and maturing. Treating PTSD entails pain and grief, and usually transforms us. Although treating PTSD is not a simple affair, its treatment can be simplified with adequate knowledge and training, but it should never become simplistic.

As we all know, PTSD is precipitated by the occurrence of a traumatic event and its embedded violence and entailed helplessness. Traumatic events do not happen in a vacuum,

however. PTSD emerges within the greater context of genetic and acquired predispositions, neural pathways underlining mental functions, schemas based on past experiences, unresolved conflicts, more or less mature defense mechanisms and coping strategies, and actual life situations. Many factors influence both the onset and the course of PTSD. In approaching PTSD treatment, it is useful for practitioners to view any clinical case as comprising predisposing, precipitating, and maintaining factors. Theoretical models are useful to sort out hypotheses about the processes at play. These factors and hypotheses will be explained throughout this book.

Before treating any given patient presenting with PTSD, two key questions need to be asked. (1) Should PTSD be treated by inviting patients to reexperience the traumatic event, or not if at all? (2) Should PTSD be treated by addressing issues such as stressors, avoidant behaviors, pathogenic cognitive patterns, unresolved affect-laden conflicts, and/or structural deficits?

These two questions can be summarized as whether PTSD treatment should be trauma-focused or person-focused, or both. Whether to use trauma-focused techniques and/or person-focused interventions may depend on the patient's capacities, but empirical evidence informs us that person-focused techniques are equally effective and provoke less therapy dropout (thus less iatrogenic effects, although the latter are almost never reported in published articles). This book attempts to answer these questions.

The main contention of this exposé is two-fold: (1) trauma-focused techniques are not as efficacious as researchers would like us to believe and they can be detrimental to individuals suffering from PTSD; (2) all major therapeutic approaches can contribute to treating PTSD, however, a comprehensive and integrative approach is required to treat successfully patients presenting with PTSD, especially if the PTSD is severe and associated with complications.

Psychodynamic, humanistic, cognitive, behavioral, and neurological phenomena are at play in psychotherapy in varying degrees, whether we recognize them are not. In a book chapter (Gaston, 1995), I have previously outlined how these ingredients can be integrated for treating PTSD. Many authors have proposed similar approaches (Epstein, 1991; Jakovljević et al., 2012; McCann & Pearlman, 1990; Smyth, 1994).

This book echoes the ways by which most practitioners conduct psychotherapy for treating patients with PTSD. Most practitioners report being eclectic in their theoretical comprehension and employing integrative approaches in treating mental disorders. This tendency toward eclecticism and integration of therapeutic approaches is pronounced amongst my colleagues. Practitioners tend to combine humanistic, dynamic, cognitive, and behavioral models. They also welcome the adjunct use of pharmacotherapy. Practitioners know that human beings are multidimensional and are at their best when their bodies, minds, and hearts are integrated.

In contrast, the therapeutic methods proposed in the scientific literature are

standardized and rigid. This is required for the conduct of a controlled clinical trial, but such conditions do not apply to clinical practice. Over the last few decades, therapeutic methods have become more and more systematic and focusing on utilizing fewer and fewer techniques. Such trend reflects a general social inclination toward technologizing or robotizing everything, including human beings. Such technical focus comprises uncalculated risks, "The surgery went very well, but the patient died!"

In academia and professional organizations, trauma-focused therapies for PTSD are prioritized and recommended. Subsequently, these potentially destabilizing techniques for treating PTSD are endorsed and demanded more and more as the treatment of choice for PTSD by organizations, governmental agencies, and insurance companies. Such trend is worrisome, although it is empirically based.

Individuals with PTSD need both time and assistance to restructure themselves after having been seriously destabilized by a traumatic event. Human beings developing PTSD are in the throes of uncontrollable inner phenomena, seemingly sprouting out of nowhere, triggering their body into painful states and their mind into greater confusion than ever. Avoidance is part of the disorder and yet they need to avoid in order to not deteriorate their condition. Alone, they are caught into a conundrum, a catch-22 condition.

Experienced practitioners do not collude with avoidance of the traumatic information, but they do encourage patients to avoid trauma-related cues until they have reduced their arousal and have the capacities to modulate anxiety and dysphoric affects. Seasoned practitioners invite patients to address experientially traumatic events only when patients are structurally ready to experience intense dysphoric affects without becoming destabilized. In this vein, experienced practitioners encourage patients who are structured-enough to experientially review their traumatic experiences in psychotherapy. Therefore, for any given patient, an informed middle ground between reexperiencing and restructuring needs to be threaded by practitioners.

* * *

We hope to illustrate how clinical and neurobiological results indicate the necessity to move away from utilizing trauma-focused techniques for treating PTSD in both research and practice. Ample empirical findings are provided, indicating that 'gentler' approaches for treating PTSD such anxiety management, traditional cognitive therapy, dynamic therapy and pharmacotherapy are as effective in clinical trials, if not more efficacious with the general PTSD population, than trauma-focused techniques.

'Above all do no harm.'

Maybe it is time to go back to our core ethical premise and promise as practitioners. Said otherwise, 'the good physician will treat the disease, but the great physician will treat the patient' (William Osler). As psychotherapists, we have borrowed from the medical model too

much, viewing symptoms rather than persons. As researchers, we have standardized psychological treatments excessively. The next 'step' of clinical trials might be to provide treatments which will have been standardized to the precise wording to be employed (exaggeration sometimes helps see a trend in a clear way).

'Proceed from periphery to the core.'

One principle of the medical model needs be remembered and applied in our practice at this time: the least potentially disturbing interventions should always be employed first in psychotherapy. We are ethically required to proceed from least damaging interventions to more potentially damaging ones, in an incremental fashion, constantly assessing the possibility of iatrogenic effects. A transition from 'gentler' interventions to 'harsher' ones should proceed if, and only if, the gentler interventions were not effective *and* conditions allow for the use of potentially destabilizing techniques. Most psychotherapists practice because they care of the human fellows. Therefore, the last thing practitioners wish is to further destabilize patients, to create iatrogenic effects. Over the last decades, such destabilizing techniques have been praised by researchers and endorsed by repeated national institutions. As the Horowitz (1976, 1986, 2001) author of *Stress response Syndrome*, pointed out, psychotherapeutic work needs to proceed from periphery to the core. The treatment of PTSD needs to proceed gradually in order to avoid unduly destabilization of the patient and severe side effects.

PTSD, Known and Less Known

Not all victims will develop PTSD following a traumatic event. PTSD is found in about 25% of victims and the Incidence of PTSD varies across types of traumatic events (e.g., Breslau et al., 1998). The variability of PTSD depends on various factors. Such factors can be classified as predisposing, precipitating, and maintaining. Predisposing and maintaining factors belong to the individuals and their environments while precipitating factors are associated with the traumatic event per se. First, let's consider the complexities of experiencing traumatic events and reexperiencing traumatic events through PTSD.

Before examining trauma-focused therapies for treating PTSD, it may be crucial to review the experience of traumatic events by human beings. Understanding the experience of PTSD is also instrumental in treating PTSD in a sensitive manner. Given the particular vulnerabilities of PTSD sufferers and the damage incurred to their sense of humanity, the consideration of the person per se should precede the use of any technique. A PTSD sufferer needs to be related to as a sentient being rather than a collaborator in completing a therapeutic task or the object of a technique. A therapeutic relationship should be established before any technique is applied, and such relation endeavour takes time, weeks to months.

The Experience of Traumatic Events

To successfully master a traumatic experience, an individual needs to have a well-rooted sense of trust in one's capacities to withstand adversity. Such ability develops over time, in an incremental fashion, after repeated successful experiences of mastery over challenging situations. When one has had such experiences facing adversity, one can more readily perceive traumatic events as a challenges rather than threats (see Chorpita & Barlow, 1998). Such previous experiences of mastery contribute to develop internal representations of self-efficacy, as coined by Bandura. These successful experiences are likely to rely on a mild propensity to react emotionality and on well-developed capacities for affect-modulation.

During a traumatic event, a sense of self-efficacy can prevent one to become behaviorally paralyzed by helplessness or to overreact with rage. To access internal representations of self as competent, one cannot become overwhelmed by intense dysphoric affects. Furthermore, one needs to be able to contain such dysphoric affects, such as fear or anger, if they emerge. After a traumatic event, one needs to steadily access internal representations of others as mostly benevolent, to avoid generalizing one's experience with the perpetrator to every stranger. Benevolent internal representations provide a sense of security in the world despite its adversity (Bowlby, 1988). However, individuals developing severe PTSD can rarely rely on such capacities.

Traumatic events involve the most brutal facts of life: unreasonable aggression, sheer helplessness, outmost vulnerability, complete disregard for one's humanity, death, will to live, core impulse to self-activate, etc. Experiencing traumatic events involves experiencing many of

these harsh realities. In turn, old wounds associated with these issues are powerfully reactivated. Having to urgently deal with present danger, the persons has also to contain a potent resurgence of the unresolved past. Together, past and present trigger such affects/arousal that the person becomes overwhelmed. Ideally, the person continues to act adaptively, but sometimes flight, anger or paralyzing freight take over, leading to less than optimal outcomes. Beyond a newly problematic outer reality to manage, the person has to bear and manage a highly disturbing inner reality, namely PTSD.

The Experience of PTSD

Traumatic events can be re-experienced in at least two usual states of consciousness, awake and asleep. Some would argue that traumatic events can also be experienced unconsciously. Consciously, individuals with PTSD relieve traumatic events through flashbacks. Unconsciously, they may react or feel as if the traumatic event is reoccurring but without a conscious association between their actual experience and the traumatic event. *For example, a woman found herself in a foetal position on the kitchen floor, again and again, and she wondered in psychotherapy what she was doing. She was unable to consciously link her behavioral symptom with her experience during the hold-up although she had curled up in a foetal position underneath a desk to avoid facing the screaming robbers bearing machine guns.*

The reexperiencing of traumatic memories can be perceptual, physiological, emotional, representational, cognitive, behavioral, physiological, and neurobiological. Within the perceptual dimension, flashbacks can be experienced through all sensory modalities. Many individuals see the traumatic event reoccurring in their minds' eye as if it were a video being superimposed over their visual field. During flashbacks, some may re-hear the same devaluating words thrown at them with forceful disgust, while others feel the cold metal of the gun barrel against their skin, and others keep on tasting semen in their mouth. Other have powerful somatic reactions.

For example, a young man was assaulted from behind with a crowbar. Being very muscular as an athlete, his body inflated his muscles to protect his neck and back where he was being hit again and again. The aggressors left being killing him, for a unknown reason. When he woke up in the hospital, five men were trying to contain his violence. Now he could fight. Realizing what he was doing and where he was, he regained control over his fighting. Beyond an extremely PTSD, suicidal thoughts, major depression, panic disorder and agoraphobia, he would faint about once a week and his body would shake as if he were having epileptic convulsions. Medical assessments detected no epilepsy. After several months of psychotherapy, he was able to face his vulnerability and anger. He reported about the continuation of these episodes and his worries about them because the last one occurred in a staircase. Upon examination, it became clear that his body also inflated during the pseudo-epileptic episode, as it did during the severe assault. Further examination of the antecedents of these convulsions revealed that they were preceded by being unfairly accused by a loved one, his mother or his girlfriend. His anger was such that it brought him back to the traumatic event when he was homicidally angry. Recognition of his anger and anger management issues were addressed in

psychotherapy. The pseudo-epileptic convulsions never came back.

Reexperiencing responses are often triggered by specific trauma-related cues, as predicted by classical conditioning. If a person is robbed at gunpoint by a man with a baseball cap on his head, with the visor turned toward the back, the person will react with anxiety whenever a man with such a positioned hat is approaching the person. News about wars will triggers veterans with PTSD into anxiety, anger, and/or sadness. This phenomenon is hallmark of PTSD.

Reexperiencing responses can also be free-floating, impending themselves beyond the person's awareness and throughout daily consciousness. They are ongoingly present, but they are not consciously associated with any trauma-related cues. *For example, an older woman without hearing difficulties was at the bank when a hold-up occurred, when four gunmen had rifles and one of them screamed: "This is a hold-up. All of you on the floor!" She saw the bank teller who was serving her become terrified, through her facial expressions, and she turned around to verify what was happening. Everyone behind her was lying face down on the floor, while some men were holding rifles. She thought to herself that it was a joke. It took one robber to scream at her few times for her to finally realize the seriousness of the situation and to lie down with the other costumers. The first day I met her, she arrived in my waiting room with her pants having been hosed down from the knees. I inquired about what had happened, and, as construction workers outside my office were redoing the sidewalk, she had just stepped into the freshly poured concrete. Interviewing her to assess the symptoms she presented, it became clear that, since the hold-up, she became severely confused every time she stepped outside of her apartment, although she had lived in it for 40 years. She had not made the conscious connection between her confusion during the hold-up and her disorientation when she walked outside.*

Other times, PTSD symptoms appear to be stemming "out of the blue" rather than related to a traumatic event. *For example, a patient of mine developed severe swelling of her hands and arms, paralyzing her movements to the point where she was unable to dress herself or to go to restroom by herself. She was a bus driver who had found a young teenager stabbed to death for no apparent reason. This somatic symptom could have reflected her disavowed helplessness or it could have been an unconscious strategy to prevent her return to work, or both. Such manifestations are reflective of PTSD as a structural problem.*

Reexperiencing and avoidance symptoms of PTSD can be stimulus-specific or generalized, like the traumatized dogs of Seligman who became intolerant to previously tolerated stressors. Many PTSD patients I have treated presented with agoraphobia and a panic disorder as co-morbid conditions, not due to fear of having panic attacks outside their homes, but because they perceived danger everywhere outside their homes. From a behavioral perspective, panic disorder and agoraphobia could be manifestations of a generalization of the threat experienced during the traumatic event. From a dynamic approach, they could be due to a projection of the unbearable anger they harbour unconsciously toward the perpetrator (the desire to badly hurt or kill someone is projected onto strangers because such terrifying inner

reality is incompatible with conscious and acceptable representations of self).

PTSD symptoms are usually experienced as ego-dystonic, that is, the person does not recognize the symptoms as being part of themselves, as reflective who they are. Patients with PTSD do not recognize themselves. Even with severe PTSD with co-morbid disorders, patients do not overtly recognize their overwhelming distress despite all the symptoms they have. As a clinician, it is always impressive to face individuals, presenting with all PTSD symptoms and severe co-morbid disorders, reject all empathic reflections of their obvious distress. Such individuals say that they are doing just fine, the problem is just symptomatic, and all of 'this' is just a hurdle. They simply want to get back to the way they were before the 'incident', as they usually point out. Indeed, individuals with PTSD tend to minimize the seriousness of the impact of traumatic events as well as the severity of their distress. Avoidance is an inherent part of PTSD, not only behavioral or cognitive avoidance but avoidance in terms of denial of the deep psychological wound.

Beyond the confusing aspects of PTSD, the multiple responses involved in PTSD tend to exacerbate each other. Intrusive symptoms and anxiety reinforces a sense of confusion and helplessness, which fosters an avoidance of environmental problems. Personal and occupational stressors worsen and anxiety augments consequently. Higher anxiety further reinforces helplessness. Etc., etc., etc.

A Vicious Cycle

PTSD is embedded with a vicious cycle. When PTSD is set in motion, symptoms themselves repeatedly invalidate one's sense of self as competent. Chronic neurobiological activation is set in place and it repeatedly attacks cortical and limbic structures, further decreasing any affect-modulation capacities. A greater sense of helplessness is engendered, further interfering with one's functioning. Over time, one's sense of self-efficacy diminishes through repeated experiences of lack of control and overwhelming emotional states. PTSD sufferers feel more and more helpless, less and less competent. Others appear to be less and less caring. Some individuals with PTSD end up isolating themselves in an effort to avoid damaging conflicts with others, they withdraw from pleasurable activities, and they withdraw from life.

Over time, as difficulties accumulate, the helplessness and vulnerability experienced during the traumatic event increases, further interfering with one's functioning. PTSD sufferers feel more and more helpless and incompetent. Conversely, others appear less and less caring. Some individuals with PTSD isolate themselves in an effort to avoid additional damaging conflicts with others. They also withdraw from activities and pleasurable activities. They end up withdrawing from relationships and life. The interpersonal and social structures are deeply affected.

Resolutions, Adaptive and Maladaptive

When PTSD emerges, the traumatic experience has not been assimilated. PTSD involves

traumatic memories, which are affect-laden and intrusive. The traumatic memories remain active, pushing their way into consciousness. Is it just because a conditioned response has been set in place? The answer is obviously 'no' given that intrusive symptoms beyond a conditioned response are paramount to PTSD: flashbacks, nightmares, dissociative re-enactments, pseudo-illusions and pseudo-hallucinations. Nowadays, most theoreticians and clinicians agree that PTSD involves a lack of assimilation of the traumatic material. Consequently, resolving PTSD requires accommodation of the pre-existing structure. Accommodation allows for assimilation to occur spontaneously and integration to between opposite schemas to happen.

Horowitz (1976, 1984, 2001), the father of our contemporary understanding of PTSD, provides the most simple and yet sophisticated answers. In our own understanding, we have seen clinically that, in PTSD: (1) the traumatic information is too discrepant with pre-existing, provoking basic schemas to be overly challenged; (2) the traumatic experience has reactivated unresolved affect-laden conflict, highly contributing to the activation of the traumatic memories; (3) the affects/arousal elicited by the traumatic event are overwhelming, rendering the system less than optimally functional; (4) diverse mechanisms (non-conscious) and strategies (conscious) are set in place to push the traumatic memories outside of consciousness in order to protect the organism against unnecessary pain until the person can face consciously the traumatic experience; (5) these defensive or protective responses are only partially effective, unable to completely and constantly keep at bay the distressing traumatic memories; and (6), therefore, the traumatic memories represent themselves again and again to the person's consciousness under various modalities (more or less obvious) in order to be recognized and integrated. PTSD is a partly adaptive response to an overwhelming experience.

Adaptive resolutions of PTSD may be summarized as involving integration and/or habituation. Let's take a look at each of these processes in order to keep them in mind as we review empirical findings about PTSD and PTSD therapies. It is important to understand these change processes in order to consider whether they are involved in the PTSD therapies, especially trauma-focused ones.

Integration requires consciousness and attention. Integration is the optimal process of resolution of PTSD. Integration happens through either assimilation or accommodation followed by assimilation. Assimilation is spontaneous and non-conscious. In contrast, accommodation requires attention and conscious efforts by considering the rejected 'bad news' embedded in the traumatic event. For the person to succeed an accommodation process, the person needs to consciously experience and own (in one way or another) the pre-existing components of their inner world which have been disturbed by the traumatic event. Such inner realities may be an overly positive schema about this world, an overly negative yet previously suppressed internal representation of a parent, an unresolved conflict around anger, a disavowed sense of vulnerability, a core issue of abandonment depression, and/or a limited capacity to modulate affects/arousal.

Habituation is the process of resolution for arousal responses based on conditioning. It is spontaneous and neurobiological. Habituation is the resolution process for classical

conditioning. From clinical experience, it appears that fear responses upon exposure to trauma-related stimuli are the least difficulties of individuals struggling with post-traumatic symptomatology. Nonetheless, approach leading to habituation is at times required in a PTSD treatment. Deactivating a conditioned response requires approaching the conditioned stimulus until one has stopped reacting to it, with fear, anger, etc. (let's remember here that anger, fear, humiliation, etc., can all provoke anxiety). Habituation is the resolution process involved here. Habituation requires exposure, under conditions recognized as safe, until the elevated arousal triggered has diminished to the baseline level. Habituation occurs naturally and is orchestrated mostly by the organism rather than conscious efforts on the part of the person. Nonetheless, conscious attention to phenomena sustaining arousal (for example, dramatic thoughts, attention to the heart racing, etc.) can interfere with habituation. Conscious attention to realities which are potentially soothing (for example, recognizing fully that this is a traumatic memory and not the reexperience of the trauma, paying attention that it happened then and there, not here and now, etc.) may also be involved in habituation. Habituation is a process and, therefore, occurs gradually. For example, with a dog phobia, the heart rate of the person will first accelerate very quickly upon exposure to a calm dog, but it will diminish gradually, over time, if all conducive factors are in place. Another example involves a more mundane reality; when shopping in a fishery, we may first intensely smell the intense fish odour, but in minutes we forget about it and cannot even smell it anymore (our physical sense of smell has habituated). Here no conscious effort was needed. But, if we are prone to disgust and become overwhelmed by the stench, we may have to leave the fishery despite all of our best efforts to disregard it. Habituation may a basic process of change in some trauma-focused therapies, but PTSD is likely to be mild and more phobia-like.

Maladaptive resolutions can also be set in motion after an overwhelming traumatic experience. Although maladaptive, these resolutions prevent the emergence of PTSD, at least for a time. When the psyche cannot manage the overwhelming traumatic experience and memories, psychosis may well follow. When the system cannot bear the traumatic experience but has a dissociative tendency, dissociation may well completely occult trauma, pushing its remembering outside of consciousness, apparently resolving the traumatic dilemma or PTSD. Epstein (1991) identifies very well the many non-adaptive modes of resolution to traumatic events, including adopting fear or anger as a *modus operandi*, embracing the trauma as a way of life, or dissociating the traumatic experience. These maladaptive resolutions are set in place in the aftermath of a traumatic event to prevent its reexperience, but at a cost.

As caring clinicians, we need to remain mindful of the possibilities that our interventions can trigger adaptive and/or maladaptive resolutions of PTSD. Our clinical mistakes as well as the clinical mishaps of other therapists inform us that individuals with PTSD can be damaged. Worse, our apparent successes of inducing partial PTSD remission may not pass the test of time. After months or years, PTSD relapses. Unsuccessful therapies seem to be due to an adherence to a technical approach or a dogmatic model. Supportive therapies may not sufficiently address core traumatic issues, leaving PTSD at times to simply follow its natural course. Employing trauma-focused techniques leads practitioners to be more at risk at inducing iatrogenic effects.

Trauma-focused and supportive ingredients may bring about unwarranted changes in those we are trying to help. Let's take the example of debriefing, a very popular intervention offered in the aftermath of traumatic events. Although debriefing has been claimed to prevent the incidence of PTSD by Mitchell, one session of debriefing has been shown by meta-analyses to repeatedly not improve PTSD symptoms (Emmerick et al. 2002; Rose et al., 2001). More worrisome, debriefing was shown to produce negative effects (symptomatic, functional, and eve financial deteriorations) over time in participants 3 years after a traffic accident (Mayou et al., 2000). Debriefing contains a potent trauma-focused component which is applied right after the occurrence of traumatic experience.

Another ingredient of debriefing aims at normalizing post-traumatic responses. Such educational and supportive intervention can be ill-applied and become detrimental. McFarlane (1988) has shown that firefighters receiving debriefing, in comparison to others receiving no interventions, showed more delayed PTSD than the others. The normalization of post-traumatic symptoms had been too emphasized, and firefighters understood that any post-traumatic symptom was just normal and should be subdued. For months, traumatized firefighters used an intellectual defense against their traumatic wounds, subduing the response into a subclinical PTSD. After months, this defense became exhausted; post-traumatic cues triggered full-blown PTSD or PTSD symptoms simply forced their way into consciousness through a natural decay of the defense of intellectualizing.

Why a trauma-focused intervention such as debriefing is still used in a large scale fashion is baffling to seasoned PTSD psychotherapists, clinically and ethically (Mayou et al., 2000; Rose et al., 2001). Rose et al. recommended that debriefing should cease and that a more appropriate model should be concerned about 'screen and treat' PTSD. The popularity of debriefing is particularly worrisome given that meta-analytic findings indicate clearly a multidimensional deteriorative impact of debriefing on participants in one session. Debriefing contains an important trauma-focused ingredient. In this book, our hope is to circumvent the replication of such phenomenon with regard to trauma-focused techniques for treating PTSD. Despite researchers' claims, a large-scale utilization of trauma-focused techniques with most PTSD sufferers would certainly be unwarranted and potentially detrimental. Carefulness and caution are our allies in preventing iatrogenic effects.

'Remember to remember' is apparently not the solution, especially not in the aftermath of a traumatic event. Trauma-focused techniques may unintentionally overwhelm patients with limited capacities to modulate arousal and affects. They may trigger such overwhelming responses, affective and neurobiological, that they interfere with the possibility of any habituation or conscious work on the traumatic experience. Inversely, trauma-focused techniques may produce apparent and sudden remission of PTSD. From experience, these techniques seem to unintentionally trigger some patients into endorsing defensive mechanisms against traumatic pain, defenses such as dissociation, in an attempt of the psyche to shield itself from unbearable affects. As with other techniques, trauma-focused methods ones may induce neither integration nor habituation, but rather trigger severe destabilization.

PTSD as Structural Disorder

Before examining PTSD as a structural disorder, let's look at PTSD as a phenomenon beyond the fear response, but as a phenomenon involving anxiety at its core.

Beyond Fear Response

In the academic literature, PTSD is often seen as a fear response or phobia. According to Lang's theory of bio-informational theory, fear responses are neural pathways containing representations of the feared stimuli, fear, and basic meanings such as the pain associated with these stimuli and responses. Fear responses of anxiety-disordered individuals would include basic pathological meanings. To inhibit a fear response, two conditions are required: first, the fear response must be activated and, second, fear-antagonistic information must be provided to allow for a new memory, neural connections, to be formed. Exposure-based treatments such as PE aim at providing these two conditions: they elicit a fear response and provide an opportunity for corrective information within the context of a therapeutic relationship, which is hoped to lead to habituation (Foa & Meadows, 1997). This model, focusing on PTSD as a fear response, leaves many questions unanswered, however.

First, fear responses are never extinguished. Although their phenomenological expressions cannot be at times reactivated upon repeated exposure to conditioned stimuli in animals, neurobiological evidence shows that fear responses are never extinct. Consequently, clinical interventions such as prolonged exposure or systematic desensitization may inhibit the expression of fear responses in humans, but they do not eliminate the underlying neural basis (Metcalfe & Jacobs, 1996).

Second, viewing PTSD as a fear response implies that PTSD needs to be approached clinically as a phobia. Consequently, trauma-focused methods are recommended by some. Indeed, meta-analytic studies have shown that trauma-focused techniques such as prolonged exposure (PE) are efficacious in reducing PTSD symptoms (Bisson et al., 2007; Bradley et al., 2005; Powers et al., 2010). In average, only 50% half of selected participants show a remission of PTSD (Schotten…). When partial versus complete remission are considered, only 15% of completers obtained complete remission from PE (Schnurr et al., 2007). Dropout rates are high (Schotten…) and they are higher in trauma-focused therapy in comparison to others (Barrera et al., 2012). The majority of participants continue to have substantial residual symptoms (Bradley et al., 2005). In addition, in a meta-analysis found that the exposure component of cognitive-behavioral therapies added no efficacy to PTSD remission rate (Barrera et al., 2012). Finally, trauma-focused techniques such as PE were shown to be simply as efficacious as much gentler approaches such as anxiety management, traditional cognitive therapy (Powers et al., 2010) and dynamic therapy (Brom, Kleber, & Defares, 1989). Together these findings highlight two major points: (1) PTSD is not optimally treated by exposure techniques; and (2) PTSD should not be clinically approached as a phobia.

Third, studies examining the predictors of the efficacy of PE reported only one robust predictor: the initial severity of PTSD (Van Minnen et al., 2002). Such finding highlights the need to employ trauma-focused techniques for mild PTSD only, that is, a PTSD resembling a phobia. Whenever symptoms are numerous and severe, PTSD is first and foremost a structural disorder, involving all dimensions of a human being. This statement is especially true whenever one of the first three reexperiencing PTSD symptoms in the DSM is present: namely, flashbacks, nightmares, and pseudo-hallucinations/non-psychotic delusions/dissociative re-enactments.

Now let's consider a clinical example about the complexities potentially involved in PTSD. A complicated case presenting with severe PTSD and comorbid disorders (severe depression and conversion disorder) saw a complete resolution of all symptomatology after a comprehensive, integrative psychotherapy lasting 18 months in which PE was not employed. The traumatic event consisted of witnessing the stabbing to death of a passenger in a bus. The patient was a bus driver. Few years later, this patient decided to get a more relaxed job than driving a municipal bus and she was transferred to work as a subway conductor. This time, a suicidal man jumped over the rail and landed on the windshield of her cabin, breaking it and killing himself. This second traumatic event provoked a new PTSD, but without the resurgence of any reexperiencing symptoms associated with the stabbing and death of a teenager. At her work site, they insisted upon the use of PE over few sessions as practiced at the Employee Assistance Program. Her PTSD symptoms receded partially after 6 sessions and she returned to work. Years later, she experienced another traumatic event; she was caught in a serious car accident involving forty cars and she had physical wounds. Interestingly, beyond the flashbacks involving the car accident, the only other flashbacks she experienced were related to the suicide in the subway. No reexperiencing symptoms reminded her of the stabbing and killing. This case illustrates how the PTSD symptoms treated by PE were reactivated by new traumatic event and how the PTSD fully resorbed by an integrative psychotherapy remained in full remission. The integrative treatment allowed the full integration of the traumatic information by accommodating the psychological structure of the person presenting with severe PTSD, comorbid disorders and major functional limitations.

So let's remember that PTSD is not a phobia. This point is crucial because techniques for treating PTSD actually favored by the academic community involve reexperiencing a traumatic event as if PTSD was a phobia. Phobias include a feared object and a fear response, along with the avoidance of the feared object. Although it is reasonable to recognize that the phenomenon of classical conditioning is involved in PTSD, it operates only at the periphery of PTSD, unless PTSD is mild and defined by DSM-IV criteria (especially symptoms B.4, B.5, C.1, and C.3). Out of the 20 PTSD symptoms in DSM-5, only few symptoms reflect a manifestation of classical conditioning. Becoming psychologically and/or physiologically distressed upon exposure to trauma-related cues and its avoidance can reflect the presence of a conditioned response. However, flashbacks, nightmares, pseudo-hallucinations, negative cognitions, guilt, risky or self-destructive behaviors, etc. cannot be explained away by a fear response. PTSD is a phenomenon involving much more than a fear response. Nonetheless, PTSD does comprise anxiety features.

Anxiety in PTSD

Before DSM-5, PTSD was classified as an anxiety disorder in the DSM-IV (American Psychiatric Association, 2007), but such classification was challenged. Some wanted to define PTSD as a dissociative disorder, while others argued that PTSD should stand apart. Although the latter categorization was favoured in DSM-5, strong epidemiological findings support the association between PTSD and anxiety. Pre-existing anxiety disorders have been found to predispose individuals to developing PTSD after a traumatic event (Kessler et al., 1999). PTSD is also highly associated with anxiety disorders as comorbid complications (…). Viewing PTSD as anxiety-based is significant for defining treatment goals and its processes.

In general, seasoned practitioners consider the anxiety level associated with any given PTSD and intervene accordingly. Baseline anxiety can be addressed in a variety of ways, stemming from experiencing a benevolent relationship to practicing a relaxation technique, regaining control over stressors, solving problems, taking psychotropic medication, resolving intrapsychic conflicts, etc.

Many neurobiological, environmental, and psychological factors are involved in shaping an individual's baseline anxiety. An elevated baseline anxiety predisposes to PTSD. In turn, PTSD increases an individual's anxiety level and contributed to maintaining such high anxiety. All of these factors need to be considered in psychotherapy in order to alleviate PTSD symptoms and its complications.

Neurobiologically speaking, baseline anxiety is based on the arousal level of the amygdala, the center for survival drives, affects, anxiety, etc. (Metcalf & Jacobson, 1996; Nemeroff …). Baseline anxiety is in part genetically determined by temperament at birth and in part by environmental factors (Schwartz et al., 2003).

According to Chess & Thomas (1986) longitudinal studies, 20% of humans are born with an anxious temperament at varying degrees. When faced with a threatening situation, such infant quickly goes into high gear of amygdala arousal and fear takes hold of their higher mental functions. As infant, any novel toy or situation triggers crying. As adults, such individuals tend to react with anxiety to novel situations. For example, during a hold-up, some tellers run away screaming as soon as they realize that a hold-up is happening. Unfortunately, their behavior increases the likelihood of being hurt or killed, but such flight pattern seems to be outside of their control as they are then governed by fear. Although some temperaments are so severe that they can hardly be changed, most can be modified according to Chess & Thomas (1986). Consequently, the therapeutic focus may be on reducing baseline anxiety, through practicing relation or meditation techniques regularly, modifying anxiety-provoking cognitions and therapeutic soothing, for persons presenting with PTSD and an anxious temperament, and possibly with pre-existing anxiety disorders.

Baseline anxiety is also environmentally fashioned. Early experiences of helplessness, rather than control over one's situation, can predispose individuals to develop a high anxiety baseline, anxiety disorders, and reduced performance at diverse tasks (Chorpita & Balow, 1998). Such heightened arousal predisposes, in turn, to developing PTSD. Having experienced

multiple traumatic events also increases substantially the risk of developing heightened anxiety and PTSD (Breslau et al., 1998). While many factors trigger higher levels of arousal, one major protective factor of PTSD consists of the perceived presence of adequate social support (Kaniasty, 2005). Inversely, the lack of adequate social support thus heightens PTSD.

In psychotherapy for PTSD, practitioners need to consider the person's baseline anxiety, to determine whether the arousal is trauma-related or temperament-based and to intervene accordingly, to assist the person in regaining control over basic life circumstances and challenging situations, to encourage the person to solve ongoing problems, and to promote reaching out for adequate social and affective support.

PTSD as a Heterogeneous Phenomenon

PTSD is not a simple mental disorder and it carries many other mental disorders along with it. In samples of veterans with PTSD, comorbidity was as high as 99% (Weathers, Litz, & Keane, 1995). PTSD is often found to be comorbid with major depressive disorder, substance abuse or dependence, panic disorder, agoraphobia, obsessive–compulsive disorder, generalized anxiety disorder, social phobia, specific phobias, and bipolar disorder (APA,2000; Hoge et al., 2004).

Many factors converge to predispose, to precipitate, and to maintain PTSD and its co-morbidity. Features of the traumatic event, the environmental responses are involved in determining PTSD. The initial severity of PTSD and its course over time also define any given PTSD.

Features of trauma events account for a small proportion of the variance in PTSD (Yehuda & McFarlane, 1995). For example, while PTSD is present in 47% of rape victims three months later (Rothbaum et al., 1992), PTSD affects approximately 25% of the population (Breslau et al., 1991). The intensity of the violence embedded within traumatic events is thus predictive of PTSD severity (Breslau et al., 1992; Jones et al., 2001), but more factors contribute to PTSD's etiology and course.

Post-traumatic environmental conditions have an impact of PTSD symptoms. Survivors of the WWII holocaust placed in foster homes later presented a lower quality of life and more psychiatric symptoms than others (Lev-Weisel & Amir, 2000). A history of family instability is also associated with increased prevalence of PTSD (King et al., 1996), whereas social support is associated with lower levels of PTSD symptoms (Solomon et al., 1988).

PTSD changes over time. Some studies report a decrease in PTSD symptoms over time as in rape victims (Rothbaum et al., 1992), while others note an increase (Southwick et al., 1995). PTSD can also be delayed (Koren et al., 1999), suggesting that PTSD can be suppressed for a while and later triggered into manifestation. Neurobiological sensitization and kindling effect can together account for delayed PTSD (McFarlane, 2010). PTSD can be long-lasting and even permanent as in WWII holocaust survivors 55 years later who were still experiencing psychological distress, including PTSD symptoms, depression, anxiety, somatization, and anger

(Amir & Lev-Weisel, 2003).

The severity of PTSD influences its own course. In a longitudinal study comprising a community sample, Perkonigg et al. (2005) observed that 52% of PTSD remitted during a 4-year period, while 48% showed no remission. The initial severity of PTSD predicted outcome; non-remitters had initially more severe PTSD than remitters.

In a detailed study of a very large sample of the Gulf War veterans, Orcutt et al. (2004) investigated prospectively the course of PTSD symptoms over 7 years. A two-group model emerged, suggesting two distinct courses of PTSD. Those with initially mild PTSD showed little change over time, comprising 57% of the sample, while those with severe PTSD symptoms, comprising 43% of sample, showed a significant deterioration of PTSD over time. As it will be shown below, such findings are reflected in neurological studies of PTSD.

The initial severity of PTSD might well be the marker for its perseverance and deterioration over time, as it is the case for most mental disorders. There might be two basic trajectories in PTSD: (1) initially mild PTSD with no comorbidity allows for spontaneous remission or status quo; (2) initially severe PTSD, especially with comorbidity, becomes deteriorates over time. At this point, the question of the aetiology of PTSD severity needs to be addressed.

What factors contribute to the initial severity of PTSD? One hypothesis would be that mild PTSD, as diagnosed by DSM-III or DSM-IV, is fear-based and reflects mostly a phenomenon of classical conditioning. Another hypothesis would be that mild PTSD reflects the presence of well-developed capacities for modulating intense affects and the presence of well-differentiated and well-balanced schemas of self, others, and life. Moderate to severe PTSD is likely to reflect the presence of structural deficits and vulnerabilities: limited capacities for affect-modulation and core unresolved conflicts. Such structural deficits and vulnerabilities predispose to developing PTSD, beyond the features of the traumatic event, although they interact with them. This latter is particularly sound if PTSD is associated with comorbid disorders, functional limitations, and a personality disorder.

Structures Involved in PTSD

PTSD is thus a multidimensional phenomenon, involving the person, the environment, and their interactions. As PTSD involves all aspects of a person, it can be regarded as a structural problem. This is particularly true for the PTSD diagnosis in DSM-5 (American Psychiatric Association, 2013, in which more pervasive symptoms were added. As a structural disorder, PTSD stems from a breaking down of core issues in a person, not peripheral ones.

A successful treatment is likely to be comprehensive and integrative. Structural deficits predispose to PTSD and contribute to PTSD's deterioration and maintenance over years. Over time, PTSD itself creates structural damages reflected in long-standing neurological damages (Freeman et al., 1998; Pederson et al., 2004; Schuff et al., 1997; Schuff et al.,2001),

psychological complications (Breslau et al., 1992; Breslau et al., 1998), interpersonal disruptions (…), occupational dysfunctions (…), and chronic physical consequences (McFarlane, 2010). Structural damages can be such that the International Classifications of Mental and Behavioral Disorders (ICD-10; World Health Organization, 1993) recognizes a diagnosis of 'enduring personality change after catastrophic experience' (pp. 129-131). Structural deficits predisposing to PTSD are neurobiological and psychological.

Neurobiological Structures

Many neurobiological sites are involved in PTSD. The amygdala is responsible for the arousal of an individual, from alertness to anxiety and panic. The amygdala stimulates the release of glucocorticoids, the stress hormones. The threshold of activation of the amygdala varies from individual to individual; some will face intense stressors before becoming aroused to a level of experiencing anxiety, while others will become anxious readily in response to minute stimulation such as a child playing loudly. In parallel, some brain sites modulate stress and thus the activation of the amygdala and the subsequent arousal of the organism. The two main sites are the hippocampus (the 'cool cat' of the limbic system which is involves in putting things in perspective, inducing habituation, and encoding non-emotional elements of memories) and the prefrontal cortex, particularly the cingulate anterior involved in modulating affects and affect-based behaviors and in perceiving events through basic schemas. In return, glucocorticoids regulate the neurons of the hippocampus and the prefrontal cortex.

Individuals with long-lasting PTSD present usually structural abnormalities and functional dysregulations of the amygdala, the hippocampus, and the prefrontal cortex. Inversely, these structural abnormalities and functional dysregulations are likely to predispose individuals to PTSD. Such effects are observed in the amygdala, the hippocampus, and the prefrontal cortex (Sherin & Nemeroff., 2011).

The amygdala has been found to present an increased baseline activity and increased reactivity to trauma-related cues, which promotes hypervigilance and impairs discrimination of environmental threats. Prolonged exposure to stress leads to high levels of glucocorticoids in laboratory animal, which in turn leads to damages in the hippocampus. In humans, sustained glucocorticoid exposure, such as in repeated traumatization and/or long-lasting PTSD, has been shown to have adversely impact the hippocampus, including a reduction in volume. Because a reduced hippocampal volume has been repeatedly found in individuals with chronic PTSD, it is now viewed as a hallmark feature of PTSD. Sites of the prefrontal cortex have also shown a reduced volume in individuals with PTSD. Such volume reduction dysregulates executive functions, especially in the anterior cingulate. A reduced volume of the cingulate anterior impairs the deactivation of fear responses and reduces one's capacity of affect-modulation (Sherin & Nemeroff, 2011). Basic schemas are thought be encoded in the amygdala, hippocampus, and the prefrontal cingulate anterior. As schemas are mostly based on personal experiences, neurobiology brings us to consider their manifestations at a psychological level.

These neurobiological structures will be examined at length in this book. Empirical findings will inform us that PTSD presents with and/or induces neurobiological vulnerabilities and limitations. In turn, these vulnerabilities and limitations will inform us to mostly use gentle methods for treating PTSD rather than trauma-focused ones.

Psychological Structures

Personality Features. In American community samples, 6% to 10% of individuals have been identified as presenting a personality disorder as defined by the DSM (Samuels, 2011).. The prevalence of personality disorders varies across socio-demographic groups. Personality disorders are strongly associated with clinical disorders in the community, including PTSD, mood disorders, anxiety disorders, and substance use disorders, as well as with functional impairments (Samuels, 2011).

A history of adverse experiences, including childhood abuse, has been found to be related to borderline, self–defeating, narcissistic, histrionic, sadistic, and schizotypal traits (Norden, Klein, Donaldson, Pepper, & Klein, 1995). Physical abuse (but not sexual abuse) has been linked to antisocial personality traits (Norden, et al., 1995).

In a longitudinal study of borderline personality disorder and PTSD in veterans (Axelrod, Morgan, & Southwick, 2005), which showed that pre-deployment BPD symptoms were associated with PTSD symptoms at 6 months post deployment, and that self-reported trauma exposure and PTSD symptoms were associated with post-deployment BPD symptoms. The authors 'concluded that traumatic experiences and PTSD in adulthood can lead to BPD.

Many patients with PTSD come in psychotherapy presenting with a personality disorder (Samuels, 2011). In a sample of cocaine dependent individuals seeking therapy (n = 91), 43% had lifetime PTSD and higher rates of co-morbid disorders and personality disorders (Back et al., 2000). As many as 80% of PTSD patients in a special treatment unit also present with a personality disorder (McFarland, 1985). Another study found that 76% of treatment-seeking patients with combat-related PTSD met criteria for borderline personality disorder (Southwick, Yehuda, & Giller, 1993).

In a large sample of firefighters exposed to a bushfire disaster (n = 469), McFarlane (1989) found that neuroticism and a past history of treatment for a psychological disorder were better predictors of PTSD severity at termination than the degree of trauma exposure or the number of losses sustained. The findings highlight how patient's structural characteristics predispose to developing PTSD and comorbidity.

Attachment Patterns. Personality structures can also be defined along the lines of attachment theory. Infants with secure attachment represent 55% of the general population, leaving 45% of the population with an insecure attachment disorder. Insecure-avoidant attachment occurs in 23% of the general population while infants with insecure-resistant (ambivalent) attachment consist of 8% of the general population. An NIH report indicates that

the remaining 15% suffer from an insecure insecure-disorganized attachment (Benoit, 2004).

Attachment patterns develop very early in infancy and are defined by the parents' attachment patterns and the interaction of parents with the infant (Bowlby, 1988). Prospective studies have shown that parental attachment styles before birth of a child predict later infant attachment (Fonagy, Steele, & Steele,1991; Steele, Steele, & Fonagy, 1996). A prospective study of attachment style and trauma in adults that showed that pre-existing attachment style affects the way in which people cope with trauma (Mikulincer & Florian, 1995). The infant experiences how others can be trusted.

Secure attachment appears to mitigate the adverse impact of abuse on later psychological functioning (Roche, Runtz, & Hunter, 1999). Individuals with secure attachment tend to have fewer PTSD symptoms (Mikulincer, Florian, & Weller, 1993; Mikulincer, Horesh, Eilati, & Kotler, 1999). Secure attachment is related to fewer PTSD symptoms, past and present, and better adjustment (Solomon, Ginzburg, Mikulincer,Neria, & Ohry, 1998; Zakin, Solomon, & Neria, 2003).

Insecure attachment patterns were found to predispose individuals to PTSD and other mental disorders in a large sample of trauma victims (n = 1577) (Armour et al., 2011). Attachment styles were classified as fearful (18.6%), preoccupied (34.5%), and secure (46.9%). Following a traffic accident involving a whiplash, individuals presenting with a secure attachment pattern showed the lowest scores on PTSD, depression, and anxiety disorders. Subjects with a fearful attachment pattern obtained the highest PTSD scores (Armour et al., 2011). Insecure attachment patterns (anxious and avoidant) were related to more short- and long-term distress (Solomon et al., 1998). Insecure attachment was the strongest predictor of the severity of all PTSD symptoms (reexperiencing, avoidance, and hypervigilance) (Dieperink, Leskela, Thuras, & Engdahl, 2001).

Traumatic events can predispose to developing or worsening attachment patterns. Epidemiological study showed that 58% of middle class U.S. adults suffered at least two types of traumas as children, including physical and sexual abuse, physical and emotional neglect in newborn, and eight other types of traumas. Such traumas put children into a chronic state which is biologically proven to shut down the organism's capacity for feelings of attachment (Benoit, 2004).

In psychotherapy for PTSD, the personality structure and attachment patterns of individuals with PTSD thus have to be considered in order to intervene appropriately and effectively in psychotherapy. The therapeutic relationship, or alliance, is particularly important in treating people with PTSD and a personality/attachment disorder. Specialized models for treating PTSD do not suffice because they ignore personality issues and attachment needs/fears. While PTSD needs to be considered in its own right, its consideration needs to be counterbalanced by therapeutic acknowledgement and work on personality and attachment issues. Indeed, given that (1) one person out of ten has a personality disorder, (2) one person out of four presents with an insecure attachment pattern, and (3) these two are factors

predispose to PTSD, practitioners specializing in PTSD need to also be competently trained in diagnosing and treating personality/attachment disorders.

Psychological Defenses. Defenses are psychological means of dealing with wishes, needs, affects, or impulses that the individual experiences as unpleasant or inacceptable (Perry & Cooper, 1997). Psychodynamic theory posits that defenses evolve in the course of an individual's psychological development (Vaillant, 1992). Some defenses have been shown to be more adaptive than others (Offer, Lavic, Gothelf, & Apter, 2000).

Speanburg and colleagues (2003) who found that improved global functioning was associated significantly with increased the operation of mature defenses and decreased the working of immature defenses over a 3 to 7 year follow–up. The sample in this study included 48 participants with treatment-resistant disorders, including 34% with PTSD, who received residential psychodynamic psychotherapy and some follow–up psychotherapy for varying lengths of time. The onset of more mature defenses is related to the conscious appraisal of the use of less mature defenses, as suggested by psychodynamic models.

Defense mechanisms are particularly at play in PTSD given the intense emotional surge happening during the traumatic event and in association with the subsequent destabilization. Traumatic events can disturb the homeostasis of human beings and reduce their ability to use mature defenses (Punamaki, Kanninen, Qouta, & El-Sarraj, 2002; Vaillant, 1971). After torture, the more severe PTSD is, the more immature were the psychological defenses are employed (Punamaki, 2002).

Indeed, many patients presenting with PTSD also present with an already-existent limited capacity for modulating affects, a feature of personality/attachment disorders. Psychological defenses are preconscious operations aimed at warding off unacceptable experiences (wishes, emotions, and impulses). Individuals with PTSD can hardly experience intense dysphoric affects without becoming destabilized, overtly (as with a borderline disorder or an insecure ambivalent attachment disorder) or covertly (as with a schizoid disorder or an insecure resistant attachment disorder). Such individuals easily reach a heightened level arousal and a confused sense of self. Planning for adequate behavior thus becomes difficult.

Internal representations of self and others. Beyond neurological deficits and less mature defenses toward successful affect-modulation, it is postulated that the psyche of the personality-disordered individuals is often composed of powerful malevolent internal representations and weak positive internal representations, of both self and others (Horowitz, 1984; McCann & Pearlman, 1990). Let's see if empirical findings support these theoretical assertions based on clinical observations.

Findings on the association between attachment patterns and PTSD severity highlight that insecure attachment predisposes to developing PTSD after a traumatic event. Insecure attachment patterns have been shown to be established at one year old and to persist throughout one's life (Bowlby, 1988; Grossman, Grossman, & Waters, 2005). Insecure

attachment patterns derive from experiences with parents in early years and, therefore, they are deep-seated and not readily accessible. With infants presenting with insecure attachments, parents behave in a laboratory setting in malevolent ways toward the infant (abandoning, rejecting, hostile, etc.) rather than benevolent ways, or a mixture of both. For attachment patterns to persist over a lifetime, core internal representations of self as unworthy, unlovable, burdensome, etc. have been installed, along with internal representations of others as abandoning, rejecting, hostile, etc.

In cognitive-behavioral therapy, schemas or beliefs about oneself and others are considered. Many contend that negative schemas about self and others are involved in PTSD. Regarding the association between schemas and PTSD, the empirical evidence is unclear.

Dunmore, Clark, and Ehlers (2001) also found that negative beliefs about self, along with negative views of work, were the highest correlated predictors of developing PTSD. Gaston, Brunet, Kosycki, and Bradwejn (1998) found that MMPI scores of respondents with acute PTSD reflected a positive view of self, others, and the world, while MMPI scores of respondents with chronic PTSD reflected a negative view of self, others, and the world. However, Muller, Sicoli, and Lemieux (2000) found that a negative view of oneself was the predictor of PTSD symptomatology, but they also found a negative view of others to not be associated with PTSD.

In a large study with American journalists (*N* = 906) participated in a study on the mediational of schemas in PTSD (Pyevich, Newman, & Daleiden, 2003). Results indicated that greater exposure to traumatic events was associated with PTSD symptoms and negative cognitive schemas. However, cognitive schemas only partially accounted for PTSD symptoms, rendering the full cognitive mediational model unsupported.

PTSD patients sometimes present with negative schemas of self and others, but they sometimes endorse positive views. Experienced clinicians might notice that positive schemas are often psychological defenses employed by patients with PTSD, rightfully so in order to function, a defense against acknowledging how much they feel insecure in the world. Whether patients present with negative schemas which are readily acknowledged or deep-seated internal representations which are often operative beyond conscious, their disturbance and emphasis by traumatic events reflects how PTSD comprises structural damages.

In summary, PTSD develops more often in individuals presenting with high baseline anxiety, limited capacities for modulating affects, personality disorders, insecure attachment patterns, immature psychological defenses, and negative representations of self, others and the world. These features reflect structural deficits. In clinical practice, observant psychotherapists can notice that these pre-existing structural deficits have been amplified by traumatic events. With PTSD, greater stress is put upon preexisting psychological structures, and individuals revert to less mature defenses to contain affects and impulses. Individuals with preexisting structural deficits are more likely to develop PTSD and a more severe PTSD, and they are likely to have severe comorbidity and functional limitations. In psychotherapy, all of these contributing factors need to be addressed to resolve PTSD, for PTSD to enter a fullest

remission possible. Constricting psychotherapy to trauma-focused techniques is thus at best limited, and potentially detrimental.

Nonetheless, academic authors emphasize the easiness of treating PTSD with trauma-focused techniques, without the need to considering patients' vulnerabilities. They also emphasize the almost absence of severe iatrogenic effects following trauma-focused techniques. Such assertions are worrisome because the internal and external validity of controlled clinical trials are questionable, especially regarding to the generalizability of findings to the general population of PTSD sufferers. Enthusiastic conclusions about trauma-focused techniques are at times even at variance with the data reported in articles. Let's look at the neurobiological evidence supporting the need to use trauma-focused techniques in a cautious and gentle fashion for treating PTSD.

Neurophysiological Underpinnings of PTSD

In this chapter, neurophysiological hypotheses about PTSD will be reviewed. It will be outlined how individuals can be neurophysiologically predisposed to developing PTSD and how PTSD itself can create serious neurophysiological consequences. For practitioners, knowing and understanding the neurophysiological effects of PTSD can allow us to patient and sensitive toward PTSD sufferers, careful and supportive toward their suffering. Behavioral models instruct us about the contribution of classical and operant conditionings. Information theory introduces cognitive and dynamic models of PTSD, guiding us toward deep pathogenic beliefs, unresolved conflicts, and interfering defense mechanisms. Mostly, neurological research related to attachment theory informs us of the need to provide an attuned relationship to PTSD sufferers, to develop an alliance by offering an empathic attitude and anxiety-reducing interventions, an alliance from which challenging interventions can later bear fruits. Throughout this chapter, an emphasis is placed onto how each of these major psychotherapeutic models does contribute a unique and valuable piece of the puzzle of PTSD.

In 1941, Kardiner noted that PTSD sufferers continue to live in the emotional environment of the traumatic events they have experienced. This unconscious "emotional environment" or "arousal" appears to be at least partially based on neurobiological processes. Traumatic memories are acutely conscious when individual with PTSD endure re-experiencing symptoms, but trauma-related states are usually subconscious and neurologically manifested. Neurological and psychophysiological findings provide us with incredible information to be truly empathic toward the silent suffering of individuals with PTSD.

Our actual knowledge of the neurobiological processes involved in PTSD allows us to derive clinical implications from these empirical findings. Such clinical implications are not in line with the actual proposed treatments for PTSD by academia and others, which tend to be trauma-focused. The clinical implications of neurobiological findings orient us toward caution and the need to work progressively in psychotherapy with PTSD, in respect of their neurobiological and psychological capacities and limitations.

This is not to say that PTSD can be reduced to a neurobiological phenomenon. This is also not to say that neurobiological-oriented interventions, such as psychotropic medication, are sufficient or even necessary to successfully treat PTSD, although they can be helpful.

Neurobiological findings about PTSD afford practitioners guidance toward a better understanding of the complexities presented by any given patient with PTSD, beyond appearances and subjective reports as PTSD patient can heavily use denial. For example, neurobiological findings can help practitioners to understand and to recognize that some PTSD patients cannot tolerate the anxiety provoked by a premature interpretation about one's vulnerability or the overwhelming arousal created by a trauma-focused method. Psychotherapists can consequently direct their therapeutic interventions toward a more anxiety-reducing and supportive stance. Neurobiological findings can also guide practitioners in

elaborating treatment plans in line with patients' capacities to modulate arousal and affects.

In the field of PTSD, there are at least two main hypotheses regarding the neurobiology of PTSD. The first hypothesis views PTSD as an arousal disorder, while the second considers PTSD as a pathological manifestation of the inability to modulate arousal (Everly, 1993; McFarlane and Yehuda, 1996; van der Kolk & Saporta, 1993). Each hypothesis has been amply supported empirically. Viewing these two hypotheses as complementary is instrumental in fully comprehending and treating PTSD.

As a rule of thumb, when two competing empirically-supported hypotheses are presented in the empirical literature, it is fruitful to view them as complementary and to integrate them into a larger theoretical model.

PTSD as Arousal Disorder

Originally, PTSD was viewed as an arousal disorder. Individuals with PTSD are indeed more anxious than others and are sometimes too anxious to enjoy life and to function properly (Everly, 1993; McFarlane and Yehuda, 1996; van der Kolk & Saporta, 1993). Arousal, activation, and anxiety are terms which are used to represent the same phenomenon, although anxiety is mostly employed to refer to the conscious experience of a high arousal level.

In clinical practice, it is easy for practitioners to notice that individuals with PTSD are more anxious than others. These patients are ongoingly anxious and react with high anxiety to trauma-related cues, but their reactions are not always apparent to practitioners. Individuals with PTSD, especially recent PTSD, often cover their anxiety and distress by using defense mechanisms such as denial, minimization, isolation, etc. It is thus important to ascertain how individuals with PTSD experience anxiety, beyond their spontaneous self-reported anxiety.

Psychophysiological Arousal

Arousal/anxiety can be assessed by different markers. In PTSD studies, psychophysiological activation and reactivity is often evaluated using heart rate, skin conductance, blood pressure, EEG, etc. However, heart rate is now considered the psychophysiological marker of PTSD (e.g. Blanchard et al., 1982).

The notion that PTSD is an arousal disorder level is supported by many psychophysiological studies. Higher baselines of arousal are repeatedly observed in PTSD subjects in comparison to controls. Findings generally indicate that trauma-exposed individuals present an elevated baseline arousal, but individuals with PTSD display even higher levels of psychophysiological activity, namely an accelerated heart rate. Let's look first at subjective arousal or anxiety.

Self-reports of anxiety indicate that PTSD subjects tend to subjectively experience more anxiety than others. For example, Blanchard et al. (1982) administered the State-Trait Anxiety

Inventory and found that veterans with PTSD scored significantly higher than controls. In civilians, Gaston et al. (1996) compared MMPI scores of individuals with acute or chronic PTSD to individuals with a panic disorder. Higher elevations on five of the ten MMPI clinical scales (including the scale reflective of anxiety) were associated with chronic PTSD only, and not acute PTSD and panic disorder. As indicated by epidemiological findings, PTSD symptoms, including anxiety, worsens over time. Even when compared to psychiatric inpatients and outpatients, individuals with PTSD report higher anxiety as Keane et al. (1984) observed that veterans with PTSD produced more elevated scores on all MMPI scales, except for Mf, than veterans with other psychiatric disorders.

Heart rate is an excellent indicator of psychophysiological responses to traumatic events. Davidson & Baum (1986) reported elevations in basal heart rate in residents living within 5 miles of the Three Miles Island nuclear power plant after the disaster, whether they had PTSD or not. Individuals with PTSD, however, consistently show higher basal heart rates than healthy controls (Blanchard et al., 1991). In a study of levels of 24-hour ambulatory heart rate, Buckley et al. (2004) found that veterans with chronic PTSD had higher resting heart rate than veterans without PTSD, and the former also had higher blood pressure in association with dysphoric affects. Therefore, both exposure to a traumatic event and PTSD contribute to elevate heart rate.

Following exposure to a traumatic event, it is of interest to compare anxiety levels of individuals with and without PTSD. A meta-analysis (Buckley & Kaloupek, 2001) indicated that PTSD is associated with elevated basal heart rate and diastolic blood pressure in comparison to trauma-exposed subjects without PTSD. Buckley et al. (2004) compared very chronic PTSD subjects (12 and more years) with less chronic PTSD (8 and less years), and they found that more chronic PTSD subjects had larger variations in their basal heart rate than less chronic PTSD subjects. PTSD chronicity thus impacts basal heart rate, along with heart rate reactivity. In short, when PTSD does not resorb, it becomes worse over time and impacts the body negatively. Elevated cardiovascular activity may be, unfortunately, one of the negative consequences of chronic PTSD.

When physiological reactivity to trauma-related cues in PTSD is examined, researchers find a marked elevation of heart rate during a trauma-related stimulation, just as in trauma-focused techniques. Following a combat-related stimulation, combat veterans with PTSD showed higher heart rate in comparison to combat veterans without PTSD and combat veterans with an anxiety disorder (Shalev & Rogel-Fuchs, 1993). In and of itself, PTSD thus contributes to marked elevations in psychophysiological reactivity upon exposure to trauma-related stimuli. Furthermore, individuals with PTSD demonstrate a failure at discriminating stimuli of differing relevance, reacting to more or less innocuous stimuli (images of fearful or angry faces) as if they were threatening (Dickie et al., 2011; McFarlane et al., 1993). Such tendency to overinterpret environmental cues as threats renders PTSD sufferers susceptible to ongoingly react to ‘simple’ stressful stimulations.

Landmark Study of Blanchard and colleagues

A landmark study was conducted by Blanchard et al. (1982). This study illustrates the psychophysiological reality of individuals with chronic and severe PTSD. These researchers compared the psychophysiological arousal of Vietnam combat veterans with severe and chronic PTSD with age-matched non-veteran controls, under diverse conditions of stimulation (listening to calm music, doing mental arithmetic, and being exposed to the ambush scene of the war movie Platoon taking place in Vietnam). Various psychophysiological indicators of arousal were measured, including heart rate. Four main findings emerged from this sophisticated study.

First, baseline heart rate varied across the groups. Veterans with PTSD showed higher resting heart rates than controls.

Second, during the presentation of the ambush scene, all groups presented an elevated heart rate, but veterans with PTSD presented an higher heart rate elevation than controls, with an average increased heart rate of 11 bpm, beyond the elevated heart rate of controls. Researchers then used heart rate elevations as a discriminating variable between groups, and they correctly classified 91 % of veterans with PTSD and 100% of the controls.

Third, during the presentation of the highly violent ambush scene of the movie Platoon, only the non-PTSD subjects reacted with the usual phasic psychophysiological response, a reaction observed whenever a stressful stimulus is presented to subjects in experiments. Upon stimulation, the heart rate of the non-PTSD subjects quickly increased, to reach a maximal but moderate level of arousal, before gradually subsiding to its original baseline level in a timely fashion. In contrast, in veterans with PTSD, their heart rate quickly increased at a very high level, remained at a very high level for an unusually long period, and decreased in an unusually slow fashion. This observation points to the limited capacity of individuals with severe PTSD to modulate arousal.

Fourth, Blanchard et al. (1982) also examined the numbing response, a phenomenon recognized as a PTSD symptom at a psychological level. After the trauma-related stimulation, the researchers tested the numbing reactions of subjects by using needle pricking and measuring endorphin levels. After exposure to the ambush scene of the war movie Platoon, PTSD subjects were not physically sensitive to the pain induced by needle pricking, while controls were still physically sensitive. This observed physical numbing appeared to be consequent to a spontaneous neurobiological anaesthesia. Only in PTSD subjects, endorphins levels in blood had increased significantly during the viewing of the ambush scene. In subjects with serve and chronic PTSD, endorphins were heavily secreted, reaching a level similar to providing 8 mg of morphine. To understand the significance of this finding, one can contrast the morphine dosage given to cancer patients entering the terminal phase of their untreatable illness; cancer patients with unbearable pain receive 5 mg of morphine.

In summary, the study of Blanchard et al. (1982) identified four basic neurophysiological phenomena associated with PTSD. First, PTSD sufferers display an elevated baseline arousal, which supports the hypothesis that PTSD is an arousal disorder. Secondly, they are neurophysiologically activated by trauma-related stimuli, especially in terms of heart rate

activation, which supports the hypothesis that PTSD is an arousal disorder and the hypothesis that PTSD involves classical conditioning. Third, the sustained heart rate activation observed for prolonged period after a trauma-related stimulation indicates a limited capacity for affect-modulation in individuals with severe and chronic PTSD. Fourth, the arousal experienced by PTSD sufferers can be so high at times that a large amount of endorphins is secreted to counterbalance the unbearable pain. The physical numbing associated with PTSD thus appears to be consequent to neurobiological overactivation, while psychological numbing could still be due to defense mechanisms.

Reading the results of Blanchard et al. (1982) illustrated for me the pain endured by my patients, beyond what I could have imagines and they could have reported. From considering these findings, I could imagine the inner disorganization of my patients with PTSD, their unbearable distress, and their physical pain experienced upon exposure to traumatic cues. It certainly led me to become even more sensitive and patient toward these individuals. It made me more cognizant of their limited capacities and, thus, more cautious in using trauma-focused techniques.

Paradoxical Responses Associated with Dissociative Tendencies

Individual differences exist in people's response to traumatic events and to PTSD, especially dissociation. Practitioners need to consider this factor by itself in order to treat PTSD efficaciously. Elevated physiological reactivity is not consistently observed across all groups of individuals with PTSD (Koopman et al., 2004), which leads practitioners to consider again individual differences, especially a tendency toward dissociating. The lack of physiological arousal, even the presence of de-arousal, can be attributed to dissociation in some individuals, a psychological defense mechanism associated at times with PTSD. Griffin et al. (1997) reported that rape victims with PTSD displaying high scores on a dissociation scale showed a reduced heart rate when they were asked to discuss their traumatic experience, in contrast to those with low dissociative scores who displayed an increased heart rate, as subjects with PTSD usually do. Such results were replicate by Werner & Griffin (2012) with rape victims and by Sack et al. (2012) with victims of multiple traumatic events. Dissociation is thus a major feature to assess in PTSD treatment, as added in the PTSD diagnosis of the DSM-5 (APA, 2013).

Responses of individuals with PTSD and dissociative tendencies contrast with elevated heart rate consistently measured across samples of veterans with PTSD. Some individuals may be more susceptible than others to employ dissociative mechanisms during both traumatic events and their recalls of traumatic events, such as victims of sexual abuse in childhood. Some traumatic experiences may also be more conducive to dissociating during the event as well as during its recall.

PTSD and Neurological Arousal

In PTSD studies, neurophysiological arousal is determined by measuring the level of catecholamines (e.g., norepinephrine and dopamine) or cortisol in saliva or urine. Cortisol is a

metabolite of glucocorticoids, and glucocorticoids are secreted by the surrenal glands following a message sent by the brain. According to Selye (1956) and Sapolsky (2000), glucocorticoids are the neurobiological marker of chronic stress. In neurological studies, the structural qualities of cerebral sites (amygdala, hippocampus, prefrontal cortex cingulate anterior, etc.) are assessed by MRI of volume or thickness, while the activity of neurological sites is measured by fMRI.

Neurobiological studies strongly suggest that severe psychological trauma can cause alterations in the neurobiological responses to stress for PTSD subjects, both immediately after the traumatic event and years after its occurrence. These alterations can contribute to the numerous symptoms presented by individuals with PTSD (Southwick et al., 1997). A literature review indicated that norepinephrine levels were higher in urine of PTSD subjects in comparison to healthy controls and psychiatric patients, using 24-hour urine samples taken throughout the day in response to meaningful stimulation and restful level of autonomic arousal (Southwick et al., 1997). Such results indicate that PTSD is associated with elevated neurophysiological arousal.

Other studies also strikingly illustrate how PTSD is an arousal disorder. In these studies, researchers injected individuals with yohimbine, a chemical compound enhancing arousal. For example, when yohimbine is injected in individuals with panic disorder, many react by having panic attacks, one after another, until yohimbine is metabolized few hours later (Charney et al., 1987). While such reaction was not observed in individuals with major depression, schizophrenia, obsessive-compulsive disorder, and generalized anxiety disorder (Charney et al, 1990), yohimbine induced panic attacks in 70% of PTSD subjects. Yohimbine also provoked post-traumatic flashbacks in 40% of PTSD subjects (Southwick et al, 1993). So, a chemically-provoked neurobiological activation leads to extreme arousal elevations in PTSD, inducing panic attacks as well as flashbacks.

Clinically, these findings suggest two considerations. First, as PTSD symptoms can be exacerbated by increased neurophysiological arousal, it is therapeutically wise for PTSD patients to avoid unnecessary exposure to environmental stimuli provoking heightened arousal such watching televised news and violent movies. Second, individuals with PTSD need to understand the importance of limiting their intake of arousal-provoking substances, such as coffee and sodas, because these substances can heighten arousal and increase PTSD symptoms unnecessarily.

Selye (1956), a stress pioneer, hypothesized that the amount of cortisol released during stress provides an index of the stressor severity. Indeed, there is clear empirical evidence of a direct and linear relation between cortisol release and stressor severity; greater stress leads to more cortisol secretion (Mason et al., 1968). In PTSD, however, the data on cortisol are not directly compatible with Selye's stress model because the relation between cortisol secretion and stress is quite complex. Let's see if we can simplify.

Individuals with PTSD have been found to present lower cortisol levels than psychiatric patients and healthy controls, a rather opposite finding to Selye's stress research indicating

higher cortisol levels are present in stressed individuals. The former finding emphasizes the fact that PTSD is a post-traumatic stress disorder and not just a stress reaction. In medicine, a trauma occurs to the physical body when a violent accident causes a serious physical bodily injury. Allegorically, stress can be represented by bruises incurred in a car accident, while traumatic stress is the broken arm (mild PTSD as defined in DSM-IV) up to the broken spine with a head trauma (severe PTSD with co-morbid disorders).

Although counter-intuitive at first, Southwick et al. (1997) suggest that lower cortisol level in blood and urine in PTSD reflects the chronically heightened sensitivity of the HPA axis (hypothalamic-pituitary-adrenal) observed in PTSD. In a study comparing PTSD subjects with individuals having major depression and healthy controls, the circadian release of cortisol over a 24-hour period was lower in PTSD throughout the day,. However, in PTSD subjects, there was a higher reactivity of cortisol release; relative to their low baseline cortisol level, PTSD subjects tended to show large fluctuations in cortisol levels throughout the day, indicating a more active HPA axis in PTSD (Yehuda, 1994). Furthermore, lower cortisol levels in PTSD can be explained by an increase number of cortisol receptors in the brain, reuptaking the cortisol secreted in the blood, thus leaving lower levels in blood and urine. Indeed, veterans with PTSD were found to have more glucocorticoid (cortisol) receptors than non-psychiatric and psychiatric subjects (Yehuda et al., 1993). As brain receptors of any given neurotransmitter increase in numbers, the neurotransmitters are reuptaken, leaving less in circulation. In other words, the brain adapts to the presence of high cortisol levels in individuals with PTSD, by producing a mechanism by which cortisol molecules are retrieved from blood circulation.

Taken together, findings on the neurobiological arousal in PTSD demonstrate a unique stress response in PTSD. Individuals with PTSD have a higher arousal baseline as reflected in higher basal heart rate. Trauma-related cues induce a heightened reactivity of the sympathetic nervous system in individuals with PTSD. Low levels of basal cortisol, coupled with more numerous receptors for reuptaking glucocorticoids, suggest a heightened reactivity to stress in individuals with PTSD, with the HPA axis having become extremely sensitive to stress hormones. As individuals with PTSD are ongoingly stressed, as shown by their elevated basal heart rate, the brain adapts over time to heightened levels of glucocorticoid secretion by developing more receptors to reuptake them.

These physiological and neurobiological findings indicate an increase sensitivity to stress in PTSD, which is consistent with the hypothesis of hypersensitivity in PTSD which will be explained later on. Indeed, it is well know that high anxiety is associated with a reduced threshold of arousal (it takes less stimulation to become more anxious). This punctual hypersensitivity, however, develops into a neurobiological hypersensitivity which is chronic, long-lasting, and difficult to revert although possible.

PTSD as Arousal-Modulation Disorder

The findings of Blanchard et al. (1982) led to the adoption of the hypothesis that PTSD is an arousal and the hypothesis that PTSD is a pathological manifestation of the inability to

modulate arousal (Everly, 1993; McFarlane & Yehuda, 1996; van der Kolk & Saporta, 1993). Simply stated, individuals with PTSD experience greater activation than non-PTSD individuals and they have a harder time calming themselves down when they are further activated. Individuals with PTSD can be highly activated by simple life stressors and trauma-related stimuli, the latter usually provoking an even higher arousal.

As demonstrated by Blanchard et al. (1982), when individuals with severe PTSD are activated by trauma-related stimulation, their heart rate can reach levels so high that it takes a very long time for them to come back to the previous baseline. One can say that, upon trauma-related exposure, veterans with severe PTSD had difficulties modulating their arousal, manifested by a dramatic heart rate increase, and were incapable of normally modulating arousal afterward. Simply expressed, one can say that veterans with severe PTSD had a hard time soothing themselves during and after experiencing an intense traumatic reminder. No wonder these individuals avoid such exposure!

In my practice, I have met several patients with severe to extreme PTSD who, after exposure to a trauma-related cue, became aroused at such high levels that it took them hours, if not few days, to reach a bearable level of arousal. One of my patients stayed in her room for few days after each random exposure to a trauma reminder. Obviously, psychotropic medication can be quite helpful to those individual in order to protect them against experiencing such peaks of arousal.

In reaction to very elevated arousal, the brains of individuals with PTSD release endogenous opiates in times of severe arousal. For example, Pitman et al. (1990) found that veterans with PTSD had greater levels of endogenous opiates than healthy controls after they were shown combat-related stimulus. This endogenous analgesic response was induced by an exposure to trauma-related cues. Therefore, psychophysiological and neurobiological arousal can be so high at times in individuals with PTSD that the organism responds by releasing opiates to produce analgesia to protect against unbearable pain.

In psychotherapy, practitioners need to attend to the arousal level of patients with PTSD and focus their interventions at both reducing the baseline arousal and preventing flaring up of arousal through preventive avoidance of trauma-related cues. Reducing arousal can be achieved by the learning and practice of anxiety management techniques. Psychotropic medication can also be employed and it is preferable than an endogenous secretion of opioids. Paroxetine (Paxil) and citalopram (Celexa), both SSRIs, have been found to reduce PTSD symptoms (English et al., 2006; Ipser & Stein, 2012) and to augment the volume of the hippocampal, thus the number of hippocampal neurons (Thomaes et al., 2014).

Reduced Performance Under Heightened Arousal: Yerkes-Dodson Law

The arousal-modulation hypothesis for PTSD is quite complementary to the arousal hypothesis. They are interrelated neurobiologically and their interaction can be explained by one of the laws in psychology.

The Yerkes-Dobson law provides an understanding of the neurophysiological processes involved in PTSD as both an arousal disorder and an arousal-modulation disorder. It informs us that the relationship between anxiety and performance follows an inverted-U curve. The Yerkes-Dobson law indicates the following: at low levels of anxiety, individuals perform a task minimally, if not at all; at moderate levels of anxiety, individuals perform optimally; and at high levels of anxiety, individuals perform again minimally, if not at all. If PTSD sufferers undergo their daily living at high levels of anxiety, as indicated by studies on basal heart rates, their performances in any sphere of life activity is likely to be reduced and even hampered, according to the Yerkes-Dobson law.

Relaxing or calming oneself down is task, a performance, in and of itself. Therefore, individuals with severe PTSD, associated with high baseline levels of anxiety, will have a hard time to reduce their ongoing arousal through the use of a task such as a relaxation technique. Relaxation requires utilizing mental functions hampered by anxiety. Here again, psychotropic medication can be quite useful in reducing anxiety to lower levels allowing psychotherapy work to proceed and unfold.

When we mention performance, we usually think of a task involving questions on an IQ test. Performance also involves the functioning of neurobiological processes supporting mental operations. Beyond a moderate and optimum level of anxiety, anxiety interferes increasingly with mental functioning. The more PTSD patients present with high baseline arousal, the less they have access to their mental and neurological functions involved in reducing arousal, especially if arousal is triggered by trauma-related stressors. Many mental functions are involved in the reduction of anxiety, as well as in PTSD symptoms. If hampered by very high anxiety, mental functions cannot be put to work to resolve PTSD. As it will be discussed further, the functions most at risk during high arousal levels are the most sophisticated ones, that is, those embedded in frontal and pre-frontal cortex, such as those responsible for containing and reducing arousal, those who are partly required for psychotherapy to be effective. The poor get poorer.

The Yerkes-Dobson law explains why veterans with severe PTSD in the Blanchard et al.'s (1982) study were at the mercy of their racing heart rate after the exposure to the ambush scene of the war movie Platoon. As they experienced very high levels of anxiety during the visioning of the violent scene, the performance of both mental and neurological functions involved in arousal-modulation were diminished if not cancelled, rendering them incapable of

calming down for a while. The more one is overwhelmed by arousal, anxiety, and affects, the less one can perform mentally and behaviorally.

The impact of high anxiety is well known by practitioners specialized in treating anxiety disorders. When patients are in the throes of having a panic attack, relaxation techniques are rendered useless; at best, focusing on breathing calms their immediate distress and soothes them by distracting them from the pain associated with the panic attack. Once it flares up, a panic attack needs to follow its course.

At a less extreme level, a high level of baseline anxiety will not be reduced by a one-time use of a relaxation technique. Even though patients may engage in the exercise, the anxiety will not be affected at first. It takes the implementation of an ongoing practice of a relaxation technique, over several weeks or months, to obtain the expected benefits. At first, the relaxation technique will have to be performed when patients are least anxious during the day in order to achieve palpable and encouraging results.

Similarly, when anxiety is high to very high in patients with PTSD, it is reasonable to expect mental functions to be less accessible and to function less well in psychotherapy. In my clinical experience, practitioners often need to turn to other types of interventions to reduce arousal. Indeed, many patients with severe PTSD are likely to decrease their heightened by taking an appropriate medication, by resolving ongoing external stressors, by welcoming the attuned relationship offered by the psychotherapist, by regaining control over their outer and inner worlds, and by obtaining adequate social and affective support.

The Yerkes-Dodson law can also elucidate the diversity of reactions during a traumatic event. Our performance during a trauma event will depend on the strength of our mental functions at play. The stronger a mental function is prior to the occurrence of a traumatic event, the less its functionality will be hampered by arousal during a traumatic event.

During a traumatic event, individuals can adequately perform the various motions required by the situation, such as giving money to the robbers during a hold-up. Others may have difficulties in organizing their thoughts and containing their arousal. Some do not even have the capacity to contain themselves in order to simply give the money to the thief during an armed robbery. Indeed, some of my patients ran to the restroom to hide, while others attacked the robber, irrespective of possible severe consequences. These last two reactions reflect Cannon’s model of flight or fight.

Regrettably, our most sophisticated mental functions are most fragile. Planning a response and anticipating its consequences are sophisticated function in comparison to a response or flight or fight. The hindrance to higher mental functions is brought about by high arousal.

In psychotherapy, it may be easier for patients with PTSD to engage in strategies involving behaviors before engaging in interventions involving higher mental functions such as

reflecting and putting things in perspective. In my clinical experience, patients with severe PTSD engage more easily at first in behaviors aimed at reducing stressors, which in turn reduces anxiety. Afterward, they can attempt the practice of a relaxation technique. The removal of stressors reduces anxiety, encouraging patients and rendering them more amenable to engage in more psychological interventions to further reduce their anxiety.

Factors Influencing Arousal and Arousal-Modulation

When practitioners are well trained in diagnosing personality disorders, it becomes evident that most patients with severe PTSD have pervasive structural difficulties impacting their relationship to self and others. Such structural disorders can be viewed as attachment disorders (Bowlby, 1988) or disorders of the self (Masterson, 1985) or as personality disorders according to the DSM. Clinically, patients' limited capacity to modulate arousal and affects is usually reflective of a structural feature such as temperaments, personality and attachment.

Temperaments

Temperaments also play a significant role in the capacity to modulate arousal. According to Chess and Thomas (1986), there are three basic temperaments: easy, slow-to warm-up (anxious), and difficult (feisty or angry). Children with heightened temperaments (slow-to-warm-up or difficult) present an inherent difficulty at modulating arousal, either in terms of anxiety or anger. Temperaments are hard-wired, genetically determined, and present at birth. They can be modified or attenuated by environmental experiences to allow individuals born with heightened temperaments to live enjoyable and functional lives. People do not always need to be imprisoned by biology. When adequately accompanied by caring parents, 75% of children with an anxious temperament get over their anxiety and succeed in approaching novel objects and situations, according to research conducted by Kagan who called these children inhibited (Kohnstamm et al., 1989). In psychotherapy for PTSD, work could be required at the level of temperaments of patients when life-long difficulties in modulating anger or anxiety are displayed.

Considering temperament in psychotherapy, it is interesting to cite findings of a study highlighting the environmental impact and beyond genetically-determined temperaments. Meany (2001) has studied the activation level of young mice according to the mother's temperament. In laboratories, there are different types of mice, including mice genetically-determined to be calm or anxious. Meany (2001) took genetically-anxious mothers and gave them offsprings of genetically-calm mothers, and vice versa. Therefore, mice raised offsprings with temperaments opposite to theirs. Researchers later measured the responses of offsprings to stressors. Results showed that environment had a greater impact than genes. The genetically-calm mothers induced, in genetically-anxious offsprings, a greater capacity to remain calmer when confronted to stressors than the genetically-calm offsprings raised by genetically-anxious mothers. The anxious mother succeeded in activating the neurobiological system of the young genetically-calm mice so that the latter responded to stressors with more anxiety than the genetically-anxious ones soothed by calm mothers. Therefore, Meany (2001)

showed that, in mice, mothers' temperaments did fashion the stress response of offsprings beyond their own genetically-determined temperaments. Maybe a bold leap can be taken here to underline the possibility that such phenomenon can also occur in mother-child relationships, as well as in long-term psychotherapy, spanning months to years. This has been my experience as a mother of an infant born with an anxious temperament and as psychotherapist of many patients born anxious.

Attachment Patterns

Let's focus first on attachment disorders. In my clinical experience as psychotherapist and supervisor, practitioners can expect that, at least, half of individuals presenting with severe PTSD will have a pre-existing attachment disorder. It is of interest to acknowledge the role played by attachment disorders because they are based on anxiety, being called by Bowlby (1988) "insecure" attachment patterns. Attachment disorders do not necessarily lead to the development of an anxiety disorder, but attachment disorders do predispose to the development of anxiety disorders (Brimariu & Kerns, 2008).

Schore (2003) has well demonstrated how individuals with attachment disorders have a limited capacity to modulate affects. An underdeveloped capacity to modulate affects is likely to be both a predisposing factor to developing PTSD and a maintaining factor as well. In PTSD, the experience of a traumatic event has involved high levels of anxiety and intense dysphoric affects. A limited ability to modulate arousal and thus affects is more likely to throw individuals with insecure attachment patterns into PTSD. When intense affects cannot be modulated successfully, they are suppressed and their management is avoided. Consequently, individuals become overwhelmed by arousal. Others dissociate and remain relatively calm, almost too calm, but they suffer consequences later, from having dissociated from themselves, others, and life.

To resolve PTSD, one needs to have sufficient capacities to face and to modulate highly unsettling affects. Such affect-modulation capacity is hampered in individuals having developed an attachment disorder because they have not been attuned and soothed as infants and toddlers (Perrin, 2011). In psychotherapy, work will need to consider attachment patterns and their associated features. Ways of relating to self and others, based on internal representations of self and others, developed from interpersonal experiences in early childhood, need to be sufficiently altered through an attuned therapeutic relationship, to allow for the development of a good-enough internal representations of self and others, to foster the capacity to modulate arousal, which will in turn assist them in soothing themselves and engaging in more trustworthy relations with people (Bowlby, 1988).

According to Masterson (1985), disorders of the self as seen as emerging from an attempt to avoid experiencing abandonment anxiety at a time when infants cannot modulate such anxiety on their own, that is, while they are psychologically or physically abandoned by their mother, caregiver. Again, in this model, anxiety is at the core of structural disorders such as schizoid, narcissistic, and borderline disorders of the self. Oftentimes, clinicians need to

recognize and therapeutically consider a disorder of the self, even if patients come in psychotherapy for help regarding PTSD, because disorders of the self tend to hinder the effectiveness of the techniques recommended for treating PTSD in well-structured individuals (Gaston, 2014).

Self-Medication

Sometimes their affect-modulation capacities are so limited that PTSD sufferers self-medicate. For some PTSD sufferers, the endogenous secretion of endorphins can be insufficient to counter the pain. Consequently, they resort to external means for numbing themselves; they turn to psychoactive substances to reduce pain, both psychological and physical. As Edit Piaf answered when she was asked by a physician the reason behind her use of opioids, she answered: "To stop the body from screaming." Edith Piaf, a famous French singer in the mid-twentieth century, suffered many traumatic events throughout her life.

In a sample of cocaine dependent individuals seeking therapy, structured clinical interviews revealed that 43% had lifetime PTSD. These patients had higher rates of co-morbid disorders and personality disorders (Back et al., 2000). Not only is PTSD correlated to substance use disorders, but PTSD deterioration precedes substance use.

Ouimette et al. (2010) found that changes from subclinical to full-blown PTSD was associated with an 11% increase in alcohol dependence symptoms, a 29% increase in cocaine dependence symptoms, and a 94% increase in opioid dependence symptoms in the following week. In a clinical trial with cocaine-dependent and heroin-dependent outpatients, complex PTSD symptoms were found to interfere with treatment outcomes for substance abuse, independent of demographics, psychological distress, baseline substance use, and treatment modality (Ford et al., 2007). This study suggests that PTSD can be causative in abusing psychoactive substances. In contrast, when PTSD is successfully resolved, substance use decreases or even disappears. Finally, Ouimette et al., (2003) found that PTSD patients with substance use disorders attending PTSD therapy and a 12-step program for substance abuse had better remission rate for their substance abuse at 5-year follow-up than those who rejected treatments. Patients who stayed longer treatments were more likely to be remitted 5 years later. Taken together, these findings support the hypothesis that individuals with PTSD use psychoactive substances in a strategy of self-medication due to the pain associated with PTSD, including very high arousal levels.

Kindling Effect in PTSD

To kindle means to activate, to arouse, to set on fire. When we kindle a fire, we blow oxygen into the fire and the fire gains strength. The kindling hypothesis suggests that the same principle applies to the nervous system. The more neurons are activated, again and again, the more neurons are aroused further and further, and the more easily they are aroused.

For a sound understanding of PTSD patients, it is crucial to understand the kindling

hypothesis because the kindling of the nervous system of PTSD patients into higher and higher greater arousal has several consequences, including long-term neurobiological alterations associated with various dysfunctions. To best understand the kindling hypothesis, it is useful to first review a classical laboratory experiment.

Let's imagine two groups of neurons, A and B. Each neuron is implanted with an electrode. The two groups of neurons are stimulated differently. At time 0, their level of activation is measured and observed as being similar. At time 1, only neurons A are electrically stimulated with a given voltage, followed by the cessation of the electrical stimulation. The subsequent level of activation in neurons A was found to be higher than in neurons B. After a while the activation level of neurons A goes back to its original baseline, which is the same as in neurons B. Thus, before the second electrical stimulation at time 2, neurons A had regained a similar lower level of activation as neurons B. At time 2, both neurons A and B are activated this time, receiving same amount of electrical stimulation received at time 1. Neurons B become aroused at the same level than neurons A at time 1, but neurons A become twice as much aroused at time 2 than at time 1, although they receive the same voltage as neurons B. Basically, this experiment demonstrates that neural activation is additive over time. The more neurons are activated over time, the more they are prone to be activated and the more they become activated the subsequent time. Previously active neurons become more aroused after each stimulation even though they have previously regained the initial arousal level. Translated to PTSD, the Kindling hypothesis explains why epidemiological studies (Breaslau et al., 1998) keep find that the more people are exposed to traumatic events, the more they are likely to develop PTSD. Stress is additive and post-traumatic stress is particularly additive, neurologically speaking.

Neural kindling is associated with long-term potentiation (LTP), a persistent increase in the strength of a synapse. LTP is widely considered one of the major mechanisms by which the brain learns and memorizes. Under experimental conditions, a series of short and high-frequency electrical stimuli are applied to a synapse. As a result, the synapse is strengthened or potentiated, more ready to receive the next information, for minutes to hours. In living animals, LTP occurs naturally and can last from hours to days, months, and years. LPT occurs in both the amygdala and the hippocampus, as well as in many neurobiological structures. Given that neurons exist in an interrelated system of neural pathways and associated mental functions, what does the kindling hypothesis mean for PTSD? To answer this question, the kindling hypothesis needs to be first set against the background of the phenomenon called habituation.

At first, the kindling hypothesis appears to be at odds with the phenomenon of habituation. Following a sensory stimulation of the nervous system, habituation is expected to be set in motion, that is, reversing the neural activation back to its original level. Habituation consists of activation reduction over exposure, while kindling consists of activation augmentation over exposure. In my research experience, when two competing hypotheses ar e empirically supported, they are usually complementary to each other within a larger theoretical framework.

The kindling effect is complementary to the habituation process, as one or the other occurs depending on the conditions in place. At low levels of stimulation, habituation is likely to occur but, at high levels of stimulation, neural activity is likely to be kindled into higher levels of activation. This hypothesis is in line with findings showing that mild PTSD symptoms remit easily while severe PTSD symptoms become chronic and further deteriorate into other disorders such as major depression, generalized anxiety, etc. Differential effects of kindling and habituation can be understood by reviewing the different responses in the hippocampus and the amygdala under varying degrees of stress.

Capacity for Habituation as a Protection

Habituation is an important concept in the field of anxiety. It refers to the spontaneous deactivation of the amygdala arousal after it has been stimulated. When people can habituate to new stimuli, the nervous system quickly restores itself to the baseline level of activation. During stressful situations, habituation allows individuals to have access to their well-developed mental functions to continue to perform sophisticated tasks. If one is "rich" in habituation capacities, one is able to remain calm enough and to sooth oneself, reducing one's anxiety or even remaining poised in the face of highly stressful stimulation. Such individuals are unlikely to develop neurobiological hypersensitivity, a phenomenon further described.

An example can hereby illustrate the phenomenon of habituation. Let's imagine that you are working at your desk and, outside your office, construction workers are suddenly using gas-powered hammers. You are first alerted by the first loud noise and you turn your attention to it to understand what it is, where it comes from, and whether you need to be concerned by it. As the loud noise is repeated over and over, you might soon disregard its occurrence because you now know the non-threatening quality of the situation. Your brain will kick into gear, automatically and unconsciously, to de-activation of the neurobiological processing of the loud noise. Consequently, you will be able to refocus your attention away from the recurrent loud noise, refocusing your attention onto the task at hand. Your brain will have habituated. The stimulus will have been recognized and appraised as non-threatening, and the neurobiological activation associated with the occurrence of loud noises will have ceased. Your brain will not be activated further and further upon its repeated occurrence. You will have shown a capacity to habituate to loud noises within this situation and, therefore, you will not be bothered by them. Your brain will have stopped its unnecessary activation and will have regained its baseline level of arousal, spontaneously employing the mechanism of habituation. The rich get richer.

Other individuals might not have the capacity to habituate to such stressful situations. They might be unable to distract themselves from the repeated loud noises and their nervous system will becomes more and more activated by them. They could even become neurobiologically aroused to such a level that they will be thrown into a state of irritability or distress.

If we go back to the Yerkes-Dobson law regarding the inverted-U relationship between

anxiety and performance, we can understand that, as individuals are thrown into high levels of arousal, their capacity to turn their attention away from the sensory stimulation is reduced, a task which would have reduced their neurobiological arousal. Due to this limited capacity to habituate, their brains will be activated at higher and higher levels if the loud noises do not stop. Few options are then left. Some stay in the situation and endure the arousal triggered by the noises, as some people "tough it out". Some leave the premises because they care not to be unnecessarily disturbed. Some wait a while and leave only when their neurobiological activation has become so high that they cannot concentrate anymore on their task at hand, leaving them in painful state of arousal. Some consciously utilize de-activation strategies such as concentrating on their breathing or reminding themselves that they are safe despite the noise. These strategies may be successful or not. For these individuals, any strategy involving the repeated activation of their brains, beyond their capacity to modulate arousal, will lead to neurobiological hypersensitivity, rather than habituation. The poor get poorer.

In the treatment of simple phobias, individuals become anxious upon exposure to anxiety-provoking stimulation and can respond to prolonged exposure (flooding, immersion) therapy. Because individuals with PTSD react intensely to trauma-related stimuli, some have argued that the empirical findings with anxiety disorders, such as simple phobias, support the use of trauma-related exposure for treating PTSD (Rabois et al., 2002). However, individuals with PTSD differ from individuals with simple phobias, in terms of their habituation capacity. Indeed, PTSD is a structural disorder while simple phobias are based on a circumscribed phenomenon of classical conditioning.

Psychophysiological studies have repeatedly found abnormalities in the habituation capacity of individuals with PTSD (Butler et al., 1990; Kozak et al., 1998; Paige et al., 1990; Shalev et al., 1993). Individuals with PTSD react with high arousal upon trauma-related exposure and they display limited capacities to habituate, that is, they remain at high levels of arousal for prolonged periods. One study specifically examined the relation between subjects' initial arousal upon trauma-related stimulation and outcome of prolonged exposure or PE (Van Minnen & Hagenaars, 2002). Improved subjects displayed a capacity to habituate in the first session and over the course of treatment. After treatment, improved PTSD subjects showed a lessened arousal upon trauma-related exposure, while non-improved subjects showed higher arousal. These findings are in line with the Yerkes-Dobson law. Subjects showing a capacity for habituation improved their PTSD in exposure therapy, while others did not habituate upon exposure to trauma-related cues.

Another variable influences subjects' responsivity to prolonged exposure or PE, the initial severity of PTSD. Van Minnen et al. (2002) examined patients' data files from controlled clinical trials and found a strong relation between pre-treatment PTSD severity and outcome in PE. Initial severity of PTSD was the only predictor of outcome in PE. The less symptomatic subjects were before treatment, the more they benefited from PE and, inversely, the more symptomatic subjects were initially, the less they benefited from PE. This finding is concordant with the Yerkes-Dobson law stipulating that moderate arousal will allow habituation to take place, but high arousal will interfere with habituation. Let's underline here a well-known fact:

initial severity of symptoms is the strongest predictor of outcome across all psychotherapy modalities and across all mental disorders.

Practitioners can consider two basic groups of individuals with PTSD. On one hand, individuals with mild PTSD who could benefit from any type of a therapy because they have few PTSD symptoms, present moderate basal arousal, are not highly reactive to trauma-related exposure, and can quickly show habituation in treatment. On the other hand, individuals with severe PTSD may not benefit from trauma-focused therapy because they tend to have many PTSD symptoms, to be highly reactive to trauma-related exposure, and to display a limited capacity for habituation.

A third subgroup of individuals with PTSD needs to be recognized by practitioners in order to design adequate a treatment plan for any PTSD sufferers, namely dissociators. Dissociators display a particular reactivity to trauma-related stimulation. Upon stimulation, they react with reduced arousal, such as lowered heart rates, rather than heightened arousal as others PTSD sufferers (Griffin et al., 1997; Werner & Griffin, 2012). Dissociators are likely to require specific interventions addressing dissociation for psychotherapy to be effective in reducing or eliminating their PTSD.

To respond adequately to each individual presenting with PTSD, practitioners need to consider each patient in his or her own complexity. There are at least three subgroups of individuals with PTSD to be recognized because they respond differently to trauma-focused exposure: those presenting with mild to moderate PTSD, those presenting with severe and extreme PTSD, and those presenting with PTSD and a clear tendency to dissociate. In my clinical experience, this categorization has been very useful because it quickly orients us toward the most effective therapeutic interventions.

Long-Term Hypersensitivity

When habituation fails to reduce arousal following repeated stimulations, hypersensitivity develops. As it was demonstrated in the experiment with neurons receiving electrical stimulation, the kindling hypothesis stipulates that repeated activation produces additive effects of arousal. In humans, after repeated and excessive stimulations (beyond the individual's threshold for habituation), neurons become activated at higher and higher levels, causing hypersensitivity. The phenomenon of hypersensitivity resides in the amygdala, the center for both survival drives and affects.

Many empirical studies provide evidence regarding hypersensitization in PTSD. In laboratories, repeatedly shocked animals display heightened reactivity to lesser shocks, responding as if the shocks were more intense. Shocked animals release amounts of norepinephrine consistent with a much intense stressors. The animals' reactivity is also found to be a function of the severity of the initial shock; the greater the initial shock is, the more shocked animals react to innocuous stressors (Southwick et al., 1997).

In humans, the magnitude of combat exposure has been repeatedly associated with a greater incidence of PTSD (Kulka et al., 1990) and exposure to childhood traumatic events is related to PTSD symptoms (Putnam, 1993). Finally, the severity of PTSD symptoms increases over time once they are established (Breslau et al., 1998). If individuals are highly anxious, they will have a hard time soothing themselves, calming themselves, and putting things in perspective during a very stressful situation. As stressful situations reoccur, hypersensitivity develops because arousal-modulation capacities are unable to constrict arousal within an acceptable range. Stressful situations will push arousal to higher and higher levels, in an incremental, additive fashion.

The kindling hypothesis provides an explanation for a very interesting clinical phenomenon observed in a particular group of crime victims, bank tellers.* From my clinical experience in treating bank tellers having developed PTSD, these women can tolerate the first few hold-ups without developing PTSD, but, as hold-ups keep on occurring, the likelihood that they develop PTSD increases. Why? The kindling hypothesis stipulates that, after each hold-up, the baseline arousal will be heightened and, after a while, it is likely to remain at high levels. This phenomenon represents the cumulative quality of the kindling effect. Over time, their brains will have been kindled by a repeated exposure to hold-ups into higher levels of baseline arousal and into hypersensitivity, leading to further neurobiological arousal. One day, another hold-up happens and this one becomes the one too many, provoking such a high level of arousal that mental functions are seriously abated and the traumatic event makes its mark upon the emotional memory system of the individuals, forcing them into PTSD. These bank tellers become overwhelmed and cannot contain the resulting activation associated with this latest hold-up. They present with PTSD and co-morbidity, and they cannot work in such high stress situations anymore, at least for the time being.

** This example applies only if bank tellers do not present with borderline personality disorders, which would interact with their limited arousal-modulation capacities to create PTSD at the first hold-up. In my clinical practice, individuals with borderline personality disorders have be known to develop PTSD more readily after one hold-up only, than individuals with narcissistic or schizoid personality disorders because persons with borderline disorders tend to react with high emotional intensity and to seek help more readily than other personality disorders.*

Hypersensitivity is not only induced by the traumatic events, but hypersensitivity can be facilitated by PTSD in and of itself. The reexperiencing symptoms, such as flashbacks, are event, in and of themselves, capable of producing heightened and excessive stimulation of the amygdala. Each time a flashback occurs, the individual is thrown into a higher arousal level. Over time, repeated arousal causes hypersensitivity. This self-reinforcing quality of PTSD explains why some individuals can seriously deteriorate over the years although they initially displayed moderate PTSD. When PTSD is not treated promptly, effectively, and adequately, all psychotherapy modalities may become obsolete for resolving PTSD later in life because neurobiological structures may have become so deteriorated that they will be impaired irreversibly.

Hypersensitivity as Vulnerability

The kindling hypothesis also suggests that the more anxious individuals are, the more likely they will develop PTSD when they are confronted to a traumatic event (McFarlane, 2010). Epidemiology studies support this hypothesis because it has been found that people with pre-existing anxiety disorders have a higher likelihood for developing PTSD after the occurrence of a traumatic event than individuals without anxiety difficulties (Breslau et al., 1998). People who have experienced previous traumatic events are also more likely to develop PTSD than others (Kulka et al., 1990; Putnam, 1993). And, people who have developed PTSD previously are more likely to develop another PTSD upon the occurrence of a new traumatic event (assuming that they were not treated adequately or not at all).

Many individuals with PTSD do not have well-developed capacities of habituation, especially if their PTSD is severe. Indeed, they are likely to have developed PTSD, at least in part, because of their limited capacities of arousal-modulation and the associated hypersensitivity (McFarlane, 2010). Some people have been so frequently exposed to excessive stressors throughout their lives that any relatively intense stimulation throws them into hyperarousal. Such high arousal counteracts their limited capacity for arousal-modulation, creating a vicious cycle.

In summary, neural kindling can lead to hypersensitivity. Hypersensitivity represents a consequence of PTSD, but it is also a vulnerability for developing PTSD. The more hypersensitized people are, the more likely they will develop PTSD following exposure to a traumatic event (McFarlane, 2010) and the severe the PTSD is likely to be. As this vicious cycle sets in and amplifies itself over time.

Long-Term Neurobiological Alterations

Hypersensitivity can cause long-term alterations in neural excitability, which is associated with vitality loss in various brain areas. Excessive and repeated stimulation can be toxic to neural substrates. This “excitatory toxicity” would be caused by glucocorticoids, huge molecules associated with chronic stress. Although they are secreted by the adrenal glands, glucocorticoids cross the blood-brain barrier and damage neurons (Sapolsky, 2000).

The more complex and sophisticated neurons are, the more fragile they are; the more they can be damaged by glucocorticoids. The most sophisticated neurons compose the frontal and pre-frontal cortex as well as the hippocampus. These areas allow arousal-modulation, thus in PTSD resolution (Metcalfe & Jacobs, 1996).

Prolonged stress can result in changes in the hippocampus, both functional and morphological, via an increased secretion of glucocorticoids (Sapolsky, 2000). In animal studies, a glucocorticoid-mediated atrophy of the hippocampus was found when animals were stressed for several weeks. The atrophy was reversible, however, within weeks after the cessation of the stressor (Sousa et al., 2000). Such studies do not inform whether traumatic stress, such as

chronic PTSD, leads to reversible alterations or permanent ones.

In humans, hippocampal atrophy was observed in individuals with long-lasting PTSD. Using magnetic resonance imaging (MRI) technology, several studies indicated that individuals with long-lasting PTSD had reduced hippocampal volume by 5%-25% compared to normal controls, but not all studies showed a reduced hippocampal volume (Pederson et al., 2004). Differences in methodology in these studies could explain this divergence in findings.

The functionality of the hippocampus can be measured by another methodology called magnetic resonance spectroscopic imaging (MRSI), which assesses the neural vitality rather than volume. This technology (MRSI) detects neural death by assessing N-Acetyl Aspartate (NAA) concentrations in neurons before their death leads to structural atrophy. At least three studies found reduced NAA concentrations in participants with PTSD, in comparison to normal controls (Freeman et al., 1998; Schuff et al., 1997; Schuff et al.,2001). Effect sizes between groups were also larger with MRSI methodology in comparison to MRI.

The hippocampus is implicated in the control of stress responses, declarative memory, and contextual aspects of fear conditioning. Not surprisingly, the hippocampus is one of the most plastic regions in the brain. Hippocampal volume reduction (a reduction in dendritic branching, a loss of dendritic spines, and an impairment of neurogenesis) in PTSD may reflect the accumulated toxic effects of repeated exposure to increased glucocorticoid levels or increased glucocorticoid sensitivity. Recent evidence also suggests that decreased hippocampal volumes might be a pre-existing vulnerability factor for developing PTSD. Indeed, hippocampal deficits may promote activation of stress responses and failure to terminate stress responses, and such deficits may also contribute to impaired extinction of conditioned fear as well as deficits in discriminating between threatening and non-threatening cues. Studies using functional neuroimaging have further shown that PTSD patients have deficits in hippocampal activation during a memory task (Bremner et al., 2003).

In contrast, neurons located in the amygdala, a key neurological center involved in survival and arousal, remains unaltered by large secretions of glucocorticoids. Such discrimination of action by glucocorticoids makes ontological sense because organisms need to focus on survival before being concerned with arousal reduction.

Findings on the neurobiological alterations associated with intense stress and PTSD point to obvious clinical implications. Practitioners need to be aware that higher mental functions can be altered by heightened arousal and chronic stress. Practitioners need to choose interventions suitable to patients' capacities for arousal-modulation. At the beginning of therapy, practitioners need to appraise whether patients can modulate arousal and dysphoric affects. If not, practitioners need to help patients develop such abilities, through the therapeutic relationship and supportive intervention, before employing any method provoking anxiety. Such concern is especially true when PTSD is severe, long-standing, and complicated by co-morbid disorders, because the higher mental capacities of such patients are likely to have been hampered or damaged over time. As the arousal baseline diminishes, individuals have

greater access to reflective capacities, allowing them to better participate in psychotherapy.

Clinical attention needs to be paid to reducing anxiety in PTSD patients in order to prevent hypersensitivity. Nonetheless, at the beginning of therapy, some interventions potentially provoking anxiety, such as empathic reflections of patients' distress, might need to be employed in order to assess whether patients can tolerate some minimal anxiety. From patients' responses, practitioners can identify how patients protect themselves against psychological pain, such as using minimization or amplification of affects. To prevent unnecessary arousal, anxiety-provoking interventions should be brought into psychotherapy gradually, moving from peripheral concerns to core trauma-related issues (Horowitz, 1984). Practitioners work from least anxiety-provoking interventions to more anxiety-provoking ones, assessing patients' arousal level and arousal-modulation capacities after each intervention and adapting technique choice accordingly. It is always best to provide patients with interventions at a tolerable dosage (Horowitz, 1984).

Free-Floating Helplessness

Learned helplessness occurs after repeated traumatization, as studied by Seligman (Peterson et al., 2004). After dogs had been conditioned to associate a sound with electrical shocks, they ended up reacting to electrical shocks as if nothing mattered, failing to try to escape anymore. These dogs had learned helplessness. Seligman employed a trauma paradigm with animals and noticed that the resulting behaviors were expressions of anxiety and depression. He labelled this set of reactions "learned helplessness".

In many studies on learned helplessness, dogs were put in two conditions. In the first condition, dogs were given electrical shocks, but they were given a means to stop them from occurring. In the second group, dogs were given electrical shocks, but they could do nothing to stop them from occurring. The dogs in the first condition quickly recovered, but the helpless dogs stopped trying to do anything. All dogs were later put in a room divided in half by a low barrier. They were warned by a flash of light that an electrical shock was coming, allowing them to avoid it by going into the other half of the room. The dogs in the first condition escape by jumping over the fence, while the helpless dogs simply crawled down and cried even though they could now escape. Previous repeated experiences of lack of control over aversive stimulation can produce helplessness.

The helpless dogs also failed to use previously successful escape strategies and they engaged in generalized behavioral inhibition. Aversive stimulation led to opioid and non-opioid forms of analgesia, stomach ulcerations, immunosuppression, and lowered tumor resistance. Compulsions and addictions emerged in some dogs: once the aversive stimulation was terminated, some dogs tried to escape compulsively, and some drank alcohol whenever it was provided. These dogs displayed difficulties similar to those associated with chronic PTSD.

One observation is remarkable: only two-thirds of animals developed helplessness and trauma-based complications. Like humans, not all animals are predisposed to become severely

destabilized by repeated traumatic events. Some even appear to be immune to developing marked post-traumatic vulnerabilities.

In humans, Seligman proposed characteristics as protection from developing learned helplessness. People resilient to learned helplessness tend to view aversive situations as due to factors other than their own. They view causes to be specific to the situation at hand and temporary. Such individuals do not generalize their experience.

Studies on learned helplessness have been numerous. They highlight the importance of recognizing signs of learned helplessness in patients who have endured repeated traumatization. For example, it is not infrequent to encounter women, who have been sexually abused as children, engaging in abusive relationships with men. Dynamic and cognitive interventions may be useful in assisting these women in regaining control over their lives, such as addressing the thorny problem of the internalized perpetrator (Herman, 1992). Behavioral interventions, such as assertion training, can be needed to assist these women in countering the interpersonal inhibition embedded in learned helplessness.

Neurobiological Systems of Arousal-Modulation in PTSD

Let's begin this section with a caution. Neurobiology is the fashion in psychology and psychotherapy. Knowing and understanding neurobiological findings are, however, not about intervening at neurobiological level or about bending psychotherapeutic interventions to effect specific neurobiological changes per se. Such comprehension is here to inform practitioners of the need to go easy with PTSD, to proceed gradually from the periphery to the core, and to respect patients' limitations concerning the capacity for affect-modulation. In psychotherapy, brief of prolonged, the primary focus should always be the person, the suffering human being before us. The etymology of the word 'patient' refers in Greek to 'one who suffers' (the virtue patience requires one to bear suffering). Psychotherapists should be careful to not fall into the growing fashion of using words reflecting a dehumanizing of human beings. Now let's see how neurobiological findings can inform us to better care for individuals presenting with PTSD.

The neurobiological system underlying arousal-modulation (including affect-regulation) comprises many areas of the brain. For the sake of clinical understanding and purpose, a simplified view of well-delineated brain areas will be adopted hereby. The major neurobiological systems underlying arousal-modulation are the limbic system and the pre-frontal cortex.

Everly (1993) proposed that the phenomenological epicenter of PTSD resides in a functional hypersensitivity of the limbic system. More specifically, arousal and hypersensitivity occur in the amygdala-hippocampal nuclei and their efferent projections. These glands, the amygdala and the hippocampus, are systems capable of treating and storing information. They are involved in learning and memory. There is an amygdala and a hippocampus in each hemisphere of the brain. For PTSD, the glands located in the right hemisphere are of interest. The following sections are based on Everly's (1993) and Metcalfe & Jacob's (1996) works.

Amygdala

The right amygdala is responsible for survival and comprises instinctual drives such as thirst, hunger, reproduction, fight, and flight. The amygdala is the neurobiological center for arousal. The first affect produced by the amygdala is a state of vitality, essential for functioning in life. The amygdala produces simple states in which we feel good or bad. The affects are pleasure, interest, fear, anger, sadness, disgust, and shame.

The amygdala is thus the center where the emotional component of any given memory is located. Let's remember that memories consist of a co-activated set of neural pathways in different brain areas. Some memories are mostly affective while others are mostly cognitive. A memory involving affects will involve neurons in the amygdala.

For each stimulus selected to receive attention, the amygdala assesses its basic

meaning, that is, whether the stimulus is a good or bad for the organism. If it is assessed as good, the organism is propelled to approach and, if it is assessed as bad, the organism is propelled to avoid. To trigger particular responses, the amygdala relies on its memories encoded either genetically or experientially. We are born with inherent knowledge embedded within the amygdala, but our experiences fashion responses further as the amygdala treats novel information. New associations are formed within its neural structure, which later serve as a basis for interpreting subsequently information.

From experiences, the amygdala records main features of a stimulus, its meaning, and its affective responses. A subsequent conditioned stimulus can trigger the retrieval of the information encoded in the amygdala about the unconditioned stimulus. Thus, classical conditioning occurs within the amygdala which encodes it.

The information encoded by the amygdala has particular qualities. It is fragmentary, unintegrated, emotional, and behavioral, including the fear-provoking features of traumatic events which are directly linked to fear responses. These amygdala-based memories are thus highly emotional, inflexible, fragmentary, and unintegrated. The amygdala-based memories are quickly stored and quickly retrieved, which is an advantage for survival. These resemble traumatic memories.

When an amygdala-based memory is triggered, the memory is often experienced as an intense affect, along with possible fragments of sensory representations; for example, internally viewing the barrel of a gun and experiencing intense fear all at once. Nonetheless, amygdala-based information is subject to the control processes of the pre-frontal cortex.

Let's remember that the information provided by senses will first go to the thalamus, with the exception of olfactory sensations which are directly transmitted to the amygdala. This phenomenon can provide an explanation as to why olfactory sensations are so often associated with intense dysphoric affects in PTSD. Olfactory memories are powerful in destabilizing individuals and are difficult to eradicate in psychotherapy.

LeDoux (1995) and Davis (1992) have extensively studied this phenomenon in the amygdala. They have shown that selective lesions to the amygdala in rats eliminate the expression of fear and the ability to learn conditioned fear. LeDoux (1995) has traced the informational pathways leading into and out of the amygdala. One pathway is the connection between the thalamus and the amygdala; this pathway is quick and non-cognitive. If this connection is severed, new information coming from the thalamus cannot elicit a fear response. A second pathway is more circuitous and cognitive, involving feedback from the hippocampus and the cortex which allow for the suppression of a fear response.

Most practitioners are cautious, recognizing the limitations of a classical conditioning model to account for the complexities observed in PTSD. Nonetheless, it is useful to recognize that fear responses, and possibly other emotional responses, are never extinct and that cortical structures and neural pathways need to connected, enhanced, or built in order to inhibit

amygdala-based responses in PTSD.

Hippocampus

Complete memories also comprise actions and thoughts, which are put in perspective in terms of time and space. Such memories involve the activation of the right hippocampus. The right hippocampus is of interest for our understanding PTSD because it is involved in autobiographical memories. Anatomically, the right hippocampus is next to the right amygdala, which is of prime importance because the secretion of neurotransmitters by the amygdala will directly and quickly affect the functionality of the hippocampus.

The hippocampus is also involved in processing and encoding information, as the amygdala, but the type of information treated and encoded by the hippocampus is quite different. The hippocampus records the action sequence of autobiographical events (from a to z, so to speak). The gist of the biographical memories is encoded in a sequential, oftentimes complete, coherent, and unemotional fashion in the right hippocampus. Of outmost importance for PTSD, the memories recorded in the hippocampus comprise the spatial-temporal context of the traumatic event, that is, where and when the event took place. In the right hippocampus, the encoded information is cognitive, complex, neutral, integrative, and subject to be controlled by the pre-frontal cortex. Although its processes operate at an unconscious level, the hippocampus is the basis for our capacity to put things in perspective, to be the witness of our experiences.

Importantly, the hippocampus is involved in memory consolidation, allowing active affect-laden memories to become long-term memories and thus cease to present themselves intrusively onto neurobiological processes and/or one's conscious awareness. McClelland et al. (1995) proposed that new memories are initially represented within the hippocampus, and that during the course of consolidation, they become interwoven into a network of existing related memories in the neocortex. This interweaving process incorporates new memories and typically requires modification of the pre-existing network structure to add the new memories. It appears clearly that, as the hippocampal functioning is essential for new memories to be created in psychotherapy and as the hippocampal activity is hampered by heightened arousal, psychotherapy needs to aim at reducing patient's arousal in the amygdala.

In severe PTSD patients, Dickie et al. (2013) found that initial hippocampal activity predicted PTSD severity after 6 to 9 months of ongoing psychotherapy, that is, at time when 65% of patients were already in PTSD remission. As suggested above, PTSD improvements in psychotherapy depended, partly, on the initial functioning of the hippocampus. Furthermore, changes in hippocampal activity correlated with changes in PTSD, over 6 to 9 months. As PTSD improved over the course of therapy, the activity in the hippocampus improved.

Fortunately, hippocampal atrophy and functional deficits reverse to a considerable extent after treatment with selective serotonine reuptake inhibitors or SSRIs (Bremner & Vermetten, 2004).

Amygdala-Hippocampus Tandem

Let's focus on the functions of the right amygdala and right hippocampus during a traumatic event. The hippocampal activity records that the traumatic event happens here (location) and now (date), while the amygdala produces affects informing us how we feel about it. While the amygdala is the center for arousal and affects, the hippocampus is devoid of emotional reactivity. The amygdala's responsivity is extremely quick as the organism's survival, and the hippocampus puts things in perspective. The information stored in the amygdala is inflexible and records only fragments of the information (for example, after a hold-up, the memories stored in the amygdala retain the most emotional components of the event such as the barrel of the gun pointed at you), while the hippocampus records events in a sequential and coherent fashion (what you were doing as the robbers arrived, then what you did after they left, etc.). The hippocampus records information in a complete fashion while the amygdala records fragmented bits and pieces, those associated with arousal. We could think of the amygdala as providing the music in a movie and the hippocampus the visual.

Located next to the amygdala, the hippocampus is a counterbalancing and complementary system. It is a cool system dampening the activity of a hot system. As explained by Metcalfe and Jacobs (1996), the amygdala is the hot center for information processing and storing, while the hippocampus is the cool center for information processing and storing. The amygdala and the hippocampus are complementary. When everything goes well, they function in concert. Encoding processes happen in parallel in the amygdala and the hippocampus. However, the encoding processes in the hippocampus are dependent on the activation of the amygdala. The amygdala can be at times activated to such levels that it momentarily interferes with the functioning of the hippocampus.

The functioning of the amygdala and its secretion of neurotransmitters is proportional to the amount of stimulation provided. Expressed as a correlation, there is a straight line between the amount of external (or internal) stimulation and the amount of activity in the amygdala. In contrast, the functioning of the hippocampus follows an inverted-U curve, just like the association between performance and anxiety in the Yerkes-Dobson law. Indeed, an understanding of the relative functioning of the hippocampus in relation to the different levels of activation of the amygdala corresponds to the Yerkes-Dobson law.

At low to moderate levels of stress, both amygdala and hippocampus are performing proportionally to the level of stimulation; the amygdala and the hippocampus are equally active. Beyond moderate levels of stress, the relation changes. From moderate to extreme levels of stress, the response of the amygdala continues to directly follow the amount of stimulation, but, in contrast, the response of the hippocampus diminishes gradually. Why? At high arousal levels, the amygdala secretes such amounts of neurotransmitters that they start to interfere with the functioning of the hippocampus. The hippocampus becomes "under attack" and its functions gradually diminish as stress augments until it shuts down.

During extreme traumatic events, the activation of the amygdala can be so high that it

seriously dampens the functioning of the hippocampus, thus limiting people's capacities to put things in perspective and record the traumatic event in a complete and coherent fashion.

In PTSD, traumatic memories are located in the amygdala, along with dysphoric affects, rendering them destabilizing as in flash-backs. The emotional charge associated with traumatic memories can be overwhelming. The traumatic components encoded in the hippocampus remind the individual that the traumatic event occurred then and there. Such contextual components cannot be relegated to the past as long as the emotional components are activated in the amygdala. In contrast, every time an individual has access to higher mental functions lowering arousal in the amygdala and processing the traumatic information, a new perspective can be gained. The traumatic memories become integrated into the larger system of long-term memory.

Clinical examples

Clinically, the role of the right hippocampus is crucial. Consequently, its functionality needs to be stimulated in psychotherapy. The hippocampus is responsible for putting things in perspective, to step aside from emotional responses in order to observe, to become conscious and to reflect upon a situation. Sterba (1934) suggested that the working alliance in psychotherapy requires a capacity to oscillate between experiencing and observing. From a dynamic perspective, the hippocampus can be viewed as the kernel of the observing ego. So, when the amygdala has become hypersensitized over time and the functionality of the hippocampus has been hampered by repeated traumatization over years, individuals have limited capacities to observe themselves and to soothe themselves. Practitioners thus need to help reduce the activation of the amygdala (discussed in a later section). Mostly, clinicians need to address patients' issues in a calm, reflective, and supportive way. These strategies can go a long way, but their effect takes time.

Let's examine a clinical example of an individual whose amygdala's repeated activation has overwhelmed his higher brain functions, so much that it has rendered him hypersensitized to a point of no possible return to his original baseline of functioning (unless, maybe, he devotes the rest of his life to only practicing meditation). A fifty-year-old man was sent for an evaluation to have his mental condition assessed, including the presence of a PTSD, for insurance purposes. Although the evaluator has seen more than five hundred individuals already in evaluation for PTSD, she was struck to see a tall and strong man present with such overt vulnerability. It was as if this man had no more psychological capacities to hide his heightened arousal in order to protect his self-esteem, no more energy to save his honor. His teary eyes ongoingly showed fear, reflecting a sense of diffuse but real danger. His eyes were pleading for help and yet he shared his complete discomfort at being viewed so damaged. His hands shook lightly but incessantly. His body was curved forward as if he was attempting to protect himself from a blow which would not come. He could answer questions about himself and symptoms, but his tone was soft, hesitant, and slow. He kept repeating, "I can't do it anymore!" He presented with an extreme PTSD, along with anxious and depressive co-morbid disorders. He was completely dysfunctional, barely making it through every day. Taking his

history, the evaluator learned that this man used to be a very active member of his community, volunteering in many organizations. His childhood was uneventful, but he was raised in a familial and a cultural climate in which a man needs to be strong at all costs. He had worked for 30 years as a police officer. He had seen many car wrecks in which people's bodies were dismembered, crashed, etc. His life has been in danger several times throughout his career. He intervened onto many horrific scenes, the last involving a basement where a teenager had blown his brains out with a rifle; the impact of the bullet was so strong that body was left headless and the walls were covered by blood and flesh. To protect the family from this horror, he and his colleague had cleaned the whole room. He reported that he functioned well for years despite all of these traumatic events, until this last event. It was the last drop. His brain had been neurobiologically hypersensitized by repeated traumatic events over the years, but he had consistently denied any distress, probably to be able to continue to function as if nothing could destabilize him, needing to be strong at all costs. His psychological make-up led him to neglect himself for all those years. He started to take care of himself only when he was forced, when his denial broke down under the strength of arousal. Now, his induced neurobiological alterations appeared to be so deep that they could be irreversible. The practitioner told him that, given what he experienced over the years, he was now disabled and would never work again. This man was saddened but relieved when he learned that the evaluator would write these conclusions in her evaluation report. This man was already taking adequate medication, which efficacy was limited, and had been seen in psychotherapy for two years. According to his verbal report, he had a good relationship with his therapist who was comprehensive and whom he trusted. The psychotherapist seemed sensitive and flexible enough to provide this damaged man with a gentle presence, affective support, stress management at times, and a willingness to share empathically the patient's experiences of deep psychological and physiological pain. With such patients, therapeutic expectations need to be very limited. Practitioners need to be respectful of the severe limitations presented. In this way, we honor the beauty of human beings, ours and theirs.

The amygdala-hippocampus relation can be understood through some reexperiencing symptoms of PTSD. During flashbacks, individuals usually demonstrate a capacity to be aware that the event is not reoccurring at this very moment, although the event may be replayed as a superimposed film in their mind and intense dysphoric feelings may be experienced. The hippocampus is strong enough to allow individuals to remember that the traumatic event happened then and there, not here and now. The arousal of the amygdala is not overriding the functioning of the hippocampus.

An example of a dissociative flashback might be useful at this point. A veteran consulted after participating to numerous peace missions. He presented with a very severe PTSD and co-morbid disorders. Whenever he heard a loud noise, he went back to moments of his military engagement during which he heard artillery shots and his life was threatened. During dissociative flashbacks, he could be anywhere and, every time, he grabbed an object which could be turned into a weapon and he went hiding behind a sofa or a bush, waiting to be attacked and ready to defend himself. After several minutes of dissociative flashback, in which he was sure that he was back in a war zone, he woke out to realize where he was and the actual

absence of danger. Shame would usually ensue. For months, the therapeutic focus was mostly on helping this man to reduce his arousal.

During dissociative flashbacks or pseudo-hallucinations, the amygdala-hippocampus relation appears to be different. The amygdala seems to be more activated and the hippocampus more abated, reducing access to the spatial-temporal mnemonic aspects of the traumatic event. Clinically, it could be said that, during dissociative flashbacks, individuals temporarily lack access to their observing ego. Indeed, there is a lack of discrimination between the inner and outer worlds; the inner experience is so strong that it becomes projected onto the outer world. Although they feel or behave as if the traumatic event and a related aspects of the event are reoccurring during dissociative flashbacks, individuals are able to regain access to higher mental functions afterwards, to regain sufficient perspective to know that it did not happen. If they would think that it truly happened, they would be experiencing a psychotic episode. So, after dissociative flashbacks, people can recover from the overwhelming nature of the experience. Nonetheless, practitioners need to consider patients presenting with dissociation flashbacks or pseudo-hallucinations as more destabilized or more fragile than others displaying only flashbacks. Practitioners need to determine whether these two symptoms, dissociative flashbacks and pseudo-hallucinations, are function of pre-existing structural damages or post-traumatic responses such as a wish to kill or even a wish to repair.

Let's now examine what a pseudo-hallucination might entail. As he was driving a public bus in a big metropolis, a driver was assaulted from behind by a late teenager who stabbed him with a knife repeatedly in the chest, damaging his heart. Paramedics arrived very quickly and his life was saved by surgeons. Most of the stabbing wounds needed to left open because they have to heal from within toward the outside of the body. He sat in a lazyboy chair for weeks to allow the deep wounds to heal. A social worker came to visit, wanting to ask him to meet with the perpetrator in order to forgive him in an effort to rehabilitate him. This victim responded with tremendous anger, "You are lucky that I cannot move because I would do to you exactly what he did to me. Then we would see how forgiving you would be!" The impact of that visit was the topic of psychotherapy for several sessions. His rage had increased to such a degree that he had a pseudo-hallucination, luckily only after his stabbing wounds were healed. One day, he opened a closet and saw the perpetrator jumping at him from within the closet; he fell backward on the floor and physically fought with the hallucinated perpetrator as if he was really there. Suddenly, he realized that he was in his house, alone, and that he was fighting with no one. Before this experience, the practitioner was mostly able to simply provide affective support to this wounded man as well as validating his anger. After this pseudo-hallucination, the extent of his rage could be further worked through in psychotherapy, including the helplessness he experienced the night of this gratuitous murder attempt.

Amygdala and Hippocampus Over Time

The development of the amygdala and the hippocampus occurs differentially. At birth, the amygdala is completely functional, but the hippocampus has just started its development. The hippocampus will mature from birth to about four years of age. The clinical implication of

this reality is of outmost importance because it puts in perspective the necessity to assess the environment in which patients were raised to start having an idea about the hippocampal functionality.

Infants and toddlers living in highly emotional environments will develop high baseline arousal of the amygdala because emotional stress will have repeatedly stimulated the amygdala into higher and higher arousal. As we know, not only emotional stress is disruptive, but neglect is an even greater factor of stress, fostering an ongoing secretion of glucocorticoids.

When the activity of the amygdala has been heightened at a very young age, the development of the hippocampus has been hindered. As the amygdala's arousal increases, the burgeoning hippocampus is prevented from developing to its fullest. A highly activated amygdala can well hinder a developing hippocampus by secreting large amounts of neurotransmitters.

In psychotherapy, these individuals can be helped, but it will necessarily take time. Their condition is likely to be ameliorated by the use of an appropriate medication such as an SSRI, but they will also require a soothing relationship offered by the therapist. Psychotherapeutic work with such individuals will require supportive, anxiety-reducing interventions. Practitioners need to refrain from stimulating the amygdala of these patients, while fostering the hippocampal development by helping them to put things in perspective, by helping them to look at issues more objectively. A psychotherapist's quiet presence can be calming for these individuals. Calmly repeating their discourse can allow such patients to apprehend issues without heightened emotionality, thus fostering hippocampal development. Assisting them in practicing relaxation or meditation techniques can also be of help because they could develop a relaxation response which could be applied as an antagonistic response to their chronically high activation. However, oftentimes, very distressed individuals have a hard time practicing such techniques on their own because their anxiety is very high. Furthermore, the solo practice of such techniques involves self-activation and a sense of aloneness, which can trigger anxiety abandonment in individuals with attachment or self disorders. Gradually, yet slowly, the hippocampus of these overly activated individuals could be rendered more functional. In parallel to these interventions, psychotherapy also needs to focus on these patients' relational models to self and others because these are likely to be malevolent and anxiety-provoking.

Other patients with dysfunctional family react by presenting an exaggerated level of stoicism. They appear unemotional and detached from others, like individuals presenting with an avoidant insecure attachment pattern. In the scientific literature, there is another term for these people, they are called repressors. Repressors are known to report very little anxiety on subjective questionnaires, but their baseline level of glucocorticoids is much higher than normally. Although repressors can appear to be in control over their emotions and thus to have a normally activated amygdala, they do not show enough emotionality to infer a capacity for affect-regulation. Well-trained and experienced clinicians know to not infer that the outer presentation of these patients is not a reflection of their inner arousal. At the beginning of psychotherapy, emotionally-charged themes are avoided with such individuals. The

establishment of a safe and reliable therapeutic relationship is facilitated with the help of supportive interventions, affect-regulation techniques, problem solving, and a gradual interpretation of defenses against the experience of emotions. In my clinical experience, a specialized training in treating attachment disorders of disorders of the self is required (i.e., Masterson, 1989).

There are individual differences, of course. One could say that not all children from such stressful families will develop arousal difficulties such as hypersensitivity. Their inherited temperaments will provide the genetic basis determining the level of amygdala arousal at birth. Some individuals are born with a relatively poised temperament, the "easy children" as defined by Chess and Thomas (1986), while others are born with either anxious temperaments (sometimes identified as "slow-to-warm-up" or inhibited) or anger-prone temperaments (sometimes labeled as "difficult" or feisty). Anxious and angry temperaments are manifestations of inherited heightened arousal in specific areas of the amygdala: anger area and anxiety area. Due to their temperaments, they are easily thrown into high levels of arousal. With anxious or feisty temperaments, even when they are parented by competent and caring adults, these infants take much longer to become appeased when they are distressed than infants with an easy temperament. If these infants are born within dysfunctional environments, their amygdalas are likely to become activated at very high levels because the baseline arousal is already high at birth and, furthermore, dysfunctional parents do not provide appropriate soothing to their infants when they are in distress (Bowlby, 1988).

In sum, the first goals of psychotherapy with highly distressed PTSD patients who present with limited capacities to sooth themselves are as follows: to reduce their baseline anxiety; and to increase their self-soothing capacities. In this way, practitioners deactivate the amygdala and activate the functioning of the hippocampus, neurologically speaking. Many interventions can be helpful within the context of a supportive stance, therapeutically speaking. As the amygdala is deactivated, the hippocampus regains greater functionality and higher mental functions can kick into gear, which fosters further the hippocampal activity. These higher mental functions are involved in appraising situations, in planning as well as in arousal-modulation and self-soothing. These higher functions are located in the pre-frontal cortex, part of the cortical system.

Anterior Cingulate Cortex

The anterior cingulate cortex (ACC) appears to be particularly area involved in affect-modulation and affect-based behaviors (Bust et al., 2000). The anterior cingulate cortex was shown to be involved in modulating affects and emotional behaviors in mood disorders (Drevets et al, 2009). More specifically, the ACC is divided in two main parts, dorsal and ventral. The dorsal ACC is known to be involved in cognitive appraisal, while the ventral ACC is known to be involved in emotional inhibition (Etkin, Egner, & Kalisch, 2011).

Given the importance of having sufficient arousal-modulation during a traumatic event, PTSD is likely to develop in individuals who possess limited capacities for self-soothing. Indeed,

PTSD severity at intake was found to be associated with the thickness of the ventral ACC at intake (Dickie et al., 2011).

In psychotherapy, to gain a new perspective upon one's reaction to a traumatic event, there needs to be more than just cognitive work on the conceptualization of the traumatic event. Interventions also need to contain elements of compassion such as when we say "Given that you have endured much abusive authority as a kid from your mother, it is understandable that your emotional reactions during the hold-up has been as intense as it was and you refused to submit to the robber." The activation of the right pre-frontal cortex by empathic interventions is of outmost importance in psychotherapy for PTSD because it is the right pre-frontal cortex which is mostly responsible for self-soothing and thus de-activating the amygdala. The provision of only cognitive comments (here not to be understood as an intervention pertaining to cognitive therapy only) is likely to develop an intellectual understanding. The new cognitive understanding will remain unattached to the emotional component of the traumatic memory and, therefore, it is likely to become just another defense against experiencing the distress associated with the traumatic memory. Cognitive interventions such as cognitive restructuring, interpretations, or educational comments are likely to bear fruits if they are embedded within the greater scope of soothing, of providing a holding environment as suggested by Winnicott (1965) or an empathic and non-judgmental relationship as proposed by Rogers (1951).

Pre-Frontal Cortex

The pre-frontal cortex is highly sophisticated. Ontologically, it is the most recently developed area of the brain. Depending on the hemisphere in which it is located, the pre-frontal cortex performs different functions. The left pre-frontal cortex provides sophisticated functions such as language, analysis, and planning. The right pre-frontal cortex comprises functions such as arousal-modulation and representations of self and others (Schore, 2003; Preston & Eichenbaum, 2013).

The encoding of internal representations or schemas depends on the functioning of the pre-frontal cortex over time. Very young children experience the world in a more direct way than adults. In the first few years, they interact with their world from an experiential point of view. Over time, cognitive functions mature into a capacity for mental representations. Around the age of five, the information they have stored from cumulative experiences is organized into prototypical mental representations or schemas of self, others, and life. As a consequence, in any given situation, school-age children start appraising external reality according to their prototypical representations to decide a course of action. Children do not need fully experience every situation. If internal representations or schemas are strongly established and affect-laden, they often act as filters, letting in only the event-related information fitting their own information and keeping out any novel information. Such inflexible schemas are self-validating. They partake in the self-maintaining cycle of severe PTSD.

Affect-modulation is partly dependent on the functioning of the right pre-frontal cortex

(Schore, 2003). The development of this cortical area is in turn dependent on the quality of the relationships offered to infants by caregivers. If one has been soothed early in life, one has developed self-soothing capacities, arousal modulation capacities (Schore, 2003).

Let's examine the roles of the left and right pre-frontal cortex of a well-structured person during a traumatic event. During an armed robbery, for example, the left pre-frontal can tell individuals that a hold-up is happening, inform that it is required to remain calm because it is a dangerous situation, know that if one remains calm one has a greater chance to be remain alive and unhurt, and decide on an appropriate course of action given the numerous factors involved in this emotionally charged situation. One is likely to say to oneself, "Oh my God, I'm in danger. Let's see what to do." In this same situation, the soothing representations embedded in right pre-frontal cortex can allow to modulate the arousal elicited in the amygdala, now highly triggered by the individual being suddenly been confronted by an agitated individual brandishing a weapon. By modulating the arousal of the individual, the right pre-frontal cortex allows the individual to access the left pre-frontal functions capable of assessing the situation and providing a solution. Such mental work will lead to a reasonable course of action, such as giving the money rather than throwing it at the face of the robber or screaming out of fear. Furthermore, it is likely to prevent the occurrence of PTSD following such a traumatic event. Such cognitive mental work emanating from the left and right pre-frontal cortex will further deactivate the amygdala and contain its activation. An individual reacting in this way has received good-enough parenting and has succeeded in responding adaptively to difficult challenges in life.

Let's imagine that the pre-frontal cortex of an ill-structured person, whose internal representations are mostly of non-caring, aggressive, or helpless caregivers, and whose arousal is easily provoked and whose arousal-modulation capacities are limited. This individual's reaction to a hold-up is likely to be "Oh, my God, I will be killed!", and it would be automatic. By appraising a dangerous situation as a threat is likely to lead an individual to experience heightened arousal of the amygdala, and vice versa. Ideally, the arousal and emotional responses will not be overwhelming and will allow the individual to access higher mental functions and take appropriate actions. It is very adapted for individuals to be able to react internally with heightened arousal externally remaining composed and doing what's best to protect one's life.

So, even during an extremely traumatic event, if the arousal in the amygdala is not sufficiently modulated by the a well-developed right pre-frontal cortex, a person cannot quickly and strongly respond by using well-established functions, such as the capacity to integrate facts located in the hippocampus and the capacity to prepare an adapted response located in the left pre-frontal cortex. One is left with an overactive amygdala, interfering with both encoding and integration of the cognitive and emotional elements of the traumatic event. Such phenomena are likely to induce PTSD.

The highly sophisticated pre-frontal functions of individuals with PTSD are hampered by

a high baseline anxiety. This phenomenon is clearly demonstrated by studies in which PTSD sufferers' scores are lower on IQ tests than other groups. It is not because they are intrinsically less intelligent than others, but their performance on intelligence tests, as for any other stressful situation, is likely to be reduced due to a high baseline anxiety, which is in turn likely to be heightened in anxiety-provoking situations such as taking an IQ test.

Let's examine this PTSD manifestation in a young man presented with an extremely PTSD, along with co-morbid disorders (depression with suicidal ideations and precise plan, multiple daily panic attacks, full-blown agoraphobia countered by running everywhere, and weekly pseudo-epileptic convulsions as conversion disorder) presented with difficulties at completing his college courses successfully. His grades had fallen by forty percent after the occurrence of a potentially lethal assault during which he was attacked from behind. After this assault, he continued his life as if nothing had happened, including working at a convenience store to pay his student fees. At the store, an aggressive and sadistic hold-up happened, throwing him further into more overwhelming symptomatology, forcing him to consult. During the first year of psychotherapy, he was understandably worried about his future because his intellectual abilities had significantly reduced due to his high baseline anxiety interfering with his concentration and retention abilities. A conditioned response coupled with the projection of his immense anger also made him focus in class on every man in a classroom, rather than the lecture, because he expected that anyone of these men could assault him at any moment. He counted only on his intellectual abilities to get himself out of a low socio-economic condition. In psychotherapy, he was informed of the relation between anxiety and mental functioning and he was reassured about the permanence of his intellectual abilities. To do so, the practitioner needed to know about the neurobiology of PTSD. When his extremely severe PTSD was resolved, along with major depression, panic disorder and agoraphobia, his college grades went back up to A, which allowed him to be accepted by a school of medicine.

Clinical Implications

PTSD is a complex disorder and its treatment needs to be comprehensive, especially when PTSD symptoms are severe and accompanied by co-morbidity. Theoretical models and therapeutic methods are maps while the real terrain of clinical practice is always more complex. Neurobiology informs us of phenomena such as hypersensitivity and pre-frontal depletion in PTSD, which indicate the need to consider in psychotherapy the capacity for arousal-modulation in patients presenting with severe PTSD and co-morbid disorders. It is up to us, practitioners, to combine the neurobiological findings associated with PTSD and the clinical observations of the therapeutic complexities with PTSD. While the avoidant tendency embedded within PTSD calls practitioners to employ therapeutic methods involving an experiential approach of the traumatic material, neurobiological findings presented in this book suggest that some or many PTSD patients cannot engage in trauma-focused techniques without suffering further complications. As Horowitz (1984) suggested, psychotherapy for PTSD needs to always proceed at a tolerable dosage. If there are functional or structural deficits, clinicians attend to those before ever revising the traumatic event experientially, if ever.

In previous sections, arousal has been equated to anxiety, but arousal refers to specific neurophysiological reactions as well as associated dysphoric affects. Indeed, anxiety and dysphoric affects are produced by the brain center involved in survival, the amygdala. The kindling hypothesis suggests two phenomena reflecting the neurobiological processes involved in PTSD. First, individuals with PTSD are aroused, if not very arousal, in an ongoing fashion. Second, they have limited capacities for reducing anxiety whenever they are aroused beyond their elevated baseline. These phenomena have concrete clinical implications.

In psychotherapy for PTSD, practitioners need to assess the arousal level of PTSD patients. They need to consider the severity of symptoms, the degree of dysfunctionality reported, and the behaviors displayed in therapy. Clinicians cannot solely rely on verbal reports because patients with PTSD tend to disown their distress and minimize their internal destabilization. Patients' capacities to modulate anxiety and dysphoric affects, which are the phenomenological expressions of neurobiological arousal, need to be considered in therapy in terms of patients' reactivity to external situations as well as therapeutic interventions. Two major goals of psychotherapy for PTSD is to help patients to find ways to reduce baseline anxiety and to develop a greater capacity to modulate intense dysphoric affects.

As a wonderful supervisor told me years ago: "As long as people are in a panic, there can be no psychotherapy." They are many ways to help to reduce anxiety and to foster affect-modulation: taking medication, resolving external stressors, practicing a relaxation technique regularly, avoiding trauma-related stimuli, etc. Additional elements, such as unresolved conflicts, malevolent internal representations, and personality disorders, often contribute to heightened anxiety baseline and need to be addressed with patients presenting PTSD. Therefore, offering a safe relationship, developing a therapeutic alliance, and providing empathic statements and educational information as well as interpretations and

confrontations, are also required.

Enhancing on Coping Skills

Identifying coping skills employed by a traumatized individual prior and during a traumatic event is helpful because it informs indirectly about the person's functionality. Although some individuals may have been inwardly driven to run away or to assault the perpetrator during a hold-up, they were able to refrain themselves from doing so. Most likely, these individuals were able to successfully access inhibitory systems located in the pre-frontal cortex to appraise the situation, modulate affects and plan for adaptive actions. As these individuals can inhibit their reactive responses, they had time to figure out what was happening and chose a least-damaging course of action. In the midst of a traumatic event, a thoughtful response may be one of collaboration and not of confrontation, or vice versa. Often, victims just have few seconds to react.

For example, an older gentleman was attacked on the street. His first internal reaction was to run away, but he realized that he could not outrun the younger aggressor. Consequently, he froze, hoping to not activate the violence of the aggressor further. He heard himself plead for his life, which was in and of itself quite distressing for him. In the meantime, he kept repeating to himself "Think, think, think". While attempting to think about the best course of action, he assessed the non-verbal messages emanating from the aggressor and realized that the latter intended to kill him. So, the gentleman decided that he had to try to defend himself physically with all his strength. Upon this quick realization, he started to fight back. Fortunately, he was strong enough to overcome the assailant. Later on, when the policemen heard his story, they reinforced his decision to fight, commenting that this assailant was out to kill. For the next few days, this gentleman dealt with his emotional reactions and realizations on his own and in isolation given that he is an introvert, attempting to integrate the information embedded in the assault. Luckily, he had enough psychological strength to face his own vulnerability and mortality, realities which were forced upon him by the aggressor. This man did not develop PTSD. This story suggests that this man had higher mental capacities to inhibit the amygdala arousal during the assault, allowing him to reflect upon the situation and to decide which course of action was best in order to act purposefully and successfully. This man was rich in affect-modulation capacities.

In psychotherapy, practitioners benefit from assessing a person's impulse control and affect-modulation capacities in order to determine a most favorable course of therapy, along with the possibility of medication. Coping skills need to be identified at their worst and at their best in order to ascertain the person's regression under PTSD and progressive capacities toward a highest functioning. Detecting defense mechanisms employed by the person suggests ways of intervening, as dissociation requires different interventions than projective identification.

Lowering Anxiety Threshold

The hypothesis that PTSD is an arousal disorder informs us that we need to intervene firstly in a

fashion conducive to reduce arousal in patients with PTSD. Given that PTSD symptoms tend to augment in frequency and intensity as arousal increases, it is important to assist patients to lower their daily level of arousal. To do so, practitioners can utilize various techniques directly aimed at reducing baseline arousal, such as relaxation techniques and pharmacotherapy. Patients can also be encouraged to reduce their exposure to anxiety-provoking stressors and trauma-related stimuli. To reduce arousal, the therapeutic relationship can be a major asset and, in some personality-disordered patients, it can be the only one (this factor will be discussed later).

Stressors can be situations such as pressure at work or caring for children. Arousal-provoking products are items such as coffee, sodas, illicit drugs, etc. Returning where the traumatic event happened or visiting similar locations (such as a shopping center for a victim of hold-up) can provoke unnecessary arousal.

In research, PTSD arousal and reactivity symptoms (Cluster E in DSM-5) are usually viewed as expressions of heightened arousal. In other words, they are produced and maintained through neurobiological arousal. Consequently, relaxation techniques are recommended by some authors (e.g. Rabois et al., 2002) and pharmacotherapy by others (Ipser & Stein, 2012). However, it would be premature, if not misleading, to think that only behavioral or pharmacological interventions can serve the purpose of lowering arousal in PTSD.

The kindling hypothesis implies also that the arousal threshold for higher arousal will be lowered. Said differently, it will take less stimulation to activate the amygdala further into higher arousal. A hypersensitized brain will kick into increasingly higher levels of arousal over time if repeated and excessive stimuli are presented repeatedly, and the arousal baseline will also increase over time, leaving individuals more and more anxious.

An increase of baseline anxiety allows for a pernicious cycle of positive feedback to be set into motion. As baseline anxiety increases, the arousal threshold for anxiety is lowered and, the next time, less stimulation is required to provoke further arousal. In other words, the more anxious we become, the less stimulation it takes to trigger us into higher levels of anxiety. The more anxious we become, the more sensitive we are to any stressful situations. So, a hypersensitized person requires less stimulation for the amygdala to become activated.

This feedback loop explains, partially, why individuals end up developing PTSD after repeated exposure to traumatic events. This feedback loop also explains the daily reality of PTSD sufferers who cannot tolerate previously tolerated stressful situations. Indeed, it does not take much stress for individuals with severe PTSD to experience a large increase of anxiety. Many things bother them from now on, while did not affect them before PTSD developed.

Preventing and Decreasing Hypersensitivity

To reduce anxiety levels, many approaches can be undertaken. Traditionally, clinicians have mostly attempted to reduce anxiety with the use of relaxation techniques, while others focus

on the therapeutic relationship. Pharmacotherapy is also an option.

Selective serotonin reuptake inhibitors, SSRI, represent another effective way to reduce the baseline anxiety level. Its use is limited, however, because such medication has side effects in some individuals and medication does not treat PTSD per se although it is very useful in reducing the anxiety embedded in PTSD (Ipser & Stein, 2012). This intervention is particularly good when PTSD sufferers are so overwhelmed with anxiety that they cannot function or sleep properly. The reduction of their anxiety through the intake of a SSRI allows for the better functioning of their mental functions, which permits problem resolution, putting things in perspective, reaccessing loving feelings, practicing a relaxation technique effectively, etc. The more advanced mental functions. Furthermore, the intake of a SSRI during an extended period of time seems to allow for the development of new synaptic connections (i.e. Nasser et al., 1998), a long-term positive alteration of the brain, which counters the negative long-term alterations created by the kindling effect. If we dare to take a mechanical metaphor to explain the impact of the SSRI on the brain, we can think of a SSRI as a way to put oil within a motor to allow its parts to function well together.

Anxiety levels can also be reduced by altering stressors, that is, by identifying problems and implementing an effective solution. If a patient has an interactional problem with the claims adjuster, it is important to review with the patient what the problem seems to be, how the claims adjuster can be approached differently, and encourage the patient to implement such novel strategy to resolve this problem in order to modify the response of the claims adjuster; if the patient succeeds in altering such a problematic situation, it is a major gain because it alters a stressor and help the patient gain further self-confidence in regaining control toward the outer world, which counters the deep-seated sense of helplessness entrenched when PTSD is active. If the patient is truly overwhelmed by the situation, becoming in a panic or rageful state when it is addressed in therapy, then the psychotherapist might have to intervene directly in a modeling fashion which will likely reduce the stressors and foster the alliance, but such intervention should be implemented only when it is clear that the patient will only complicate matters further if he intervenes himself. For example, a patient with very severe PTSD and who has had a terrible relationship with an absent but domineering father could not even hear out what his male claims adjuster was saying to him, but would go into rage. The therapist called to explain the situation to the claims adjuster and see if the regular phone contacts could be postponed for a while. The claims adjuster first reacted, saying that he had to do his job, but then calmed down when the psychological status and limitations of the patient were explained with compassion. Because regular phone calls were necessary, a solution was found in that a female claims adjuster would enter the scene and call the patient, rather than her male counterpart, and so help not deteriorate the psychological condition of the patient further. Here we try to alter situations or conditions.

Another way of reducing anxiety level relies upon the identification of the situations provoking anxiety in the patient and helping the patient avoiding them. The reduction of anxiety is brought about indirectly by insuring that the patient will not get exposed unduly to trauma-related stimuli, or as rarely as possible. For example, if a patient has been victim of a

hold-up at a bank, the anxiety level is likely to be heightened severely through a classical conditioning association when this patient does grocery shopping and waits in line at the cash register line, knowing too well the possibility of being victim again of a hold-up whenever money is handled in a public place. The probability is extremely small, but such reasoning belongs to the rational mind or the left hemisphere, and is not substantial enough as a reality to organize the patient's experience at this moment of exposure to a trauma-related stimuli. The experience of the past hold-up is reactivated in a way that the patient knows "viscerally" that a hold-up could occur and start reacting neurobiologically almost as if it were occurring or re-occurring, which belongs to the amygdala, the center of emotionality in the limbic system. The emotional response overrides the rational one. So we cannot change the reality that to eat, one needs to do grocery shopping, but one can send a loved one to do the grocery shopping instead or can order food delivery from the grocery whenever possible. Here one does not try to change reality but try to withdraw or avoid anxiety-provoking situations which are unchangeable.

Functional analysis is very valuable in identifying anxiety-provoking situations. Whenever a patient's anxiety level has been heightened during the week, it is useful to identify with the patient the moment at which the anxiety was increased, the situation at hand before the anxiety increase, and the qualities of that situation which contributed to increase the anxiety level of the patient. It can be that the patient has watched a television program about the dramas at an emergency room during the previous week and it triggered a classical conditioning response of high anxiety. Or it can be that the father of the patient came to visit and the patient felt belittled again, developing resentment toward the father and having a long standing anger resurface, which provokes an activation of the limbic system and, if suppressed, can produce a high level of anxiety. Some patients also watch the television news every night, almost as if they want to challenge themselves and prove to themselves that they do everything as before, that they can face anything as before. However, they end up having a heightened level of anxiety and, consequently, have a hard time falling asleep and are likely to have nightmares.

As previously mentioned, anxiety can also be brought about by the simple occurrence of noises. In a study in a sleep laboratory, subjects with PTSD were randomly exposed to loud noises while they were sleeping, were subsequently awaken, and were asked about the content of their immediate dream. They were usually dreaming about the traumatic event which had led to their PTSD. The experimenter also woke up the PTSD subjects in a random fashion without the presentation of a loud noise, and the PTSD subjected then reported not dreaming about the PTSD-related traumatic event. As we all know, a loud noise will often times activate our nervous system into heightened. For PTSD sufferers, loud noises send them further than just a mere activation of their nervous system; they can be propelled in such a heightened level of anxiety that PTSD reexperiencing symptoms are triggered, which further activates their brains and bodies. The clinical implications of this study is that PTSD sufferers should be exposed to calm, non-noisy, conditions so that their nervous systems can deactivate slowly and, mostly, not be reactivated constantly by a loud environment. As the reader will remember, an exaggerated startle response to the exposure of sudden loud noises is an arousal PTSD

symptom. Psychotherapist will aim at reducing the occurrence of this arousal symptom in order reduce the overall level of anxiety of patients. For example, ideally, a parent of small children who is suffering from PTSD should receive help in handling the children so that the parent is less exposed to the kids' screaming or crying which would activate the parent's nervous system. If such patient were to gain access to babysitting from a loved one or have the opportunity to send few hours per day her young children to a nursery, her anxiety level could decrease, which is a neurobiological aim of this intervention, but her sense of worth would increase along with her sense of being loved and cared for, which are important psychological realities allowing the patient to feel less estranged from others, the world. Furthermore, for any PTSD sufferer, just having the television set at on ongoingly throughout the day would increase their arousal level, and it is noteworthy to remind ourselves that 25% of Americans keep their television set at on all day long. It can be unsettling to turn it off for some people at first, but the gains are likely to be substantial although this procedure is quite simple. Also it is now well known that television watching among very young children leads to a heightened probability of developing a problem of hyperactivity, and American children watch an average of four hours of television per day. Needless to say that the parents of those kids are listening to these four hours of noise emanating from the television set. Furthermore, beyond the noise level and the speed of the images and sounds of television, it is known that people who watch the news on television are in general more anxious and more depressed than those who abstain from this activity. Those are all good reasons to reduce television watching or listening for PTSD sufferers. Maybe, for those individuals who require noise in their house to not feel alone and not be thrown into the high distress sometimes associated with aloneness, putting on quiet music enjoyed by the patient could be a solution. Each patient can find a manageable solution or alternative.

To resolve stressors or avoid stressful situations seems to be pretty simple, but any experienced psychotherapist knows too well how difficult it can be for some persons to confront difficult situations or to acknowledge a limitation. The psychotherapeutic work must thus be directed at the inner reality of the patient, using an introspective approach such as humanistic or dynamic interventions, to help release the resistances of the patient toward resolving anxiety-provoking problems.

There can be a growing consensus that PTSD is more than an arousal disorder. Most likely, it is both an arousal disorder and a pathological manifestation of the inability to modulate arousal, which hypotheses go hand in hand when considered through the lens of the Yorkes-Dobson law. Viewing PTSD through both hypotheses comprises important clinical implications. Along helping individuals with PTSD to reduce their arousal, any practitioner will want to help them develop greater capacities of affect modulation. Such capacities entails reducing one's activation whenever it has been triggered, as well as containing affect intensification whenever arousal kicks in. The way I am phrasing the above therapeutic tasks, one could infer that the therapeutic solutions are necessarily and only technical, if not only behavioural. Such an inference would be fashionable and adapted to today's trends in psychology, but it would be excluding many powerful if not essential ingredients of efficacy, such as a safe relationship.

Acknowledging Vulnerability

Interestingly, patients with PTSD often deny being in psychological distress. Individuals may come in evaluation for psychotherapy after a traumatic event presenting with all PTSD symptoms along with severe co-morbidity, but they will deny any psychological destabilization. Obviously, before we can fully work on resolving the factors involved in their anxiety, it is necessary to help those PTSD patients go beyond the denial or minimization of their distress. One cannot work on something if one does not acknowledge it.

PTSD patients also regularly challenge themselves to overcome anxiety-provoking situations in which they put themselves intentionally. They need to prove to themselves that they are the same as they were before the traumatic event. Their environment usually supports this attitude, requiring them to be strong again. Evidently, these individuals with PTSD display personality features associated more with a narcissistic flavor than a borderline disposition.

For example, such individuals can present with severe PTSD, agoraphobia and panic attacks, but they will bring themselves to shopping malls despite their dread and the physical pain provoked by the elicited anxiety and they will seat in the middle of the mall asking themselves to stay until they calm down – the latter part of their self-imposed in vivo flooding 'treatment' is internally said with much anger and impatience. Naturally, after few minutes they will have a panic attack but they will keep on seating hoping that they will finally overcome their newly found vulnerability which is more intolerable psychologically than the experience of panic attacks. They can repeat this flooding treatment many times and cannot realize that it will not work this way; they have functioned in life using their willpower before the onset of PTSD and they expect to be able to continue to do so. At first, practitioners can help those individuals simply by instructing them about the neurobiology of PTSD and the long-term consequences of repeated activation. These patients can come to realize that they are damaging their brains and they will cease this exposure strategy to protect them. If the cessation of this unnecessary exposure were to be explained as their own sake, they would have a hard time stopping it because they tend to treat themselves harshly in life and paying attention to themselves in kind, compassionate way would be quite destabilizing internally. The latter intervention would be premature at the beginning of therapy but it will required later on to move in that therapeutic direction so patients can remodel their self-representations in a way more adapted to the human condition.

Considering the two neurobiological hypotheses derived from the kindling phenomenon, it is possible to see that PTSD sufferers are more fragile than other patients, which suggests that any treatment plan should take into consideration their fragility. Practitioners particularly need to intervene with any individual presenting with PTSD while considering *de facto* a neurobiological fragility beyond their discourse because their newly found fragility is at odds with their perception of themselves; they view themselves as stronger than they have become, too often forcing themselves to accomplish tasks previously easy for them such as doing grocery shopping. So often PTSD sufferers come in psychotherapy saying that everything is well and that they are doing fine and when they are asked, 'How are you

today?' To use psychoanalytical terms, their condition is ego dystonic and they employ denial or minimization.

After having assessed their PTSD symptomatology, practitioners can gently reflect an inferred psychological distress. Some patients can dismiss any empathic reflection of their inferred distress, but they may accept a reflection of their possible discomfort. If this intervention is not accepted, their minimization of their distress can be gently interpreted or they can be gently confronted about the dissonance between their usual sense of self ("Everything is fine.") and their trauma-related self ("I am overwhelmed by so many symptoms and am afraid of becoming crazy."). The impact of the last two interventions needs to be monitored closely in order to assess if patients were capable of modulating the anxiety provoked by them. Indeed, practitioners need to recognize the signs of the neurobiological fragility of patients, over and beyond their verbal reports pertaining to their anxiety. Obtaining information about their anxiety level and their activities throughout the day can also assist in assessing realistically their baseline level of anxiety and their capacity to calm themselves down.

A clinical example may illustrate well the implications of not assessing and respecting the patients' capacities to modulate arousal. A woman had a very serious car accident on her way to work few years ago. Although she was not hurt physically, she developed PTSD immediately after the occurrence of the traumatic event. The psychologist she consulted encouraged her to continue driving to work every day, which corresponds to one and a half hour each way, and to do so until the anxiety disappears completely. Viewing herself as a strong person, she undertook this assignment with resignation and drove to and fro work for three more years. Over time, her anxiety and PTSD symptoms worsened to the point that she became unable to perform her work and had to quit her job. She ended up staying at home, stewing in anxiety and trying to repair herself. One can quickly see the unnecessary losses involved in this situation. Obviously, the psychotherapist was counting on the process of habituation to kick in and most likely viewed her PTSD as a specific phobia rather than a structural disorder. Given the unduly exposure which worsened her anxiety rather than reducing it as the theory of classical conditioning would imply, her nervous system endured undue activation in a chronic manner, which worsened her symptomatology and dysfunctionality. This woman told me her story after one of my lectures on PTSD during which I addressed the necessity to cater to the neurobiological fragility of PTSD sufferers. With tears in her eyes and obviously shaken by hearing my statement, she said that every professional simply advised her to 'get back on the horse', which is a common advice to victims of accidents. Having done horseback riding for many years and jumped up to five feet with a horse, I knew what falling off a horse entailed. I told her that it is fine to get back on the horse if you only got a bruise and were temporarily shaken, but it would be the worst thing to do if your hip bone was broken because it would just become more damaged and healing would be prevented.

This example illustrates well how PTSD is structural disorder and not a specific phobia, and that simply confronting one's fear by exposure will not be beneficial. Such testimony is the sad proof that exposure, especially non-therapeutically controlled exposure, is not a panacea.

PTSD treatment requires a therapeutic approach which is multidimensional, possibly including a period of rest and avoidance after the occurrence of a traumatic event to avoid further activating an already overly stimulated nervous system. There is a fine line between therapeutic exposure and promoting hypersensitivity. Practitioners can develop a capacity to discriminate when each therapeutic strategy is applicable and likely to be beneficial.

Another traditional approach could also be destabilizing for PTSD patients. Catharsis or experiencing affects for the sake of experiencing them in the hope that they will consequently go away, is not effective and can also lead to hypersensitivity. It can be of importance to re-experience the emotions related to traumatic events, but only if they are experienced without overwhelming patients and in an attempt to consciously access the messages embedded in the intense dysphoric emotions. The newly found information about oneself and others can then be appraised and integrated within patients' greater scheme of mental representations. For example, fear can be the carrier of our inherent vulnerability as humans and anger can point to our denied helplessness at orchestrating major events in our lives.

Clinicians know that psychological factors are at play in both precipitating and maintaining PTSD. For example, one arousal symptom, namely hypervigilance, can be understood as a generalization of the anxiety response, but it can also be comprehended as a projection of unbearable anger. Indeed, is hypervigilance almost always disappears clinically when PTSD patients fully acknowledge and integrate their overwhelming anger and homicidal wish toward the perpetrator. As patients own these reactions in psychotherapy, I have witnessed, again and again, that they stop projecting their anger and homicidal wish onto strangers and, consequently, they cease to anticipate being assaulted by others. In turn, this resolved conflict allows them to calm down tremendously. Resick and Gerrol (1988) has found that PTSD severity is correlated to detachment, anger, guilt, confusion, humiliation, and betrayal before it is related to anxiety

Encouraging Prophylactic Avoidance

To reduce anxiety, PTSD sufferers often need to employ avoidance strategies, by reducing their exposure to trauma-related stimuli because it would activate their nervous system unnecessarily. As Horowitz (1984) recommended, practitioners should not entice PTSD patients to simply stop avoiding, but they should orient them to using avoidance strategies in a conscious and adapted manner.

For example, a bank teller who developed PTSD following an armed robbery at her work place, employed avoidance in a non-discriminative way. She stopped going grocery shopping due to the reactivation of her PTSD symptoms at the supermarket, and such avoidance was both protective and beneficial to her because it was within the confines of her life situation. But, she has also stopped going out of her house completely, which involved not seeing her friends anymore. Therapeutically, it was important that she resumed seeing her friends at her house because it provided her with needed affective support and a sense of belonging to life. Consequently, her anxiety level diminished and she was able to venture outside her house.

Another example further highlights the usefulness of prophylactic avoidance. A patient presenting with PTSD after a recent armed robbery had also been raped at 20 years old by a friend while she was drunk after a New Year's party. Since the rape, she had been reading at night before going to sleep those 'yellow' newspapers in which violent crimes are reported with graphic details and pictures. In psychotherapy, she complained of having nightmares every night, but made no connection with her self-imposed exposure to trauma-related cues. After investigation, the therapist realized that the patient exposed herself intentionally every night to sordid crimes stories in order to activate herself in the hope that she would remain aroused enough to see any new danger coming and possibly counter it. Naturally, the patient refused at first to cease this practice. An invitation to simply to do a trial cessation of exposure was finally accepted by the patient when the neurobiological consequences of her behavior was explained. That night, she slept well without nightmares. She was surprised and threw away all those newspaper. She avoided going out of her house unless she was obliged to do so, feeling more in control in her house than outside. This avoidance was detrimental to her social life which was now absent. As events can appear at first as negative can turn out to have beneficial effects, she was victim of a breaking-and-entering in her apartment while she was away. Now that her sense of security could not be associated anymore with her apartment, she contacted old friends and resumed having a social life.

Avoiding consciously and adaptively allows preventing an unproductive activation of the patients' nervous system, which unnecessarily fosters the emergence of more reexperiencing and hyperarousal symptoms of PTSD. In psychotherapy, it is a matter of finding a temporary balance between avoidance and approach. Practitioners aim at helping patients function at their maximum capacity without deteriorating their psychological condition through unnecessary exposure, repeated increases of anxiety, unnecessary reexperiencing symptoms, and consequent stimulation into hypersensitivity. Practitioners also aim at eliminating any avoidance which unnecessarily restricts patients' functioning and quality of life. Again such discrimination is learned through clinical experience when trials-and-errors are guided by an understanding of the neurobiology of PTSD.

Fostering Experiences of Control

Anxiety can also be provoked by a sense of lack of control, environmental isolation, interpersonal conflicts, etc. High levels of arousal can thus be reduced by helping PTSD patients regain control over their inner and outer worlds, which includes reaching out to others for social and affective support.

Anxiety levels are intimately linked with a sense of control (Chorpita & Barlow, 1998). Therapeutic interventions for PTSD need to aim at helping patients regain control over themselves and their lives, that is, over their inner and outer worlds. Chorpita and Barlow (1998) demonstrate how the experience of a lack of control in childhood leads to a sustained sense of helplessness and anxiety. In one remarkable study reported by them, some young primates were provided with an ongoing access of water and food during the day, which led

them to better perform on problem solving tasks in comparison to their counterparts which were subjected to random access to water and food, representing a complete lack of control over one's basic needs (those primates achieved tasks least successfully), or a rigid scheduled access to water and food, representing a middle ground of controllability through predictability (those primates completed tasks better than those deprived of any control, but less well than those having complete control over their basic needs).

A factor influencing post-traumatic responses consists of our past experiences. Different elements of any experiences are recorded in the pre-frontal cortex, the hippocampus, and the amygdala. During a traumatic event, internal representations with elements in these three areas are available for comparison with the situation at hand. As the brain works through associations, the affects evoked by traumatic events trigger memories already embedded in the amygdala charged with the same affects.

A major aspect of past experiences consists of whether they entailed helplessness or mastery. If individuals have has successfully experienced a variety of challenging situations in the past and succeeded in obtaining a desirable outcome, they may be in good shape to encounter a traumatic situation successfully. For example, if individuals experienced past experiences encompassing helplessness but also an acceptable outcome, they might spontaneously retrieve that mnemonic information of mastery and use it to access anew a sense of control during the traumatic event, which would guide their course of action. Expecting a successful outcome, these individuals' capacities for affect-modulation are likely to kick into gear during a threatening situation and thus contain the activation of the amygdala (Chorpita and Barlow, 1998). Ideally, memories of past successful reactions comprise a diversity of actions, from the capacity to fight for one's life to the capacity to surrender whenever an action would increase damages; the path of least resistance may be the way at times.

In contrast, if individuals have experienced intensely charged moments of helplessness in the past, these associated affects are likely to be reactivated during a traumatic event. Consequently, they are likely to spontaneously access those memories of helplessness, interfering with their appraising and planning potential, which will activate the amygdala further. For example, individuals who have intensely experienced helplessness at the hands of a parental authority figure are likely to develop PTSD after the occurrence of only one traumatic event if they were to be rendered utterly helpless during the event.

One's reaction to a traumatic event can also be determined according to one's actual life situation. Some horrible situations "hit home" more than others. For example, medical professionals will at one point encounter situations in which they will witness wounds inflicted to a young child; those who are most likely to develop PTSD are those who have young children, especially if the wounded child presents characteristics similar to those of their children.

Nonetheless, many individuals have gone through intensely traumatic experiences, and they have not developed PTSD in their wake. Some humans even seek intense life-challenging experiences. Certain individuals react to traumatic as challenges and seem to grow

psychologically from them.

Finally, the presence of an adequate social support provides a corrective experience to the individual in contrast to the traumatic experience, favors a remission of symptoms through providing a sense of security due to an experience of togetherness which counters the profound loneliness suffered by people with PTSD, and reactivates an impression of control over one's environment which has been found to lessen one's stress response.

Providing a Therapeutic Relationship

Patients with PTSD often have difficulty trusting other people and developing effective interpersonal relationships, such as the veterans in the study conducted by Okey et al. (2000) who longed for closeness while fearing rejection, and who tended to lash out at others as a result. Psychodynamic psychotherapy offers these patients a chance to develop awareness of their interpersonal patterns, experience a different type of relationship, and make important changes in their interpersonal functioning (in Schottenbauer, 2008).

At an interpersonal level, the provision of a safe relationship also contributes to lowering anxiety. In psychotherapy, the therapeutic relationship has been found repeatedly to be the best predictor of outcome, after pre-treatment symptomatology, across mental disorders, including PTSD (Horowitz et al., 1984) and across therapeutic modalities (Gaston et al., 1991; Gaston et al., 1995; Gaston et al., 1998), even in pharmacotherapy (Weiss et al., 1997).

Beyond any technique, providing a safe relationship to a human being is about the best anxiety reducer there can be. For example, we know kids with attachment disorders present much higher physiological activation upon the leaving of their mothers, than kids presenting with secure attachment patterns. Bowlby has accurately labelled those attachment disorders as insecure, highlighting the associated anxiety, although the anxiety is not always experienced consciously, and at times not at all. Individuals presenting with the most severe attachment disorders, avoidant insecure attachment disorders, are those known to react with the highest levels of anxiety upon separation from an attachment figure and to present as a baseline the highest levels of cortisol, although they report experiencing very little if no anxiety (these are called "repressors"). The therapeutic alliance has been shown to be the best predictor of psychotherapy outcome, along with pre-treatment symptom severity (Gaston et al., 1998).

If we would be inclined to then proclaim that providing a safe relationship would therefore be sufficient, we would be incorrect in my opinion, restricting ourselves and our efficacy. Although it is a necessary start, there is much to do beyond providing a safe relationship to a patient, and for many reasons. First, the provision itself of a safe relationship does not entail that the patient will experience the offered relationship as safe. Individuals with insecure attachment disorders do not experience their relationship with anyone, including us, as safe. With those individuals presenting with PTSD, practitioners need to help them develop such a capacity to experience a safe and secure connection to others, in order to

reduce their baseline anxiety and help them develop a greater capacity for affect modulation.

To do so, practitioners need to provide an attuned and contingent response to the emotional needs of patients. Schore (2003) has shown that, by responding to their infant's needs, secure mothers foster the development of a neurobiological structure responsible for affect modulation, as observed in securely attached infants. This structure involves a highly active pathway of connections between the right pre-frontal cortex and the amygdala. The right pre-frontal cortex is responsible for affect modulation, or self-soothing, through its connection with the amygdala in the limbic system, the brain center responsible for affects and drives. The development of a strong neuronal activity within the right pre-frontal cortex and its functional association with the amygdala is completely dependent upon experience.

A capacity for self-soothing is not genetically programmed to spontaneously mature at one point in time. One's capacity for affect modulation is completely based on the contingent and attuned nurturing received by the infant from a parental figure (ideally the mother), except for the blueprint that is present in the brain and be used to be this tremendous highway of connectivity which affords affect modulation to an individual. Knowing how the brain is plastic, we can reasonably infer that the provision of an attuned and safe relationship by a psychotherapist to a patient can help reduce the baseline anxiety of this patient and can help develop a capacity for self-soothing within that individual, but only in the long term.

Good-enough psychotherapists are emotionally attuned to their patients and available to their patients' needs, not to conscious and defensive needs but to needs of the real self. As the traumatized self is embedded within the real self, it can be seen and responded by an attuned psychotherapist. Beyond pre-treatment symptom severity, the therapeutic alliance has been found to be the major predictor of psychotherapy outcome (Gaston, 1995), including for PTSD (Horowitz et al., 1984) and pharmacotherapy (Weiss et al., 1998).

Fostering a Secure Attachment

Persons with insecure attachment find it difficult to utilize relationships as a source of emotion regulation, which complicates their efforts to cope after traumatization (Allen, 2001; Allen et al., 2001). Dieperink and colleagues (2001) caution that it is important to differentiate symptoms related to trauma from those inherent in the individual's attachment style and to focus on interventions that may improve the working alliance of individuals who have insecure attachment. Attachment style has been shown to relate to the quality of the therapeutic alliance (Diamond, Stovall–McClough, Clarkin & Levy, 2003) and the outcome of psychotherapy (Fonagy et al., 1996) (in Schottenbauer, 2008).

With regard to attachment, research on psychodynamic psychotherapy has shown that client attachment style changes through therapy. Specifically, this research has shown a shift from insecure to secure attachment states of mind, and from less secure (e.g., unresolved or insecure) to more secure (e.g., cannot classify or mixed) attachment states of mind on the Adult Attachment Interview after object–relational psychotherapy (Diamond et

al., 1999; Diamond, Clarkin, et al., 2003; Diamond, Stovall–McClough, Clarkin, & Levy, 2003; Levy, Clarkin,&Kernberg, 2004). This finding is notable, since most studies have found attachment to be relatively stable over time (e.g. Levy, Blatt, & Shaver, 1998; Main, Kaplan, & Cassidy, 1985) (in Schottenbauer, 2008).

How are mothers with an insecure attachment pattern behaving? They demonstrate soothing behaviors when her infant is in distress, behaviorally and emotionally. They are attentive to auditory signs of distress in their infant, respond immediately whenever the infant indicates distress through vocal signals, and pick up the child while talking in an empathic and responsive tone of voice. They indicate that they are emotionally attuned to the expressed distress, but they also remain calm. They gradually lower the emotional pitch in their voice and verify along the way if the infant's expressed distress follows this reduction; if not, these mothers match up their voice again closer to the distressed tone of their infant, but at a lesser degree to also communicate calmness. In this way, they modulate their infants' affects. The mothers' response needs to be enacted behaviorally and emotionally.

Studies have shown that such mother lower their vocal pitch gradually and, if the child's distress signals are lessened, they will continue to lower her vocal pitch of distress, demonstrating to the infant their emotional attunement and soothing the infant (Siegel, 1999). It is not the words which are important, but the music in the mother's voice. The mother's voice cannot be neutral to be effective in soothing her infant and, conversely, the mother cannot indicate in her voice that she is herself in distress. A study on the vocal pitch of various types of mothers responding to their distressed infants has shown that the soothing mothers produced a vocal pitch exactly half way between neutrality and the distress level of their infants; these mothers indicated vocally that they were emotionally attuned to their infants' distress while they were also not overwhelmed by such distress.

It is as if the pre-frontal cortex of the mother is influencing the pre-frontal cortex of the infant. If every time or almost an infant is in distress, his mother reacts in such a caring and unconditional way, three major learning experiences happen. Firstly, the infant's amygdala will not be activated at high levels of distress in a regular fashion, thus leveling the ceiling of activation in the amygdala. Secondly, the child learns that others will respond if he needs which creates a securing permanent expectation in the child. Thirdly, the child develops his own capacity for self-soothing, located in his now well-developed right pre-frontal cortex which can now become activated as soon as the amygdala is triggered into activation. Thus, the development of a soothing function in the right pre-frontal cortex is environmentally based and thus not hard-wired at birth (Schore, 2003).

In psychotherapy, to develop a capacity for self- soothing in patients, psychotherapists need to be emotionally attuned to their patients, employ discrete vocal strategies to indicate their attunement and to induce the gradual deactivation of the amygdala whenever patients become unnecessarily distressed. Because psychotherapists usually intervene with individuals, children or adults, who can understand language, the choice of words will be important to reflect cognitively the patients' emotional state, but the quality of the voice will also influence

the development of a soothing mental function. Psychotherapists need to endorse an attuned and caring attitude which will be reflected in the choice of their words and the tone of their voices whenever patients present with limited affect modulation capacities.

Psychotherapy involves creating new memories. There is now considerable evidence that new memories are not simply transferred to the cortex, but rather assimilated into neocortical memory networks called schemas (or representations of self and others; Bowlby, 1988). To do so, neural pathways or structures need to be modified and elaborated (Preston & Eichenbaum, 2013).

While new memories are initially represented within the hippocampus and other areas, during the course of consolidation, they become interwoven into a neocortical network of already-existing related memories. This process requires the modification of the pre-existing neural structures to add the new memories McClellan et al. (1995). As psychotherapy unfolds, one gains access to higher mental functions and new memories are formed, replacing trauma-based memories.

In psychotherapy, when practitioners help patients to put things in perspective with a calm tone of voice, even if it is simply reminding them of the place and time of the traumatic event such as “You experienced that hold-up three months ago, at the convenience store where you work”, they invite patients to reflect on their experience, to become observers. With the use of such interactive moments, practitioners may help deactivate the amygdala and increase the hippocampal functioning of patients. When practitioners restructure the understanding of a traumatic event, they entice the left pre-frontal cortex to come into play to make new cognitive associations with the emotional memory, such as “It was a terrible thing that happened to you and you did the best you could under such circumstances” in contrast to what patients say about themselves such as “I did nothing. I was such a jerk!” The tone of voice associated with interventions is central to foster a soothing impact.

Reappraising Beliefs

The traditional model of Beck and Emery (1985) was developed to explain anxiety disorders. It contains propositions which could at times be applied to PTSD, but within limits. First, fear reactions emanate from the appraisal of a threat. Second, this threat appraisal involves the activation of a pre-existing cognitive schema of fear, so it involves an activation of a consistent self-reinforcing information structure, a pathological one because it is unfitting with reality. Third, this pre-existing schema leads to both a selective attention focusing on the evidence which is consistent with the fear schema and a selective inattention neglecting to consider the evidence that is inconsistent. Fourth, any ambiguous information about a possible threat brings the person's attention toward obtaining further evidence of threat, hereby creating a positive feedback loop between anxiety and attended stimuli. Finally, this feedback loop triggers eventually a fear response of escape and avoidance. In research, anxious subjects have been shown to have an attentional bias toward threat cues.

This cognitive model of anxiety was developed for anxiety disorders such as specific phobias, panic attacks, social phobias, generalized anxiety. In PTSD, traumatic events reinforce at times cognitive schemas of fear, but the main problem with this cognitive model is that fears subsequent to traumatic events are based on reality and not on fantasies. Therefore, the anxious symptoms of individuals with PTSD are not far away from a possible reality, a reality they have experienced.

Epstein (1991) proposed a cognitive-experiential self-theory for PTSD. This model appears, in my experience, to be better suited to understand and treat PTSD In its entirety. In this model, everyone constructs a personal theory about the self and the world. Each personal theory is developed through an interaction between experiencing and conceptualizing. If everything goes well, through a reiterative process of assimilation and accommodation, one's conceptual system (personal theory) becomes more and more differentiated (complex) and integrated (association between its diverse elements). If the self-theory is unable to fulfil its functions under stress, anxiety arises and disorganization ensues if coping fails.

Epstein proposed that one's personal theory performs four functions, each associated with positive and negative beliefs. The first function entails maintaining a favorable pain-pleasure balance (as in Freud's model), with beliefs about the benevolence vs. malevolence of the world. The second function entails maintaining a coherent theory of the self and the world (as in Rogers' model), with beliefs about the world is meaningful (predictable, controllable, just) vs. meaningless (capricious, uncontrollable, and unjust). The third function entails maintaining self-esteem (as in Adler's and Allport's models), with belief about the self as worthy (competent, lovable, good, powerful, and attractive) vs. unworthy (inadequate, unlovable, bad, helpless, and unattractive). Let's note here that these first three functions with associated beliefs are derived from Janoff-Bulman's model (1989). The fourth function entails maintaining relatedness (as in Bowlby's model), with belief about people as trustworthy and worthy relating to vs. untrustworthy and unworthy relating to.

Most importantly, Epstein's model stipulates that PTSD arises when one is unable to carry out one of these functions. The personal theory then shifts in the negative zone at a preconscious level. In PTSD, most of these basic functions and associated beliefs are under assault, thus forcing a substantial shift into the negative at a preconscious level and, consequently, requiring a reformulation of one's basic views about the self and the world.

As suggested by some cognitive-behavioral therapists, is it sufficient that the more sophisticated neurobiological structures, such as the prefrontal cortex and the anterior cingulate cortex, are made by techniques to inhibit trauma-based emotional responses for treating PTSD? Clinically, the answer appears to be no even though some techniques appear to be successful at reducing PTSD by temporarily inhibiting emotional responses (Gaston, 2016). However, inhibiting is at the opposite end of the spectrum from making conscious. Research has shown that dynamic interpretations accurately reflecting patients' unconscious beliefs are related to significant improvements at termination and follow–up (Norville, Sampson, & Weiss, 1996; in Schottenbaeur, 2008). Dynamic interpretations entail making conscious and holding

the emotional pain associated with the belief in order to get its embedded message and integrate it. Facing reality also involves learning to bear the difficult aspects of life which cannot be changed.

In psychotherapy for PTSD, considering the functions outlined by Epstein is very useful when the issue is at center stage. Clinicians can aim at increasing a patient's pleasure, especially with trustworthy others, in order to rebalance the world view toward less malevolent and more benevolent beliefs. Clinicians can also ascertain that psychotherapy is not only about painful issues and so foster pleasant affects in therapy. To help someone regain a sense of meaningfulness in life, practitioners can identify problematic issues in the person's life and assist in resolving them, which enhances a sense of the world as controllable and predictable and thus decreases feelings of helplessness. To assist a person in countering a negative sense of self, practitioners may wish relate to this person with a positive regard and unconditional acceptance, along with fostering favorable relationships outside of therapy. Practitioners can help a person with PTSD differentiate between people who are trustworthy versus untrustworthy ones, but they need to be themselves worthy of trust first and foremost. Practitioners can also have to allow and foster the development of a secure attachment in the person toward them, if the person comes in therapy with insecure attachments.

As Epstein underscored, human beings can enjoy life but within limits in this world, they can have some control over events but only within limits, they can feel good about oneself but within limits given that our shadow can recede but does not go away, and they can depend on and trust others but only so much. In therapy, identifying whether a person has been operating from any of these negative beliefs since the occurrence of traumatic event can have a tremendous clinical impact if clinicians foster experiences countering these beliefs. Altering cognitions or beliefs is not sufficient to resolve PTSD. Patients need to work through their diverse dysphoric emotions (anger, disgust, shame, etc.) and states (hopelessness, helplessness, etc.) associated with the above prominent negative beliefs, beyond their face value, in order to resolve PTSD at its core.

Working Through Emotions

Treating PTSD by solely focusing on fear responses could be misleading as many more emotional responses are induced by a traumatic event. Fear is only one of many affective responses potentially involved in PTSD. Any comprehensive treatment for PTSD needs to consider all emotional reactions triggered by a traumatic event in a given individual, conscious and non-conscious emotions.

All dysphoric emotions can be triggered by a traumatic event. They are called dysphoric or negative given the suffering associated with the. To fully understand the emotional responses, therapists need to obtain a detailed description of the traumatic event per se, of course, but they also need to know the details of the situation preceding and succeeding the event. By doing so, clinicians can identify the emotions potentially experienced, consciously and non-consciously, as well as the reactions of the person.

The emotions usually activated by a traumatic event are the following: anger, rage (anger mixed with helplessness), shame, disgust, guilt, fear, and sadness. In PTSD, the main problematic states of consciousness involve helplessness, vulnerability, detachment, and confusion (the latter two are defense mechanisms). In a sample of 256 victims of rape and theft, Resick Gerrol (1988) found an association between PTSD severity and diverse emotions and states, one month after the occurrence of the traumatic event: detachment (dissociation), anger, guilt, confusion, humiliation, sense of betrayal and anxiety. Note that fear is not included in the problematic responses.

In his theory of emotions, Tomkins identified many types of dysphoric emotions: fear, sadness, anger, disgust, and shame (Nathanson, 1994). This theory based on observation and experimentation, highlights how our emotions act as amplifiers of meanings, indicating that something significant has happened to us. Any emotion carries a particular message. To grasp the meanings embedded in an emotion, we need to live the emotion fully, without becoming overwhelmed or inhibiting it by fear of becoming overwhelmed.

Anger

Anger is an emotion often triggered during a traumatic event, but it is usually kept in check, controlled, in order to ensure survival. Afterwards, anger becomes problematic can be either over-controlled or under-controlled.

Over-controlled anger is present whenever the victim does not feel anger at the perpetrator or the person responsible of the event. One's anger is inhibition and rejected, even though such anger would be more than reasonable given that the person has developed a deep wound consequent to the actions of this person, namely a PTSD. In such circumstance, the victim is worried by the magnitude of the anger, possibly associated with murderous and/or sadistic wishes.

Anger-based desires are usually repressed and then projected onto others. Beyond hypervigilance, the person can develop panic attacks and agoraphobia based on repressed anger. Sometimes, the person even develops pseudo-hallucinations of persecution. This immense anger desires can also be somatized, attacking the body of the person instead of others, such as Crohn's disease or pseudo-epileptic convulsions, severe swelling, or paralysis. In my clinical experience, these somatic manifestations should be interpreted; they disappear when the deep=seated psychological wound and anger are fully recognized by the person.

In psychotherapy, over-controlled anger can be addressed in many ways in order to its recognition, emergence, and transformation. Clinicians can educate about anger: "We have not learned to differentiate between anger and aggression. Research has shown that these two reactions are not linked at all. Anger is an emotion informing that one has been violated or our expectations were inadequate to the situation. In contrast, aggression is a behavior by which a

person attempts to gain an advantage and control over another person. Anger is a legitimate emotional reaction to a gratuitous aggression.'' Validation can also be employed: "Given what happened to you, I would understand if you were anger toward this person". Importantly, clinicians need to emphasize that anger does not make one similar as perpetrators of gratuitous violence. Self-disclosure is useful to indicate that such anger and related wishes are not illegitimate and do not transform one into a monster: "By seeing victims in therapy and hearing about such gratuitous aggressions, I sometimes feel a strong desire to kill one aggressor or another, but I do not do it. So people can harbor such feelings without acting upon them." Clinicians can also use gentle confrontations, such as therapeutic astonishment, to help the person recognize anger: "I do know that you report having no anger whatsoever toward the perpetrator, but the contemptuous words you employ reveal the contrary." Interpretations are valuable tools to bring anger to consciousness: "I understand that you report having no anger because you may have important worries about being angry." Most importantly, clinicians need to contain their countertransference. In a counter-transferential reaction, therapists can become angry at perpetrators due to their own issues, robbing victims of their own anger. Yet, therapists can also become afraid, consciously or unconsciously, of victims' anger and wish to aggress, hurt, or kill. Victims can also employ projective identification to disown their anger and murderous wishes; they reject them and project them in therapists, rendering therapists susceptible to experiencing these reactions as if they belong to them and not the person in front of them. Such unconscious strategy of projective identification is always helpful if therapists can recognize that these reactions do not belong to them but the patient. Projective identification is often employed by the psyche of individuals with PTSD, which provides a window for therapists into the inner realities of the patient.

For patients, the goal is to fully recognize their anger and associated impulses/wishes, while maintaining a benevolent attitude toward oneself. In this way, the unconscious free-floating anger ceases to act on its own and the person embraces the deep psychological wound provoked by the perpetrator. The ultimate goal is to go beyond such anger because it constitutes a link with the perpetrator, keeping this 'figure' alive in the psyche of the victim. The goal is let go of anger toward the perpetrator, after fully recognizing and living it. Only then can anger give way to the deep-seated sadness of having been so hurt.

Forgiving can then emerge naturally, of its own momentum. Forgiving is more about letting go of anger than about forgetting the wrongs done to us. Pretending that nothing happened and the perpetrator is not responsible is a fallacy. The latter is naïve. Otherwise, forgiving is just another defensive strategy employed to cover up anger and murderous wishes, just as resentment is s defensive mechanism against suffering. A victim once said: "It is not to me to forgive; this is God's job."

Under-controlled anger is usually basis on an inherent tendency of the person to react with anger. Let's remember that Pavlov's dogs reacted to aversion with either fear or anger. Beyond a temperamental disposition, an emphasis on anger expression can also be a defensive maneuver to cover-up a sense of vulnerability and helplessness. Such under-controlled is mostly encountered in men, but it can be present in women.

In psychotherapy, under-controlled anger needs to be addressed in various ways. First, such anger requires recognition and validation: “I understand that you are angry given what happened to you.” An angry person is not always conscious of being angry, of carrying anger inside. Therefore, it is important for therapists to reflect the presence of angry feelings when individuals raise their voice, emit a wishful threat toward a person, engage in contemptuous attitudes, etc. Second, anger needs to be linked to unwanted consequences, actual or possible. Therapists need to inquire about negative consequences provoked by the under-controlled anger. The consequences of angry outbursts need to be acknowledged and clearly identified, before offering any solution, in order to assist the person to become an observer rather than be overtaken by anger. Third, individuals have to choose consciously as to whether they truly wish to express anger in an under-controlled way, usually a destructive way, or not. After becoming conscious, people are usually saddened by the impact of their anger on others and themselves. Fourth, victims can be encouraged to find ways to express their disagreement or anger in non-destructive ways: namely, to focus one’s attention on recognizing the presence of anger, to remind oneself on the spot that verbal or physical violence is not a response leading to a satisfactory outcome, to leave the scene by exiting behaviorally (it is not about clamming up inside oneself), to focus on attention on breathing deeply and slowly, to remind oneself that no much can be done anyway to rectify the situation at this very moment (it is better to come back on this issue, if ever, when one is calm), to avoid paying attention to anything stimulating one’s anger, to even resort to tell oneself “This is not so important”, etc. Fifth, clinicians need to bring the person’s attention onto what lies behind their anger. As anger is a protective response, therapists can interpret the underlying vulnerability, gently and empathically. The goal is again to go beyond the anger response to reach and acknowledge the deep-seated psychological pain.

Most importantly, clinicians need to acknowledge and contain their reactions toward the patient’s anger, namely, their countertransference. Therapists can afraid of such angry and thus afraid of patients with PTSD. A therapist may desire that patients subdue their anger because such anger attached with murderous wishes, which unsettles the therapist. If this is the case, therapists should not be working with victims; they need to resolve their own issues first and foremost. Practitioners should be comfortable-enough with the possibility that they can also be victimized in life as it can occur to anyone, and thus contain any unwarranted fears of being assaulted by patients with PTSD. Practitioners should also be comfortable-enough with their potential for victimizing others and remain benevolent toward themselves in this regard.

Needless to say, therapists should be confident in their capacity to differentiate someone’s real intention to assault versus someone’s expression of anger or distress. One way is to be aware of one’s own capacity for intense anger and one’s own murderous potential; if therapists are not afraid of their own, they should be afraid of such potential in their patients. In a preventive mode, clinicians should always sit near the door, allowing their exit to not be blocked by an overwhelmed patient. It remains though that dangerosity requires at times a quick assessment. Usually, the presence of danger can be ascertained by an unwavering crescendo of anger, unabated by empathic comments. In such circumstances, therapists would be well-advised to simply say that they need to leave the premises for a moment. If left alone a

person becomes violent toward objects, emergency services need to be called. If a physical aggression cannot be avoided, please defend yourself.

Shame

Shame is an emotion aimed at severing one's attentional focus from one's action. Shame is associated with a large amount of blood brought to the brain, explaining why one's face becomes red when we experience shame. This amount of blood temporarily overwhelms and thus limits one's higher mental functions (Nathanson, 1992). Shame is usually experienced with a sense of having done the wrong thing, of being at fault. Usually, the person withdraws within oneself, severing the relating with others.

In psychotherapy, shame can be identified and validated as a legitimate response: "I understand that you feel shame under such circumstances; almost all of us would do." However, such interventions are limited in their efficacy. Shame is powerful and brings a sense of self as being abject for others. Consequently, shame can only be appeased within the context of a relationship in which the other person, hereby the therapists, views the person as valuable, offering empathy and positive regard. In this way, the person experiences deserving benevolent attention. Through experience of another's benevolence, the impression of being rejectable can be dismissed by a counter-experience.

Disgust

Disgust is an emotion often neglected in psychotherapy. Almost all of our theories dismiss it. First, it is important to differentiate disgust from contempt, the latter being an attitude which entails a negative evaluation and a subsequent rejection. Physically, disgust offers very significant information by indicating that something or someone is harmful for the organism and needs to be expulsed or not incorporated. Psychologically, disgust indicates that something needs to be expulsed from within or should not be internalized. Usually, the thing to be expulsed is oftentimes a negative parental figure. Thus, such things to be rejected can be hurtful words, a condescending attitude, the seminal liquid left by a rapist, etc. Even by conveying a psychological reality, disgust can be experienced physically with nausea.

Usually, individuals are unsettled when they experience disgust toward someone. Yet, disgust is very useful because it indicates that this relationship is harmful and should not be continued; this relationship contaminates the person with negative attributes.

A person can always cleanse oneself of malevolent attitudes or internalized figures, cognitively and emotionally. At times, negative internalized objects remain alive because one agrees with the beliefs conveyed by them. At other times, they remain alive inwardly because the person clings onto an insecure attachment to a damaging figure. The former can be worked cognitively, but the latter requires long-term psychotherapy providing a secure attachment with a benevolent therapist.

Disgust can also operate as defense mechanism, as any other emotion. Sometimes, disgust protects oneself from getting closer to someone, by fear of being hurt. Therefore, therapists should be vigilant to distinguish the role played by disgust in any context.

Guilt

Guilt is a feeling, more than an emotion, based on the impression of having done something bad or, at best, a mistake. Guilt also involves psychologically attacking oneself. Such attack emerges from internalized malevolent figures. In PTSD, guilt feelings are almost always a defense mechanism against helplessness.

A traumatized person will usually prefer to having done a wrong thing because such contention implies that the right thing could have been performed. The right thing usually entails having prevented an assault, robbery, a death, etc. The psychological maneuver of guilt prevents the person to recognize one's sheer helplessness; nothing could have been done to prevent the traumatic event from occurring. Unless one is truly responsible for the event, the only power a victim has during a traumatic event is to attempt to reduce any damages, if ever possible. Hindsight reinforces guilt, but prevents the experience and recognition of one's helplessness in life.

In therapy, many techniques can assist in undoing feelings of guilt. First, therapists can inquire about the reality basis for one's sense of responsibility regarding the unfolding of the traumatic event. Such information is necessarily for latter interventions. After ascertaining for themselves that guilt is only feeling and not a valuable sense of responsibility, clinicians can demonstrate therapeutic astonishment at the guilt feelings harbored by PTSD patients. Then, these feelings can be contrasted cognitively with the absence of evidence regarding their basis. Therapists need to underscore that the person has accomplished during the event what could be done given the circumstances. It can also be useful to inquire what the person would have wished to do, instead of what was done, and examine the consequences of desired actions (for example, during a robbery, throwing the money in the face of a thief is likely to have worse consequences than giving the money).

Practitioners can proceed to interpret the guilt feelings as a defense against helplessness: "I understand you feel guilty because you can thus imagine that you did something wrong, which implied that a right thing could have been done, which prevents you from experiencing the anxiety associated with the fact that nothing could be done satisfactorily, that you were helpless. However painful and unsettling helplessness is, being helpless at times in life is unavoidable. Being human, we are limited."

Again, projective identification can be at play. Helplessness appears to be the most intolerable feeling for human beings. Thus, the sheer helplessness of PTSD patients is often projected onto therapists. In response, clinicians may react by feeling helpless at helping the person rather than recognizing that such feeling is a projection. Ideally, therapists recognize feelings of helplessness as a projection and employ this information to address the issue of

helplessness with the person. Therapists need to learn to deal with helplessness, recognize that they can be helpless at times in life and in therapy; otherwise, they will fight and deny such feelings, forfeiting change in the traumatized person. Clinicians need to bare helplessness in therapy, while remaining competent, in order for the person with PTSD to identify with such adaptive response.

Eventually, the person let go of guilt feelings by recognizing the helplessness embedded in the traumatic event. Eventually, mistakes done by victims during a traumatic event end up being acknowledged by them, without therapists having to underline mistakes (actually, therapists should never identify out loud 'mistakes' victims have made because it greatly interferes with the therapeutic alliance). When therapy is competently offered and successful, the person addresses spontaneously at the end of therapy, when PTSD is remitted, the misleading behaviors which have contributed to trauma. Then, the person considers the future avenues for preventing such tragedy.

For example, a young man consulted with PTSD after being hit many times by men with machetes. He was lucky to have survived. At the onset of the assault, this homosexual man entered a city park which was renowned for outdoors sexual encounters between homosexual strangers. This park was also very well known for recently having gangs of heterosexual men patrolling with machetes and attacking homosexuals in order for this sexual practice to stop in this public place. This young man went to the park anyway, pretending to himself that he did not look like a homosexual. At the end of therapy, he recognized having been imprudent and even pushing the limits of reality in his disfavor. He could decide to engage in more cautious actions from now on. The therapist never mentioned his dangerous choice, but acknowledged the need for all of us to entertain the limits imposed by outer reality.

Fear / Anxiety

Fear is oftentimes viewed as being at the core of PTSD. This conceptualization is mostly adopted by behaviorists. However, according to clinical experience of mine and others, PTSD is associated with many emotions. Furthermore, anger is usually neglected in the theorizations about PTSD in favor of fear. Fear is a more socially and personally acceptable emotion, nowadays, than anger. Although fear is likely to have been experienced *during* the traumatic event, a person developing PTSD usually experiences anxiety afterward, not fear. Anxiety is a reflection of the anticipation that something distressing is likely to happen, inwardly or outwardly. In the psychodynamic model, anxiety is considered to be the intrapsychic indicator that a suppressed or repressed material is pressing into consciousness; the operative defense mechanism is failing and the rejected material is not yet recognized consciously. Anxiety can also be induced by pathogenic cognitions about possible distressing occurrences.

In my clinical experience, anxiety associated with PTSD is often a defense mechanism against anger. As mentioned previously, the anger of victims can be so great that it entails sadistic and/or murderous desires, which are profoundly unsettling to most of us. Such anger is overwhelming for the conscious self of the person. Consequently, the person cannot keep it

into consciousness and, thus, the murderous anger is projected onto others, usually strangers. As a result, individuals with PTSD develop hypervigilance by which the wish to assault is projected onto strangers.

Usual techniques for addressing anxiety are many. Firstly, it is important to empathically acknowledge the presence of anxiety and its associated suffering, especially of the person identifies with anxiety. Then, the therapist facilitates a differentiation between anxiety/fear, which is an internal phenomenon, with the actual absence of an external danger, in order to help the person to refocus internally and differentiate inner reactions from external circumstances. The person can learn and practice relaxation techniques and cognitive techniques of selective attention, thought stopping, or cognitive restructuring. The therapist can address the inherent difficulties and dangers of life as being at times unavoidable and encourage the person to ponder whether the person can live with this reality, consciously, without denying or generalizing. In a timely manner, the therapist interprets anxiety as a defense against an understandable anger and anger-based desires. In clinical practice, it can be impressive how quickly disturbed sleep, hypervigilance, concentration difficulties, etc., disappear whenever anger and anger-based desires are fully acknowledged and owned. Interestingly, nightmares in which the person is chased are automatically transformed into dreams in which the person chase an aggressor. As soon as the underlying aggressive desires are recognized by the person, these dreams disappear along with the PTSD.

Whenever anxious symptoms do not disappear following an integration of previously repressed anger, exposure techniques can be undertaken along with the use of attentional focusing on external circumstances versus internal anxiety. The attenuated anxious responses are then based on a learning process; the person learned to be anxious and this has become an automatic response. Unlearning can be done with exposure techniques in very few occasions, if not only one. Moreover, the person can usually do it alone given that anxiety has been attenuated and is not likely to be overwhelming upon exposure to anxiety-provoking stimuli such as walking alone on the street, taking the bus, going to the supermarket, etc. If exposure and cognitive techniques are used prematurely (that is, without addressing and resolving anger-based issues), these may be successful in reducing the anxious symptoms but through inhibition rather than resolution or unlearning. Clinical experience has shown repeatedly that people treated with exposure techniques prematurely oftentimes become ongoingly exhausted, having to employ neurobiological energies to sustain the continual inhibition of anxious responses; they can become functional, or quasi-functional, but exhaustion is at the center of their lives.

Some sequelae may remain, especially if the person was assaulted from behind. The therapist can expect that the person may well continue to sit against a wall in any public place. However, the person may well continue to enjoy restaurants, streets, theaters, etc., despite this preventative behavior.

Sadness

Sadness could be categorized into two types: floating sadness and deep-seated sadness.

Floating sadness is associated with depression moods. Such sadness is not therapeutic and needs to be considered as the manifestation of a severance, as a detachment away from one's true self, others, and life. Floating sadness is also associated with PTSD, with such severance allowing for a non-feeling, but also a non-feeling of oneself and life.

In terms of interventions, the therapist can make associations between the person's conscious self and the person's experience in the true self, with the help of a benevolent attitude, empathic reflections, and an encouragement to be kind toward oneself. Links can also be offered by the therapist regarding the person's way of relating to others, by welcoming the person in psychotherapy, by encouraging the person to self-disclosure and feel safe by doing so, and by reaching to benevolent others). To undo the severance between the person and the environment, the therapist can encourage the person to address unresolved problems, to avoid anxiety-provoking places and others, at least temporarily, and to engage in activities with others outside one's home). Finally, the relationship of the person toward life needs to be mended, and the therapist can encourage the person to engage in pleasurable activities, ideally with others, and the therapist can also smile and laugh in therapy, in a timely manner, so that therapy is not only a place of suffering and reflecting, but also a place where all of life can occur.

Deep-seated sadness, in contrast, is manifestation of psychological distress (see Tomkins theory of emotions). Deep-seated sadness, without panic, is linked to a loss. Its experience is therapeutic in and of itself because such sadness belongs to a core feeling of the true self. During the experience of such sadness, the person allows oneself to feel psychological suffering. In a contained way, the person accepts to present to one's distress and pain, a psychological wound. This attending allows for the identification, acceptance, and then integration of the sadness-provoking material.

To foster the emergence of deep-seated sadness, the therapist can interpret defense mechanisms protecting the person's conscious self from experiencing, along with inferring the avoided pain. If the person's responds favorably by better attending to the inner world, the therapist can welcome emphatically the sadness emerging in the person, through words facilitating consciousness and soothing. The therapist can also provide a poised emotional attunement, resonating with the experienced feelings from the standpoint of a calm and soothing presence. Such poised attunement facilitates the letting go of illusions, capacities, relationships, material objects, naiveté, loss of time and life (by having stayed unnecessarily in unhealthy relationships or situations). In this way, the pressing and almost unbearable aloneness emerging with the deep-seated sadness is counterbalanced thanks to the emphatic accompaniment provided by the therapist, a caring and yet differentiated human being.

Altering Defense Mechanisms

The intensity of emotional reactions provoked by traumatic events trigger the use of less mature defenses.

Punamäki, Kanninen, Qouta, and El–Sarraj (2002) examined the role of psychological defenses in moderating trauma and psychological consequences of trauma. These authors found that Palestinian men who had been tortured were more likely to have PTSD symptoms of vigilance, avoidance, and intrusion if they used defenses mechanisms. High levels of torture were significantly related to a lower use of mature defenses; however, high levels of torture were not significantly related to use of immature defenses. Immature reality-distorting and immature reality-escaping defenses were associated with high incidence of PTSD symptoms, while mature defenses were associated with low levels of PTSD symptoms. This suggests that while victims with premorbid access to higher-level defenses may be better able to cope with trauma and therefore exhibit fewer symptoms than patients who typically rely on lower-level defenses, trauma may itself weaken an individual's ability to use higher-level defenses. One other study, however, found no differences in use of mature and immature defense mechanisms between trauma victims with and without PTSD (Birmes et al., 2000, reviewed in Schottenbauer, 2008).

Schottenbaeur (2008) reported that a large-scale longitudinal study found that psychodynamic therapy was associated with improvements in functioning not only during therapy, but also years after the end of treatment as indicated by continuing decreases in symptoms (Blomberg, Lazar, & Sandell, 2001; Sandell et al., 2000). This finding suggests that psychodynamic therapy may be related to changes within the person that allow continuation of improvement after treatment has ended. A typical psychodynamic intervention includes interpreting defense mechanisms and warded-off wishes and fears.

In severe PTSD, a reversal from mature to immature defense mechanisms seems to occur. A vicious cycle is triggered. When individuals are activated into very high levels of anxiety, anxiety can compromise higher mental functions, including mature defense mechanisms. Previously warded-off affective responses become activated. Individuals can then experience even higher anxiety, while they have even less access to mature defenses. This interference with the efficiency of one's defense mechanism appears to be a function of the arousal level (remember the Yorkes-Dobson law).

At the extreme severity of the spectrum of this phenomenon, patients are constantly afraid, feel utterly vulnerable and under a possible imminent attack (in fact, they are being attacked internally by their amygdala which is fully activated and left unchecked). Their level of anxiety is so high that not much seems to go through this curtain of anxiety and bring a sense of well-being. Oftentimes, such individuals present with multiple clinical disorders in addition to severe PTSD, a personality disorder, attachment disorder and/or disorder of the self. The defense mechanisms employed as oftentimes projection, projective identification, and somatization. Although these individuals have mostly relied upon immature defense mechanism or suppressive structures to feel better, they now need to build new structures in psychotherapy to regulate affects and resolve their PTSD.

For others, the clinical picture is not so bleak. They may present with mild to moderate PTSD, along with the re-emergence of painful realities of their lives, which they had successfully

suppressed for many years. These people possess some capacities for affect-regulation, and immature defense defenses of suppression have not been their way of fending off painfully experiences. Indeed, their affect-regulation capacities appear to gradually reduce the strength of their defense mechanisms, and this gradual process renders the experience of the affective responses less painful as they are titrated into consciousness at tolerable dosage.

In addition, some individuals with PTSD can dissociate from trauma-related cues and memories to protect themselves from intense physiological arousal. Practitioners need to be cognizant of the possible occurrence of dissociation in PTSD. Dissociation is an effective but immature defense mechanism. Clinically, it is important to understand that dissociation is a mechanism effective in reducing physiological arousal and psychological pain. Inviting patients to let go of dissociation as a defense mechanism may lead to their relinquishing of this protective manoeuver, but it is likely to lead to greater arousal and intense discomfort. Practitioners thus need to acquire specialized competencies in both identifying and working with subtle dissociative manifestations, along with the unsettling consequences associated with its cessation.

At the other end of the spectrum, neurobiology informs us that some individuals with PTSD can react so intensely to trauma-related cues that their organism needs to release endogenous opiates to counter the unbearable arousal (see Blanchard et al., 1982). This mechanism is very adaptive during the occurrence of a dangerous situation, allowing for survival behaviors to be set in motion. Clinically, this empirical fact was very useful to me as a psychotherapist; it showed me dramatically how painful PTSD can be, especially exposure to trauma-related cues. It emphasized the need to proceed gradually, from the periphery to the cores issues as proposed by Horowitz (1976, 1984, 2001), to avoid overwhelming exposure to trauma in psychotherapy. It also emphasized the need to avoid trauma-related cues or situations until one can apprehend them without becoming disorganized. It highlighted the need to adopt a caring and careful attitude toward patients with PTSD as their suffering is greater than they usually report. Knowing that trauma-focused methods, or even trauma-related interventions, can trigger pain so intense as to trigger a release of endogenous opiates informs practitioners to be cautious in their approach to trauma-related material, monitoring subtle reactions in patients as they proceed.

Before new affect-regulation capacities are put into place, medication is often a valuable adjunct treatment modality. Psychotropic medication reduces anxiety and provides some relief. The reduction of anxiety allows for neuropsychological structures to further develop.

Resolving Conflict

An intrapsychic conflict exists because a part of oneself is rejected. The conflict exists between the conscious, acceptable self and an unacceptable emotionally-charged part of the self. The latter is too painful or unsettling to embrace consciously. Such a conflict is oftentimes called an “old wound”. Are affectively charged memories such as “old wounds” rekindled by traumatic

experiences through an associative process? Do these “old wounds” need to be addressed in psychotherapy and thus de-activated?

Neuropsychological phenomena do not happen in a vacuum as it seems to happen in neurons stimulated by electrodes in a laboratory, but neurobiological phenomena exist in networks of connectivity. Brain processes are associative by nature. Systems theory informs us that no phenomenon exists in a vacuum and that all phenomena of a complex system are related in one way or another in human beings because we are complex systems. Transposed to PTSD, systems theory explains why individuals with PTSD repeatedly remind practitioners that the recent traumatic event has reevoked “old wounds” which they thought were resolved and relegated to the past.

Horowitz (1976) pointed out that traumatic events fuel through associative processes unresolved warded-off affect-laden conflicts, otherwise called “old wounds”, and these need to be resolved to eliminate PTSD symptoms. During a traumatic event, it is likely that affect-laden memories are fuelled, and thus reactivated, if the same affects are intensely triggered by the traumatic event. Because these previously warded-off affective memories are now more intensely activated, they are likely to participate in the ongoing activation of trauma-related affects maintaining the PTSD. When this dynamic model applies, trying to deactivate only the fear responses associated with PTSD is likely to be an ineffective strategy. All these affective responses of PTSD will to be addressed in psychotherapy and de-activated in one way or another, usually through dynamic work such as “working through”. Working through would involve acknowledging these previously warded-off, affect-laden conflicts through exploring and interpreting, and re-associating the individual’s negative representations of self and others with positive representations of self and others.

Finally, practitioners also know that heightened arousal can be caused by internal stimulation, and not only external trauma-related stimuli. Clinicians also know that unresolved intrapsychic conflicts can provoke and maintain PTSD in some individuals. As anger and its associated homicidal wish are repressed and projected onto others, the fear of being assaulted at any moment fosters high levels of arousal and hypervigilance in some PTSD patients. After much restructuring therapeutic work, psychotherapists can empathically suggest the presence of frightening anger harboured by patients unconsciously. If well received by patients, practitioners can interpret the maladaptive defense of projection to cover up the anger and homicidal wish, which were unbearable until now, and invite patients to accept and own these inner realities. Because such work on anger resolution is central to alleviating many PTSDs, detailed clinical examples are presented later on.

Developing New Structures

The self-soothing, affect-modulating system can be involved in de-activating fear responses. When it is well developed, it kicks in as soon as the organism becomes in distress. Such system is environmentally-dependent because its development relies on empathic, soothing interactions with others. People who display secure attachment patterns also have well-

developed self-soothing capacities. These capacities are located in the right pre-frontal cortex and are found to be highly connected with their amygdala. Because this neural pathway is one of the major consequences of adequate nurturing, of good-enough mothering, it is reasonable to wonder whether the development of a good-enough therapeutic relationship can foster the development of self-soothing capacities in patients with limited dispositions at this level.

Associated with this affect-modulation capacity are internal representations of self, others, and life as predominantly benevolent. Upon activation of distressing affects in the amygdala, these positive representations are activated and counterbalance the difficult experience triggering dysphoric reactions within the amygdala. The left pre-frontal cortex come to help here by, for example, comparing the present situation with past ones which leads to choosing a plan of action. Upon experiencing a flashback, for example, the actions of the left pre-frontal cortex could entail telling oneself this: “It is painful right now, but there is actually nothing that I can do to prevent the flashback from occurring given that it is already on its course, but I can reduce the pain by partly disengaging myself from the emotional dimension, and note its occurrence, even observe its unfolding, while reminding myself that this flashback is not going to last forever, that my internal actions, right now, are de-fuelling its strength, especially given that it is a memory and not the re-occurrence of that event which was terrible but is not over”). If they reflect a successful internal action, the last comments of this internal dialogue have used the operation provided by the hippocampus, by putting things in perspective in terms of time and space, and by engaging oneself in the hippocampal perspective, so to speak, as a connected but observing self. The sense of self has been provided by the cingulated anterior which allows us to represent ourselves and experience ourselves through time, giving us a sense of continuity rather than being forever stuck in the present experience. For the hippocampus and the cingulated anterior to come to the rescue of an individual in the throes of a painful flashback, those neurobiological structures need to have been developed, which mostly happens when one has had a good-enough life, full of reassurance, pleasures and challenges.

Conclusion

Favouring the use of trauma-focused techniques may be a defense of some practitioners against experiencing helplessness, their own and their patients' helplessness.

There may be something inherent to PTSD leading some practitioners to adopt a trauma-focused method, a more technological and hazardous approach, in a *sine qua non* fashion. For instance, the very intense nature of PTSD is a double-edged sword for its treatment. While the intensity of reactions associated with PTSD can help practitioners to readily identify the various factors at play, PTSD tends to provoke intense reactions in others, including clinicians. Psychological pain can be so great that practitioners can be enticed to protect themselves and, therefore, engage in counter-transferential reactions, unbeknownst to them. One of these protective strategies is to avoid addressing the traumatic experience at any depth and thus avoid experiencing pain with the patient. The intense dysphoric emotions and destabilizing realizations associated with traumatic events are avoided. Another protective strategy is to overemphasize the need to reexperience traumatic events, hereby adopting an active and trauma-focused mode to counter the helplessness embedded in PTSD.

Previously, traumatic events have been overlooked as mostly irrelevant by psychotherapists. The pendulum has swung in the opposite direction. Nowadays, reexperiencing traumatic events has become quite fashionable, in clinical practice and controlled clinical trials, a phenomenon of concern to me and others.

Some PTSD experts have insisted that it is essential to remember traumatic events, "Remember to remember!" After having learned debriefing, some practitioners import this trauma-focused technique in psychotherapy and, therefore, invite patient to retell their traumatic story over and over as if it were therapeutic. I dare to disagree and one clinical vignette is exemplary of the need for some people to not discuss their traumatic event.

In an attempt to seek an expert opinion, a psychologist reported to me how she had been proceeding in psychotherapy with a woman who had never even reported a traumatic event. The psychologist knew of the horrible traumatic event because by reading about it in a report when the woman was referred to her. Two years earlier, this woman had arrived home to find her three children, her mother, and her husband shut dead in the basement. She was left alone. The husband had been depressed and had shut the others family members before killing himself. Two years later, the woman had not yet mentioned this traumatic reality in psychotherapy. She was simply trying to reorganize her life without decompensating. The psychologist had the impression that a psychotic episode could be triggered at any moment if the therapist would intrude by bringing forth this horrible event and the patient's ensuing losses. I agreed with her. In the meantime, a strong therapeutic alliance was being established and the patient was restructuring her life gradually. Maybe the woman would find the strength one day to discuss this traumatic event, but maybe not. This psychologist showed a profound respect for this woman, allowing her to proceed according to her own psychic capacities.

The actual emphasis, or overemphasis, on trauma-focused therapies for PTSD in clinical trials may be an attempt to protect ourselves from the sheer helplessness embedded in PTSD. At their core, traumatic events entail a shattering experience of helplessness. In my supervisory experience, patients with PTSD struggle with helplessness so intensely that few practitioners dare to empathically accompany them in experiencing this aspect of the trauma. Along with a deep suffering and an altered functioning, patients with PTSD bring into psychotherapy a free-floating sense of helplessness, which can overwhelm both patient and practitioner. As PTSD patients struggle to avoid facing their own sense of helplessness, they "project" it onto the psychotherapist. Such projection is most uncomfortable and it can throw clinicians into a reaction, enticing the practitioner to employ an active trauma-focused technique aimed at countering any feeling of paralysis. The practitioners' own sense of helplessness in life may well be awakened; we know intimately that what happened to the patient in front of us can well happen to us. When we treat individuals with PTSD, we know, deep down inside, that we can also be rendered helpless, helpless in terrifying ways. Helplessness can be terrifying for most of us.

Helplessness is an inherent part of the human condition, but it is acutely painful and oftentimes overwhelming. When I started treating individuals with PTSD, I was impressed by the extent to which patients would do almost anything to avoid feeling helpless, as if this would cause them the greatest human suffering. This intense struggle with helplessness is, unfortunately, compounded by our societal ideologies. We live in a world informing us daily that we are successful if we control everything within the perimeter of our lives. We experience failure whenever we lose control over our situations. Avoiding helplessness is central to our Western society. Consequently, practitioners are well equipped to disavow helplessness and its profoundly destabilizing effects.

Whenever helplessness is disavowed by practitioners, the patients' traumatic experiences cannot be fully acknowledged. The core experience of helplessness is left unrecognized, and its integration to the psychological structure is forsaken. To allow full integration of traumatic experiences, practitioners need to become aware of their own helplessness and of their own struggle toward helplessness. Practitioners need to afford experiencing helplessness within themselves and with patients. By embracing helplessness as part of their human experience, practitioners allow patients to fully embrace their own human condition, including vulnerability and dysphoria. As clinicians, we need to bear helplessness.

How can practitioners intervene to treat PTSD *without* experiencing helplessness? How can practitioners intervene *while* experiencing helplessness? How to experience helplessness without becoming paralyzed? How to be confronted with helplessness without trying to eradicate it? Is the goal of PTSD therapy to only regain a sense of control, at all costs? Or is the goal integrating traumatic experiences into the whole psychological structure, embracing helplessness and regaining control?

Remarkably, trauma-focused treatments involve bringing individuals with PTSD back into reliving the most excruciating moments of a traumatic event. Such endeavour provokes high levels of arousal, if not extreme, although PTSD is now known to involve a limited capacity for arousal modulation. Nonetheless, proponents even suggest that practitioners apply trauma-based therapies to all patients with PTSD. They insist on claiming that the great majority of individuals with PTSD will not sustain harm from the use of trauma-focused methods. It appears that they take for granted that all individuals with PTSD have the neuropsychological capacities to sustain the dizzying internal activation provoked by an intense, if not overwhelming, re-experiencing of a traumatic event. Such claims are often based on conclusions of controlled clinical trials and meta-analyses, but these are inherently limited to a very small portion of persons with PTSD. Samples include only self-selected individuals and persons fitting the stringent research criteria: that is, PTSD without co-morbid disorders or personality disorders, etc. In clinical trials, remission rates are often not reported, along with complete and partial remission rates, dropout rates, side effects, and efficacy results containing the data pertaining to dropouts. Empirical evidence ends up informing us that cognitive-behavioral therapies can be efficacious in treating PTSD, but only in some patients and to certain degrees and for a certain length of time.

Maybe fragile individuals with PTSD just do not want to participate in controlled clinical trials, or they drop out early after having experienced negative side effects or to protect themselves from experiencing such effects. These subjects do not come back to tell researchers what they have experienced. Unfortunately, such realities are rarely acknowledged by proponents of trauma-focused techniques, leading them to overstate claims of efficacy which are not generalizable to real-life clinical practice. Iatrogenic effects are rarely reported in the literature, although some have the courage to do so (e.g. Brunet, 2002; Pitman et al., 1991). During 10 years, I provide courses of continuing education specialized in PTSD across the USA. Many practitioners informed me of negative consequences associated with PE or EMDR. Their patients had been previously severely damaged by PE or EMDR (suicidal attempts, psychotic episodes, bulimic episodes, relapse of substance abuse, self-mutilations, and even homicidal threat toward the therapist during an EMDR session). Clinicians care about their fellow human beings and wish to avoid any iatrogenic effects. We all make mistakes. When we make mistakes, we can sometimes repair them. Let’s learn from our mistakes and reduce them to a minimum. Let’s use the gentlest techniques with those who are most vulnerable.

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