

EMDR THERAPY OF PANIC DISORDER AND AGORAPHOBIA: A REVIEW OF THE EXISTING LITERATURE

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Abstract

Objective: The article outlines the state of the research on EMDR therapy of Panic Disorder (PD) and Agoraphobia.

Method: Qualitative analysis of the existing literature.

Results: One pilot study (Faretta 2013) as well as single and series of individual case reports suggest that EMDR therapy is effective in eliminating symptoms of panic and agoraphobia, whereas two previous studies with placebo controls (Feske and Goldstein 1997, Goldstein et al. 2000) failed to show comparable outcomes. Evaluating these discrepancies, an analysis of the two controlled studies reveals a failure to use the Adaptive Information Processing (AIP) model and to apply the treatment plans for PD/A described in the successful case reports. On the other side, the positive case reports and the pilot comparison study suggest that, when working with a comprehensive case formulation based on the AIP model, 12 up to 19 EMDR sessions can be effective to accomplish a full recovery from PD and Agoraphobia.

Conclusions: While research on EMDR therapy for PD/A is still at an early stage, these preliminary findings support the need for controlled studies to systematically evaluate the efficacy of EMDR therapy for PD in comparison with other accepted PD treatments.

Key words: Panic Disorder, Agoraphobia, Adaptive Information Processing, Panic Attack

Declaration of interest: none

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1. Definition of PD and Panic Attack

Panic disorder (PD) is an Anxiety disorder characterized by recurrent and unexpected panic attacks (DSM-5 – criterion A; American Psychiatric Association 2013), that is to say a pervasive sense of fear that becomes extremely intense within a few minutes. During a panic attack, an individual experiences symptoms that range from somatic manifestations – palpitation, sweating, trembling or shaking, sensations of shortness of breath, sensations of choking, chest pain or discomfort, nausea or abdominal disease, dizziness or light-headedness or faintness, chills or hot flushes, tingling sensations (paresthesia) – to psychological features – sense of unreality (derealization) or of detachment/estrangement from oneself (depersonalization), fear of losing control or “going crazy”, fear of dying.

These episodes can take place whether the person

is feeling calm or anxious, leading to the distinction between expected and unexpected panic attacks, depending of the presence (or absence) of recognizable triggers (e.g. driving alone on the highway; taking an exam). Expected panic attacks do not exclude PD, but more than one unexpected episode is required to correctly employ this diagnosis.

The second clinical feature of PD (criterion B) is the development of concerns (at least one month long) about the possible occurrence of new panic attacks and/or the adoption of related dysfunctional behaviors, such as avoiding physical exercise, or situations which are unfamiliar to the subject.

Co-occurrence with Agoraphobia is quite frequent, so that the past DSM edition (DSM IV-TR; American Psychiatric Association 2000) indicated two kinds of Panic disorder: “with” or “without agoraphobia”. In the present edition, where it is remarked that PD often

presents comorbidity with other Anxiety disorders, and especially with Agoraphobia, the two diagnosis are described as independent. Comorbidity is also frequent with Depressive disorders, Somatic symptom disorder and substance misuse.

PD is associated with high levels of social, working and physical disability. In the general population of United States and of some European Countries, PD affects 2-3% among adults and adolescents (DSM-5; APA 2013). The average onset age is 20-24 years (DSM-5; APA 2013). When not treated, PD tends to become chronic, even though it can be characterized by significant oscillations in symptomatology: some people may experience sporadic crises, spaced apart by years of remission, whereas others show severe and continuous symptoms. Remission is therefore often incomplete, and recurrence is frequent.

PD increases suicidal ideation and risk, and is associated with frequent medical examinations and Emergency Department (ED) admissions (DSM-5; APA 2013). The economic costs of this disorder are both direct (for the national health service) and indirect (absences from the work place, eventually leading to unemployment or, for younger individuals, to scholastic drop-out). Due to the extent of its psychological, social and economic consequences, PD has been investigated thoroughly, with special attention to treatment efficacy.

2. Evidence-based treatment approaches for PD

According to NICE (National Institute for Health and Care Excellence 2011), there are three treatment options for Panic Disorder in adults that have an evidence base: “psychological therapy, medication and self-help have all been shown to be effective” (NICE 2011, p. 25).

Best practice recommends that all three kinds of intervention should be offered to the patient, and his/her preference should be taken into account. The NICE guideline emphasizes that “the interventions that have evidence of longest duration of effect, in descending order, are: psychological therapy; pharmacological therapy (antidepressant medication); self-help” (NICE 2011, p. 26).

As regards the psychological intervention, Cognitive Behavioral Therapy is recommended in the optimal range of duration of from 7-14 hours in total. Specifically, NICE recommends weekly CBT sessions of 1-2 hours, for a maximum duration of 4 months treatment. NICE states that “CCBT [Computerized Cognitive Behavioral Therapy] is a promising low-intensity intervention for panic disorder that does not have yet a substantial evidence base” (National Collaborating Centre for Mental Health 2011, p. 320).

The attention to CCBT is prompted by the observation that “The evidence for the benefits of CBT delivered in a number of formats (group or individual) in the short term and long term is, however, undermined by the fact that as few as 20% of people with panic disorder treated in primary care receive CBT [...]. Not surprisingly the need to increase access to CBT has led to developments of CBT packages that require less input from therapists” (National Collaborating Centre for Mental Health 2011, pp. 286-287).

Among the articles reviewed by NICE, two studies support the use of CCBT for Panic disorder: the first (Carlbring et al. 2005) compared 10 individual weekly sessions of cognitive behavior therapy for PD with or without agoraphobia with a 10-module self-help program on the Internet. Forty-nine participants with

PD diagnosis were randomized. The outcomes suggest that “Internet-administered self-help plus minimal therapist contact via e-mail can be equally effective as traditional individual cognitive behavior therapy”. These results were confirmed at a one-year follow-up.

The second study (Kiroopoulos et al. 2008) compared Panic Online (PO), an internet based CBT intervention, to best-practice face-to-face CBT for people with PD with or without agoraphobia. Eighty-six participants, recruited from Victoria, Australia, were randomly assigned to either PO (n=46) or best practice face-to-face CBT (n=40). The outcomes of the internet-based CBT program were found to be comparable to those of face-to-face CBT in terms of reductions in PD and agoraphobia clinician severity ratings, self reported PD severity and panic attack frequency, depression, anxiety, stress and panic related cognitions. Participants rated both treatments conditions as equally credible and satisfying, even if ratings for compliance to treatment and understanding of the CBT material was higher in the face-to-face CBT condition.

Other study designs considered in the NICE guidelines compared CBT vs. applied relaxation (Carlbring 2003), CBT vs. CCBT vs. information control (Klein, 2006), CCBT vs. information control (Richards, 2006A), CCBT vs. waitlist control (Carlbring 2001, 2006). Richards et al. (2006a) considered CCBT + stress management vs. other active treatments: the results show that “Both CBT treatments were more effective at post-treatment assessment than the control condition in reducing PD severity, panic and agoraphobia-related cognition, negative affect and self-ratings of health. PO2 [Internet-based CBT plus stress management] was more effective than PO1 [Internet-based CBT] at post-treatment assessment on PD severity and general anxiety, although at 3-month follow-up these differences were no longer apparent”. CBT, even in the developments implying minimal patient-therapist contact as in CCBT, is therefore indicated by NICE as first choice treatment for PD.

Concerning the use of pharmacological interventions, “antidepressants should be the only pharmacological intervention used in the longer term management of Panic Disorder, with particular reference to selective serotonin reuptake inhibitors (SSRIs) and tricyclic antidepressants (TCAs)” (NICE, p. 27). Benzodiazepines are contraindicated as well as sedating antihistamines or anti-psychotics. (NICE, p. 25)

Self-help, ranked as the third evidence-based treatment option for PD, relates once again to CBT approach. Bibliotherapy based on CBT principles (including CCBT) is thus recommended, together with support groups (preferably CBT-oriented) and physical exercise.

Two recent studies (Haug et al. 2015, Nordgreen et al. 2016) pointed out CCBT limitations in terms of drop-out rates and efficacy on complex anxiety cases. More precisely, the intent of increasing patients' access to evidence-based psychological treatments for anxiety disorders through stepped care model interventions is confirmed by one of the two studies (Nordgreen et al. 2016) that compared the effectiveness of direct F-t-F CBT with a CBT stepped care model (psychoeducation, guided Internet treatment, and face-to-face CBT). Patients with PD or SAD were randomized to either stepped care (n=85) or direct F-t-F CBT (n=88). No significant differences in intention-to-treat recovery rates were identified between stepped care (40.0%) and direct FtF CBT (43.2%). Furthermore, the majority of the patients who recovered in the stepped care condition

did so at the less therapist-demanding steps (26/34, 76.5%), suggesting that CBT packages requiring less input from therapists may actually increase access to appropriate treatments. However, attrition rates were high: 41.2% in the stepped care condition and 27.3% in the direct FtF CBT condition, indicating compliance as a critical aspect of stepped care models.

The other recent study (Haug et al. 2015) points out the lower rate of response to both stepped care CBT and FtF-CBT in complex anxiety clinical cases. One hundred and seventy-three patients were recruited from nine public mental health out-patient clinics and randomized to immediate FtF-CBT or Stepped Care treatment. The results indicate that lower social functioning, higher impairment from the anxiety disorder, and a comorbid cluster C personality disorder are associated with significantly less improvement, particularly among patients with PD. According to the authors, these findings suggest that patients with lower social functioning and higher impairment from their anxiety disorder benefit less from stepped care CBT and may require more adapted and extensive treatment.

NICE data are partially contradicted by the meta-analysis conducted by Bandelow et al. (2015) on the efficacy of treatments for Anxiety disorders. The study compared the effect sizes of pharmacological, psychological and combined treatments for the three main Anxiety disorders (Panic disorder, Generalized anxiety disorder and Social phobia). A total of 232 papers, including 234 studies with 37,333 patients, were considered in the review.

The main result of this analysis is that “most psychopharmacological drugs used for anxiety disorders have markedly higher effect sizes than psychological therapies, and the gains were achieved in a shorter time” (Bandelow et al. pp.188-189). As regards the types of psychotherapy, “Mindfulness meditation yielded the highest effect size. Relaxation treatments were numerically more effective than individual behavioural treatments (CBT and exposure), which was more effective than group CBT. Exercise, non-face-to-face therapies, PDTh, EMDR and IPT showed lower pre-post effect sizes” (Bandelow et al., p.186).

Two studies investigating EMDR efficacy on Panic Disorder were included in the meta-analysis by Bandelow et al. (2015). They are presented in two articles (Feske & Goldstein 1997, Goldstein et al. 2000) that will be discussed in section 4.1. Bandelow et al. conclude by stating that “when looking at pre-post effect sizes, psychotherapies did not differ from pill placebos. This surprising finding cannot be explained by heterogeneity, publication bias or allegiance effects” (Bandelow et al., p.190). The only strong point acknowledged to psychotherapies, with particular reference to CBT, was the maintenance of therapeutic effect after the termination of treatment, “whereas patients receiving drugs experience a recurrence of anxiety symptoms after stopping medication” (Bandelow et al., p.190) The authors state that “this would offer CBT an advantage over drug treatment” and for this reason they “intend to carry out another meta-analysis to investigate whether or not this is the case”. (Bandelow et al., p.190).

Actually, few studies have examined the stability of CBT’s effectiveness over time. For example, according to de Beurs et al. (1999), while some patients recover after a brief treatment, some other need prolonged, additional interventions. Furthermore, according to Barlow et al. (2000), the combination of CBT and pharmacotherapy seems to lead to greater risk of relapse after the ending of CBT treatment.

In the cases of pharmacotherapy alone, a 5-year

follow-up indicated that only 45% of those treated achieved full remission (Woodman et al. 1999).

Ost et al. (2004) point out that “there is still much room for further development of CBT methods for PDA [Panic Disorder with Agoraphobia] because only 60% of the patients treated in RCTs [randomized controlled trials] published since 1990 have achieved a clinically significant improvement” (p. 1106). Finally, dropout rates are reported as high as 24% for exposure (Marks et al. 2004) and 26% for CBT (Bakker et al. 1999). Research also suggests that those who are more severely affected by PDA are more likely to refuse or drop out of these treatments (Hunt 2000).

An explanation to these partially contradictory results may be found by examining the etiology and function of panic symptoms in the context of PD within the theoretical frameworks of CBT and AIP models.

3. CBT and AIP conceptualization of PD and Panic Attack

The CBT model assumes that panic attacks develop from a mistaken – “catastrophizing” – interpretation of physical symptoms, e.g. dizziness and tachycardia interpreted as signs of an imminent myocardial infarction (Hofmann et al. 2007, Rovetto 2003). According to this approach, panic is not provoked by the somatic symptomatology but by the (wrong) understanding of it. The fear of a catastrophic event (stroke, heart attack, going “crazy”, and so on) amplifies the resonance of physical symptoms, which increases anxiety, creating a feedback circuit eventually giving rise to panic attack (Barlow 1988, Clark et al. 1999). Based on this conceptualization, the therapeutic intervention is primarily aimed at modifying this cognitive bias, both through “cognitive restructuring” techniques and de-conditioning (e.g. relaxation; in vivo exposure).

The Adaptive Information Processing (AIP) model, that guides the use of EMDR therapy, proposes a different interpretation of PD and panic attacks (Faretta 2013; Fernandez and Faretta 2007; Leeds, 2009, 2012, 2016; Shapiro 2001; Ural et al. 2015). Panic symptoms are considered to be the expression of dysfunctionally stored memories, which are “trapped”, so to speak, within the implicit memory network. More specifically, AIP predicts that inadequately processed adverse childhood experiences may impair resilience, increasing vulnerability to later occurring stressful experiences (Shapiro 2001). Goldstein (1995) was the first to observe that PDA patients may have disconnected formative experiences from the affective component of the maladaptive memory network. This cognitive-affective dissociation may undermine the ability to normally process stressful events, further reinforcing dissociative mechanisms as the only possible way to control overwhelming emotions.

The hypothesis that dissociation may facilitate the onset of PD and panic attacks is supported by empirical studies that investigated alexithymia, that is a personality construct that reflect deficits both in the cognitive-experiential component of emotion response system and at the level of interpersonal regulation of emotions (Taylor 2000). Specifically, alexithymia is characterized by (1) difficulty identifying and describing subjective feelings, (2) difficulty distinguishing between feelings and the bodily sensations of emotional arousal, (3) constricted imaginal capacities, as evidenced by a paucity of fantasies, and (4) an externally oriented cognitive style (Nemiah et al. 1976). Alexithymia rates have been reported to be as high as 47% (Parker et al.

1993) and 67% (Zeitlin et al. 1993) among patients with Panic disorder, compared with 13% in patients with obsessive-compulsive disorder and 12.5% in patients with simple phobia. The dramatically higher rates of alexithymia among those with PD support a role for cognitive-affective dissociation in the etiology of PD. The development of emotion regulation skills is influenced by children's attachment experiences. Alexithymia is associated with insecure attachment (Shaffer 1993, Beckendam 1977) as well as PD.

According to Ural et al. (2015), "The relationship between Panic disorder and dissociation, whose etiology is often traumatic, is well known. Panic attack has a dissociative symptom as a criterion, although the diagnosis may also be given in the absence of depersonalization or derealization" (p. 464, 2015). In their study, Ural et al. (2015, pp. 468-469) found that "Both the PAS and PDSS scores of patients with dissociative disorder revealed that these patients had more severe PD than patients without this disorder ($p < .05$). Similarly, patients with more severe symptoms of dissociation disorder had more pronounced PD ($p < .05$)". The authors interpreted these results by stating that "dissociation is a defense mechanism against traumatic memories, and this leads to emotional dysregulation, somatization, anxiety associated with panic attacks and expectation anxiety" (p. 471, 2015).

Ural et al. pointed out that "all of the childhood trauma subscales were correlated with the severity of symptoms of dissociation and PD". They emphasized "the importance of therapies targeting childhood traumatic memories among patients with a high frequency of dissociative symptoms" (p. 471, 2015). They also cited research showing that "a high dissociation level is also a predictor of a worse response to cognitive behaviour therapy in PD (Ball et al. 1997, Gulsun et al. 2007)" (p. 471, 2015).

These data are consistent with the case reports of Goldstein (1995), Nadler (1996), Fernandez & Faretta (2007), Faretta (2013) and Leeds (2009, 2012, 2016) indicating that common predisposing factors to PD involve "very early parent-child role reversals" (Leeds 2012). Leeds (2009, p. 248), therefore suggested that "we might view PDA through the lens of the model of structural dissociation just as van der Hart et al. (2006) suggest we view PTSD. [...] This further strengthens the rationale for considering EMDR therapy as an approach in cases of complex PDA for accessing and resolving hypothesized issues with the IMS – maladaptive memory network – described by Teasdale and Barnard (1993) that fail to respond to exposure and CBT".

4. EMDR treatment of PD and panic attacks: a review of the existing literature

Despite these interesting reports, the state of the research on EMDR therapy of PD is still at an initial phase and consists mainly of case studies (single and series) plus two controlled studies (Feske and Goldstein 1997, Goldstein et al. 2000), and one pilot comparison study (Faretta 2013). This review follows the chronological order of the publications in order to delineate the evolution of what is presently known about EMDR treatment of PD and panic attacks.

4.1. First phase: 1994-2007

The first panic disorder case series was published in 1994 by Goldstein and Feske, who discussed seven cases of PD (five of which also met criteria for Agoraphobia)

treated with five 90-minute EMDR sessions (plus one 60-minute treatment planning session). Their rationale for exploring the application of EMDR to PD did not consider affective-cognitive dissociation: rather, it was based on the observation that "Panic patients almost always report early panic attacks to have been traumatic and their subsequent symptoms are, in some ways, like those experienced by PTSD victims" (Goldstein and Feske 1994, p. 353). With EMDR emerging as an evidence based treatment for PTSD and other trauma- and stressful-related disorders, the authors "were intrigued enough to explore the possible effects of EMDR for panic-related memories on the clinical status of the clients with panic disorders" (1994, p. 353).

The results, based on standardized self-report data and daily self-monitoring records, showed that "all seven patients experienced decreased fear of panic attacks and behavioral gains with an absence of further panic attacks or decreased frequency of panic attacks. The greater gains were in the two patients without Agoraphobia" (Leeds 2009, p. 246). The authors reported considerable variability during the reprocessing phase, with some patients remaining focused on panic episodes, while others associated to experiences prior to the onset of PD, such as adverse childhood memories (e.g. experiencing lack of trust, helplessness or loneliness).

Although the authors underlined that none of the patients treated in the study achieved a complete resolution of PDA in just five sessions, they nevertheless concluded that "EMDR might be a powerful treatment for Panic Disorder" (Goldstein and Feske 1994, p. 360) and called for further, controlled research.

Four single case reports were published after this study, starting with Goldstein (1995) who followed up with an article that focused on complex PDA. This choice was motivated by the observation that "behavior therapy with exposure to feared situations reduced avoidance behavior for only about 50% of those with agoraphobia" (Goldstein 1995, p. 83). In order to go "beyond the barrier" of recovery, Goldstein suggested there was a need to access deeper aspects of the subjects' maladaptive memory network (described as an "implicational meaning schema" by Goldstein, in reference to the Teasdale and Barnard model of 1993) by way of EMDR. Goldstein indicated these deeper aspects involved dissociated adverse early childhood experiences.

He illustrated the procedure through the case report of a subject with complex PDA (co-morbid depression and avoidant personality disorder), successfully treated with approximately 25 sessions consisting of: psycho-education on anxiety and avoidance, coping skills training, interoceptive exposure, 12 sessions of guided in vivo driving exposure, and 9 sessions of EMDR reprocessing. Unfortunately, it is unclear which of these interventions was crucial for the positive outcome of the therapy, but in a case that presented very similar features, Fernandez and Faretta (2007) reported stable treatment gains with EMDR in approximately the same number of sessions without the need to include interoceptive or in vivo exposure.

One year later (1996), Nadler reported a two-session EMDR intervention with a patient with PD. In this case, the premise was quite the opposite of Goldstein and Feske (1994): "Sarah [the patient's conventional name] did not appear to have been traumatized by the panic itself and in spite of her assertions of confidence, her life experiences seemed prime for repressed feelings of loss and fears of imminent independence which would evoke unresolved attachment issues" (Nadler

1996, p. 3). After starting the first EMDR reprocessing session with a focus on recent experience of feeling light-headed followed by panic, childhood memories quickly emerged related to the early death of her mother and consequent feelings of grief and anger for adults' excessive expectations. Similarly, in the second session, the reprocessing started with the recent occurrence of "odd sensations" at work, but quickly shifted to aspects of her past and present relationship with her father, thus corroborating the hypothesis that, at least in some cases, PD expresses the effects of inadequately processed past feelings, attitudes and thoughts. Following these brief interventions Nadler reported that Sarah experienced a significant decrease in anticipatory anxiety and remained panic free during the six months follow-up. Nadler suggested that EMDR "may prove to be a truly patient-centered approach" (1996, p. 5). Since, as Goldstein and Feske (1994) found, "some patients experience desensitization and the alteration of catastrophic cognitions without the emergence of underlying dynamic issues, while others produce memories of earlier trauma or disturbance", then Nadler suggested that the EMDR approach could allow each patient to "engage in the type of therapeutic process needed to recover" (Nadler 1996, p. 5). This conceptualization is consistent with AIP model, which states that the "system" is intrinsically adaptive and capable of self-regulation. According to this premise, given the right conditions, the patient will spontaneously find the most useful level of elaboration for his/her recovery.

Shapiro and Forrest (1997) offered another case report about a brief EMDR intervention for PD that consisted of just two preparation and treatment planning sessions plus three 90-minute sessions of EMDR reprocessing. During the history taking session, the patient (Susan) reported that she had always been fearful of storms, but explained that they became triggers for panic attacks after her first husband died during a tornado. Once again, PD onset appeared to be related to both early contributory experiences as well as to a more recent traumatic event. Her EMDR trained clinician guided the patient to reprocess unresolved grief for her first husband and the irrational self-blame associated with it. She was also able to reprocess sense of threat that for her had characterized storms. As in the reports by Goldstein & Feske (1994) and Nadler (1996), the case of Susan suggested that even brief treatment with EMDR could significantly improve PD symptomatology. The Shapiro and Forrest report is however limited by the absence of standardized measures or follow-up information.

The final case report from the first phase of the research was published by Fernandez and Faretta in 2007. It described the case of a 32-year-old woman, "Adriana", treated with EMDR for PDA that had started when she was 20. In the subsequent eight years, her fears extended from the original fear of driving alone to include avoidance of places where it might be difficult to escape or to receive help (e.g. traffic jams, shopping, elevators). Eventually, she also became afraid of being alone, even at home. The history-taking phase identified several early contributory experiences, as well as more recent etiological events, all of which were included in the treatment plan. EMDR therapy was carried out over a total of 30 sessions: 6 sessions were dedicated to history taking and preparation; 12 sessions were used to reprocess the targets selected from past contributory and etiological events and current triggers; three sessions were used to reprocess rehearsal of future behaviors, and nine sessions were used for reviewing

and consolidating the results of active treatment phase. Follow-up data collected at 3, 6 and 12 months post treatment confirmed the stability of treatment goals achieved that included:

- elimination of anxiety and panic attacks
- resolution of avoidant behaviors
- achievement of independent functioning through the ability to be alone and drive
- elimination of agoraphobic symptoms
- insight and better understanding about symptoms and secondary gains
- development of a new self-perception.

Discussing the results, Fernandez and Faretta emphasized the central importance of an extended preparation phase (6 sessions) in order to gain an adequate understanding of the patient's history, to build a valid therapeutic alliance and to provide the patient with psycho-educational and practical tools to increase emotional self-regulation skills.

This study supported the efficacy of the EMDR approach in the short term, considering that, after the preparation phase, the resolution of panic symptoms was achieved within 4 reprocessing sessions. It also supported the ability of EMDR reprocessing to assist in both the uncovering and resolving of formative, attachment-related experiences that, just as Goldstein (1995) and Nadler (1996) had previously pointed out, appear to play a significant role in the etiology of PDA. Fernandez and Faretta emphasized the importance of developing a treatment plan founded on the EMDR phobia protocol (Shapiro 2011), including an adequate preparation phase and sufficient EMDR reprocessing of targets related to: "(a) events that set the foundation for the pathology; (b) first experience of fear, anxiety, or panic; (c) worst experience; (d) most recent experience; (e) current triggers; and (f) future templates" (Fernandez and Faretta 2007, p. 60). In contrast to the CBT approach, the authors suggest a role for in vivo exposure "only after the etiological events have been processed and the fear largely resolved" (2007, p. 59).

The first controlled study of EMDR treatment for PD was published in 1997 by Feske and Goldstein. Forty-three outpatients with DSM-III-TR (*Diagnostic and Statistical Manual of Mental Disorders*, 3rd Ed., revised; APA 1987) diagnosis of Panic Disorder were included in the study; all but two also met criteria for agoraphobia. Participants were randomly assigned to one of three conditions: (1) six EMDR reprocessing sessions, (2) six sessions of treatment similar to EMDR therapy but without eye movements (*EFER – Eye Fixation Exposure and Reprocessing*), (3) waiting list. The six treatment sessions consisted of one information-gathering session, followed by five EMDR or EFER sessions (one 2-hour and four 90-minute sessions).

The research design developed by Feske and Goldstein failed to compare EMDR with another known effective psychotherapeutic treatment but compared to the same treatment without the eye movements. This was related to the authors' purpose of examining the importance of the eye movements for the outcomes of the therapy. It is possible that the authors' focus on the question of the extent to which eye movements might contribute to EMDR treatment effectiveness led the authors to neglect what constitutes an adequate trial for EMDR treatment of PDA as previously described by the developer of EMDR (Shapiro 2001) and in the study by Fernandez and Faretta (2007).

There were several serious limitations in the research design regarding treatment plan. There was

no preparation phase in the EMDR of EFER condition, and just one history-taking session to identify targets, without a sufficient opportunity to develop a therapeutic alliance. Targets selection focused on “anxiety-provoking memories, such as the first and worst panic attack, life events that the client identified as related to the panic disorder, and anticipated panic episodes” (Feske and Goldstein 1997, p. 1028). However, targeting of current triggers, such as unpleasant body sensations – an essential element of a complete EMDR treatment plan – were deliberately excluded on the grounds that this would be similar to *in vivo* exposure. There was no effort to identify or target possible adverse childhood experiences such as separations, traumas, or inappropriate parental interactions.

The results at post-test indicated those in the EMDR condition showed more gains than the waiting-list on all measures, and all the effect sizes were large. Compared with EFER’s, EMDR clients improved more on two of five primary measures: log Agoraphobia-Anticipated Panic-Coping and Generalized Anxiety-Fear of Panic. “However, EMDR was no more effective than EFER in reducing Social Concerns-General Anxiety, Physical Concerns, or log panic frequency” (1997, p. 1029). Subjects in the EMDR condition did not maintain their initial improvement on the first two measures three months after the treatment.

Feske and Goldstein concluded that, while their study “is the first to demonstrate EMDR’s advantage over a WL procedure in the treatment of an anxiety disorder other than PTSD [...], these findings do not indicate whether EMDR effects are greater than those of a credible placebo or as large as those of other treatments for panic disorder with extensive evidence bases” (1997, p. 1033).

In spite of a research design that failed to compare EMDR to a previously recognized first-line treatment such as exposure therapy, Feske and Goldstein recommended that EMDR should not be used as a first-line treatment for PDA. Methodological limitations of the Feske and Goldstein study suggest caution in the interpretation of their results. While the authors provided good evidence for procedural fidelity within the EMDR reprocessing sessions (phases 3, 4, and 5) the overall treatment plan and the number of sessions did not meet the treatment standards set by the authors of the previous successful single case reports (Fernandez and Faretta 2007, Goldstein 1995, Nadler 1996, Shapiro and Forrest 1997) and suggested by EMDR’s developer (Shapiro 2001).

According to the behavioral literature, 7 to 15 sessions of CBT are needed to achieve stable results among PDA population (Nadler 1996). A recent meta-analysis by Cuijpers et al. (2016) confirms this data, indicating that 6 to 15 CBT sessions are effective for the treatment of Panic disorder, with an average of 9,75 sessions. While EMDR may ultimately be found to be a more efficient treatment for PDA than CBT, a research design without a preparation phase and only five EMDR reprocessing sessions of such limited scope can hardly be considered a valid test EMDR’s effectiveness for PDA.

The second controlled study of EMDR on PDA was published by Goldstein et al. in 2000. The research design revisits limitations in Feske and Goldstein’s 1997 study, with 46 PDA patients randomly assigned to a waiting-list, EMDR treatment or a credible placebo control condition known to be ineffective for PDA. The placebo condition, “association and relaxation therapy (ART) consisted of 30-45 minutes of progressive muscle relaxation training followed by

30-45 minutes of association therapy. The question of the intent behind this research design arises concerning the choice of contrasting an EMDR condition with a placebo condition, where “a more useful study would have directly compared EMDR to a credible alternate known effective treatment such as cognitive therapy or exposure” (Leeds 2009, p. 253).

Treatment in Goldstein et al. (2000) consisted of six 90-minute sessions. The first session was dedicated to assessment – information gathering regarding symptoms, history and course of the disorder, as well as descriptions of memories of the first and the worst panic attack. The other five sessions were devoted to treatment with either EMDR reprocessing or ART. “Throughout treatment, therapists in both conditions were prohibited from using interventions outside the realm of the protocol such as anxiety management training, cognitive restructuring, *in vivo* exposure, and exploration of intrapsychic issues” (Goldstein et al. 2000, p. 949), thus this research design also omitted the preparation phase and the selection of targets related to adverse early experience that had been found to be crucial for the resolution of PDA symptoms in several of the previous individual case reports on PDA.

Not surprisingly, the results of this study were even more critical regarding the EMDR condition. “EMDR was significantly superior to waiting list on panic/agoraphobia severity and on the diary factor ($p < .05$) but not on the cognitive factor, controlling for pretest panic frequency ($p > .10$). Furthermore, EMDR did not show greater improvement over waiting list on number of panic attacks ($p > .10$),” (Goldstein et al. 2000, p. 951). In comparing the EMDR condition with the ART condition, “No differences between treatment groups emerged ($p > .10$).” (2000, p. 951) and EMDR patients “fared no better than those in the attention-placebo group” (2000, p. 953). These null results were stable in the brief (1 month) follow-up.

The authors concluded by stating that “In light of the availability of treatments with solid efficacy evidence, the results of this investigation do not support the use of EMDR for treatment of panic disorder with agoraphobia” (2000, p. 955). On the other hand, in seeking to explain the weaker findings in Goldstein et al. (2000) compared both to those of Feske and Goldstein (1997) and Goldstein (1995), Goldstein later stated that “The first order of business in therapy is to provide a lot of structure, reassurance and to focus on concrete anxiety management skills. In the early stage of therapy, perhaps they [PDA patients] are not ready to engage in a process that is as emotionally provocative as EMDR” (Shapiro 2001, p. 363). This observation appears especially relevant in the light of the fact the Goldstein et al. (2000) study’s selection criteria included patients with a more severe agoraphobia than those of the 1997 research by Feske and Goldstein.

The two controlled studies by Feske and Goldstein (1997) and Goldstein et al. (2000) were later included in a meta-analysis conducted by Bandelow et al. (2015), which stated that EMDR therapy showed lower pre-post effect sizes than Mindfulness and other relaxation treatments for the resolution of anxiety disorders. Considering that these two studies achieved good fidelity to the standard EMDR procedural steps (for phases 3, 4, and 5) but failed to offer an adequate treatment plan in terms of (a) an insufficient number of sessions needed for preparation and development of rapport, (b) neglecting adverse childhood experience related to PDA symptomatology, (c) avoided reprocessing current stimuli and triggers such as unpleasant physical sensations, and (d) omitted future templates, Bandelow

et al. (2015) statements about EMDR therapy's (lack of) efficacy need to be considered with skepticism.

4.2 Second phase (2009-2016)

Based on a review of the clinical data summarized in the previous findings, Leeds (2009) developed two model treatment plans for EMDR therapy for PD and PDA. The first (Model I) was indicated for the cases of PD without Agoraphobia, or any other co-occurring anxiety disorder such as GAD, or OCD, or Complex PTSD (DESNOS), or a personality disorder. The second (Model II) was recommended for cases of PD with Agoraphobia, or PD with a co-occurring anxiety disorder such as GAD, or OCD, or Complex PTSD (DESNOS), or a personality disorder.

There are three main differences between the Model I and Model II treatment plans. The first difference is in the preparation (Phase 2), which may need to be more extended in Model II treatment plans. One or more resources may need to be installed for self-soothing, self-acceptance, or connection before or after beginning reprocessing core maladaptive memory networks of etiological experiences from childhood. The second difference involves decisions about when to start reprocessing early contributory targets of perceived abandonment, misattunement, humiliation, fear, and early parent-child reversals. In the Model II treatment plan these early targets are only addressed after patients have made symptomatic gains with reductions in the frequency and severity of their panic attacks and when patients acknowledge the essential relevance of addressing these early contributory memories and are ready to do so. The third difference involves an option for the installation (Phase 5). With PDA patients, when the desensitization (Phase 4) is incomplete and when the SUD has dropped, not to a 0 or 1, but only to a 2, 3, or 4, Leeds suggests (2009, p. 265) clinicians consider the option of moving on to an abbreviated installation (Phase 5) and installing a modified positive cognition.

In the final phase of treatment Leeds (2009, p. 260) suggests that in addition to the use of the Future Template clinicians consider installing "one or more resources to represent emergence and consolidation of new sense of self, free of avoidance of core maladaptive memory network".

Leeds (2009, 2016) presents two case examples treated with EMDR for Panic disorder without Agoraphobia. Both cases – Hannah, 17 years old and Justin, 20 years old – presented with subclinical social anxiety, and both had contributory experiences in childhood involving chronically stressful relationship with a caregiver. These two characteristics led to the choice of Model II treatment plan.

Hannah had been suffering from panic attacks since one year. The episodes took primarily place in the classroom and especially during exams. The onset of the disorder went back to the previous spring, while she was living in New York with her mother and stepfather.

Over the course of the sessions emerged the picture of a troubled relationship between Hannah and her mother. Hannah explained that she had a stomach ulcer the previous year. She had complained of stomach pains, nausea and sudden urges to vomit, but her complaints were minimized by her mother. Only when Hannah's symptoms got worse was her disease eventually taken seriously and cured, but soon afterward she developed PD symptoms. After twelve sessions (2 for history taking and preparation, 5 for the reprocessing of panic attacks, 2 for the reprocessing of contributory

experiences, and 3 for reevaluation and consolidation), Hannah made a full recovery from the panic attacks.

In accordance with the preliminary findings of Nadler (1996), Shapiro & Forrest (1997) and Fernandez & Faretta (2007), Hannah's case highlights the rapidity of EMDR therapy effects that can occur in cases of PD, even when it presents co-morbidity with another anxiety disorder (in this case, subclinical SAD) and a C type personality disorder (in this case, Avoidant personality disorder). On the other side, it illustrates the interrelations between etiological and contributory experiences in the development of PD: "Although she had many stressful experiences with her mother in her early years [contributory factors], Hannah had never suffered from panic attacks until she became ill with an ulcer that made her vulnerable to sudden unexpected bouts of nausea and vomiting [etiological factors]" (2009, p. 285). The distinction is relevant in order to delineate an appropriate treatment plan: on the basis of this case formulation, it was possible to start treating Hannah's PD by targeting and reprocessing the *etiological* experiences (symptoms of stomach ulcer) and the triggers associated with panic attacks, and then address the *contributory* experiences in the early relationship with her mother.

The second case report (Leeds 2009) is about Justin, a junior college student who had experienced panic attacks and insomnia for 8 years. The history taking session pointed out some relevant information about Justin's past: his parents divorced when he was 4 and since then he had lived with his mother and stepfather, with whom he had "a difficult relationship, for a while" (2009, p. 286). The therapist started to develop a treatment plan with the idea to first address Justin's earliest memory of a panic attack but this hypothesis was discarded at the 2nd session, when Justin – who had a depersonalization episode that same day – referred to a bad motorcycle accident he had at the end of previous year. While he reported no physical damage after flying through the air after hitting a car and landing in a shopping cart corral, he did experience a strong peri-traumatic episode of depersonalization. After this his anxiety worsened, and he began to experience depersonalization episodes. The symptomatology change after the motorcycle crash made apparent that Justin met criteria for both preexisting PD and Posttraumatic stress disorder (PTSD).

The treatment plan was modified in accordance with the new diagnosis, giving priority to the recent trauma of the crash in order to address the new and debilitating symptom of depersonalization. Pre-existing panic attacks were postponed until after this first target was resolved. After 18 sessions, Justin reported full recovery from both PTSD and PD symptoms. In the follow-up session that took place a month later the results were stable. The author interprets this positive result as derived from a case formulation built on the Adaptive Information Processing (AIP) model, that guided the non chronological sequencing of targets. "Only after significant gains on the worst symptom of depersonalization did we focus attention on addressing the history of his panic attacks. Later, we returned to addressing his residual motorcycle-driving anxiety" (2009, p. 294).

These two case reports suggest that EMDR therapy of complex PD can be effective using the Model II approach, with a gradual transition to reprocessing contributory events. Given these conditions, EMDR therapy can sometimes be effective in a relatively small number of sessions (seven reprocessing sessions, in Hannah's case). The author concluded by underling the

importance of providing sufficient preparation and trust building when treating cases of complex PD.

The first study to compare EMDR therapy with an evidence-based treatment for PD (CBT) was published in 2013 by Faretta. This pilot comparison study (N=19) contrasted 12 EMDR sessions with 12 CBT sessions by means of four outcome measures that were administered at pretreatment, posttreatment, and 1-year follow-up. Participants met DSM-IV-R criteria for PD with or without Agoraphobia; they were excluded if they presented with psychological comorbidity (complex PD) or serious somatic diseases. The four measures employed were: State-Trait Anxiety Inventory (STAI-Y1; Spielberger 1989); Panic-Associated Symptom Scale (PASS; Argyle et al. 1991); Marks-Sheehan Phobia Scale (MSPS; Sheehan 1983); Symptom Checklist-90-Revised (SCL-90-R; Derogatis et al. 1973).

Assignment to EMDR or CBT treatment was not random because patients spontaneously selected therapists by using internet resources to look for professionals that could help them (on the EMDR Italy Association Website or the Associazione Italiana di Analisi e Modificazione del Comportamento e Terapia Comportamentale e Cognitiva [AIAMC] Website). Among the seven therapists involved, three used CBT and the other four used EMDR. All therapists were overseen by an experienced supervisor (a CBT or an EMDR supervisor) to monitor fidelity of treatment.

EMDR therapy followed the 8-phase model described by Shapiro (1999, 2001) with the following additions (Fernandez and Faretta 2007; Leeds 2009, 2016): during Phase 2 (Preparation), psychoeducation on panic was included; as regards Phase 3 (Reprocessing of targets), past events included background stressors to first panic attack (if any were identified), first panic attack, worst panic attack, most recent panic attack, contributory childhood experiences of perceived abandonment, humiliation, fear, and early parent-child reversals. Reprocessing of current stimuli focused on external and internal cues associated with panic attacks. Future templates (for coping with external and internal cues) were implemented in order to help the patients rehearse successfully coping with these triggers in the future. For CBT patients, the specific guidelines for Panic disorder (NICE 2011) were followed. CBT intervention included: a diagnostic and assessment phase; a psychoeducation phase; a relaxation and breathing techniques phase; an imaginal exposure phase; a generalization phase, and homework.

The results indicate that 12 sessions of therapy with either EMDR or CBT were effective in the treatment of PD with or without Agoraphobia. At the end of the 12 sessions, no subject in the EMDR group still met criteria for PD and only one subject in the CBT group did. As regards Agoraphobia, initially two subjects in the EMDR group and five in the CBT group at pretreatment met criteria for Agoraphobia. After 12 sessions, no patient in the EMDR group or in the CBT group met full criteria for Agoraphobia, although some agoraphobia symptoms were still reported within the CBT group. The results of this study suggested that the treatment of PD (both with and without agoraphobia) with EMDR appears to be as effective as CBT, and that EMDR may be more effective in reducing frequency of panic attacks. "The EMDR group showed a decrease in the number of panic attacks [per week] from pretreatment $M = 1.47$ (0.59) to posttreatment $M = 0.10$ (0.22), maintained at the 1-year follow-up $M = 0.00$ (0.00); similarly, the CBT group showed a decrease in numbers of attacks from pretreatment $M = 1.19$ (0.72)

to posttreatment $M = 0.59$ (0.49), also maintained at follow-up $M = 0.56$ (0.47). A repeated measure ANOVA showed significant TimeXTreatment interaction for PASS (frequency of panic attack), $F(1,217, 20.695) = 7.119$, $p = .011$, $\eta_p^2 = .295$, which indicated that EMDR group differed in how the frequency of panic attacks changed over time (T0, T1, T2)" (Faretta 2013, pp. 127-128).

The stability of results in the EMDR condition over time stands in contrast to the findings of controlled study by Feske and Goldstein (1997), which stated that the positive EMDR posttreatment effects were not maintained at a briefer (3 months) follow-up. Also in contrast to Feske and Goldstein (1997) and Goldstein et al. (2000) findings, Faretta's pilot study tracked "a continuing decrease in frequency of panic attacks for participants with PD or PDA in the EMDR condition at follow-up that was significantly greater than that found in the CBT treatment group" (2013, p. 131).

A potential explanation to these diverging outcomes may be found in the differences in the development of the treatment plans in the EMDR condition. In accordance with Fernandez and Faretta (2007), Leeds (2009, 2012, 2016), and Nadler (1996), Faretta's findings corroborate the hypothesis that possible adverse and/or stressful early experiences represent a pivotal aspect in the treatment of PD. The Faretta (2013) EMDR condition treatment plan included reprocessing of early contributory experiences, as well as desensitization of later etiological factors. It also included an extended preparation phase and rehearsal through the future templates.

A recent case report by Bhagwagar (2016) also supports the theoretical framework and EMDR treatment model for PD. The purposes of the Bhagwagar study were: (1) to validate Leeds's Model II for EMDR treatment of PDA; (2) to test the hypothesis that resolving adverse childhood experiences can resolve PDA; (3) to verify the maintenance of EMDR treatment effects at 6 months, 1 year, and 5 years. The patient, Pam, was a 30-year-old working professional who met criteria for PDA (DSM-IV-TR, 2000). She was referred by a physician from a local hospital, who had conducted multiple medical tests to rule out the possibility of a cardiac disorder. Her presenting complaints involved mood problems (sadness and lethargy), sleep disturbance, and somatic symptoms, including palpitations, tremors, sweating, chest pains, and dizziness, which could not be explained by medical examinations. Two months before being referred for EMDR treatment, Pam experienced a panic attack for the first time while traveling to work by public transport. She thought she was having a heart attack and sought medical intervention. She continued to experience similar physical sensations every few days, and over the next two months she developed anticipatory anxiety, especially when she had to travel. Pam was treated by the author in a private practice setting for a total of 17 sessions, which were structured in accordance with Leeds's Model II treatment plan. History taking (Phase 1) revealed adverse childhood experiences with an alcoholic father as contributory factors to the development of PDA. Pam's panic attacks were apparently triggered by exposure to current drinking by Pam's husband (etiological factors) which activated maladaptively encoded adverse memories from Pam's childhood related to her father's drinking.

The preparation phase (2) extended from session two to six and included psychoeducation on panic and EMDR, filling out a daily anxiety chart and thought log, in-session analysis of the chart and log reports, teaching

of diaphragmatic and square breathing, sensory focusing, the calm/safe place exercise, Resource Development and Installation (RDI) and a family session for education on anxiety management skills.

Selected memories were reprocessed with EMDR therapy from session seven to ten. Following Leeds's model II treatment plan, the first memory to be reprocessed was the experience that therapist and patient recognized as the background stressor antecedent to the first panic attack – finding husband drunk and passed out in the apartment basement. The ninth session involved the successful reprocessing of the memory of the first panic attack, which was also reported as the worst episode. The PC from this session “I can handle it” reached a VoC of 7, and was used to reinforce the installation of positive future templates traveling on the train or using other forms of local city transport. In the tenth session, they addressed a recent panic attack, while home alone. After this, the patient took a 3-week break to reestablish her working routine. When she returned, Pam reported more stability and less anxiety. She stated that her panic attacks were greatly reduced, both in frequency and intensity, but that “she still had notable disturbance around her husband’s drinking” (2016, p. 268). Therapist and patient decided it was time to address contributory childhood experiences, and these were reprocessed in sessions 11 through 15. After some additional preparatory work with RDI, a memory of an episode related to her father’s drinking and parent-child role reversal was targeted. This memory was not completely reprocessed, and following this session Pam experienced a major panic attack while she was at work.

After some further stabilization work in session twelve, reprocessing the memory of her father coming home in a drunken rage and breaking household furniture was completed in session thirteen with the help of interweaves. For example, when reprocessing was ineffective her therapist asked questions in order to help her to differentiate between the duties of an adult and of a child. With completed desensitization, the patient had insights that led to a revised PC “I had no choice then, but I have a choice now”, which was installed with a VoC rating of “7”. Reprocessing of other memories involving parent-child role reversal was completed in sessions fourteen and fifteen. Session sixteen was dedicated to current triggers, including both external and interoceptive cues. In the seventeenth session therapist and patient continued with future templates to resolve areas of residual avoidance. Pam was asked to imagine approaching fearful situations such as traveling on the local train, having guests socially drinking at her home, and so on.

After this session, Pam reported feeling calm, cheerful and confident both at work and with her family. She was panic-free for a month and reported an absence of significant agoraphobic symptoms. These results were stable over time and, similarly to Faretta’s findings (2013) at the 1-year follow-up, actually showed further positive trends in the follow-ups at 6 months, 1 year and 5 years long after the end of the treatment including a growing sense of self-confidence. This trend may be understood as another benefit of EMDR therapy that, in accordance with AIP model, “resets” the system and reinstates its natural function, that is the cognitive-affective integration of the meaning of life experiences.

Discussing the results, Bhagwagar underlines the value of the “three-pronged” EMDR approach, which strategically interpolates the time-line of selected targets into the development of the treatment plan. “Sequencing targets in a manner that allows for initially reprocessing background stressors, followed by the

first, worst, and most recent panic attacks and finally targeting of childhood contributory events allows a chain of associations to emerge spontaneously, thus permeating the entire memory network” (2016, p. 270). The future template was used at several points in the treatment plan on situations that could arise in the future. Imagining how she could feel, act, and respond in a feared situation, the patient transferred her newly acquired perspective and management skills to the future. “Reduction of such avoidance probably allowed the new learning to start integrating rapidly into an overall positive schema and emerging sense of self” (2016, p. 271).

The emphasis in Bhagwagar (2016) on implementing the full three-pronged approach is in accordance with Nadler (1996), Fernandez & Faretta (2007), Faretta (2013), and Leeds (2009, 2012, 2016) who all recommend the inclusion of contributory experiences and future templates in the treatment plan. Based on these preliminary findings, it appears that EMDR therapy for PD and complex PD can be efficacious and can produce stable gains as long as it is applied with the full three-pronged protocol incorporating all eight phases originally described by Shapiro (2001), and successively adapted by Leeds (2009, 2016) to include sufficient preparation and to address early contributory experiences when the patient is ready.

5. Clinical implications and recommendations for future research

The state of the research on EMDR treatment of PD is still at an early stage. In the first phase (1994-2007) there was one case series (Goldstein and Feske 1994), four case reports (Goldstein 1995, Nadler 1996, Shapiro and Forrest 1997, Fernandez and Faretta 2007), and two controlled studies (Feske and Goldstein 1997, Goldstein et al. 2000). The second phase (2009-2016) includes two case examples (Leeds 2009), one pilot comparison study (Faretta 2013), and one case study (Bhagwagar 2016).

The pilot study, and single and series of individual case reports suggested that EMDR therapy is effective in eliminating symptoms of panic and agoraphobia, whereas the two published controlled studies (Feske and Goldstein 1997, Goldstein et al. 2000) failed to show comparable outcomes. Evaluating these discrepancies, an analysis of the two controlled studies reveals significant problems in the design and implementation of the treatment plan that fails to incorporate indispensable elements of the AIP model. In both studies, the selection of the targets focused exclusively on the memories of panic attack experience *per se*. First and worst panic attacks, life events related to panic (background stressors), and anticipated panic episodes were addressed, whereas targets related to triggers – especially internal cues – and adverse childhood experiences – especially those related to attachment issues – were neglected. In addition, insufficient attention was paid to the development of the therapeutic alliance, to skills building, and to rehearsal of future confrontation with feared situations.

A different case formulation informed Faretta’s pilot comparison study (2013), as well as the case reports by Nadler (1996), Shapiro and Forrest (1997), Fernandez and Faretta (2007), Leeds (2009), and Bhagwagar (2016). In all these reports, after reprocessing of targets related to panic attacks, contributory childhood experiences were also addressed. This is in accordance with Shapiro’s (2001) description of the AIP model,

which proposes that maladaptively encoded stressful childhood experiences form the basis for impaired resiliency in the processing of later occurring stressful events, possibly leading to different kinds of disorders (among the others, Anxiety disorders, Trauma- and stress-related disorders, Dissociative disorders).

The inclusion of the contributory factors in the reprocessing phase, together with an extended preparation phase, addressing current triggers, and implementing future templates, define the treatment plans applied to PD and PDA case reports with positive outcomes. Moreover, the stability of these results over time contrasts with the lack of stability of the slight EMDR improvement found by Feske and Goldstein (1997) at the three-months follow-up, suggesting that EMDR “three-pronged” approach is crucial for a complete recovery from PD. In other words, in order to gain positive and stable results with EMDR therapy, the diachronic temporal perspective including past, present, and future seems to be as relevant as the synchronic one (the experiences of panic attack and their effects in the here and now).

The “three-pronged” approach does not require prolonged time for treatment but does require a carefully planned therapeutic intervention. When working on the basis of a comprehensive case formulation based on the AIP model, the positive case reports and pilot comparison study indicate that a number of sessions between 12 and 19 is usually effective, with a medium of six reprocessing sessions per therapy.

Considered as a whole, the scientific literature regarding EMDR therapy of PD/A indicates the need to pursue the well-designed research on the topic, as the preliminary data suggests EMDR therapy to be effective not only in reducing the frequency of panic attacks but also in accomplishing a complete and lasting recovery from the disorder. In particular, further controlled studies are needed to systematically evaluate the efficacy of EMDR therapy for PD: these researches should have statistically significant samples, random assignment to treatment conditions, comparison with a first line treatment, and supervision provided by an independent evaluator for every tested condition. In order to monitor the stability of EMDR effects over time, research designs should call for follow-ups at six months, one year, and possibly more.

Because CBT is presently considered a first line treatment for PD, controlled comparisons between EMDR therapy and CBT would be especially useful. Given the hypothesized role of dissociation in the etiology of complex cases of PD and PDA, it would also be interesting for research designs to include among the independent variables the degree of subjects’ affective-cognitive dissociation. EMDR efficacy on simple as well as complex PD (comorbidity with Agoraphobia, or a co-occurring Anxiety disorder such as GAD and SAD, or PTSD, or Depersonalization/Derealization disorder, or a type C Personality disorder) should also be comparatively evaluated. Since the positive case reports and the positive pilot controlled study were based on the principles described in Leeds’s Model II EMDR adapted protocol (2009, 2016), it should be employed as a standard in future controlled comparison studies.

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