Psychotherapy Research

ISSN: 1050-3307 (Print) 1468-4381 (Online) Journal homepage: http://www.tandfonline.com/loi/tpsr20

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**To cite this article:** Richard Gray, Denise Budden-Potts & Frank Bourke (2017): Reconsolidation of Traumatic Memories for PTSD: A randomized controlled trial of 74 male veterans, Psychotherapy Research, DOI: 10.1080/10503307.2017.1408973

**To link to this article:** https://doi.org/10.1080/10503307.2017.1408973

Published online: 14 Dec 2017.

*Psychotherapy Research*, 2017
https://doi.org/10.1080/10503307.2017.1408973



**EMPIRICAL PAPER**

# Reconsolidation of Traumatic Memories for PTSD: A randomized controlled trial of 74 male veterans

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*(Received 28 March 2017; revised 18 September 2017; accepted 13 November 2017)*



**Abstract**

**Design:** A randomized waitlist-controlled design ($n = 74$) examined the efficacy of Reconsolidation of Traumatic Memories (RTM) among male veterans with current-month flashbacks and nightmares. Volunteers were randomly assigned to immediate treatment (three 120-minute sessions of RTM), or to a 3-week waiting condition before receiving the RTM treatment. Blinded psychometricians evaluated the symptoms at intake, 2 weeks, and 6 weeks post. Wait-listed participants were re-evaluated and then treated. Sixty-five volunteers completed the treatment. **Results:** Of those treated, 46 (71%) lost DSM diagnosis for post-traumatic stress disorder (PTSD) by one of the following definitions: 42 persons (65%) were in complete remission (PTSD Symptom Scale Interview (PSS-I) $\leq 20$ and DSM criteria not met). Four others (6%) lost the DSM diagnosis or were otherwise sub-clinical by dichotomous criteria (PSS-I $< 20$ and absence of flashbacks and nightmares) but non-ambiguous on the PTSD Checklist Military Version measures. Within-group RTM effect sizes (Hedges' $g$) for PSS-I score changes ranged from 1.5 to 2.2. The between-group comparison between the treatment group and the untreated controls was significant ($p < .001$) with an effect size equivalent to two standard deviations ($g = -2.121$; 95% CI [−4.693–0.453]). Patient satisfaction with the intervention was high. **Conclusions:** RTM shows promise as a brief, cost-effective intervention for PTSD characterized primarily by intrusive symptoms.

**Keywords:** post-traumatic stress disorder (PTSD); randomized trials; reconsolidation; waiting list

**Clinical or methodological significance of this article:** The article provides evidence to support a fast (5 hours or fewer) robust intervention for PTSD characterized by intrusive symptoms including current-month flashbacks, nightmares, and accompanied by sympathetic arousal in response to trauma narratives. The intervention is well tolerated and has demonstrated efficacy up to one year.

## Introduction

### Current Interventions for Post-traumatic Stress Disorder

The United States Veterans Health Administration (VA) currently supports several psychotherapy interventions for post-traumatic stress disorder (PTSD) at its medical centers. These include Prolonged Exposure (PE) and Cognitive Processing Therapy (CPT (Bisson, Roberts, Andrew, Cooper, & Lewis, 2013; Fernández, Bavassi, Forcato, & Pedreira, 2016; Goetter et al., 2015; Goodson et al., 2011; Resick, Williams, Suvak, Monson, & Gradus, 2012; Steenkamp & Litz, 2013; Steenkamp, Litz, Hoge, & Marmar, 2015). These treatments have well-documented efficacy for both male and female veterans including combat, sexual, and other traumas. Several other less well-supported treatments are also provided in VA facilities (Eftekhari et al., 2013; Monson et al., 2006; Schnurr et al., 2007; Tuerk et al., 2011). Finley et al. (2015) found that PE and CPT account for up to 50% of treatment hours for VA outpatient treatment providers in Veterans Affairs outpatient clinics. Nevertheless, lengthy treatment regimens and other difficulties, including negative expectations and perceptions of trauma-focused cognitive behavioral treatments (TFCBTs) make completion difficult, resulting in high dropout rates

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(Kim, Britt, Klocko, Riviere, & Adler, 2011; Najavits, 2015; Pietrzak, Johnson, Goldstein, Malley, & Southwick, 2009; Steenkamp et al., 2015). Treatment efficacy in most studies is measured in modest reductions in symptoms scores with treatments providing low rates of recovery from the PTSD diagnosis. In many studies, the number of persons who have lost or retained the diagnosis is not reported (Bisson et al., 2013; Steenkamp et al., 2015; Steenkamp & Litz, 2013). There is a continuing call for innovative approaches to the treatment of PTSD (Barrera, Mott, Hofstein, & Teng, 2013; Bisson et al., 2013; Fernández et al., 2016; Goetter et al., 2015; Goodson et al., 2011; Steenkamp et al., 2015; Steenkamp & Litz, 2013). Here, we report a randomized controlled trial (RCT) of 74 male veterans employing the Reconsolidation of Traumatic Memories (RTM) protocol, a new treatment for PTSD.



## The RTM Intervention

RTM is a brief, systematic, non-traumatizing, TFCBT derived from neuro-linguistic programming (NLP) techniques. It is closely related to the Visual Kinesthetic Dissociation protocol (Gray & Liotta, 2012) and the Rewind Technique (Muss, 1991, 2002). It is distinct from them in its intentional reliance upon the syntax of reconsolidation to enhance outcomes (Gray & Bourke, 2015; Gray, Budden-Potts, & Bourke, 2016; Gray & Liotta, 2012; Tylee, Gray, Glatt, & Bourke, 2017). RTM may represent an alternative to current cognitive and extinction-based interventions (Gray & Bourke, 2015; Gray & Liotta, 2012). RTM focuses upon the PTSD symptoms that are expressed as immediate, phobic-like responses to triggering stimuli (flashbacks) and repeated nightmares, or night terrors that regularly disrupt sleep and often make it impossible to return to sleep in a normal manner. Nightmares and flashbacks must be related to one or a few identifiable traumatic incidents. Patients without significant intrusive symptoms related to identifiable traumatic exposure are inappropriate to the intervention (see "Methods" for further explanation).

According to Schiller and Phelps (2011), the syntax of reconsolidation requires the following elements in the described order: (1) reactivation of a consolidated, long-term memory using a brief or unreinforced reminder cue; (2) after reactivating the memory, apply the treatment or treatments that are aimed at modifying the memory; and (3) test for the effects of the intervention(s) after the end of the treatment(s) and after the window of labilization has closed (typically 24 hours). RTM uses the same basic paradigm or syntax, even though it is usually implemented over a period of several treatment sessions: (1) a consolidated trauma memory is reactivated using a brief or incomplete narrative as the activation cue; (2) after reactivating the memory, treatment or treatments are applied that are aimed at modifying the memory. In our case, these are structural changes related to safety information about the memory content; (3) test for the effects of the intervention(s) after the end of the treatment(s) and after the window of labilization has closed (typically 24 hours). In RTM, this is done by having the client retell the narrative (in part or in full depending on how far the treatment has proceeded) and providing a Subjective Units of Distress (SUDs) score for the new retelling (Gray & Bourke, 2015; Nader & Einarsson, 2010; Tylee et al., 2017).

The RTM protocol initiates each treatment session with a brief, controlled retelling of the target trauma. That narrative is stopped as soon as signs of sympathetic arousal are observed (e.g., changes in breathing position, muscular tone, lacrimation, flushing, voice tone, etc.). Besides ensuring that the patient is not re-traumatized by the exposure, this interrupted evocation is expected to initiate the reconsolidation mechanism (Agren, 2014; Forcato et al., 2007; Gray & Liotta, 2012; Kindt, Soeter, & Vervliet, 2009; Lee, 2009; Schiller et al., 2010; Schiller, Kanen, LeDoux, Monfils, & Phelps, 2013; Schiller & Phelps, 2011). In RTM, the reminder takes the form of a limited exposure to the trauma memory as described by Lee, Nader, and Schiller (2017). Research regarding this modification considered that an intervention for PTSD might require a stimulus of broader applicability than a simple Conditioned Stimulus (CS) – Conditioned Response (CR) response system. The successful use of modified Unconditions Stimulus (UCS) presentations to evoke the reconsolidation mechanism has been used to treat (as cited by Lee et al., 2017): Conditioned fear in rats using a shock of lesser intensity than the original UCS shock to trigger labilization (Liu et al., 2014); Luo et al. (2015) who illustrated the same capacity of a priming (limited) dose of cocaine to block reconsolidation in addicted rats thereby reducing cocaine seeking; Dunbar and Taylor (2016) who hypothesized that PTSD sufferers whose initial trauma was their victimization in Hurricane Katrina, experienced reduced PTSD symptoms after exposure to a less violent Hurricane; and Xue et al. (2017), in a smoking reduction study using limited exposure to nicotine to evoke cravings in their test of a reconsolidation disruption model using propranolol. In all cases, the limited presentation of the UCS successfully evoked labilization.

In accordance with experimental frames developed in preclinical literature, this brief, incomplete, or unreinforced reminder is believed to render the traumatic memory subject to change for a period of from 1 to 6 hours (Nader, Schafe, & Le Doux, 2000; Schiller et al., 2010). After termination of the narrative exposure, and ensuring that the client is calm and fully oriented to present circumstances, those patients are then presented with healthy dissociative distancing experiences and exercises that dereify the present experience of the target event. These exercises are hypothesized to modify the perceptual structure of the remembered traumatic event. As these changes provide relevant, new information about the target event, and its current level of threat, it is believed that, in accordance with reconsolidation theory (Agren, 2014; Fernández et al., 2016; Forcato et al., 2007; Gray & Liotta, 2012; Kindt et al., 2009; Lee, 2009; Schiller et al., 2013; Schiller & Phelps, 2011), those changes are incorporated into the structure of the target memory. After treatment, the event becomes available to declarative memory without evoking the strong pathological emotion characteristic of PTSD. Partial memories are often restored to more complete narratives and the perspective within the memory typically shifts to a more distant, third-person status. Except for the widening of scope, a change in perspective, and the addition of forgotten details, memory content remains unchanged.

Although the content change in the narrative has not yet been objectively measured, nearly all clients and clinicians observe an increase in detail and fluidity as the treatment progresses. The appearance of spontaneous reappraisals of the meaning of the target memory in the client's life story is typically observed after significant reductions in SUDs and increased restoration of detail. This suggests that such reappraisals are a result of the treatment, not its cause (Gray et al., 2016; Gray & Bourke, 2015; Gray & Liotta, 2012; Kindt et al., 2009; Schiller & Phelps, 2011; Tylee et al., 2017).

The RTM protocol is distinct from other TFCBTs, in that exposure to the trauma memory is not the central effector of treatment change. Here, the brief exposure narrative serves to initiate a period during which the structure of the trauma memory is destabilized and during which new information can be incorporated into the structure of the target memory (Agren, 2014; Fernández et al., 2016; Forcato et al., 2007; Gray & Liotta, 2012; Kindt et al., 2009; Lee, 2009; Schiller et al., 2013; Schiller & Phelps, 2011). RTM is not an exposure or extinction-based intervention. Previous examinations of the RTM protocol (Gray & Bourke, 2015; Tylee et al., 2017) have been careful to distinguish between the neurological mechanism of creating extinction memories and the use of extinction training, using unreinforced presentations of the trauma stimulus. The two are different and we believe that the concepts are often wrongly used interchangeably. The bulk of early reconsolidation studies used extinction training within the reconsolidation window. In these cases, the result represented new learning about the original memory, not enhanced extinctions (for a review, see Auber, Tedesco, Jones, Monfils, & Chiamulera, 2013). As indicated by temporal boundary conditions, divergent protein synthesis pathways and other distinctive features, most authors have concluded that reconsolidation is not extinction (Alberini, 2005; Johnson & Casey, 2015; Kredlow, Unger, & Otto, 2016; Monfils, Cowansage, Klann, & LeDoux, 2009; Nader & Einarsson, 2010; Pedreira, Perez-Cuesta, & Maldonado, 2004).

Differentiating RTM from extinction more specifically, we note that there is no extended conscious focus upon the index memory insofar as it continues to evoke observable autonomic response. Those responses are terminated as soon as they are observed. Throughout the movie procedure (as detailed below), the trauma memory is presented through three levels of dissociation, and perceptual modifications are applied to ensure or reinforce the memory's loss of immediacy. At every instance where the client expresses discomfort, whether verbally or non-verbally, the process is immediately interrupted (see Table I).

According to Pedreira et al. (2004), (1) neither reconsolidation nor extinction can occur until after the offset of the evoking stimulus. (2) There are clear temporal constraints upon the length of exposure that will support reconsolidation rather than extinction. While this length has not been quantified, the sequencing of results indicates that a longer or more intense presentation of the stimulus produces extinction, while reconsolidation is only evoked by a much briefer exposure. We believe that our effort to terminate the narrative and other stimuli at any point where sympathetic responses may be evoked (i.e., at the observable initiation—often unnoticed by the client—of autonomic responses; see Coccoz, Sandoval, Stehberg, & Delorenzi, 2013), satisfies this temporal constraint. (3) Insofar as each "exposure" represents successively lower levels of arousal in the client, this new safety information (Clem & Schiller, 2016) supports the transformation of the memory via reconsolidative updating, rather than the creation of a new extinction memory. Insofar as our interventions do not produce a series of extinction events (see below), it is possible that each presentation of the dissociated (unreinforced or non-traumatizing) memory begins a new

Table I. The RTM process outline.

1. The client is asked to briefly recount the trauma.
2. Their narrative *is terminated as soon as autonomic arousal is observed.*
3. The subject is reoriented to the present time and circumstances.
4. SUDs ratings are elicited.
5. The clinician assists the client in choosing times before and after the event (bookends) as delimiters for the event: one before they knew the event would occur and another when they knew that the specific event was over and that they had survived.
6. The client is guided through the construction (or recall) of an imaginal movie theater in which the pre-trauma bookend is displayed in black and white on the screen.
7. The client is instructed in how to find a seat in the theater, remain dissociated from the content, and alter their perception of a black and white movie of the index event.
8. A black and white movie of the event is played and may be repeated with structural alterations as needed.
9. When the client is comfortable with the black and white representation, they are invited to step into a two-second, fully associated, reversed experience of the episode beginning with the post-trauma resource and ending with the pre-trauma resource.
10. When the client signals that the rewind was comfortable, they are probed for responses to stimuli which had previously elicited the autonomic response.
11. SUDs ratings are elicited.
12. When the client is free from emotions in retelling, or sufficiently comfortable (SUDs $\leq 3$), they are invited to walk through several alternate, non-traumatizing versions of the previously traumatizing event of their own design.
13. After the new scenarios have been practiced, the client is again asked to relate the trauma narrative and his previous triggers are probed.
14. SUDs ratings are elicited.
15. When the trauma cannot be evoked, and the narrative can be told without significant autonomic arousal, the procedure is over.

*Notes*. Table is adapted from Gray et al. (2016). The RTM protocol for the treatment of PTSD: a randomized waitlist study of 30 female veterans. It is used with the permission of the authors.



round of reconsolidation on that version of the memory. That is, the newest version of the memory becomes the target memory as the intervention proceeds and the final result is a fully dissociated memory built up of successive reconsolidative events—Lee et al.'s (2017) "snowball" evocation.

While the reduction in the response to the index memory might conceivably reflect extinction, our results have been remarkably free of the indicia of extinction-based relapse (e.g., spontaneous recovery, contextual renewal, etc.) at 6 months and have now been observed to persist over a period of 12 months (Tylee et al., 2017). This would seem to eliminate the argument that our results are extinction based upon any level.

The cognitive elements of the intervention are well known. In RTM, they are distributed throughout the process as the means used to adjust the present-time salience and apparent reality of the target memory or the patient's response to it (Gray & Liotta, 2012). Although here derived from the discipline of NLP (Andreas & Andreas, 1987; Bandler, 1985), the removal of color, the distancing, dissociation from the memory, as well as the cognitive restructuring of the memory, are all standard elements of cognitive/perceptual psychology. Flattening the picture, removing the color, and dissociation were observed by Moore, Mischel, and Zeiss (1976) in their famous marshmallow experiment. The effects of dissociation and distancing were reported by Ayduk and Kross (2010; see also Kross & Ayduk, 2011). Codispoti and De Cesarei, 2007; De Cesarei & Codispoti (2006, 2008, 2010) have reported on the subjective modification of the size and perceived distance of remembered images.

Restructuring the trauma-related imagery is a fairly common cognitive intervention (Arntz & Weertman, 1999; Germain, Shear, Hall, & Buysse, 2007; Lu, Wagner, Van Male, Whitehead, & Boehnlein, 2009). It was first designed for the treatment of PTSD but more recently has been used to treat PTSD-related nightmares.

The crucial element here is the use of the cognitive elements of the intervention during the labilization window which is, for our purposes, the core of the reconsolidation phenomenon. This model has been established in both preclinical and clinical studies with great consistency. During this period, thought to last from 1 to 6 hours (Nader, 2003; Nader et al., 2000; Schiller et al., 2010), information about the target memory that is new, or novel, that provides safety information, or information that changes the status of the threat, may be introduced into the structure of the memory (Agren, 2014; Fernández et al., 2016; Forcato et al., 2007; Kindt et al., 2009; Lee, 2009; Schiller et al., 2013; Schiller & Phelps, 2011). It can then be integrated into the structure of the original memory within about 24 hours. This leads to a fast, largely permanent change in the index memory such that the memory is de-reified and loses its present-time urgency.

RTM is targeted specifically at the intrusive symptoms of PTSD, especially when they are experienced as sudden, uncontrollable, sympathetic responses either to the trauma narrative, elements of the narrative, or stimuli known to elicit flashbacks and nightmares. This represents an automatic and unconscious response style which some authors have identified as being particularly susceptible to alterations through "reconsolidative modification" (Kredlow et al., 2016). The centrality of flashbacks and nightmares and the automaticity of response are crucial indicators for the use of the protocol. If they are absent, the protocol is inappropriate (Gray & Bourke, 2015; Gray & Liotta, 2012; Tylee et al., 2017).

## Studies of RTM Efficacy

There have been two previous waitlist-controlled studies of RTM and one pre–post pilot that included a small ($n = 5$) waitlist control. Both RCTs evaluated the protocol using the PTSD Symptom Scale Interview (PSS-I) and PTSD Checklist Military Version (PCL-M) at intake and at 2-week follow-ups and the PCL-M either alone or with the PSS-I at 6 weeks post and later follow-ups. The pilot study used PCL-M alone (Gray & Bourke, 2015), while the other two employed both PCL-M and PSS-I (Gray et al., 2016; Tylee et al., 2017). Two of the studies applied RTM to male veterans. The third study (Gray et al., 2016) examined 30 female veterans, 21 of whom suffered from some degree of Military Sexual Trauma. All studies obtained high effect sizes and significant loss of diagnosis. Up to 90% of treatment completers reported a complete absence of flashbacks and nightmares after the last treatment.

Gray and Bourke (2015) examined a population of 30 male veterans using a 30-point admission baseline score (PCL-M) for participation. They found a mean intake score of 61.7 for all subjects. Control subjects ($n = 5$), after a 3-week wait period, were also given the intervention. Pooled results of all participants post-treatment showed a mean reduction of 33 points in trauma severity, with a final mean PCL-M score of $28.8 \pm 7.5$ at 6 weeks or the last reported measure. Hedges' $g$ at 6 weeks post showed a 2.9 standard deviations (*SD*) difference from intake to follow-up (CI 99% [26.05, 33.71]). These results were significant at the .001 level. Comparisons of untreated controls and experimental subjects at an equivalent post-treatment time points were statistically significant ($p < .001$). An informal follow-up reaching approximately 75% of treatment completers indicated that those gains were maintained at 6 months post (R. Gray, personal communication, August 5, 2016).

Tylee et al. (2017), in a study of 30 male veterans, reported a mean reduction of 39.8 points (cumulative intake Mean = $66.5 \pm 8.27$) for all treatment completers, with a final mean PCL-M score of $26.8 \pm 13.08$ at 6 months. Hedges' $g$ for all treatment completers at 6-months post indicated a 3.59 *SD* difference in effect from intake to follow-up (CI 99% [22.06, 33.54]). Comparisons of the waiting-list control group at baseline 2 with the treatment group at 2 weeks post-treatment yielded an efficacy measure equivalent to nearly 4 *SD* ($g = 3.99$; CI 95% [−0.355, 7.252]). Data from a one-year follow-up indicated that these improvements were maintained for a full year after treatment. Twelve-month mean PCL-M scores for treatment completers, with 81.5% reporting, were 20.9 (±4.2), a reduction of 46.5 point.

In their study of 30 female subjects, Gray et al. (2016) reported a mean reduction of 48.21 points (cumulative intake Mean = $71.06 \pm 6.82$) for all treatment completers, with a final mean PCL-M score of 22.85 (±6.17) at 6 weeks. Hedges' $g$ for all treatment completers at 6 weeks post indicated a 7.05 *SD* difference from intake to follow-up (CI 95% [19.95–8.046]). When PCL-M scores for the treatment group at 2 weeks post was compared to the waiting-list control at baseline 2 (an equivalent time point), the efficacy of the intervention showed a difference greater than two standard measures post-treatment (Hedges' $g = 2.603$, CI 95% [−0.038, 7.243]). Clinical improvement in the PTSD symptoms was determined using standard levels of change in PCL-M scores (Schnurr et al., 2007; VA National Center for PTSD, 2014). Response to treatment was regarded as clinically significant for improvements in the PCL-M scores of greater than 20 points (Monson et al., 2008). Loss of diagnosis (VA National Center for PTSD, 2014) was defined as a total PCL-M score of <45 points and the absence of flashbacks, nightmares, and reactivity to the target memory. Full remission was defined as a total PCL-M score of less than 30 (Castillo et al., 2016; VA National Center for PTSD, 2014).

For both RCT investigations, loss of diagnosis was determined based upon a combination of standard DSM criteria (scoring below threshold on symptom inventories while failing to endorse all three symptom clusters at the required levels). This accounted for 65% or more of the results in all three studies. A second criterion included scoring below the dichotomous clinical threshold for PTSD as defined for the primary measure (PSS-I ≤ 20, PCL-M ≤ 45) while showing no autonomic reactivity to relevant stimuli and reporting a total loss of nightmares and flashbacks. Using these combined measures, loss of diagnosis was above 90% for all three studies.

### Purpose of the Study

The current research extends prior investigations of the RTM protocol by using a larger sample size, employing an intent-to-treat (ITT) analysis, and by focusing upon the PSS-I scores, a diagnostic indicator of current PTSD diagnosis, rather than PCL-M. With these modifications, we hope to discover whether RTM can once more produce results consistent with prior studies in a population of male US veterans. We examined the immediate treatment outcomes and sustained treatment effects at 2 weeks and 6 weeks post among volunteers in immediate treatment, untreated waitlist, and among patients who were treated after completing a 3-week waiting period. The economy of the intervention and the robust nature of its outcomes are attributed to the intentional evocation of the reconsolidative mechanism. These mechanisms appear to be conserved across species (Agren, 2014; Pedreira et al., 2004; Schiller & Phelps, 2011). Alberini (2005) reports observations of reconsolidation in mollusks (aplysia and pond snails), medaka fish, two kinds of crabs, chicks, mice, rats, and gerbils. Besnard, Caboche, and Laroche (2012) add nematodes (*Caenorhabditis elegans*), drosophila, bees, and rabbits to the list. It has also been observed in non-human primates (Diaz-Mataix, Ruiz Martinez, Schafe, LeDoux, & Doyere, 2013) and human subjects (Forcato et al., 2007, 2013; Kindt et al., 2009; Kindt & van Emerik, 2016; Meir Drexler et al., 2014; Oyarzún et al., 2012; Schiller et al., 2010; Schiller & Phelps, 2011; Soeter & Kindt, 2015). Previous work has suggested that RTM can produce reductions in intrusive symptomatology that remain stable over a period of at least one year (Tylee et al., 2017). These earlier findings led us to hypothesize that RTM would produce clinically significant symptom reductions using standard measures of PTSD symptoms (PSS-I, PCL-M), and that these symptom reductions would remain stable over time.

## Methods

The methods and study design here follow the same parameters as those described in Gray and Bourke (2015), Gray and Liotta (2012), Gray et al. (2016), and Tylee et al. (2017). They differ from the methods reported by Gray and Bourke (2015) only in that the number of treatments is reduced from five to three and that the PSS-I was substituted as the principal measure of change.

### Ethical Approval and Safety Measures

The study protocol and informed consent were approved by the New England Independent Review Board (NEIRB). All personal identifying and Health Insurance Portability and Accountability Act (HIPAA)-sensitive information was held in strict confidence. Following NEIRB guidelines, the protocol and all aspects of participation were reviewed with participants and signed informed consents were obtained from each. If any participant had had significant emotional difficulties during the study, an immediate intervention would have been administered by the licensed clinician on staff. If necessary, the participant would have been referred to his psychiatrist or primary care physician or for emergency treatment. No need for such emergency treatment arose.

### Study Design

The RTM protocol for the treatment of PTSD was evaluated using a randomized, waitlist-controlled design (see Figure 1). Participants were admitted to the study in cohorts of 10 and then randomly assigned to treatment and control groups (5 each) using a list of random numbers generated using Microsoft Excel 2016. Clients were assigned to treatment conditions by site managers according to a randomized list generated by study personnel at another location.

For clarity of reporting, we refer to follow-up time points during which symptoms were evaluated, based

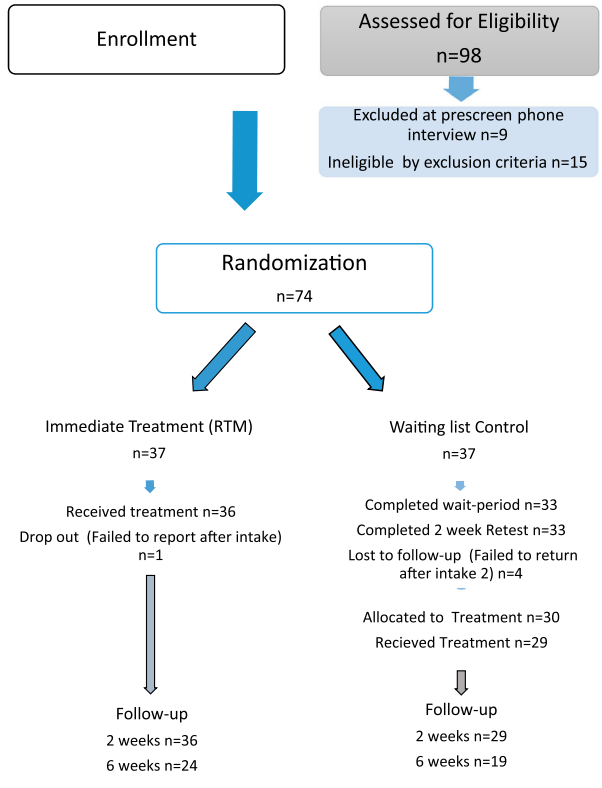


Figure 1. Participant flow chart.

on the number of weeks elapsed since the completion of the intake measures and the post-treatment period. Intake evaluations were performed for all participants on study week 1. The treatment group began treatment on the same week. RTM was administered across a period of 2 weeks. Participants received three 120-minute treatment sessions separated from each other by a minimum of 24 hours over the course of 1–3 weeks. Post-treatment evaluation of symptoms was performed 2 weeks post-treatment and 4 weeks later (reflecting the 2- and 6-week follow-up time points). Control participants also had intake assessment during week 1 and were then informed that they would wait several weeks before receiving treatment. On study week 5, control participants were re-evaluated by psychometricians blinded to the study condition. Control participants were then offered the same intervention schedule, and their symptom scores measured 2- and 6 weeks post-treatment. All assessments were provided by psychometricians blinded to the study condition from which the subjects were drawn. All treatments and evaluations were performed in private office suites dedicated to the study.

## The RTM Protocol

The RTM protocol is a brief cognitive intervention with a minimal, non-traumatizing exposure to the original trauma memory at the beginning of each session. It was administered in 3 sessions of up to 120 minutes each.

The intervention proceeded as shown in Table I (reproduced with permission from Gray et al., 2016).

Full details of the intervention are available from the corresponding author.

## Inclusion and Exclusion Criteria

The following inclusion and exclusion criteria closely follow Gray and Bourke (2015), Gray et al. (2016), and Tylee et al. (2017).

Inclusion criteria: Baseline symptom assessments for PTSD above commonly used diagnostic thresholds (PCL-M $\geq$ 45, PSS-I $\geq$ 21; VA National Center for PTSD, 2014) at intake. A valid PSS-I-based diagnosis requiring that prospective participants endorse symptom clusters in accordance with DSM-IV (American Psychiatric Association [APA], 1994) criteria (endorsing at least 1 question from the intrusive cluster, 3 from the avoidant cluster, and 2 from the hypervigilant cluster). Two clients provided ambiguous responses to the PSS-I screens. These included PSS-I symptom score readings greater than 20 but failure to endorse all 3 symptom clusters for a valid DSM diagnosis. As these clients had provided parallel PCL-M evaluations, those responses were compared to the ambiguous PSS-I evaluations. The concurrent PCL-M evaluations provided high intake scores and clearly endorsed all three symptom clusters. As a result, these patients were accepted as possessing valid diagnoses of PTSD. Participants must have reported PTSD symptoms including intrusive, instantaneous, phobic-type responses to triggering stimuli and observable sympathetic arousal either while recounting the index trauma or triggering flashback-related stimuli. Volunteers must have reported at least one flashback or nightmare during the preceding month. Those meeting intake criteria were reimbursed up to $200 for travel expenses. Reimbursements were disbursed on a per-visit basis. Exclusion criteria: Possession of a comorbid DSM-IV Axis I or II disorder sufficiently severe as to intrude upon the participant's ability to cooperate with treatment; prospects adjudged by the interviewer or clinician as being incapable of sustained attention were excluded. Sustained attention, as here defined, is used to eliminate, e.g., those who may be severely under the effects of licit or illicit pharmacological agents at the point of interview, those with severe attentional difficulty (as experienced in severe ADD/HD, schizophrenia, and other psychiatric diagnoses), and the floridly psychotic. We have found that persons diagnosed with PTSD that include attentional difficulty are generally sufficiently motivated to complete the assigned tasks. Excluded participants were referred to their ongoing treatment provider.

**Definitions.** *Flashbacks and nightmares*. These definitions come into play especially during intake and follow-up. Their application in those circumstances is discussed below. For all clients, we required a minimum of one flashback or nightmare during the month before admission to the study. These are defined (following Gray et al., 2016) as follows:

*Flashbacks*. involuntary re-association into the traumatic memory that includes all of the following elements: (1) a loss of orientation to the present time or context either in full or in part; (2) the traumatic event is experienced as a fully associated event: The client is 'in' the recalled event; (3) it is not only involuntary but also tends to persist as the client's current reality; (4) whether the dissociative event persists for a long period—many minutes—or does not persist for long, its emotional tone carries through past the end of the dissociative event (the re-association into the traumatic memory) so that it continues to color much of the following hours, the remainder of the day, or several days, afterwards.

Brief associations of current events with past traumas (the more common and cinematic use of the word *flashback*) that do not last long, that do not include a dissociation from the current context, and that do not have a continuing effect on the client are usually not, for our purposes, flashbacks. They are most often just normal memories.

Where there is difficulty in differentiating between a flashback and a brief memory of the trauma, the presence or absence of intrusive autonomic responses will normally be determinative.

*Nightmares*. Any dream or, more especially, a night terror that, whether consciously recalled or not, may be indicated by any of the following: (1) projects the client into the context of one or more index traumas and/or (2) results in hypnagogic imagery related to the index trauma sufficiently vivid as to result in confusion between waking and dream contexts and/or (3) results in unconscious acting out as sleep walking, speaking, or violence that can be related by emotional tone or content to the index trauma or its context, that; (4) produces lingering emotional effects that may color the following hours or days, and that often makes it difficult or impossible to immediately return to sleep.



## Client Flow

Clients were referred based upon fliers, personal referrals, and recommendations by local clinicians. All referrals and public solicitations included the restriction that prospects have current-month flashbacks and nightmares. This had the effect of prescreening many of the referrals. Recommendations by persons having experienced the intervention may have introduced a positive expectation bias. We note further that insofar as referring agencies had been advised of our criteria that clients must have both the intrusive symptomatology and be capable of conscious participation in a relatively complex set of cognitive tasks, this may have had the effect of eliminating more complex or difficult cases and biased their referrals in favor of a population more favorable to the intervention.

Of 98 original referrals, 9 were determined to be ineligible, based upon the absence of present-month flashbacks and nightmares as reported during telephone interviews. Fifteen others were excluded at intake. Rejections at intake were based upon client failure to meet DSM-IV (APA, 1994) diagnostic criteria, the lack of observable autonomic reactivity, or the inability to identify one or more discrete traumatic events (DSM-IV Criterion A). The 74 remaining volunteers were randomly assigned to treatment and waitlist control conditions: 37 to each condition. Among the 37 control group participants, 4 persons having met inclusion criteria, and completed the initial intake procedure never returned for the post-wait retest (baseline 2) at week 5 leaving 33 control subjects. In order to maintain the statistical validity of the study, results for these four persons, for whom only baseline data were available, the baseline observation carried forward (BOCF) method was used to fill out all subsequent measures. This was justified by our expectation that treatment would improve these scores. Thus, this conservative measure had the effect of including these participants as hypothetical treatment failures (European Medicines Agency, 2010; National Research Council, 2010; White, Carpenter, & Horton, 2012). No control group participants reported new traumas during the wait period.

One treatment group participant dropped out after intake. Like the control dropouts, his scores were represented by the BOCF method. Thereafter, all 36 of the remaining treatment group members completed treatment and 2-week follow-up. When waitlist control participants were offered the RTM intervention, all opted to participate. One of the control subjects dropped out of treatment citing family problems. Of the control subjects, 32 were retained for the follow-up assessments. Post-wait control treatments began on study week 6—after the end of the waitlist interval.

When persons from either group, after completing the 2-week follow-up testing, failed to report for subsequent observations, the last valid observation was carried forward (LOCF) to represent the missing data. This was also justified by our expectation that scores would either remain the same or improve for treatment completers. Insofar as all measures were simple scores or means, and the same kind of allocation was provided for those who had or had not improved, the measure follows protocols deemed acceptable by the international research community (European Medicines Agency, 2010; National Research Council, 2010; White et al., 2012). Participant flow is illustrated in Figure 1.

## Participants

The study employed a non-random convenience sample using referrals, fliers, and word of mouth to recruit male US veterans. Recruiting began during December 2015 and was completed by mid-August 2016. All treatments were completed by September 2016. Sample demographics are presented in Table II.

Thirty-nine volunteers self-identified as Caucasian, 18 as African-American, 3 as Hispanic, and 1

Table II. Demographic data.

| Category | | *n* (%) |
|---|---|---|
| Age | Mean Age = 48.6 (±13.3)<br>Median Age = 51.5 | |
| Service type | US Army | 30 (57%) |
| | US Marines | 22 (35%) |
| | US Navy | 12 (17.5%) |
| | US Air Force | 3 (6%) |
| Ethnicity | Caucasian | 32 (28%) |
| | African American | 19 (26%) |
| | Hispanic | 9 (4%) |
| | Native American | 3 (1.5%) |
| | Asian | 2 |
| | Other/multiple | 2 (3%) |

*Notes*. MST = Military Sexual Trauma; Non-MST = Non-Military Sexual Trauma. Traumas per location reflect multiple traumas per location per person. Percentages may not add to 100% due to rounding errors.

as Native American. Two others claimed multiple ethnicities. Participants had a mean age of 48.6 (±13.33) and a median age of 51.5. Thirty-nine participants served in the Army, 24 in the Marine Corps, 12 in the Navy, and 4 in the Air Force. Most traumas occurred in combat situations. Traumas reported included a mix of active duty, pre-enlistment, and post-service events Furthermore, demographic data are reported in Table III.

Although we did not collect information on comorbid diagnoses, we did collect data on current medication regimens. Among those treated, 36 persons reported the use of antipsychotics, antidepressants, or anxiolytics before and during the study. Thirteen persons reported using prescription sleep aids alone. Two were unable to specify their medications. Ten persons reported using non-psychotropic prescription drugs for the treatment of pain and other conditions, and 12 reported using no prescription medications. One person reported using medicinal marijuana alone. Twenty-one different psychotropic medications were being used by participants in multiple combinations before or during treatment. Six were encountered five or more times, these most frequently prescribed drugs were Trazadone (14 times: 8 treatment, 6 control); Prazosin (8 times: 5 treatment, 3 control), Wellbutrin (6 times: 3 treatment, 3 control), Hydroxyzine (5 times: 3 treatment, 2 control), and Seroquel (5 times: 4 treatment, 1 control). The use of prescription psychotropics appeared to be evenly distributed between control and treatment conditions (see Table IV). Clients, in general, found the drugs somewhat effective in mollifying the symptoms but reported that they did not represent resolutions for their continuing problems. Clinical observation and interviews found that there were only minimal differences between medicated

Table III. Trauma types and contexts.

| a. Trauma categories treated | | |
|---|---|---|
| Combat general | Discrete combat incidents | 65 |
| | Sets of combat traumas related to one incident | 13 |
| Non-combat deaths | Non-combat deaths or murders of non-family members | 7 |
| | Non-combat deaths of family members | 6 |
| Sexual assault | MST | 5 |
| | Non-MST | 5 |
| Accidents | Combat related | 2 |
| | Non-combat related | 4 |
| Crimes | Non-military assaults | 4 |
| | Gang-related assaults | 4 |
| Terrorist attacks | Terrorist attacks | 5 |
| Non-military legal encounters | Legal situations | 2 |
| Family related | Family violence | 4 |
| | Other domestic incidents | 5 |
| Miscellaneous | OTHER (panic attack, weather related) | 3 |

| b. Locus of incidents | | |
|---|---|---|
| | Location | Number of incidents (approx.) |
| | USA | 49 |
| | Kuwait, Iraq, Afghanistan | 47 |
| | Vietnam | 16 |
| | Europe | 4 |
| | Korea | 2 |
| | Somalia | 2 |
| | Other | 2 |

*Note*. Clients regularly reported multiple traumas spanning multiple contexts.

Table IV. Number of participants using prescribed medications by impact of medical class and experimental arm

| Group | Anti- psychotics | Anti- depressants | Anxiolytics | Hypnotics | Other non-psychotropic | THC | None | Unspecified |
|---|---|---|---|---|---|---|---|---|
| Treatment | 3 | 8 | 4 | 8 | 5 | 1 | 8 | 3 |
| Control | 2 | 11 | 3 | 7 | 3 | | 6 | 2 |

*Notes*. Medications are listed in terms of the severity of the disorder for which they are normally prescribed. Most clients used more than one medication, the strongest prescribed is the only one noted.



and non-medicated clients. No medication changes or changes in other treatments were noted either during the study treatment or during the waiting period.

We acknowledge the time since exposure as a possible point of interest, but point out that the mean and median ages of those treated suggest that the time of exposure was most frequently longer than 10 years. This is especially interesting, as boundary conditions for the labilization of fear memories, as originally discussed, indicated that older memories were thought to be more resistant to labilization than others (Fernández et al., 2016; Gershman, Monfils, Norman, & Niv, 2017; Lee, 2009; Lee et al., 2017; Milekic & Alberini, 2002; Monfils et al., 2009; Pedreira et al., 2004). Researchers who have studied the RTM protocol (Gray et al., 2016; Gray & Bourke, 2015; Gray & Liotta, 2012; Tylee et al., 2017) have consistently taken the stance that the PTSD trauma memories are constantly renewed by reconsolidation each time a flashback or nightmare is experienced, thus rendering the traumatic event a new, recent memory. This perspective is (as noted above) in line with the suggestion of Lee et al. (2017). We believe that, more often than not, our client population fits their description of persons, who, through repeated reactivations and reconsolidations of the memory through flashbacks and nightmares, have renewed and strengthened these memories through what they call the "Snowball" effect (p. 536). These renewed traumas may be treated as essentially new memories so that time since trauma becomes irrelevant.

## Psychometric Scales

The PSS-I and PCL-M were used as primary measures of symptoms at various study time points. Both scales are regularly used by the military and the VA to assess PTSD symptoms. Both tests were administered at intake for both groups, the week 5 retest for controls, and the 2- and 6-week post-tests for all participants. These were intended to document pre-/post-PTSD treatment changes as well as the consistency of change across time. In order to infer whether PTSD symptoms remitted below levels that might warrant a clinical diagnosis, commonly used thresholds were applied to these clinical scales (VA, 2014; PCL-M threshold $\geq 45$; PSS-I threshold $\geq 20$).

The PSS-I is a highly regarded structured clinical interview, on par with other structured interviews such as the Clinically Administered PTSD Scale (CAPS; Foa, Riggs, Dancu, & Rothbaum, 1993; Foa & Tolin, 2000). It is sufficiently accurate to be used as a primary diagnostic tool in the assessment of PTSD (Foa et al., 1993; VA National Center for PTSD, 2014; Weathers & Ford, 1996).

We note that the PSS-I may be analyzed using the presence or absence of specific symptom clusters or as a continuous score. Foa and Tolin (2000) indicate that the PSS-I is highly correlated with other gold standard measures. In both the initial diagnosis and post-treatment diagnosis, we have used a 20-point cut-off for PSS-I scores in combination with the presence or absence of DSM criteria (cluster endorsements). According to this measure, any score >20 indicates the presence of PTSD with an optimal clinical diagnosis for veterans and active military $\geq 45$ points. We used a total score of $\leq 20$ with concurrent failure to meet DSM-IV Criteria (failure to endorse all of the following: 1 item from the intrusive symptoms cluster, 3 items from the avoidant cluster, and 2 from the hypervigilant cluster) as indicating the loss of diagnosis.

The PCL-M (Weathers, Litz, Herman, Huska, & Keane, 1993) is a 17-item, self-report scale based upon DSM diagnostic criteria for PTSD (APA, 1994; Weathers, Litz et al., 1993). The scale can be scored dichotomously based upon total score >50 or continuously following the DSM-IV symptom criteria. PCL-M evaluations are highly correlated to the CAPS ($r = 0.93$; Blanchard, Jones-Alexander, Buckley, & Forneris, 1996; Castillo, Lacefield, C'de Baca, Blankenship, & Qualls, 2014). Scores $\leq 30$ points on PCL-M which likewise failed to meet DSM criteria were also regarded as being in in full remission. In cases where PSS-I diagnoses were ambiguous, PCL-M was used to determine the final diagnosis. At intake, a PSS-I score might have more than met the threshold scoring requirements but only endorsed two DSM categories (or a similar case), such cases were accepted for study entry if the PCL-M data were unambiguous, that is, if the total

PCL-M scores were above the clinical threshold (PCL-M ≥ 50) and clients endorsed all three DSM clusters in their responses. Two such cases were described earlier. Likewise, for the determination of loss of diagnosis, ambiguous PSS-I ratings were compared with PCL-M materials in a similar manner for a final decision.

All post-treatment evaluations were completed by independently contracted psychometricians who were not informed of the arm of the study from which the client was referred.

### Treatment Fidelity

All screening and treatment sessions were video recorded on digital media for the assessment of treatment fidelity. At the end of each day, the video recordings were uploaded to a secure HIPAA-compliant server and archived for review. Three well-practiced experts, familiar with the RTM protocol (two Ph.D.-level psychologists and one licensed, masters-level social worker), reviewed arbitrary and targeted videos of treatment sessions. Evaluations were made based upon the following elements: (a) adherence to the RTM procedure (available from the corresponding author); (b) adherence to the syntax of reconsolidation (as reflected in Gray & Bourke, 2015; Gray & Liotta, 2012; Schiller & Phelps, 2011); (c) the calibration skills used by the clinician; and (d) whether the client had been properly screened.

### Data Analysis

Following the method described in Gray et al. (2016), all analyses were performed using Microsoft Excel 2016. We performed a one-tailed student's *t*-test for groups of different variances to test for expected differences between the experimental and control groups at the point when the first post-treatment results from the experimental group could be compared to the control subjects at their post-wait re-evaluation. To test for responses to treatment within groups, we performed six paired, one-tailed student's *t*-tests comparing baseline symptom scores at study week 1 to symptom score changes at 2 and 6 weeks post-treatment. Separate analyses were performed for treatment and post-treatment control groups, and for the group of all protocol completers. To examine whether the wait-listed controls changed either spontaneously or due to other treatments during the wait period, we compared the wait-list control baselines at week 1 to their own post-wait baselines at study week 5. Similarly, 2-week follow-up results were compared against 6-week follow-ups for all treatment completers to test for decay of results over time. Hedges' *g* was used to assess the effect size for all comparisons.

As previously noted, in accordance with the ITT practice, when persons from either group, after completing the 2-week follow-up testing, failed to report for subsequent observations, the LOCF method was used to represent the missing data. This was justified by our expectation that scores would either remain the same or improve for treatment completers (European Medicines Agency, 2010; National Research Council, 2010; White et al., 2012). Similarly, for any person for whom only baseline data were available, their baseline scores were carried forward (BOCF) through all follow-up measures. This was justified by our expectation, in light of research reporting a poor prognosis for untreated PTSD (Devilly & McFarlane, 2009), that treatment would improve these scores (European Medicines Agency, 2010; National Research Council, 2010; White et al., 2012). All data are reported as means ± *SD*.

## Results

We have already noted the results from previous studies of the RTM protocol (Gray et al., 2016; Gray & Bourke, 2015; Tylee et al., 2017). Here, we predicted that RTM would reduce PSS-I and PCL-M scores as effectively as it had in the previous studies.

Using the PSS-I as the primary diagnostic instrument, we found the following: Sixty-five persons completed treatment. Of those, 44 were in total remission (44/65 or 67%; PSS-I ≤ 20 and DSM criteria not met). Another four persons provided ambiguous responses to the last PSS-I measure provided. In each of those 4 cases, the total score was at or below 20, and each person had lost 23 or more symptom score points at the last follow-up recorded (mean score decrease = 32.75). When the PCL-M responses for each of these cases were examined, it was found that each had scored below 30 on the PCL-M and that they had not met the DSM-IV diagnostic criteria after treatment. These four were therefore considered to have lost the diagnosis as well. Including these four, the number of successful cases, ending treatment diagnosis-free rises to 48 of 65 cases, or 73%. None of those designated as cleared of the diagnosis reported post-treatment flashbacks or nightmares related to the treated traumas and none showed autonomic reactivity in response to relevant probes or stimuli at follow-up. Inconsistencies between scores on the PSS-I and PCL-M inventories were minor and not unexpected. All scores are reported in Table V.

Table V. Effect sizes for all comparisons at 95% CI.

| | Comparison & terms | Measure | Term a Mean ± *SD* | Term b Mean ± *SD* | Hedges *g* | 95% CI Lower | 95% CI Higher | *p* |
|---|---|---|---|---|---|---|---|---|
| 1 | Waiting-list comparison | | | | | | | |
| | a. Treatment group at 2-week post | PSS-I | 12.92 ± 12.9 | 37.11 ± 9.387††† | −2.120 | −4.693 | 0.453 | .001 |
| | b. Wait-listed controls at baseline 2 | PCL-M | 31.459 ± 15.1 | 62.97 ± 12.13††† | 2.276 | −5.398 | 0.846 | .001 |
| 2 | RTM group | | | | | | | |
| | a. Baseline | PSS-I | 37.32 ± 6.678 | 12.92 ± 12.9 ††† | 2.348 | 0.005 | 4.691 | .001 |
| | b. 2-Week post | PCL-M | 62.92 ± 10.94 | 31.46 ± 15.1††† | 2.361 | −0.643 | 5.382 | .001 |
| 3 | RTM group | | | | | | | |
| | a. Baseline | PSS-I | 37.32 ± 6.678 | 17.41 ± 14.3 ††† | 1.550 | −1.282 | 4.383 | .001 |
| | b. 6-Week post | PCL-M | 62.91 ± 10.94 | 32.40 ± 15.735††† | 2.228 | −0.860 | 5.315 | .001 |
| 4 | Post waitlist controls | | | | | | | |
| | a. Week 1 baseline | PSS-I | 36.67 ± 6.936 | 18.46 ± 15.48 ††† | 1.750 | −0.983 | 4.83 | .001 |
| | b. 2-Week post | PCL-M | 66.216 ± 9.51 | 37.19 ± 18.10††† | 1.987 | −1.308 | 5.282 | .001 |
| 5 | Post waitlist controls | | | | | | | |
| | a. Week 1 baseline | PSS-I | 36.67 ± 6.936 | 17.84 ± 16.26††† | 1.728 | −1.121 | 4.577 | .001 |
| | b. 6-Week post | PCL-M | 66.216 ± 9.51 | 36.08 ± 19.59[a] | 1.957 | −1.572 | 5.446 | .16 |
| 6 | All treated subjects | | | | | | | |
| | a. Baseline | PSS-I | 38.5 ± 6.783 | 17.93 ± 14.7††† | 1.787 | 0.059 | 3.632 | .001 |
| | b. 2-Week post | PCL-M | 64.567 ± 10.25 | 37.18 ± 17.79††† | 1.876 | −0.463 | 4.251 | .001 |
| 7 | All treated subjects | | | | | | | |
| | a. Baseline | PSS-I | 38.5 ± 6.783 | 15.38 ± 15.23††† | 1.951 | 0.052 | 3.850 | .001 |
| | b. 6-Week post | PCL-M | 64.567 ± 10.25 | 34.24 ± 17.62††† | 2.093 | 0.230 | 4.415 | .001 |
| 8 | a. All waitlist baseline 1 | PSS-I | 38.5 ± 6.78 | 37.11 ± 9.156 [b] | 0.307 | −1.701 | 2.009 | .048 |
| | b. All waitlist baseline 2 | PCL-M | 66.21 ± 9.52 | 62.973 ± 12.14[c] | 0.295 | −2.191 | 2.780 | .024 |
| 9 | All Completers | | | | | | | |
| | a. 2-Week post-treatment | PSS-I | 17.93 ± 14.7 | 15.38 ± 15.226[d] | 0.173 | −2.293 | 2.585 | .013 |
| | b. 6-Week post-treatment | PCL-M | 37.18 ± 17.79 | 34.24 ± 17.62[e] | 0.165 | −3.869 | 4.199 | .48 |

*Notes.* All computations based upon ITT analysis. Incomplete values were replaced using the last observation carried forward (LOCF). Post-randomization dropouts were replaced using the baseline observation carried forward (BOCF; European Medicines Agency, 2010; National Research Council, 2010; White et al., 2012).

†††$p = .002$.

[a]$p = .16$.

[b]$p = .048$.

[c]$p = .024$.

[d]$p = .0135$.

[e]$p = .48$.



## Experimental Comparison

We compared the PSS-I scores for the treatment group at week 4 (their 2-week post-treatment follow-up) to the untreated wait-listed control group at week 5 (their untreated re-evaluation at the end of the wait period). As expected, the untreated waitlist participants at the end of the wait period (Mean = 37.11 ± 9.387) and treatment subjects at 2-week post (Mean = 12.9 ± 12.9) were significantly different in the expected direction ($p < .001$; Table V, comparison 1). The effect of treatment as compared to the untreated post-wait sample was equivalent to two *SD* above the post-wait control mean ($g = -2.1$; 95% CI [−4.69–0.453].

## Within-Group Comparisons

Symptom scores for experimental subjects were significantly reduced ($p < .001$) from baseline (PSS-I Mean = 37.32 ± 6.678, $n = 37$) to 2-week post (Mean = 12.9 ± 12.9; $n = 37$) and 6-week post (Mean = 17.4 ± 14.3; $n = 37$; Table V, comparison 2–3). Hedges' *g* was computed for both comparisons and showed the effect to be meaningful at the equivalent of 2.3 *SD* from baseline at 2 weeks ($g = 2.348$; 95% CI = [0.005–4.69] and to 1.5 *SD* at 6 weeks ($g = 1.550$; 95% CI [−1.282–4.383]).

Similarly, when we tested the previously wait-listed controls, who also received treatment, we found significant ($p < .001$) reductions in 2-week (Mean = 18.46 ± 15.48; $n = 37$) and 6-week (Mean = 17.84 ± 16.26; $n = 37$) post-treatment scores as compared to (initial) baseline (Mean = 36.67 ± 6.936; $p < .001$; $n = 37$; Table V, comparisons 4–5). Here too, effect sizes indicated that these changes were meaningful with Hedges' *g* pointing to very large effects ($g = 1.750$; 95% CI [−0.983–4.83 and $g = 1.728$; 95% CI [−1.121–4.577], respectively).

### Cumulative Within-Group Comparisons

Following the pattern of earlier studies, when we pooled results for all treatment completers, we compared baseline PSS-I (Mean = 38.5 ± 6.783; $n$ = 74) against 2-week (Mean = 17.93 ± 14.7; $n$ = 74) and 6-week (Mean = 15.38 ± 15.23; $n$ = 74) post-treatment follow-ups; those differences were also significant in the expected direction ($p$ < .001; Table V, comparisons 6–7). Both measures provided very large effect sizes when compared to the pooled mean baseline $g$ = 1.787 (95% CI [.059–2.632], and $g$ = 1.951 (95% CI [0.052–3.850]), respectively.

### Control Group Stability

The experimental comparison (week 4 treatment group vs. week 5 post-wait control) raised the question whether the untreated waitlist control group had made significant improvements or declines during the waiting period. We therefore compared the waitlist PSS-I results from baseline at study week 1 (Mean = 38.5 ± 6.78) to the untreated retest at study week 5 (Mean = 37.11 ± 9.156), and found the differences to be significant at the .05 level ($p$ = .048; Table V, comparison 8). This result falsified our assumption of group equivalence. However, analysis of the effect of the difference suggests that it did not have a meaningful impact ($g$ = 0.181; 95% CI [−1.242–1.603]). These differences and their implications are discussed below.

### Stability of Results over Time

As other PTSD treatments have been shown to be unstable over time (Steenkamp et al., 2015; Steenkamp & Litz, 2013), we wanted to know whether our treatment results were stable. We, therefore, compared the 2-week post-treatment scores for all subjects (Mean = 17.93 ± 14.7, $n$ = 74) with their 6-week post-treatment scores (Mean = 15.38 ± 15.226, $n$ = 74). The differences were significant ($p$ = .0134; Table V, comparison 9); however, the actual increase in mean scores (2.45) did not reach a meaningful level (≥5) and had a minimal effect ($g$ = 0.173; 95% CI [−2.293–2.585]).

Similar comparisons were made using the PCL-M. All results are reported in Table V.

## Discussion

These results indicate that the RTM protocol may be an effective treatment for PTSD. Based simply on the mean symptom scores, effect sizes indicate that, as compared to no treatment, these results may meet the need for novel treatment alternatives as reported by Steenkamp and Litz (2013) and Steenkamp et al. (2015).

### Symptom Inventories

PSS-I was used as the primary diagnostic at intake and at both post-treatment measures. PCL-M was used for confirmatory diagnosis and to resolve ambiguous cases as discussed above. Although CAPS is generally regarded as the better measure, there is an extensive literature documenting the accuracy and utility of PSS-I (Blanchard et al., 1996; Castillo et al., 2016; Foa & Tolin, 2000). In the interest of available time, the possibility of overtaxing the client, and similar considerations, we opted to use the briefer PSS-I.

Missing data for early dropouts were supplied using the BOCF method, while missing scores for persons who had completed the 2-week follow-ups were replaced using the LOCF method. These both had the effect of artificially depressing later measures of treatment change. This conservative bias is used to justify their use here (European Medicines Agency, 2010; National Research Council, 2010; White et al., 2012).

### Consistency of RTM over Time

*Variations in the present study.* We noted above that whereas we had expected little or no change for control participants between untreated baseline scores at the initial and post-wait baselines, the 1.4-point decline in the mean PSS-I score, significant at the .05 level ($p$ = .048) and 3.28-point decline in mean PCL-M scores, significant at the .03 level ($p$ = .024), was somewhat surprising. Insofar as many of the clients were receiving other care and many of them were on medication, it is perhaps inevitable that there should have been changes. There is also some likelihood of a placebo effect, in that just being seen or cared for impacted many of the waiting-list controls. It is also important to note that this investigation took place using outpatient volunteers so that all clients were living in the community or nearby shelters and all were exposed to the vagaries of everyday life. This leads the study to the border between the classical experiment and efficacy research where such changes are much more likely.

Perhaps more importantly, the observed differences were not at the 5-point level generally regarded as being meaningful (Monson et al., 2008), and the effect of the difference was minimal to non-significant

($g = 0.307$; 95% CI [−1.701–2.009]; and $g = 0.295$; 95% CI [−2.191–2.780].

Similarly, and also contrary to expectation, the 2- and 6-week post-treatment comparisons for all participants were significantly different for both PSS-I ($p = .013$) and PCL-M ($p = .48$). Nevertheless, as with the pre-treatment control comparisons, the actual effect of this 2.45-point difference in the PSS-I means was negligible ($g = 0.198$; 95% CI [−2.820–2.245]).

We noted earlier that contrary to expectations both the pre- and post-wait waitlist comparisons of the control group and the 2- and 6-week follow-up comparisons for all subjects showed significant differences from time point to time point. Earlier studies did not fully follow ITT protocols. Here, we included 5 BOCF and 37 LOCF scores for the 6-week follow-ups which have inevitably biased the later observation in an unfavorable direction. As noted, neither change had a material impact on the result, as both changes were less than 5 points—the downward limit of meaningful change—and neither had a significant effect size.

*Previous research.* The RTM protocol has been examined in three other studies. Each of the investigations used clients drawn from similar populations, using similar means, who all displayed high levels of symptomatology and current-month reports of flashbacks and nightmares. In all three studies (Gray et al., 2016; Gray & Bourke, 2015; Tylee et al., 2017), participants were required to have pre-existing diagnoses of PTSD. Their diagnoses and remissions were confirmed using PSS-I and PCL-M. Consistent with findings reported here, total remissions using standard criteria in previous studies have hovered above the 65% mark. Total clearance of diagnosis in those other studies reports a consistent loss of diagnosis above 90% but differs from the current study in its adherence to more conservative outcome measures. Prior studies included participants who scored below cut-off on the relevant symptom inventory with secession of nightmares, flashbacks, and autonomic reactivity to relevant stimuli—independent of other DSM-IV criteria—as being free of diagnosis. When examined from the perspective of standard outcome measures, all four studies are consistent in their reporting of >65% clearance of diagnosis. Other differences in outcomes may be related to the use of PCL-M as the main measure for follow-up observations in the other studies. Using an ITT analysis and strict application of only the DSM diagnostic criteria, the current research reports 73% loss of (DSM) diagnosis. Taking a more pragmatic, clinical approach, and viewing success in terms of subclinical score levels and total loss of intrusive symptoms, with positive life adjustment at follow-up, independent of other DSM criteria, we find here a total loss of PTSD centered on intrusive symptoms at ~90% among treatment completers. This is consistent with levels reported in previous RTM studies. Other differences in outcomes may be related to the use of PCL-M as the main measure for follow-up observations in the other studies.

## Identification of RTM with the Reconsolidation Mechanism

Other researchers have suggested that the enhanced efficacy and robust changes related to the RTM approach lie in its proposed mechanism of action. This has been discussed previously by Gray and Liotta (2012) and Tylee et al. (2017). This discussion follows their argument closely.

Previous studies have noted that the RTM protocol relies upon structural modifications of the trauma memory that change its immediacy and salience. Because the intervention takes advantage of the so-called labilization window associated with the reconsolidation phenomenon, the changes made to the perceptual structure of the recalled trauma experience become a stable part of the original memory. We have identified the following elements as crucial to the association of reconsolidation mechanisms with the RTM intervention. (1) The syntax of RTM (Gray & Bourke, 2015; Gray & Liotta, 2012) matches the syntax of reconsolidation. (2) Previous studies indicate that the results of the intervention tend to be long lasting and robust (Agren, 2014; Björkstrand et al., 2015; Clem & Schiller, 2016; Fernández et al., 2016; Gray & Bourke, 2015; Schiller et al., 2010, 2013; Tylee et al., 2017); they do not appear to be characterized by symptoms of clinical relapse as reflected in extinction memories including spontaneous recovery, contextual renewal, reinstatement, and rapid reacquisition (Björkstrand et al., 2015; Bouton, 2004; Kindt & Soeter, 2013; Kredlow et al., 2016). (3) RTM uses an abbreviated reminder stimulus that is too short and lacking in intensity to support extinction (Almeida-Correa & Amaral, 2014; Gray & Bourke, 2016; Gray & Liotta, 2012; Lee, 2009; Merlo, Milton, Goozée, Theobald, & Everitt, 2014; Nader, 2003; Perez-Cuesta & Maldonado, 2009; Suzuki et al., 2004). (4) Like the RTM protocol, the initiation of labilization in reconsolidation requires a novel presentation of the fear stimulus rather than a repeated or extended exposure (Almeida-Correa & Amaral, 2014; Fernández et al., 2016; Kindt & Soeter, 2013; Lee, 2009; Pedreira et al., 2004). Novelty may be represented by non-reinforcement (Agren, 2014; Perez-Cuesta & Maldonado, 2009; Schwabe, Nader, & Pruessner, 2014), changes in the duration of re-exposure (Almeida-Correa & Amaral,

2014), the presentation of safety information (Clem & Schiller, 2016), limited presentation of the actual feared stimulus (Dunbar & Taylor, 2016; Lee et al., 2017; Liu, et al, 2014; Luo et al., 2015; Xue et al., 2017), or retelling the trauma narrative in a safe clinical setting (Agren, 2014).

## Limitations of the Study

The current study is limited by several factors. These are enumerated as follows.

**Sampling technique.** The sampling technique targeted a specific subpopulation of male veterans. It used a combination of referrals and word-of-mouth recruitment, resulting in a non-random distribution of veterans which may limit the external validity of the results. That many of the referrals were made by others who reported satisfactory results from the intervention raises the possibility of expectation bias and placebo effects.

We note, however, that expectancies may have been overcome in part by the non-intuitive nature of the intervention. The imaginal nature of the intervention and its radical commitment to providing a non-traumatizing context for and experience of treatment have often been greeted with outright disbelief on the part of our participants (Gray et al., 2016; Gray & Bourke, 2015; Tylee et al., 2017). *Sample diversity*: While the sample is fairly representative (see Tables II and III), it is notable for its lack of female participants. It is also heavily weighted toward an older population with a mean age of nearly 50 years ($48.6 \pm 13.33$). Further, and as already noted previously, it is likely that the study attracted participants who were predisposed to respond well. As noted, although the study focused on a target population (intrusive symptoms primary with current-month flashbacks and nightmares), and thus has limited generalizability to PTSD more generally, its targeting is based upon clinical experience with older variants of the model that suggest that the intervention will not work for the excluded types (Gray & Liotta, 2012). We believe that, based upon clinical experience with previous versions of the intervention, RTM potentially applies to 50–75% of all afflicted by the disorder (Steve Andreas, personal communication, April 2016). Further experiments with access to larger pools of veterans will be able to test RTM's generalizability beyond our target group and in the process further clarify the boundaries of the larger subpopulation which this intervention appears to serve.

**The waiting-list control.** Waiting-list comparisons indicate, basically, that these results are better than nothing. Nevertheless, observed effect sizes (often greater than two standard measures), symptom reductions (>20 points on average), loss of diagnosis (for more than 73% of those treated), and maintenance of treatment gains to one-year post (Tylee et al., 2017) argue for its value when compared to other interventions using similar waiting-list controls (Bisson et al., 2013; Devilly & McFarlane, 2009; Foa & Meadows, 1997; Steenkamp et al., 2015; Steenkamp & Litz, 2013). An active comparison condition would have provided more generalizable results. As we have previously reported (Tylee et al., 2017), the agencies who have helped by referring clients have a great deal of difficulty finding volunteers for placebo, possibly less effective, or more noxious comparison conditions. It is with these limitations in mind that we chose the waiting-list design. We invite other researchers with better access to a base of other clients to create the required comparison trials.

**Length of the waitlist control period.** It is important to note that our 3-week waitlist protocol runs far short of more standard waitlist designs. Devilly and McFarlane's (2009) meta-analysis of 20 waiting-list studies of PTSD interventions showed a mean wait of 9.6 weeks with a 6-week median. Our short wait period may have allowed for the effects of other treatments and the normal progress of the disorder to have been expressed inaccurately and so have affected our measures. Further studies, if based on waitlist designs, should look to extend the waiting period to at least 6 weeks.

**Rebounding treatment scores.** Several clients, over the course of several evaluations, made dramatic changes in symptom scores, either improving dramatically or suddenly declining and then rebounding. Interviews with these clients found that their life situations had changed dramatically at one of the evaluation points and that those circumstances had affected their responses. This suggests the importance of framing of follow-up questions to ensure that the target trauma is the focus of their concern.

**Screening failures.** Despite high levels of remission, we note that some treatment responses varied from what was expected. RTM is expected to have some positive impact on symptom scores, even when the client does not lose the diagnosis. Three cases in the current study began with high PCL-M scores that failed to vary after treatment. When we reviewed video records of intake and treatment, all three appeared to be screening failures. They were

nevertheless included in the present analysis. One person could not identify an appropriate DSM Criterion 1 event. He had no flashbacks within the noted definition and no nightmares. He was just angry.

A second client experienced his trauma dissociatively. He dreamed about it as if through a tunnel and his "flashbacks" were experienced third person. The completely dissociative nature of his symptoms marked him as ineligible for the RTM intervention.

A third client complained of multiple traumas, but as the narratives proceeded, the events tended to merge into a broad all-encompassing anxiety. Although no direct measure was applied, the reviewer recognized the symptoms of Generalized Anxiety Disorder (GAD), without separate specific symptomatology that could he could identify as PTSD.

These cases reflect the need for a high level of expertise in the screening process. We recommend that, in the future, prospective clients be evaluated for GAD, that screeners be trained to differentiate clearly between PTSD and intense but otherwise normal anger, and to clearly identify the intrusive variety of PTSD for which RTM is best suited. Target traumas must be distinct events that can be differentiated from a broad emotional background. Experience suggests that (as with other PTSD interventions) targeting the trauma that is most closely associated by content or feeling tone to nightmares and flashbacks is often valuable in targeting the most relevant incident. In some cases, traumas appear to be linked as part of a complex memory system so that targeting one particularly salient trauma, in terms of its autonomic characteristics, can affect many associated traumas and comorbidities.

## Conclusion

These results, in line with other investigations of the RTM Protocol (Gray et al., 2016; Gray & Bourke, 2015; Tylee et al., 2017), suggest that the RTM protocol is a viable treatment modality for PTSD-related symptoms in a military population challenged by high levels of intrusive symptoms. Here, its application to a larger sample of male veterans suggests that the intervention may be an effective intervention but must be subjected to further evaluations using larger more diverse populations, and more rigorous investigative designs.

## Funding

This research was supported by funding from the State of New York (New York State Office of Mental Health) under contract number [C020338].

Geolocation. This study was made within the confines of the United States of America.

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