

Rebound Effects Following Deliberate Thought Suppression: Does PTSD Make a Difference?

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This study was designed to examine the effects of deliberate suppression of trauma-related thoughts in 44 individuals who were diagnosed with posttraumatic stress disorder (PTSD+) and 26 individuals who were not (PTSD–) following a motor vehicle accident (MVA). In an effort to resolve discrepancies in the literature, the PTSD– group was selected from the same help-seeking population as the patient group. Measures included the percentage of MVA-related thoughts, mood, perceived controllability of thoughts, and physiological arousal (heart rate, skin conductance, and two measures of facial EMG). Contrary to hypothesis, both PTSD+ and PTSD– groups showed a rebound in trauma-related thoughts following deliberate thought suppression. This rebound was associated with increases in negative affect, anxiety, and distress and diminished perceptions of controllability over thoughts. Examination of the physiological measures did not mirror the pattern noted for trauma-related thoughts, although the data suggest that suppression was associated with higher levels of frontalis EMG. The current study indicates that help-seeking individuals who are distressed about their psychological state following a serious MVA will show a rebound in MVA-related thoughts, irrespective of PTSD diagnosis. Implications for the study of thought suppression as a potential maintaining factor for trauma-related problems are discussed, with suggestions for future research.

THE THOUGHT SUPPRESSION PARADIGM represents a popular avenue for investigating the maintenance of cognitive intrusions and their role in psychopathology (e.g., Abramowitz, Tolin, & Street, 2001; Wenzlaff & Wegner, 2000). As originally observed by Wegner and colleagues (Wegner, Schneider, Carter, & White, 1987), attempting to suppress a target thought can produce an ironic increase in the occurrence of that thought. Two forms of this unexpected increase in target thoughts have been observed: (a) an immediate enhancement effect, where the frequency of target thoughts increases during the interval when suppression is occurring, and (b) a rebound effect, where the frequency of the target thought increases following deliberate suppression. This effect has been observed with personally irrelevant target thoughts (e.g., Kelly & Kahn, 1994; Wegner & Gold, 1995) as well as personally relevant thoughts (e.g., Smari, Birgisdottir, & Brynjolfsdottir, 1995). Several investigators have employed this paradigm to study deliberate thought suppression in clinical populations, particularly populations with diagnosed disorders that are characterized by intrusive thoughts such as obsessive-compulsive disorder (e.g., Tolin, Abramowitz, Przeworski, & Foa, 2002), depression (e.g., Wenzlaff, Wegner, & Roper, 1988), and posttraumatic stress disorder (PTSD; Shipherd & Beck, 1999; 2005). The current study examined emotional and psychophysiological correlates of deliberate thought suppression among individuals with PTSD and a help-seeking comparison sample.

One of the key characteristics of PTSD is recurrent, intrusive thoughts about the traumatic event (American Psychiatric Association, 1994). Several theoretical accounts posit that these intrusive thoughts signify an effort to assimilate information about the traumatic event into existing mental representations (Horowitz, 1976; Lang,

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1977). Presumably, attempts to suppress these thoughts could retard or inhibit emotional processing of the trauma and contribute to the maintenance of posttrauma intrusions (e.g., Foa & Kozak, 1986). In particular, suppression could interrupt the way that trauma survivors review trauma-related thoughts and feelings, which theoretically increases their risk for the development of PTSD. As such, past research has examined the role of thought suppression in naturalistic studies with trauma survivors. For example, Ehlers, Mayou, and Bryant (1998) assessed 967 individuals who attended an emergency clinic immediately after a motor vehicle accident (MVA) and followed these participants over the subsequent 12 months. Deliberate thought suppression emerged as a significant predictor of PTSD at 12 months, along with a collection of other demographic, psychological, and trauma characteristic factors. Thus, thought suppression may play a role in the etiology or maintenance of PTSD, based on this and related naturalistic studies (e.g., Aaron, Zagul, & Emery, 1999; Morgan, Mathews, & Winton, 1995). However, naturalistic studies do not allow precision in measurement when considering the role of thought suppression (e.g., separating the influence of repeated measurement from natural changes over time). Additionally, naturalistic studies do not allow determination of the causal relationship between variables. As such, investigators have begun to rely on the experimental thought suppression paradigm to examine in a more controlled fashion the effects of deliberately not thinking about trauma-related thoughts.

To date, four studies have been published using this paradigm with traumatized individuals, with very mixed results. Harvey and Bryant (1998) examined thought suppression in 48 newly traumatized individuals, 24 with acute stress disorder (ASD) and 24 without. All participants were inpatients who had been admitted to a trauma hospital following a serious MVA. Participants experienced three phases, each lasting for 5 minutes: First, they were told that they could think about anything; second, they were randomly assigned to either suppress or not suppress trauma-related thoughts; and third, they were instructed that they could think about anything. Both ASD and non-ASD participants who suppressed trauma thoughts showed a rebound in these thoughts during the third measurement interval, relative to those who had not received suppression instructions. In an effort to examine thought suppression over an extended time interval, Guthrie and Bryant (2000) used a similar paradigm as Harvey and Bryant (1998), only each phase was 24 hours in length. Derived from a sample of mixed trauma survivors recruited from a

trauma hospital, two groups were formed: a group of participants with ASD ($n = 20$) and a group without ASD ($n = 20$). No evidence was found for a rebound in trauma-related thoughts following deliberate suppression. Shipherd and Beck (1999) examined thought suppression in women with and without chronic PTSD following sexual assault. In this study, participants were recruited from the community for research involvement. The paradigm involved three phases, each lasting 9 minutes. All participants were asked to suppress trauma-related thoughts during the second phase, with similar instructions as those used by Harvey and Bryant during the first and third phases. Sexual assault survivors with chronic PTSD showed a rebound of trauma-related thoughts following suppression, whereas women without PTSD did not. To expand these findings, Shipherd and Beck (2005) used a slightly modified paradigm with individuals with PTSD following an MVA. In this second study, 30 PTSD patients were compared with 25 non-PTSD participants; the majority of participants were part of a larger treatment study, although a portion of the non-PTSD control group were recruited from friendship networks and ads in the community. The paradigm was expanded to include a personally relevant neutral thought target condition (e.g., thoughts about one's errands), in addition to a trauma-related thought target condition. The PTSD group showed a rebound effect following attempted suppression of trauma-related thoughts but was able to suppress neutral thoughts without a rebound. The non-PTSD group did not show a rebound effect following suppression of either neutral or trauma-related thoughts.

These findings suggest that deliberate thought suppression may play a role as a maintaining factor in PTSD, although the data from ASD patients paint a less clear picture. In considering the use of the thought suppression paradigm with clinical samples, it is salient to recognize that there are very few published studies, and within this small collection, methodological variations are apparent. In reviewing the four studies involving traumatized samples, two studies involved acute trauma survivors, with one study noting a rebound in previously suppressed thoughts for both diagnosed and control samples, while the second study failed to observe a rebound following suppression. Among the two studies that have focused on chronic trauma survivors, both reported rebound effects but only among diagnosed samples and only for trauma-relevant thoughts. Differences appear among these studies with respect to the strategies used to recruit "control" samples, including recruitment from the same medical or help-seeking population versus recruitment of

community volunteers, as well in the length of time involved in suppression. Additionally, it is important to recognize that the larger thought suppression literature is replete with mixed findings. A meta-analysis by Abramowitz and colleagues (2001) indicated a small to moderate rebound effect across studies (average $d = .30$) that varied in magnitude depending on methodological features, such as whether thoughts were assessed in writing, via a button press, or using another method.

Thus, the intent of this study was twofold. First, we sought to replicate the finding that patients with PTSD show a rebound in trauma-related thoughts following deliberate thought suppression (Shipherd & Beck, 1999, 2005). Accordingly, we hypothesized that patients with PTSD would show increased frequency of trauma-related thoughts following deliberate thought suppression, unlike individuals who had experienced a traumatic MVA but were not diagnosed with PTSD. Given differences among published studies, the methodology for this study was selected with an eye towards specific features such as the method of recruiting a control group and the length of time elapsed since the trauma. In this report, we replicated the methodology used by Shipherd and Beck (1999), with two exceptions: (a) instead of focusing on women with and without chronic PTSD following sexual assault, we focused on participants (both genders) with and without PTSD following a serious MVA, and (b) instead of relying on a community control sample, we used a control group that was seeking help for emotional problems relating to their MVA that was recruited from the same pool as those individuals with PTSD.

The second aim of this study was to examine psychophysiological concomitants of deliberate thought suppression. Because physiological hyperarousal represents a core symptom cluster of PTSD (American Psychiatric Association, 1994), it is plausible that deliberate thought suppression will be accompanied by reduced arousal and, conversely, a rebound in trauma-related thoughts accompanied by increased arousal, as speculated by some models of the psychopathology of PTSD (e.g., Brewin, Dalgleish, & Joseph, 1996). Specifically, Brewin et al. (1996) hypothesize that intrusive trauma thoughts may be accompanied by increases in physiological arousal. If this speculation is true, a rebound in trauma-related thoughts should be accompanied by increases in peripheral arousal. To date, only a handful of studies on deliberate thought suppression have incorporated psychophysiological measures and these studies have primarily involved measurement of skin conductance. As noted by Wegner and Gold (1995) and Wegner, Shortt, Blake,

and Page (1990), rebound of previously suppressed thoughts appears to be accompanied by an increase in skin conductance, particularly when the target thought carries emotional valence. In the present study, measures of skin conductance, heart rate, and facial EMG (corrugator and frontalis) were included to expand our knowledge of psychophysiological concomitants of deliberate thought suppression. Heart rate and skin conductance were selected because these measures have been shown to differentiate individuals with and without PTSD (e.g., Keane et al., 1998; Orr et al., 2003), in addition to being used in previous studies of thought suppression. Facial EMG measures were included as they have been noted to differentiate between veterans with PTSD and those without during combat-related imagery (Keane et al.). Because no previous investigation has studied psychophysiological responding during deliberate thought suppression in individuals with PTSD, the selection of these measures was designed to be as inclusive as possible; these measures were considered exploratory and thus no hypotheses were proposed for them.

Method

PARTICIPANTS

Seventy MVA survivors were included in this study. All participants were seeking assessment and treatment services at a university-based research clinic for help with emotional difficulties in the aftermath of an MVA. Participants were referred by chiropractors, massage and physical therapists, or physicians specializing in rehabilitation medicine or pain management. Individuals qualified for assessment if they had experienced an MVA involving actual or threatened death or serious injury and their emotional response included intense fear, helplessness, horror, or the perception that they would die (American Psychiatric Association, 1994). These features were evaluated during initial phone contact with the project and using the MVA Interview (Blanchard & Hickling, 1997). Individuals involved in minor accidents that did not satisfy Criterion A of the diagnostic criteria for PTSD were not evaluated. The sample included 53 women (76%) and 17 men (24%) and ranged in age from 18 to 65 (mean 39.5, SD 10.60). Fifty-three participants (75.7%) were Caucasian, 13 (18.6%) were African American, 3 (4.3%) were Hispanic, and 1 (1.4%) was Asian. The majority of patients ($n = 48$, 69%) reported ongoing pain complaints from injuries sustained during the MVA. In these cases, pain caused significant lifestyle limitations (e.g., inability to work), impairment (e.g., use of prescription pain medications at least 3 days/week),

or significant distress (e.g., continued health care utilization for pain). Average elapsed time after the MVA was 29.2 months (SD 75.64), with a range from 1 to 600 months. Data from individuals presenting with neurological impairment, substance dependence and abuse in the 6 months preceding the assessment, psychotic symptoms, or acute suicidality were excluded. All participants provided informed consent prior to participation.

All participants were administered the Clinician Administered PTSD Scale for *DSM-IV* (CAPS; Blake et al., 1990) and Anxiety Disorders Interview Schedule (ADIS-IV; DiNardo, Brown, & Barlow, 1994) by trained doctoral students. The CAPS is designed to assess the frequency and intensity of the 17 symptoms of PTSD in the past month, with ratings provided on a 5-point Likert scale for both of these dimensions. The CAPS was used to determine diagnostic status (PTSD+/PTSD-) in this study, using a rating of 1 or higher on the frequency dimension and a rating of 2 or higher on the intensity dimension to determine the presence of a symptom. As reviewed by Weathers, Keane, and Davidson (2001), the CAPS has excellent support for its reliability and validity. Interviews from a slightly larger sample ($N = 88$) that included the 70 participants in this report were videotaped and 35% ($n = 31$) were randomly selected and reviewed by an independent clinician to establish diagnostic reliability. Interrater agreement, reflected by the kappa statistic, was strong for PTSD ($k = .93$). The ADIS-IV was used to assess other *DSM-IV* anxiety and mood disorders. As illustrated by Brown, DiNardo, Lehman, and Campbell (2001), the ADIS-IV has good psychometric properties and is widely used as a diagnostic tool. As with the CAPS, 35% of a slightly larger sample ($N = 88$) was randomly selected to be rated by an independent interviewer. Agreement between diagnosticians was good for panic disorder ($k = .84$), social phobia ($k = .71$), generalized anxiety disorder ($k = .87$), specific phobia ($k = .73$), and major depressive disorder ($k = .84$), and moderate for panic disorder with agoraphobia ($k = .65$) and obsessive-compulsive disorder ($k = .64$).¹

In the current sample, 44 patients were classified as PTSD+ and 26 as PTSD-.² As seen in

Table 1, the groups did not differ with respect to age, the proportion of women, ethnicity, and employment status, although differences were noted with respect to the time elapsed since the MVA ($p < .03$) and pain ($p < .02$). The PTSD+ sample was assessed significantly closer in time to participants' MVA and significantly more members of this group reported chronic pain, relative to the PTSD- sample. Differences between the PTSD+ and PTSD- samples were noted on the Impact of Event Scale (Horowitz, Wilner, & Alvarez, 1979) and the PTSD Symptom Scale-Self-Report (Foa, Riggs, Dancu, & Rothbaum, 1993), as expected based on group assignment. As well, the PTSD+ sample reported higher levels of depressive symptoms on the Beck Depression Inventory (Beck, Steer, & Garbin, 1988), relative to the PTSD- sample.

PROCEDURE

The thought-suppression procedure was included as a component of a larger assessment in this study. Each participant was assessed individually after administration of the clinical interviews and questionnaires in a separate session (which was scheduled on a different day). Upon arrival for the assessment, the participant was introduced to the laboratory, a sound- and light-controlled environment with intercom communication. The physiological transducers were attached, followed by a 10-minute adaptation phase. At the end of the adaptation phase, the thought suppression procedure began, which consisted of three phases: Monitor 1, Suppression, and Monitor 2. Each phase was 9 minutes long and was preceded and followed by a 5-minute rest interval, to minimize carryover effects. Before both Monitor 1 and Monitor 2 phases, participants were asked to write down their thoughts, using the following instructions:

I would like for you to spend the next 9 minutes writing on this blank page whatever thoughts are present in your awareness from moment to moment. Your writing might include descriptions of images, memories, feelings, fantasies, plans, sensations, observations, daydreams, objects that catch your attention, or efforts to solve a problem. Do not worry about spelling or grammar.

Preceding the Suppression phase, participants were asked to deliberately suppress thoughts about their accident, using the following instructions:

I would like you to write down what you are thinking about again, exactly like you did before. Except, this time, please try not to think

¹ Other disorders were not present at a sufficient frequency in the reliability sample to compute kappa coefficients.

² Participants who met *DSM* criteria for PTSD in two of the three symptom clusters and reported clinically significant distress and interference from these symptoms were excluded from this study ($n = 18$). The decision was made to exclude these individuals given the fact that the Group variable was conceptualized as a between-group factor and it was necessary to clearly define the PTSD+/- samples.

Table 1
Characteristics of the PTSD+ and PTSD- samples

	PTSD+ (n = 44)	PTSD- (n = 26)	p
Age (years)	41.5 (7.80)	36.1 (13.65)	ns
Gender (% female)	82%	65%	ns
Months since MVA	13.9 (20.92)	55.04 (118.02)	.03
Ethnicity (% Caucasian)	75%	77%	ns
Employment status (% employed)	24%	31%	ns
Chronic pain (% yes)	79.5%	50%	.02
Impact of Event Scale–Intrusion subscale	20.6 (8.91)	5.6 (5.92)	.0001
Impact of Event Scale–Avoidance subscale	22.2 (9.38)	7.8 (8.95)	.0001
PTSD Symptom Scale–Self-Report	26.8 (11.65)	10.1 (9.48)	.0001
Beck Depression Inventory	21.5 (9.68)	10.9 (9.11)	.0001
Percent diagnosed with major depressive disorder	34% (n = 15)	8% (n = 2)	-
Percent diagnosed with additional anxiety disorders (other than PTSD)	59%	50%	ns
Percent diagnosed with Generalized Anxiety Disorder	36% (n = 16)	19% (n = 5)	-
Percent diagnosed with Social Phobia	14% (n = 6)	19% (n = 5)	-
Percent diagnosed with OCD	7% (n = 3)	0%	-
Percent diagnosed with Panic Disorder (with or without Agoraphobia)	11% (n = 5)	12% (n = 3)	-

about your car wreck, which you spoke about with the interviewer.

In order to replicate [Shipherd and Beck \(1999\)](#), writing was used as the medium for thought monitoring. Measures of the degree of suppression, subjective distress, and mood were taken immediately following each phase.

MEASURES

Percent MVA-related thoughts. Because a written thought listing procedure was used to determine the presence of target thoughts, the first step involved determination of specific “thought units” ([Cacioppo, von Hippel, & Ernst, 1997](#)). The majority of thought units were sentences, as determined by the third author. Two independent, trained raters, who were unaware of participants’ diagnostic status, coded these data. Raters were initially trained to 80% agreement with an independent coder, using thought listing records that are not included in this data set. All thoughts were coded as either MVA-related or non MVA-related. Raters were provided with a description of each participant’s MVA (drawn from the MVA Interview), in order to ensure accuracy in coding. Overall, raters were in agreement on 91% of thoughts coded, with discrepancies resolved by a third trained rater (the second author). Variation in the number of thoughts provided among individuals was controlled by calculating the percentage of MVA-related thoughts for each individual within each phase.

Mood. Mood ratings were taken after each phase, using three measures. First, participants completed the Positive and Negative Affect Schedule (PANAS; [Watson, Clark, & Tellegen, 1988](#)).

The PANAS includes two 10-item mood scales: the Positive subscale (PANAS-P) includes items that describe feelings associated with positive affect (e.g., excited, alert, inspired) and the Negative subscale (PANAS-N) includes items that describe feelings associated with negative affect (e.g., distressed, upset, scared). Each item is rated on a 1–5 scale with respect to how the participant feels “at the moment.” [Watson et al. \(1988\)](#) report evidence for convergent and discriminant validity, as well as internal consistency and test-retest stability. Second, participants were asked to rate their current level of anxiety on a 0–100 scale (0 = *no anxiety*, 100 = *as much anxiety as you can imagine*). Third, participants were asked to rate their current levels of distress, using a similar 0–100 scale.

Controllability of thoughts. Participants were asked to rate the perceived controllability of their thoughts on a 0–100 scale (0 = *no difficulty controlling*, 100 = *extreme difficulty controlling*). These ratings were taken after each phase.

Degree of suppression. Using a 0–100 scale (0 = *unsuccessful*, 100 = *completely successful*), participants were asked to rate their perceived success at suppressing accident-related thoughts. This rating was collected immediately after the Suppression phase only.

Physiological arousal. Four measures of physiological arousal were included: heart rate, skin conductance, EMG recorded from the left lateral frontalis region (the region that raises the outer and middle brow), and EMG recorded from the left corrugator region (the region that knits the brow). Heart rate was assessed using a pulse oximeter (Ohmeda 3700e) attached to the right earlobe, was sampled on-line, and was scored as beats/min. Skin

conductance was assessed from the medial phalanx of the palmar surface of the first two fingers of the nondominant hand, with Ag-AgCl electrodes, using a Coulbourn S71-23 isolated coupler (0.5 V constant voltage) and scored as micromhos. EMG was recorded using 4-mm diameter Sensor Medics Ag-AgCl electrodes attached with adhesive collars, using published guidelines for electrode placement (Fridlund & Cacioppo, 1986). For both EMG measures, electrodes were connected to a Coulbourn High Gain Bioamplifier (S75-01) set to filter signal components that were less than 90 Hz or more than 250 Hz frequency. The EMG signal then was processed by a Coulbourn Contour Following Integrator (S76-01) at a setting of 500 ms and were scored as microvolts. Each measure was sampled at a rate of 15 Hz using HS/Videograph software and visually checked for exclusion of movement artifacts. Data were averaged to form 3-min epochs in order to permit a finer-grained analysis of the physiological concomitants of deliberate thought suppression.

ANALYTIC STRATEGY

A series of ANOVAs were used to analyze these data. For all measures except the psychophysiological measures, a Group (2: PTSD+, PTSD-) \times Phase (3: Monitor 1, Suppression, Monitor 2) design was used, with repeated measures on the second factor. For the psychophysiological measures, a Group (2: PTSD+, PTSD-) \times Phase (3: Monitor 1, Suppression, Monitor 2) \times Epoch (3: first 3 min, middle 3 min, and final 3 min) design was used, with repeated measures on the second and third factors. Significant effects were followed up using the Tukey procedure, with comparison specific error terms for contrasts involving the repeated factors. Greenhouse-Geisser correction was used where the sphericity assumption was violated. Effect size was determined for each significant effect, using partial η^2 for effects derived from multiple-factor ANOVAs and r^2 for single-factor ANOVAs (which both reflect the percent of variance accounted for by each effect). Effect size was interpreted using Cohen's system (1988), wherein 2% to 12% represents a small effect, 13% to 44% a medium effect, and 45% or higher a large effect.

Results

PERCENT MVA-RELATED THOUGHTS

Examination of the percent MVA-related thoughts revealed a significant phase effect, $F(2, 136) = 31.09$, $p < .0001$, partial $\eta^2 = .31$, and a significant group effect, $F(1, 68) = 10.7$, $p < .002$, partial $\eta^2 = .14$. The Group \times Phase interaction

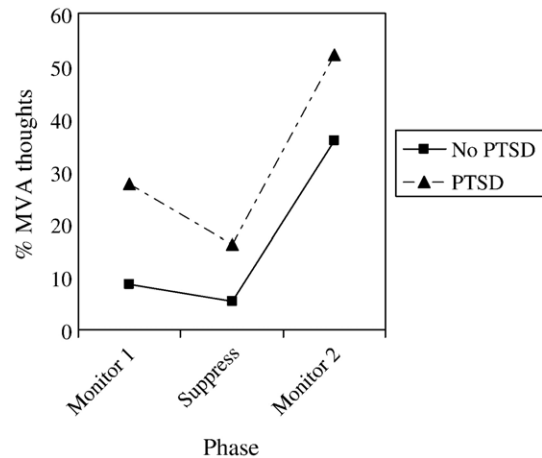


FIGURE 1 Percent MVA-related thoughts for PTSD+ and PTSD- samples, across the three phases.

term was not significant, contrary to hypothesis (see Figure 1). Follow-up of the significant phase effect indicated that participants showed significantly lower levels of MVA-related thoughts during the Suppression phase (mean 12.1%, SD 22.06) relative to the Monitor 1 phase (mean 20.5%, SD 30.04, $p < .05$). Participants showed a rebound in MVA-related thoughts during the Monitor 2 phase (mean 46.1%, SD 33.30), which was significantly different from both the Suppression and Monitor 1 phases ($p < .05$). Thus, participants appeared to suppress MVA-related thoughts during the Suppression phase, which was followed by a rebound in these thoughts during the Monitor 2 phase. Follow-up of the significant group effect indicated that the PTSD+ group reported a higher percentage of MVA thoughts overall (mean 31.9%, SD 31.11), relative to the PTSD- group (mean 16.6%, SD 19.24, $p < .05$).³

MOOD

Examination of the PANAS-N indicated a near-significant phase effect, $F(2, 134) = 2.85$, $p = .06$, partial $\eta^2 = .04$, and a significant group effect, $F(1, 67) = 8.89$, $p < .004$, partial $\eta^2 = .12$. The hypothesized Group \times Phase interaction was not significant. Follow-up of the Phase trend indicated that greater negative mood was noted during the Monitor 1 phase (mean 18.73, SD 8.33), relative to the Suppression phase (mean 17.69, SD 7.73, $p < .05$). During the Monitor 2 phase, negative mood was higher, relative to the other two phases

³These analyses were repeated, using arcsine transformation of the percentage of MVA thoughts, owing to concerns inherent with proportional data (Cohen & Cohen, 1975). Because the pattern of results was identical, the untransformed data are presented.

(mean 19.38, *SD* 8.35, $p < .05$). Follow-up of the significant group effect indicated that the PTSD+ group reported significantly higher levels of negative affect (mean 20.51, *SD* 8.47) relative to the PTSD- group (mean 15.24, *SD* 6.31, $p < .05$). Examination of the PANAS-P revealed no significant effects, although a trend was noted for the group effect, $F(1, 67) = 3.58$, $p = .06$, partial $\eta^2 = .05$, suggesting that the PTSD+ group reported overall lower levels of positive affect (mean 23.42, *SD* 8.66), relative to the PTSD- group (mean 27.29, *SD* 8.59). For the anxiety measure, a significant phase effect, $F(2, 136) = 6.02$, $p < .003$, partial $\eta^2 = .08$, and a significant group effect, $F(1, 68) = 9.12$, $p < .004$, partial $\eta^2 = .12$, were noted. For the distress measure, a significant phase effect, $F(2, 136) = 10.83$, $p < .0001$, partial $\eta^2 = .14$, and a significant group effect, $F(1, 68) = 10.99$, $p < .001$, partial $\eta^2 = .14$, also were noted. For both of these measures, a nonsignificant reduction in affect was noted between the Monitor 1 (anxiety mean 37.64, *SD* 27.65; distress mean 34.76, *SD* 30.12) and Suppression (anxiety mean 35.13, *SD* 29.72; distress mean 34.34, *SD* 31.00) phases, with a significant increase in negative affect noted during the Monitor 2 phase (anxiety mean 45.46, *SD* 32.21; distress mean 47.13, *SD* 33.83; $ps < .05$). As with the other mood measures, the PTSD+ group reported higher levels of anxiety (mean = 38.69, *SD* 29.89) and distress (mean 46.84, *SD* 32.29), compared with the PTSD- group (anxiety mean 27.53, *SD* 26.04; distress mean 25.04, *SD* 25.41; $ps < .05$).

CONTROLLABILITY OF THOUGHTS

A significant phase effect, $F(2, 136) = 6.88$, $p < .001$, partial $\eta^2 = .09$, was noted for the controllability measures. Follow-up of the phase effect indicated that participants reported the least amount of difficulty controlling their thoughts during the Monitor 1 phase (mean 36.04, *SD* 32.55), which increased significantly during the Suppression phase (mean 44.20, *SD* 33.48) and again during the Monitor 2 phase (mean 51.59, *SD* 34.48; $ps < .05$). Additionally, a significant group effect, $F(1, 68) = 8.00$, $p < .006$, partial $\eta^2 = .11$, was reported, indicating that the PTSD+ group reported significantly more difficulty controlling their thoughts (mean 50.57, *SD* 32.61) than the PTSD- group (mean 32.73, *SD* 32.33, $p < .05$).

DEGREE OF SUPPRESSION

Comparison of the perceived success at suppressing MVA-related thoughts during the Suppression phase indicated that the PTSD+ group reported significantly lower ratings (mean 44.09, *SD* 31.7),

relative to the PTSD- group (mean 75.08, *SD* 27.7, $t = 4.13$, $p < .0001$, $r^2 = .20$).

PHYSIOLOGICAL MEASURES

Examination of the heart rate data indicated only a significant phase effect, $F(1.54, 103.39) = 18.50$, $p < .0001$, partial $\eta^2 = .22$, which revealed that heart rate during the Monitor 1 phase was significantly higher (mean 79.38 beats/min, *SD* 11.83) relative to the Suppression phase (mean 77.04 beats/min, *SD* 10.93, $p < .05$) and the Monitor 2 phase (mean 77.21 beats/min, *SD* 10.58, $p < .05$). Examination of skin conductance revealed a marginally significant Group \times Epoch interaction, $F(1.77, 116.59) = 2.89$, $p = .06$, partial $\eta^2 = .04$, and a significant epoch main effect, $F(1.77, 116.59) = 13.52$, $p < .0001$, partial $\eta^2 = .17$. Follow-up of the interaction trend indicated that for the PTSD+ group, responding during the middle 3 mins of each phase was significantly higher (mean 0.47 micromhos, *SD* 0.65), relative to the first 3 mins (mean 0.40 micromhos, *SD* 0.62, $p < .05$) and the final 3 mins (mean 0.42 micromhos, *SD* 0.65, $p < .05$), which did not differ. For the PTSD- group, responding during the middle (mean 0.45 micromhos, *SD* 0.57) and final 3 min epochs (mean 0.45 micromhos, *SD* 0.52) phases were significantly higher than the initial 3 min epoch (mean 0.36 micromhos, *SD* 0.53, $ps < .05$).

Examination of frontalis region EMG revealed a marginally significant Group \times Phase interaction, $F(1.22, 83.33) = 3.08$, $p < .07$, partial $\eta^2 = .04$, and a significant Phase \times Epoch interaction, $F(1.93, 131.49) = 6.06$, $p < .003$, partial $\eta^2 = .08$. Examination of the Group \times Phase interaction trend indicated that the PTSD+ group showed increased responding of frontalis EMG during the Suppression (mean 2.37 μ V, *SD* 0.12) and Monitor 2 (mean 2.36 μ V, *SD* 0.15) phases, relative to the Monitor 1 phase (mean 2.33 μ V, *SD* 0.13, $ps < .05$). In contrast, the PTSD- group showed reduced responding during the Monitor 2 phase (mean 2.31 μ V, *SD* 0.24), relative to Monitor 1 (mean 2.37 μ V, *SD* 0.15, $p < .05$) with responding during the Suppression phase falling in between (mean 2.35 μ V, *SD* 0.16). Follow-up testing failed to indicate any between-group differences for this trend. Examination of the Phase \times Epoch interaction indicated that during the Monitor 1 phase, frontalis EMG responding significantly increased during the last 3-min epoch ($p < .05$; see Figure 2). In contrast, during the Suppression and Monitor 2 phases, responding slightly but significantly decreased across the three 3-min epochs ($p < .05$). Comparison of the three phases at each epoch indicated that responding during the Suppression phase was

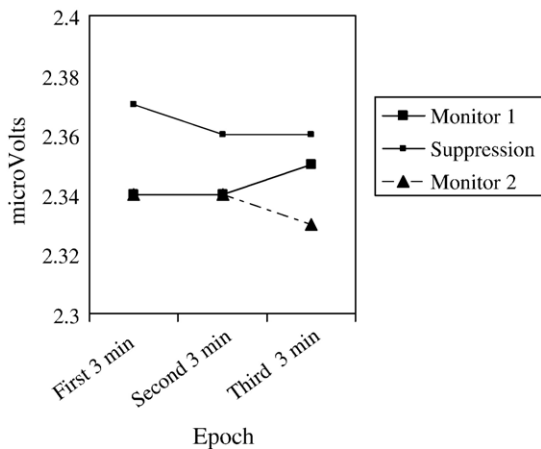


FIGURE 2 Frontalis EMG across the three phases of the procedure, shown in 3-min epochs.

significantly higher, relative to Monitor 1 and Monitor 2, for the first and second 3-min epochs ($p < .05$). During the third 3-min epoch, only the Suppression and Monitor 2 phases differed significantly ($p < .05$), with higher responding during the Suppression phase. Examination of the corrugator region EMG did not indicate any significant findings.⁴

Discussion

Contrary to hypothesis and to previous studies in our lab, a rebound in trauma-related thoughts was noted following deliberate thought suppression in individuals without chronic PTSD in this study. Much like previous studies using this procedure (Shpherd & Beck, 1999, 2005), individuals diagnosed with chronic PTSD also showed a rebound in trauma-related thoughts following suppression. In both groups, this rebound was associated with increases in negative affect, anxiety, and distress. As well, perceptions of controllability over thoughts diminished in the interval following deliberate suppression. Examination of the physiological measures suggested that suppression was associated with higher levels of frontalis EMG, although these data did not mirror the pattern noted for trauma-related thoughts and mood.

In considering these findings, it is important to highlight that this study included a help-seeking control sample. Although the PTSD- sample did not report sufficient symptomatology to receive a

diagnosis of PTSD, participants were distressed in the wake of their traumatic MVA and sought psychological assessment. In previous studies involving the thought suppression paradigm with chronic PTSD patients, the control groups have been recruited expressly for research participation and/or have been paid for their involvement, suggesting that they may have been less distressed in the wake of their trauma or interpreted the purpose of the thought suppression procedure differently. In the present study, the thought listing procedure was incorporated as part of the overall psychological assessment, possibly changing the way that participants interpreted its intent. Of note, it is salient that the findings of the current study resemble results reported by Harvey and Bryant (1998), who recruited their control sample from the same inpatient hospital environment as their ASD sample. Although direct comparison of these two data sets is difficult given differences in analytic approach, both studies found an increase in trauma-related thoughts following deliberate thought suppression (relative to baseline) irrespective of diagnosis status. Both studies used relatively brief (5 to 9 min) intervals for the thought suppression procedure but differed in how participants recorded trauma-related thoughts during the procedure, a methodological feature that has been highlighted as relevant in explaining differences in effect sizes across thought suppression studies (Abramowitz et al., 2001). As seen in Figure 1, the rebound during Monitor 2 (relative to baseline) that was observed among PTSD- participants in this study is notable and occurred even when the data were reanalyzed to only include participants who were free of other anxiety and depressive disorders in the PTSD- subsample. Thus, contrary to related studies, the current data suggest that rebound following deliberate suppression of trauma thoughts may be influenced by factors such as whether the participant is help-seeking and how the thought suppression procedure is perceived. These data suggest that there is considerably more to learn about the various parameters that influence the outcome of deliberate thought suppression among trauma survivors.

When considering the pattern of physiological arousal that was found in the current study, it is notable that thought suppression was accompanied by increased responding in frontalis EMG, a response that is akin to “frowning” one’s brow. This response was most salient during the first 6 minutes of each phase, suggesting that this was the interval when thought suppression was accompanied by greater tension in the frontalis region. Perhaps it is during this time when the greatest

⁴ We also conducted a major subset of the analyses a second time, controlling for diagnostic comorbidity. Participants in the PTSD- group who were free of all other anxiety and mood disorders ($n = 13$) were specifically compared with the PTSD+ group. The pattern of results was basically the same. Interested readers may contact the first author for more details concerning these analyses.

effort at suppression is being put forth, a hypothesis that requires continuous assessment during the various phases of this procedure. In contrast with studies by Wegner and colleagues (Wegner & Gold, 1995; Wegner et al., 1990), rebound of previously suppressed thoughts was not accompanied by increased skin conductance. Rather, a trend was observed wherein the PTSD+ group showed a different pattern of skin conductance responding from the PTSD- group, irrespective of whether they were deliberately suppressing trauma-related thoughts or not. Because of the exploratory nature of these measures in this context, it seems prudent to avoid overinterpretation of trends, pending replication. Additionally, heart rate diminished across the duration of the procedure; thus, the reduction in heart rate noted during the Monitor 2 phase appears to be part of a general habituation response to the experimental setting and the procedure in general. Thus, contrary to current models of PTSD which propose that intrusive thoughts about the trauma are accompanied by physiological hyperarousal, the current study did not observe a surge in peripheral autonomic arousal to accompany the increase in trauma-related thoughts during the Monitor 2 phase. Although physiological hyperarousal is a component of chronic PTSD and has been documented to occur in the laboratory to stimuli such as audiotapes or vivid imagery (e.g., Keane et al., 1998), it would appear that this response is not manifested as autonomic surges in response to trauma-related thoughts. Current data on biopsychological aspects of chronic PTSD suggest that the disorder is marked by enhanced negative feedback inhibition of cortisol on the pituitary and, possibly, elevated reactivity of the HPA axis (e.g., Yehuda, 2003). The result of these processes is that cortisol is tonically suppressed but sensitized to overreact to acute challenge. Although data examining cortisol and select catecholamines (e.g., epinephrine, norepinephrine) among individuals with MVA-related PTSD have only partially supported this hypothesis (e.g., Hawk, Dougall, Ursano, & Baum, 2000), the current results suggest that momentary shifts in heart rate and skin conductance may not necessarily accompany intrusive trauma-related thoughts. Additionally, it is possible that chronic levels of anxiety are not closely associated with autonomic arousal, unlike briefer anxiety states (e.g., Hoehn-Saric & McLeod, 1988). Clearly, there is a need for more refined studies that attempt to integrate cognitive, emotional, and biopsychological aspects of PTSD, with an eye towards including measures such as cortisol and epinephrine.

So, what do these data tell us about deliberate thought suppression among individuals who are distressed in the wake of a serious MVA? Could thought suppression play a potential role as a factor that maintains intrusive thoughts among individuals with PTSD? The current study unfortunately adds to the mixed data that have accumulated to date. Across these studies, it is clear that under some circumstances, deliberate thought suppression results in a rebound of trauma-related thoughts among individuals who are acute as well as chronic survivors of traumatic events. In some studies, this rebound is uniquely associated with PTSD diagnosis; in others, it is not specific to diagnosed samples. In the current study, the rebound in MVA-related thoughts was accompanied by negative affect, anxiety, and distress, although these interrelationships have not been seen in other studies (see Shipherd & Beck, 1999, 2005). Clearly, greater understanding of the circumstances when deliberate thought suppression will result in an ironic increase in the target thought is needed, particularly as these processes relate to psychological distress and impairment. The current study suggests that help-seeking individuals who are distressed about their psychological functioning in the aftermath of a serious car crash will show a rebound of trauma-related thoughts following deliberate suppression (relative to baseline), irrespective of PTSD diagnosis. Perhaps what underlies the rebound effect is the individual's general level of distress about the trauma. This speculation would account for the increased negative emotions that occurred in the current study (but have not been seen in studies employing control samples that were recruited using different strategies). When combined with naturalistic reports of the importance of deliberate thought suppression in the prediction of PTSD (e.g., Ehlers et al., 1998), it seems reasonable to conclude that deliberate thought suppression is a relevant process in delaying emotional recovery after a traumatic event.

In considering this study, it is relevant to discuss its limitations. Although a sample with clinical features was desired for this replicative effort, the sample sizes in the current study are not large, particularly for the PTSD- sample. A larger sample would have permitted additional analyses to examine predictors of the magnitude of rebound following suppression and potentially could have revealed individual difference factors that account for variation in the degree of rebound. In this effort, it might be useful to examine the amount of time that has elapsed since the traumatic event, as

deliberate thought suppression perhaps may have different effects when applied at different times posttrauma. In particular, posttrauma symptoms are more prevalent closer in time to a traumatic event (e.g., Blanchard & Hickling, 1997; Rothbaum, Foa, Riggs, Murdock, & Walsh, 1992). It is possible that the use of deliberate thought suppression early on (<6 months) after a trauma may have different effects than when used by individuals with more chronic PTSD. Second, it is possible that these results reflect unique methodological features of this paradigm. For example, the instructions that preceded the Monitor 2 phase did not explicitly cancel the previous suppression instructions. It is possible that some participants could have misunderstood the instructions and continued to suppress during the Monitor 2 phase. Likewise, the three instructional sets were not matched for length nor for mention of the MVA, which could have influenced these findings. As well, it is possible that inclusion of a 5-minute rest interval following each phase could have resulted in a loss of data. Although this was necessary to minimize carryover between phases in the physiological measures, this methodological feature could have influenced the obtained results. Third, the current study made no effort to examine basic processes that underlie deliberate thought suppression and its effects. It is possible that a shift in research focus could benefit our understanding of this issue. In particular, future studies perhaps could move away from examining whether rebound occurs following thought suppression and focus instead on the strategies that participants employ when asked to deliberately suppress trauma-related thoughts. Alternatively, examination of specific suppression techniques (e.g., deliberately thinking about another topic, redirecting one's focus) potentially could reveal greater information about how deliberate suppression of trauma-related thoughts can ironically backfire. Lastly, future work would benefit from inclusion of a broader array of physiological measures, including possible measures of endocrine function.

This study has some important clinical implications, particularly when placed among related studies in the field. The current results, along with naturalistic reports, suggest that thought suppression appears to be one mechanism that maintains emotional distress, anxiety, and perceptions of uncontrollability among trauma survivors. Clearly, greater study of when thought suppression "backfires" and when it does not would be helpful, particularly in developing programs designed to prevent the development of trauma-related emotional problems.

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