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BRIEF REPORTS

The Additive Benefit of Hypnosis and Cognitive–Behavioral Therapy in Treating Acute Stress Disorder

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This research represents the first controlled treatment study of hypnosis and cognitive–behavioral therapy (CBT) of acute stress disorder (ASD). Civilian trauma survivors ($N = 87$) who met criteria for ASD were randomly allocated to 6 sessions of CBT, CBT combined with hypnosis (CBT–hypnosis), or supportive counseling (SC). CBT comprised exposure, cognitive restructuring, and anxiety management. CBT–hypnosis comprised the CBT components with each imaginal exposure preceded by a hypnotic induction and suggestions to engage fully in the exposure. In terms of treatment completers ($n = 69$), fewer participants in the CBT and CBT–hypnosis groups met criteria for posttraumatic stress disorder at posttreatment and 6-month follow-up than those in the SC group. CBT–hypnosis resulted in greater reduction in reexperiencing symptoms at posttreatment than CBT. These findings suggest that hypnosis may have use in facilitating the treatment effects of CBT for posttraumatic stress.

Acute stress disorder (ASD) describes posttraumatic stress reactions that occur in the initial month after a traumatic experience (Bryant & Harvey, 2000). The major goal of this diagnosis is to identify acutely traumatized individuals who are at high risk for developing posttraumatic stress disorder (PTSD). Prospective studies have indicated that approximately 80% of people who initially meet criteria for ASD subsequently develop chronic PTSD (see Bryant, 2003). The predictive ability of the ASD diagnosis provides an opportunity to provide early intervention to those individuals who are most likely to develop PTSD.

Early intervention studies for posttraumatic stress have indicated moderate success in preventing PTSD. Foa, Hearst-Ikeda, and Perry (1995) provided recent sexual and nonsexual assault victims with four sessions of either cognitive–behavioral therapy (CBT) or repeated assessments. At 2-month posttrauma, 10% of the CBT group met criteria for PTSD compared with 70% of the repeated-assessments group. In the first treatment study of ASD, civilian trauma survivors were allocated to either CBT or supportive counseling (SC; Bryant, Harvey, Sackville, Dang, & Basten, 1998). CBT comprised education, prolonged imaginal exposure of the traumatic memories, in vivo exposure, anxiety management, cognitive restructuring, and relapse prevention. This study found that 67% of those who received SC suffered PTSD at 6-month follow-up compared with 20% of those who received CBT. In a replication study, ASD participants were allocated to either SC, a group comprising prolonged exposure and cognitive therapy (PE–CT), or a combination of PE–CT and anxiety management (PE–CT + AM; Bryant, Sackville, Dang, Moulds, & Guthrie, 1999). At 6-month posttrauma, there were fewer cases of PTSD in the PE–CT (18%) and PE–CT + AM (23%) groups than in the SC (62%) group.

It has been proposed that the optimal technique for treating ASD is hypnotherapy because the focused attention and heightened involvement associated with hypnotic techniques may breach dissociative symptoms that characterize ASD (Spiegel, 1996). The potential use of hypnosis is underscored by evidence that ASD participants are more hypnotizable than trauma survivors who do not develop ASD (Bryant, Moulds, & Guthrie, 2001). The potential benefit of using hypnosis in combination with CBT is indicated by evidence that providing hypnosis in combination with CBT leads to greater clinical gains than providing CBT alone (Kirsch, Montgomery, & Sapirstein, 1995). One study compared the effectiveness of desensitization, hypnotherapy, psychodynamic therapy, and a wait-list control group for posttraumatic stress (Brom, Kleber, & Defares, 1989). Hypnotherapy was intended to facilitate emotional processing of trauma memories through behaviorally oriented strategies. This study found that all three active treatments were comparably effective. This study's conclusions were limited by (a) the lack of independent assessments, (b) inadequate specification about therapy components, and (c) the absence of independent treatment fidelity checks. Considering the long historical connections between hypnosis and trauma response (e.g., Janet, 1907), it is surprising that there are no adequately controlled treatment outcome studies of hypnotherapy in ASD or PTSD (see Cardeña, Maldonado, van der Hart, & Spiegel, 2000).

In the current study, we aimed to conduct a controlled outcome study that evaluated the additive benefit of combining hypnosis with CBT in the treatment of ASD. Our premise was that a hypnotic induction preceding exposure, combined with sugges-

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tions to engage in the exposure exercise, would facilitate emotional processing of trauma memories. Accordingly, we compared (a) CBT, (b) CBT combined with hypnosis (CBT–hypnosis), and (c) SC. We hypothesized that CBT–hypnosis would lead to more symptom reduction than CBT, which in turn would result in more symptom reduction than SC.

Method

Participants

Participants were 87 (53 women, 34 men) consecutive civilian trauma survivors who were referred to the Westmead Hospital PTSD Unit (Sydney, Australia) following nonsexual assault ($n = 48$) or a motor vehicle accident ($n = 39$). All participants met the criteria for ASD defined in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV;* American Psychiatric Association, 1994). Exclusion criteria included history of psychosis, organic brain syndrome, substance dependence disorder, current suicidal ideation, history of childhood sexual abuse, or an age of less than 17 years or more than 60 years. Initial assessments of potential participants were initially screened by telephone, and full assessments were conducted only on participants who did not report any exclusion criteria on telephone screening. There were 109 participants assessed, and 22 were excluded because they declined therapy ($n = 6$), were markedly suicidal ($n = 8$), or had a history of substance dependence ($n = 8$).

Assessments

Prior to diagnostic interview, participants were assessed for hypnotic suggestibility with the Stanford Hypnotic Clinical Scale for Adults (Morgan & Hilgard, 1978–1979). This assessment involves a hypnotic induction followed by five demanding hypnotic suggestions; scores greater than 3 indicate generally high levels of hypnotic suggestibility. Independent clinicians subsequently diagnosed ASD using the Acute Stress Disorder Interview (ASDI; Bryant, Harvey, Dang, & Sackville, 1998). This structured clinical interview is based on *DSM–IV* criteria for ASD, contains 19 dichotomously scored items that relate to ASD symptoms, and provides a total score of acute stress severity (range = 1–19). The ASDI possesses sound test–retest reliability ($r = .95$), sensitivity (92%), and specificity (93%) in a sample of motor vehicle accident and nonsexual assault survivors. Participants were also administered the Impact of Event Scale (IES; Horowitz, Wilner, & Alvarez, 1979), the Beck Depression Inventory—2 (Beck, Steer, & Brown, 1996), and the Beck Anxiety Inventory (BAI; Beck & Steer, 1990). To obtain an estimate of verbal intelligence, we also administered the National Adult Reading Test to the participants (Nelson, 1982). PTSD diagnosis was identified by the Clinician Administered PTSD Scale—2 (CAPS–2; Blake et al., 1995). The CAPS–2 is a structured clinical interview that indexes the 17 symptoms described by the *DSM–IV* PTSD criteria. Each symptom is rated on a 5-point scale in terms of the severity and frequency of the symptom in the past week. At the completion of Session 1 and after the treatment rationale had been explained, participants rated their confidence in the treatment (1 = *not at all confident*, 7 = *extremely confident*) and the perceived logic of the treatment (1 = *not at all logical*, 7 = *extremely logical*).

This study followed each of the gold standards described by Foa and Meadows (1997) in their recommendations for treatment outcome research for PTSD. Accordingly, assessments were conducted at pretreatment, posttreatment, and 6-month follow-up by independent clinicians who were unaware of the treatment group of the participants. Blindness was maintained by ensuring that clinicians who conducted assessments did not have access to (a) participant notes, (b) treatment allocation of participants, or (c) supervision discussions of therapy sessions. To ensure initial ASD assessments were conducted reliably, two independent clinicians conducted 20% of the initial assessments. The interrater reliability of the two assessments in terms of diagnostic decision was 100%. All measures were administered at each assessment, except that the ASDI, therapy confidence, and logic ratings were conducted only at pretreatment, and the CAPS–2 was administered only at posttreatment and follow-up assessments.

Treatments

Participants were randomly allocated to one of the three treatment groups. Randomization was conducted by a process of minimization stratified on gender, trauma type, and ASDI total score. Participants were randomly assigned according to a random numbers system, and each month Richard A. Bryant amended allocation to ensure that gender, trauma type, and PTSD severity were balanced across groups. Individual therapy was conducted by one of three master's-level clinical psychologists. All three therapists were trained to use treatment manuals and received weekly supervision from Richard A. Bryant. Therapists were also blinded to participants' hypnotic suggestibility level. Treatment comprised five once-weekly 90-min sessions with structured daily homework activities.

CBT. Session 1 focused on psychoeducation, training in breathing control, the rationale of exposure, and conducting the initial imaginal exposure. The exposure exercise occurred for 50 min and was repeated to ensure that exposure occurred for 50 min. Imaginal exposure was not audiotaped, but participants were given explicit instructions on how to complete the exercise. Following this session, participants engaged in imaginal exposure on a daily basis for homework. Session 2 included review of homework, introduction of cognitive restructuring, introduction of in vivo exposure, and a 50-min session of imaginal exposure. Following this session, participants completed imaginal exposure and monitoring of automatic thoughts as daily homework. Session 3 included review of homework, cognitive restructuring, and completion of hierarchy for in vivo exposure. Cognitive restructuring involved daily monitoring of thoughts and affective states, modifying thoughts by Socratic questioning, probabilistic reasoning, and evidence-based thinking. Following this session, participants completed imaginal exposure, cognitive restructuring, and in vivo exposure for daily homework. Session 4 included review of homework, cognitive restructuring, and review of in vivo exposure. Session 5 was identical to Session 4, with the addition of relapse-prevention strategies. Prior to imaginal exposure, 15 min of Sessions 1 and 2 were devoted to SC to ensure that equivalent time within sessions for the CBT and CBT–hypnosis groups was devoted to exposure.

CBT–hypnosis. This treatment group was identical to the CBT protocol with one exception. Prior to each imaginal exposure exercise, participants listened to an audiotape that described a hypnotic induction. The 15-min induction involved suggestions for focused attention, muscle relaxation, and deepening suggestions. The induction was modeled on the hypnotic induction described in the Stanford Hypnotic Clinical Scale for Adults (Morgan & Hilgard, 1978–1979). This was followed by 2 min of suggestions so that the participants could engage fully in the exposure exercise and experience as much affective and sensory detail as possible.

SC. The SC program comprised education about trauma and general problem-solving skills. The SC program also provided an unconditionally supportive role. Homework comprised diary keeping of current problems and mood states. SC specifically avoided hypnosis, cognitive restructuring, and exposure techniques.

Treatment Fidelity

Audiotapes of 40 therapy sessions (9% of therapy sessions) were randomly selected and rated by three independent experts in CBT. Raters listened to audiotapes and rated the presence or absence of 36 treatment components, without regard to treatment group or treatment session. Furthermore, raters indicated the quality of the therapy session on a 7-point scale (1 = *unacceptable*, 7 = *very good*). The only omission in the CBT–hypnosis treatments was that distress ratings were not obtained

during one exposure session for 1 participant. No CBT participants received hypnosis. No SC sessions included breathing retraining, exposure, or cognitive restructuring. The mean quality ratings for treatment components in CBT and CBT–hypnosis were 5.60 (SD = 1.12) and 5.80 (SD = 2.03), respectively

Results

Preliminary Analyses

A total of 87 participants entered the study; 69 completed treatment, and 69 were retained for follow-up. Planned comparisons indicated that treatment completers and dropouts did not differ on pretreatment psychopathology measures, age, National Adult Reading Test score, time since trauma, or hypnotic suggestibility. Participants who dropped out (M = 6.69, SD = 1.54) were less confident about treatment than completers (M = 7.71, SD = 1.83), $t(85) = 2.05$, $p < .05$. There was a tendency for greater dropout in the CBT (27%) and CBT–hypnosis (23%) groups than the SC (8%) group, $\chi^2(2, N = 87) = 3.23$, $p = .19$. Table 1 presents the mean participant characteristics. One-way analyses of variance of participants' characteristics indicated no differences between treatment groups.

Immediate Treatment Effects

Table 2 and Table 3 present the mean psychopathology scores for the intent-to-treat and treatment completer samples, respectively. Intent-to-treat values were derived with a last-value-carried-forward procedure to provide data for missing values that occurred because of dropout. To control for the multiple dependent variables used in this study, we conducted overall multivariate analyses of covariance (MANCOVAs) on CAPS–2 Intensity, CAPS–2 Frequency, IES–Intrusion, IES–Avoidance, BAI, and Beck Depression Inventory—2 scores for (a) posttreatment and (b) follow-up data that controlled for initial symptom severity and trauma type. Pretreatment symptom severity was controlled by entering the pretreatment score for each psychopathology measure, with the exception that pretreatment for CAPS–2 scores was controlled with pretreatment ASDI scores. Significant overall MANCOVAs were followed by analyses of covariance (ANCOVAs) of each measure and subsequent post hoc Tukey comparisons. CAPS–2 scores are only reported for treatment completers because the CAPS–2 was not administered at pretreatment.

In terms of the intent-to-treat analyses, the MANCOVA on posttreatment scores indicated a significant main effect, $F(14, 154) = 5.80$, $p < .001$. Follow-up ANCOVAs indicated main effects for IES–Intrusion, $F(2, 82) = 6.04$, $p < .005$. Post hoc Tukey comparisons indicated that CBT–hypnosis participants scored lower than SC participants on IES–Intrusion ($p < .005$). CBT participants scored lower than SC participants on IES–Intrusion ($p < .05$). In addition, CBT–hypnosis participants scored lower than CBT participants on IES–Intrusion ($p < .05$).

In terms of treatment completers, the one-way MANCOVA indicated a significant main effect, $F(12, 114) = 25.84$, $p < .001$. Follow-up ANCOVAs indicated main effects for CAPS–2 Intensity, $F(2, 64) = 8.65$, $p < .001$; CAPS–2 Frequency, $F(2, 64) = 7.33$, $p < .001$; IES–Intrusion, $F(2, 64) = 10.51$, $p < .001$; IES–Avoidance, $F(2, 64) = 8.94$, $p < .001$; and BAI, $F(2, 64) = 3.41$, $p < .05$. Post hoc Tukey comparisons indicated that CBT–hypnosis participants scored lower than SC participants on CAPS–2 Intensity ($p < .005$), CAPS–2 Frequency ($p < .01$), IES–Intrusion ($p < .001$), IES–Avoidance ($p < .05$), and BAI ($p < .05$). CBT participants scored lower than SC participants on CAPS–2 Intensity ($p < .002$), CAPS–2 Frequency ($p < .005$), IES–Intrusion ($p < .05$), IES–Avoidance ($p < .001$), and BAI ($p < .05$).

Follow-Up Treatment Effects

In terms of intent to treat analysis, the overall MANCOVA indicated a significant main effect, $F(8, 154) = 2.09$, $p < .05$. Follow-up ANCOVAs indicated main effects for IES–Intrusion, $F(2, 84) = 3.00$, $p < .05$. Post hoc Tukey comparisons indicated that CBT–hypnosis participants scored lower than SC participants on IES–Intrusion ($p < .05$).

The overall MANCOVA for treatment completers indicated a significant main effect, $F(8, 118) = 2.12$, $p < .05$. Follow-up ANCOVAs indicated main effects for CAPS–2 Intensity, $F(2, 64) = 3.73$, $p < .05$; CAPS–2 Frequency, $F(2, 64) = 3.05$, $p < .05$; IES–Intrusion, $F(2, 64) = 4.64$, $p < .05$; and IES–Avoidance, $F(2, 64) = 3.90$, $p < .05$. Post hoc Tukey comparisons indicated that CBT–hypnosis participants scored lower than SC participants

Table 1
Participant Characteristics

	CBT		CBT–hypnosis		Supportive counseling		
Variable	*M*	*SD*	*M*	*SD*	*M*	*SD*	*F*(2, 84)
Age	33.09	12.45	32.97	7.70	35.00	13.28	0.27
Time since trauma	15.75	8.79	13.54	6.71	14.04	8.42	0.66
NART	24.83	8.47	27.82	9.69	30.00	7.55	1.67
SHSS:C	3.63	1.21	3.14	1.38	3.35	1.22	0.94
ASDS	64.60	11.36	67.39	12.71	65.18	16.53	0.29
Logic rating	8.31	1.63	8.14	1.38	8.67	1.46	0.84
Confidence rating	7.21	1.88	7.45	1.78	8.00	1.74	1.32

Note. Age measured in years. Time since trauma measured in days. CBT = Cognitive–behavioral therapy; NART = National Adult Reading Test; SHSS:C = Stanford Hypnotic Susceptibility Scale, Form C; ASDS = Acute Stress Disorder Scale.

Table 2
Psychopathology Measures for Intent-to-Treat Sample

	CBT $n = 33$		CBT–hypnosis $n = 30$		Supportive counseling $n = 24$	
Variable	*M*	*SD*	*M*	*SD*	*M*	*SD*
Pretreatment						
IES–Intrusion	27.12	7.46	24.73	8.06	24.58	8.21
IES–Avoidance	21.58	9.66	24.43	9.49	19.92	9.79
BAI	24.39	11.23	27.27	11.47	28.67	13.45
BDI–2	19.97	10.01	18.40	8.39	22.04	11.77
Posttreatment						
IES–Intrusion	16.58	12.50	11.30	9.98	19.83	9.71
IES–Avoidance	11.06	12.23	15.03	13.36	18.54	11.06
BAI	14.91	13.31	15.47	12.87	20.25	14.26
BDI–2	13.24	11.83	11.37	7.34	14.96	10.92
Follow-Up						
IES–Intrusion	16.97	11.80	13.57	9.52	20.21	9.96
IES–Avoidance	14.30	12.80	16.30	12.68	18.04	11.30
BAI	15.67	13.21	17.07	12.74	21.13	15.09
BDI–2	14.61	12.31	13.57	8.78	16.29	11.95

Note. CBT = cognitive–behavioral therapy; IES = Impact of Event Scale; BAI = Beck Anxiety Inventory; BDI–2 = Beck Depression Inventory—2.

on CAPS–2 Intensity ($p < .05$), CAPS–2 Frequency ($p < .05$), IES–Intrusion ($p < .05$), and IES–Avoidance ($p < .05$). CBT participants scored lower than SC participants on CAPS–2 Intensity ($p < .05$), IES–Intrusion ($p < .05$), and IES–Avoidance ($p < .05$).

Diagnostic Outcomes

In terms of intent-to-treat analyses, there were no significant differences between rates of PTSD across the SC (50%), CBT (36%), and CBT–hypnosis (30%) groups at posttreatment. At

Table 3
Psychopathology Measures for Treatment Completer Sample

	CBT $n = 24$		CBT–hypnosis $n = 23$		Supportive counseling $n = 22$	
Variable	*M*	*SD*	*M*	*SD*	*M*	*SD*
Pretreatment						
IES–Intrusion	27.29	6.02	25.74	8.41	24.45	8.57
IES–Avoidance	20.92	8.30	22.00	7.76	19.55	10.09
BAI	24.31	10.96	27.35	12.06	29.23	13.74
BDI–2	17.75	10.14	19.48	9.38	22.50	12.20
Posttreatment						
CAPS–2 Intensity	10.88	8.27	10.83	10.16	21.36	11.28
CAPS–2 Frequency	12.08	9.41	12.35	11.86	23.59	13.29
IES–Intrusion	12.79	11.01	7.26	6.99	19.27	9.96
IES–Avoidance	6.46	8.23	9.74	8.81	18.05	11.37
BAI	11.17	11.73	11.70	11.83	20.05	14.72
BDI–2	8.50	9.54	10.30	8.04	14.77	11.39
Follow-Up						
CAPS–2 Intensity	13.08	11.08	14.09	11.52	21.18	11.85
CAPS–2 Frequency	15.42	13.61	14.83	13.22	23.23	14.64
IES–Intrusion	13.08	10.01	11.17	9.13	19.68	10.25
IES–Avoidance	10.92	11.17	11.39	8.48	17.50	11.61
BAI	12.21	11.91	14.04	12.67	21.00	15.62
BDI–2	10.38	11.17	13.17	9.97	16.23	12.49

Note. CBT = cognitive–behavioral therapy; IES = Impact of Event Scale; BAI = Beck Anxiety Inventory; BDI–2 = Beck Depression Inventory—2; CAPS–2 = Clinician Administered Posttraumatic Stress Disorder Scale—2.

follow-up, there were comparable rates of PTSD across the SC (58%), CBT (42%), and CBT–hypnosis (40%) groups. In terms of treatment completers, more SC (46%) participants met criteria for PTSD at posttreatment than CBT (13%) participants, $\chi^2(2, N = 46) = 6.14$, $p < .05$, and CBT–hypnosis (9%) participants, $\chi^2(2, N = 45) = 7.77$, $p < .005$. More SC (59%) participants met criteria for PTSD at follow-up than CBT (21%) participants, $\chi^2(2, N = 46) = 7.05$, $p < .01$, and CBT–hypnosis (22%) participants, $\chi^2(2, N = 45) = 6.54$, $p < .01$.

End-State Functioning

We defined good end-state functioning as being less than 12 on the total IES because this score represents the mean score of nonclinical participants (Horowitz et al., 1979). Using this criterion in the intent-to-treat sample, we found no significant differences in the proportion of participants in the CBT–hypnosis (23%), CBT (24%), and SC (12%) groups achieving good end-state functioning. In terms of treatment completers, there were no significant differences in the proportion of participants in the CBT–hypnosis (30%), CBT (33%), and SC (13%) groups achieving good end-state functioning.

Effect Sizes

The effect size of each treatment was determined by calculating the mean difference between each treatment group from (a) pretreatment to posttreatment and (b) pretreatment to follow-up and dividing the mean difference by the pooled standard deviation within each of the samples (Cohen, 1988). Following the International Society for Traumatic Stress Studies treatment guidelines for PTSD (Foa, Keane, & Friedman, 2000), we then converted effect sizes to the unbiased Hedges's *G* to correct for variations due to small sample sizes (Hedges, 1982). Table 4 demonstrates that at posttreatment, participants in the CBT–hypnosis group had larger effect sizes than CBT participants on some measures (IES–Intrusion and BAI). CBT participants in turn had larger effect sizes than SC participants. At follow-up, both CBT–hypnosis and CBT participants had larger effect sizes than SC participants.

Discussion

Both CBT and CBT–hypnosis produced marked reductions in PTSD symptoms at follow-up relative to SC. This finding is comparable with studies that have found that 20% of ASD participants receiving CBT have PTSD at follow-up compared with 67% of those who received SC (Bryant et al., 1999; Bryant, Harvey, Dang, & Sackville, 1998). This repeatedly observed pattern suggests that CBT techniques are highly effective in reducing PTSD symptomatology in those participants who are highly likely to develop chronic PTSD. The finding that 67% of people receiving SC did not display PTSD suggests that this intervention was somewhat beneficial because previous studies have indicated that 80% of this group typically satisfies PTSD criteria 6 months after the trauma (Bryant, 2003).

Contrary to our prediction, the CBT–hypnosis group did not enjoy greater clinical gains than those in the CBT group. It appears that the only difference between the two active treatments was that the CBT–hypnosis participants had fewer reexperiencing symptoms at posttreatment than participants in the CBT group. That is, combining hypnosis with exposure seems to have led to a faster reduction in reexperiencing symptoms, even though this difference was no longer apparent at follow-up. This finding can be explained by the study design that limited the use of hypnosis to facilitating the imaginal exposure of the treatment protocol. Imaginal exposure typically results in reduction of reexperiencing symptoms (Keane, Fairbank, Caddell, & Zimering, 1989). By combining hypnosis with imaginal exposure, it is likely that we limited additional treatment gains to reexperiencing symptoms. It is possible that because we used hypnosis to facilitate other components (e.g., cognitive restructuring, anxiety management, in vivo exposure), broader treatment gains may have been observed in the combined treatment group.

Why did hypnosis facilitate imaginal exposure? There are three possible explanations. First, hypnosis may have breached dissociative obstacles present in those ASD participants and allowed them to experience exposure in a more compelling manner than those participants who did not receive hypnosis. This view is consistent with commentators who have suggested that hypnotherapy is an optimal intervention because of its capacity to target dissociative responses (Spiegel & Classen, 1995). This view is not supported by the lack of correlation between Stanford Hypnotic Clinical Scale scores and reductions in IES scores from pretreatment to posttreatment ($r = .06$) or follow-up ($r = .06$). Second, social psychological theories of hypnosis posit that the major mechanism mediating hypnotic response is the increased motivation elicited by the demand characteristics associated with hyp-

Table 4
Effect Size on Outcome Measures

	Pretreatment to posttreatment			Pretreatment to 6-month follow-up		
Variable	CBT	CBT–hypnosis	SC	CBT	CBT–hypnosis	SC
IES–Intrusion	1.55 (1.04)	2.23 (1.92)	0.62 (0.58)	1.72 (1.11)	1.69 (1.42)	0.59 (0.55)
IES–Avoidance	1.42 (0.88)	1.45 (1.06)	0.20 (0.19)	1.25 (0.82)	1.50 (0.78)	0.25 (0.27)
BAI	1.12 (0.75)	2.21 (1.07)	0.60 (0.56)	1.12 (0.75)	1.09 (1.07)	0.60 (0.55)
BDI–2	0.92 (0.62)	1.04 (1.02)	0.58 (0.56)	0.79 (0.53)	1.90 (0.87)	0.12 (0.10)

Note. Original values indicate effect sizes for treatment completers; values in parentheses indicate effect sizes for intent-to-treat analyses. CBT = cognitive–behavioral therapy; SC = supportive counseling; IES = Impact of Event Scale; BAI = Beck Anxiety Inventory; BDI–2 = Beck Depression Inventory—2.

notic techniques (Spanos, 1986). This view suggests that the CBT–hypnosis participants responded with fewer reexperiencing symptoms because they were more highly motivated to engage in the therapist's requests (including imaginal exposure), and this led to more effective symptom reduction. In this context, we find it noteworthy that several studies that have found that CBT–hypnosis performs more effectively than CBT alone were largely comparable in terms of the content of their therapy strategies; the major difference in these hypnosis treatments was the use of the term *hypnosis* (Kirsch et al., 1995). This pattern may indicate that enhancing participants' motivation may be a critical reason why hypnosis is a useful adjunct to therapy outcome. Third, the inclusion of muscle relaxation in the hypnotic induction may have contributed to anxiety reduction in the CBT–hypnosis group.

We recognize that the intent-to-treat analyses did not indicate that CBT was as effective as we would have hoped. The attrition rate from the CBT groups was approximately 3 times that of the SC group. This pattern is consistent with previous applications of CBT with ASD populations (Bryant et al., 1999; Bryant, Harvey, Dang, & Sackville, 1998). Considering the importance of increasing treatment effectiveness in acutely traumatized populations, there is an important need to increase treatment completion in protocols that have proven effectiveness. Although exposure is not associated with increased dropout in PTSD (Foa, Zoellner, Feeny, Hembree, & Alvarez-Conrad, 2002), it is possible that exposure-based interventions contribute to increased treatment dropout in individuals with ASD. To our knowledge, no studies to date have investigated the effectiveness of therapies that use modalities but do not include exposure.

The conclusions from this study need to be considered in the context of several methodological issues. First, the small sample size and associated limited power may have restricted the opportunity to observe differences between the CBT and CBT–hypnosis groups. Second, the diagnosis of ASD has questionable validity (Harvey & Bryant, 2002), and our reliance on the ASD diagnosis for identifying high-risk individuals shortly after trauma may not have been optimal. Third, we did not include the CAPS–2 at the initial assessment (because we focused on diagnostic assessment of ASD, rather than PTSD), and this precluded indexing treatment effects on the full range of PTSD symptoms. Fourth, we emphasize that our use of hypnosis was very focused in this study, and future studies should use hypnosis within a broader framework to evaluate its capacity to facilitate the treatment gains provided by CBT protocols. Fifth, we did not index the extent to which assessors were blind to participants' treatment group, and this may have introduced a source of assessor bias. Finally, the last-observation-carried-forward procedure used in this study for managing missing data at follow-up may not be the optimal method to conduct intent-to-treat analyses, because it assumes that psychopathology levels in participants who dropped out remained unchanged (Everitt & Pickles, 1999).

To our knowledge, this research represents the first controlled treatment study of hypnosis with ASD and one of the few controlled outcome studies in which researchers used hypnosis to treat posttraumatic stress. The results indicate that future research on the use of hypnosis as an adjunct to CBT of posttraumatic stress is warranted. Although the mechanisms that potentially mediate hypnosis in the context of CBT are unclear, the clinical gains that may be achieved through combining hypnosis and CBT justify systematic study of the effects of combining hypnosis with the full range of CBT techniques to alleviate posttraumatic stress.

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