Compulsions in Depression: Stalking by Text Message

TO THE EDITOR: The ubiquitous nature of text messaging may result in compulsive text messaging as a clinical problem. The case below highlights the affect of compulsive text messaging and suggests that trazodone and behavioral therapy might be effective treatment, although the effect of natural recovery cannot be excluded.

A 32-year-old woman compulsively sent her ex-boyfriend text messages (repeatedly asking him to meet her, chastising him for leaving her, and expressing love for him) after he ended their relationship. He told her to stop contacting him because he wanted no communication with her. Her messages were alarming and disrupted his life. She continued, however, despite his attorney threatening legal action against her. Any effort to resist sending a message resulted in increased tension until she sent a message, which was followed by a short period of relief that "a link [between her and her ex-boyfriend] was maintained." Her growing tensions were only relieved by sending messages. Her text messages continued to increase until she spent 4 hours per day sending 30 to 40 messages, resulting in phone bills of £100 per month, and disciplinary procedures were issued by her employer for her poor work performance. Subsequently, she made no other attempts to contact her ex-boyfriend.

The patient had no personal or family history of psychiatric illness, substance misuse, obsessive compulsive disorder, or habit disorders, but her parents were very distant throughout her life. Her two previous relationships, both heterosexual, ended mutually with no difficulties.

After 6 months, her general practitioner treated her with fluoxetine, 20 mg/day. The doses were increased to 40 mg/ day. In addition, she attended six sessions of Rogerian counseling concerning adjustment to relationship loss. Since there was little response after 4 months of treatment, she was referred to psychiatric services. Trazodone, which is reportedly beneficial for compulsive behaviors (1), was substituted for fluoxetine, and a behavioral program was started (charting her text messaging, prescribing relaxation techniques to reduce tension, and scheduling time to send messages). The time between sending text messages was gradually increased. Over a period of 3 months, her compulsive text messaging gradually resolved, and she has not sent her ex-boyfriend a text message for more than 1 year.

To our knowledge, this is the first report of compulsive text messaging. Compulsive text messaging can be conceptualized as stalking—a pattern of repeated, intrusive, distressing behavior focussed on one individual that persists despite clear indications that it is unwanted (2, 3); for example, in the context of maladjustment to the termination of a relationship. Cyber stalking—using technology, including e-mail, fax, text messages, and pagers as part of stalking behavior (2)—is becoming an increasingly recognized behavior (see http://www.mincava.umn.edu/documents/commissioned/stalkingandtech/stalkingandtech.pdf). Stalking behavior has been categorized as the perpetrator following the victim (e.g., keeping a vigil outside the victim's home); the perpetrator communicating with the victim via phone, letters, and other media; and, ultimately, the perpetrator using aggression toward the victim (2). Studies of stalkers report that they are more likely to be men with female victims than vice versa (2, 3). The simple obsessional group, a classification in which there is a prior relationship between the victim and the stalker, such as in the case above, is the most common type of stalking and most likely to resort to violence (2). Disturbed childhood attachment is also commonly reported in such cases (3).

References

1. Khouzam HR, Mayo-Smith MF, Bernard DR, Mahdasian JA: Treatment of crack-cocaine-induced compulsive behavior with trazodone. J Subst Abuse Treat 1995; 12:85–88
2. Nadkarni R, Grubin D: Stalking: why do people do it? BMJ 2000; 320:1486–1487
3. Kamphuis JH, Emmelkamp PM: Stalking: a contemporary challenge for forensic and clinical psychiatry. Br J Psychiatry 2000; 176:206–209

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Less Mood Switching With Venlafaxine?

TO THE EDITOR: I'm writing in response to the recent article by Gabriele S. Leverich, M.S.W., L.C.S.W.-C., and colleagues regarding mood switches.

If the rate of threshold switches was not different between bupropion, sertraline, and venlafaxine, then the marked difference (between the three drugs) in the ratio between threshold and subthreshold switches must be because of the fact that there were fewer subthreshold switches with venlafaxine. There is some theoretical rationale for thinking that venlafaxine might be more likely to induce switches, but the fact that there were less subthreshold switches with venlafaxine does not support this theory. I believe, therefore, that Figure 3 and the discussion are somewhat misleading in that the data do not indicate a greater likelihood for venlafaxine to cause switches. Rather, the data suggest that venlafaxine is less likely to cause subthreshold switches, which is probably not a meaningful finding.

Reference

1. Leverich GS, Altshuler LL, Frye MA, Suppes T, McElroy SL, Keck PE Jr, Kupka RW, Denicoff KD, Nolen WA, Grunze H, Martinez MI, Post RM: Risk of switch in mood polarity to hypomania or mania in patients with bipolar depression during acute and continuation trials of venlafaxine, sertraline, and bupropion as adjuncts to mood stabilizers. Am J Psychiatry 2006; 163:232–239

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Dr. Leverich Replies

TO THE EDITOR: We thank Dr. Mattes for commenting on our article on the risk of switching during antidepressant treatment. Dr. Mattes is correct that there is some ambiguity in the clinical meaning of the ratio of the number of full switches to the number of subthreshold switches across different drugs. However, several factors suggest that the greater ratio of full to subthreshold switches with venlafaxine relative to the other antidepressants is a meaningful result.

The first factor is an important methodological one. If patients had one or more brief hypomanias and then progressed to having full duration or more severe episodes, only the latter, most severe episode was counted. Thus, the apparent decrease in minor or subthreshold switches appears to be related to the fact that more patients receiving venlafaxine progressed to full duration hypomanias or manias relative to the patients given the other drugs.

Also consistent with this interpretation are the findings, based on this same cohort, that were analyzed using conventional cross-sectional measures of manic severity as reported in a companion article (1). If one used a two-point or greater increase in the Clinical Global Impression Scale for Bipolar Disorder mania severity rating as indicating a clinically meaningful switch, this criterion occurred in more patients receiving venlafaxine ($p<0.01$). Similarly, if one used the criterion of a Young Mania Rating Scale score of >13 as an indication of clinically meaningful hypomania or mania, this also occurred more frequently with venlafaxine ($p=0.05$), especially in rapid cyclers. Moreover, Vieta and colleagues (2) also found higher switch rates with venlafaxine than paroxetine in a randomized open study.

Thus, the graded Young Mania Rating Scale and Clinical Global Impression Scale for Bipolar Disorder manic severity ratings used by Post and colleagues (1) and the methodological clarification that only the most severe form of manic switch observed was counted suggest that there is an increased proclivity for full switches with venlafaxine relative to the other two drugs (particularly bupropion). On the basis of both analyses, we would conclude that greater caution is warranted (particularly in those patients with a prior history of four or more episodes in the year prior to study entry) in the use of antidepressant adjunctive treatment with venlafaxine than sertraline or bupropion in the treatment of depressed patients with bipolar disorder who are already receiving a mood stabilizer.

References

1. Post RM, Altshuler LL, Leverich GS: Higher switch rates on venlafaxine than bupropion or sertraline in bipolar depression. Br J Psychiatry (in press)
2. Vieta E, Martinez-Aran A, Goikolea JM, Torrent C, Colom F, Benabarre A, Reinares M: A randomized trial comparing paroxetine and venlafaxine in the treatment of bipolar depressed patients taking mood stabilizers. J Clin Psychiatry 2002; 63:508–512

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Reduced Hippocampal and Amygdalar Volume in Dissociative Identity Disorder: Not Such Clear Evidence

TO THE EDITOR: As experimental psychologists trained in research methodology, we read the article by Eric Vermetten, M.D., Ph.D., and colleagues (1) with mixed feelings. The authors employed magnetic resonance imaging (MRI) to measure the volumes of the hippocampus and amygdala in 15 female patients with dissociative identity disorder and 23 female nonpsychiatric women. Dr. Vermetten and colleagues concluded from their data that, relative to comparison subjects, patients with dissociative identity disorder had smaller hippocampal and amygdalar volumes. They admit that their study suffers from a number of shortcomings. Most important, the patients with dissociative identity disorder were significantly older than the comparison subjects. Although group differences in hippocampal and amygdalar volumes disappeared when the authors statistically controlled for age, they try to convince the reader that their results are nonetheless valid. They argue that there is generally no age-related reduction in hippocampal and amygdalar volumes in healthy women aged 20–50. This point is reiterated in an editorial by David Spiegel, M.D. (2, p. 566), who stated that the article by Dr. Vermetten and colleagues offers "clear evidence of smaller hippocampal and amygdalar volume among those with dissociative disorders" (2). Unfortunately for Dr. Vermetten and colleagues as well as Dr. Spiegel, there have been reports that this population also exhibits significant, albeit moderate, hippocampal volume decreases in a 5-year follow-up design (3). In relation to this, there is good evidence that suffering from a major psychiatric disorder may lead to accelerated aging in middle-aged people (4, 5). Therefore, MRIs of healthy, younger individuals do not constitute the ideal control condition for those of patients with dissociative identity disorder. It is with these considerations in mind that we fundamentally disagree with Dr. Spiegel when he claims that the study by Dr. Vermetten and colleagues offers "clear evidence." It does not.

References

1. Vermetten E, Schmahl C, Lindner S, Loewenstein RJ, Bremner JD: Hippocampal and amygdalar volumes in dissociative identity disorder. Am J Psychiatry 2006; 163:630–636
2. Spiegel D: Recognizing traumatic dissociation. Am J Psychiatry 2006; 163:566–568
3. Raz N, Rodrigue KM, Head D, Kennedy KM, Acker JD: Differential aging of the medial temporal lobe: a study of a five-year change. Neurology 2004; 62:433–438
4. Houx PJ, Vreeling FW, Jolles J: Rigorous health screening reduces age effect on memory scanning task. Brain Cogn 1991; 15:246–260
5. Rabbitt PMA, McInnes L, Diggle P, Holland F, Bent N, Abson V, Pendleton N, Horan M: The University of Manchester longitudinal study of cognition in normal healthy age, 1983 through 2003. Aging Neuropsychol Cogn 2004; 11:245–279

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Dr. Vermetten Replies

TO THE EDITOR: We acknowledge that the design of every study is strongest when groups that are studied are matched for several dependent variables. However, we argue that the data disputed by Drs. Smeets, Jelicic, and Merckelbach are strong and valid. Hippocampal volume was 19.2% smaller and amygdalar volume was 31.6% smaller in the patients with dissociative identity disorder relative to the healthy subjects. Statistical rigor required us to control for the age difference (dissociative identity disorder patients [42.8 years] versus comparison subjects [34.6 years]). In doing so, our findings left only the right amygdalar volume significantly smaller across groups. However, we argue that the comparison data for the hippocampal and amygdalar volumes are valid without correction for this factor. Even though age-related structural alterations in the hippocampus have been identified, there are no