services and have ambivalent relationships with past or current caregivers who may have perpetrated these traumas. These children are often instinctively motivated to attend to the positive aspects of these relationships and are not cognitively or emotionally able to consider a balanced view of their aggressors as individuals who are deserving of punishment. Perhaps this is because of the child's dependency on adults in a stable environment, although these adults might change, and the child then develops an attachment to a new caregiver.

I have treated traumatized adolescents who are able to articulate that their caregivers hurt them and that it was wrong. Most of these youth are still very emotionally and behaviorally affected. I am unsure as to whether asking about thoughts of revenge would be helpful to the therapeutic process.

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Dr. Boylan reports no competing interests.

Dr. Horowitz Replies

TO THE EDITOR: Dr. Boylan is correct in emphasizing the difference between adults and children in the therapy process in working through revenge fantasies that may follow traumatic experiences. No one can accurately distinguish veridical memory from fantasy memory, and children are even less able than they will be as adults at knowing the difference at the time of the experience and knowing the difference on later review. The child is less oriented to review and more oriented to completing a story in a future projecting way that seems to preserve personal safety. That is probably the first priority—to help them do this—in most cases.

As Dr. Boylan states, revenge fantasies, if and when present, are likely to find displaced targets, in play with an agentic self, have more than usual destructiveness, as well as influencing direct negative behavior toward people who are "safer" to attack than the actual aggressor. Unfortunately, the "safer" individual may be the child's own self, which might be manifested through self-harming behaviors such as pulling out hair, picking off skin, or knocking the head.

When a child or adolescent displays play, fantasy, or interpersonal behavioral patterns that appear to enact revenge, it may be beneficial to encourage translation of the somatic actions into verbal statements. This may help to increase self-control and interpersonal regulation. This could be done through conversation with therapists or good caretakers. An example of such would be as follows: "I guess you are still pretty angry that you got beat up. I also might feel scared and then mad until I felt I was okay and safe again." The point is not a catharsis in the old-fashioned sense of emotional venting, but the emphasis on the "okay and safe" concept of completing a reaction to traumatization.

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Dr. Horowitz's disclosure accompanies the original article.

Evolving Treatments for Panic Disorder

TO THE EDITOR: In the Feb. 2007 issue of the *Journal*, Barbara Milrod, M.D., et al. (1) reported a randomized controlled trial in which psychoanalytic psychotherapy was superior to applied relaxation in the treatment of panic disorder. Opponents of psychoanalytic therapies often level the criticism that little (if any) research exists that demonstrates the efficacy and durability of these approaches. We therefore applaud the efforts of Dr. Milrod et al. to manualize psychoanalytic therapy and to test its effects in a randomized controlled trial.

One of the great challenges with psychiatric disorders such as panic, however, is that many different interventions can appear to work at any given time. Therefore, the scientific task is to weed out those procedures that may appear to work from those that can be trusted to work reliably, not only because these trusted procedures have passed muster in controlled trials, but also because we have some directly verified knowledge about the mechanism through which they have produced positive outcomes. Where is the evidence elucidating the mechanism by which understanding unconscious conflicts (if they exist) results in the reduction of panic and agoraphobia?

In the past 30 years, we have seen significant advances in understanding the biological, cognitive, and behavioral mechanisms of panic and agoraphobia. This research has informed (and continues to inform) the development of effective treatments for these conditions, including psychological treatments such as cognitive behavior therapy (CBT) (2, 3). For example, exposure to interoceptive cues, which is an important component of CBT, is based on experimental research showing that individuals with panic disorder are predisposed to appraise changes in physical state as dangerous (4). Moreover, studies of the individual components of CBT in the treatment of panic disorder have demonstrated that exposure produces the greatest effect (5).

We have a scientifically sound model of panic (3) as well as many controlled trials that show the effectiveness of CBT directly derived from this model (6). Thus, any psychodynamic treatment for panic disorder has to meet a rather high standard. This is not to discount psychodynamic therapy altogether, however. Indeed, interpersonal psychotherapy for the treatment of depression was developed long after CBT was an empirically supported treatment for this condition, and it is now a widely accepted treatment for depression (7). Interpersonal psychotherapy, however, is based on a psychodynamic theory in which hypotheses about mechanisms of psychopathology have been empirically tested. We therefore await research substantiating the conceptual basis of the psychoanalytic treatment for panic disorder presented in the article by Dr. Milrod et al., as well as a direct comparison of the short- and long-term effects of psychoanalytic treatment and CBT for this condition.

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The authors report no competing interests.

Dr. Milrod Replies

TO THE EDITOR: In clinical medicine, mechanisms of action are often opaque. Because we have yet to (may never, in fact) fully answer questions concerning mechanisms of effective treatments, should we avoid these treatments? To do so would be irresponsible, since these treatments have demonstrated efficacy from which patients should benefit. The late Gerald L. Klerman emphasized the primacy of outcome, pointing out that if a therapy lacks clinical benefit, its mechanism holds little interest (1). Our study demonstrated the efficacy of panic-focused psychodynamic psychotherapy for the treatment of panic disorder. It should not be confused with a study of mechanism, something we never claimed. The CBT model of treatment, whether scientifically sound or not, is irrelevant to the psychoanalytic model. Often, in the treatment of complex illnesses such as psychiatric disorders, more than one model can be useful—as Dr. McKay et al. note for depression.

Our study was more rigorously conducted than many oft-cited psychotherapy outcome studies, and this was demonstrated in its control and tracking of non-study interventions such as medications, which can blur apparent psychotherapy outcome, and in its maintenance of two levels of blindedness among independent evaluators (who were blinded to patient and therapist orientation) (2). Furthermore, our study has been prescient in evaluating the moderator effect of axis II pathology on panic outcome (3). No one should be blinded by ideology (4), a risk that cognitive theorists now face as much as psychoanalysts have in the past.

Without the equivalent of a pharmaceutical industry to provide financial backing, psychotherapy researchers must battle one another for ever-shrinking federal funds. A step forward for psychodynamic psychotherapy should not be a defeat for CBT. This has become as much of a "guild war" as any. In both cases, it is in our patients' best interest that the field remains open to and accepting of a range of treatment approaches.

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Author disclosures accompany the original article.

BK_{ca} Channel in Autism and Mental Retardation

TO THE EDITOR: We read with great interest the article by Frederic Laumonnier, Ph.D., et al. (1) in the Sept. 2006 issue of the *Journal*, as it potentially contributes to knowledge of the etiobiology of autism and may identify a novel treatment target. With the exception of two prior reports (2, 3), channel mutations have not been commonly observed in autism. Therefore, the study by Dr. Laumonnier et al. may represent one of the first on definitive mutations in a channel associated with autism. Yet, several complexities to the relationship between channel mutations, autism, and epilepsy are introduced by the data presented.

The most compelling finding of the article was the discovery and characterization of balanced translocation, which appeared to interrupt one allele of the KCNMA1 gene in the first intron in a patient with autistic disorder. The patients' parents do not carry the balanced translocation, and therefore, the fact that this translocation is *de novo* supports the notion that it may be pathogenic. Using semiquantitative reverse transcriptase-polymerase chain reaction, Dr. Laumonnier et al. showed that BK gene expression was decreased by approximately 50% in lymphoblastoid cell lines from the patient, which is consistent with the mutation leading to haploinsufficiency.

In Figure 2 of the article, the authors showed that the BK toxin, iberiotoxin (IbTx), blocked whole-cell current from the autistic patient significantly more than it did in the comparison subject, which suggests decreased activity of this receptor, presumably because of the haploinsufficiency of the genetic mutation. However, this analysis represents a somewhat ambiguous assessment of such a change, since the authors did not provide information regarding the amplitude of the