

journals from Great Britain (1,054), Canada (503), and Australia/New Zealand (57). Thus, it appears that in America, psychiatrists receive an overwhelmingly more positive view of dissociative identity disorder than their English-speaking counterparts elsewhere.

How can these differences be explained? Some have suggested that they may be due to unfamiliarity with dissociative identity disorder among non-American clinicians (3) or that the problem reflects an error on the part of Britain's influential academic establishment (4). An alternative, provocative hypothesis is that dissociative identity disorder be designated a culture-bound syndrome (5), on the theory that many American therapists and their patients are involved in a subcultural mutual belief system about the existence of dissociative identity disorder, abetted perhaps by dramatized American media presentations of people diagnosed with the disorder. Although the validity of these competing hypotheses remains to be tested, we are clearly obliged to explain these remarkable national differences in the acceptance of the dissociative identity disorder diagnosis.

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JOHN HOCHMAN, M.D.
Encino, Calif.

HARRISON G. POPE, JR., M.D.
Belmont, Mass.

Biased Book Review

TO THE EDITOR: In his recent review of *Psychodynamic Concepts in General Psychiatry* (1), Dr. Hartmann is critical of the editorial integration of the text, even though he recommends as useful over 50% of the 24 chapters. While I respect his prerogative as a reviewer to find fault with the book, I do not accept his presumption that my intent as editor was to be engaged in simply a "hopeful collecting" of the authors and their chapters. My and the coeditors' efforts were to gather together in one book clinicians of considerable expertise in their subspecialty areas—a "roster of excellent authors"—and invite them to present in clinical detail their understanding of how the psychodynamic method informs their work and heals their patients.

Dr. Hartmann's presumptiveness approaches the heart of the question about his ability to fairly review this work. This issue is acknowledged in his off-handed declaration of having "not a conflict of interest" in reviewing the book. He claims to having had a mere "previous look" at the text before publication. In fact, Dr. Hartmann had, or at least attempted to have, a great deal more involvement in the production of this text. Before publication Dr. Hartmann contacted me by phone and fax numerous times and strongly suggested that I make changes in the completed manuscript, which was shown to him by the publisher. After considering a number of his sug-

gestions and reviewing them with the authors, I did agree with some of them. I did not agree with others. Dr. Hartmann persisted. He called me again at home and was quite adamant that if I failed to make the changes he was "suggesting" to me, then perhaps it would be best that the book not be published. Most of his anger, which by now was not even barely concealed, was directed at the chapter "The Depressed Male Homosexual Patient," which fills 16 of the 497 pages of the book. He insisted that this chapter should be omitted or "entirely rewritten." In fact, the chapter was significantly edited, in part because of some of Dr. Hartmann's helpful suggestions. Still, that did not suffice, and he faxed me yet again a line-by-line critique of much of the chapter with recommendations for how those lines should be further rewritten to satisfy him. Dr. Hartmann's feelings about that one chapter, his frustration that it was not changed according to his demands, and his overall involvement with the book were so considerable that on the very day it was published he faxed me an angry letter declaring that that one chapter had ruined the entire book. I will respectfully leave it to the readers of the book to evaluate this and the other chapters and their conclusions that are derived from extensive clinical data.

I believe that the *Journal* is ill-served by Dr. Hartmann's failure to fully acknowledge his extensive attempts to influence the preparation of this book and, accordingly, to have recused himself from reviewing it.

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HARVEY J. SCHWARTZ, M.D.
Philadelphia, Pa.

Dr. Hartmann Replies

TO THE EDITOR: I think it is not a conflict of interest to dislike subsequent versions of the same book twice and to say so. I would have liked a book on psychodynamic concepts to be a better book. Although I was far from the only critic (and the history of criticisms is not precisely as Dr. Schwartz recalls), and my finding some partial values in the book is not quite as he states, to reply at length to his complaints would be to give further and undeserved publicity to an inadequately edited and overall not very good book. One could recommend a few of the chapters, but the book remains a missed opportunity.

LAWRENCE HARTMANN, M.D.
Cambridge, Mass.

Homicidal Behavior in Drug Abusing Psychiatric Patients

TO THE EDITOR: The recent report of Ihsan M. Salloum, M.D., M.P.H., and colleagues (1) of greater homicidal behavior in psychiatric patients with concurrent alcohol and cocaine abuse is very interesting, particularly because combined use has not been previously linked to greater violence. On the contrary, controlled laboratory studies have suggested that alcohol-induced behavioral impairment is attenuated by stimulants (unpublished 1990 study of D.A. Beezley-Smith) and cocaine (2). Furthermore, in a study of psychiatric emer-

gency room patients (3), we found low rates of aggression in patients whose urine toxicology results indicated cocaine use (regardless of simultaneous detection of alcohol), as opposed to high rates of aggression in patients whose results indicated alcohol use only (3).

Methodology might contribute to the differences found in our two studies. Most importantly, Salloum et al. studied a selected population of patients admitted to a specialized dual-diagnosis inpatient unit whereas our study was based on a random sample of psychiatric emergency room patients with substance abuse.

Whereas the study of Salloum et al. benefits from the more detailed clinical assessment possible during an inpatient stay, no mention is made of the use of toxicological testing to confirm the substance group into which patients were classified. Results of toxicology screening in our study suggested that comprehensive clinical evaluation frequently missed polysubstance use (3).

A recent study suggests that combined use of alcohol and cocaine characterizes a subgroup of severe alcoholics (4). The excess violence found in patients with combined abuse might be due to greater severity of the alcohol abuse, with concurrent cocaine abuse being merely an indicator of severity of alcohol use. This alternative explanation of findings, not mentioned by the authors, emphasizes the importance to control for severity of use in efforts to disentangle differential clinical effects of combined drug use.

While the study of Salloum et al. is a welcome addition to the literature, more studies are needed to achieve a clear understanding of the clinical effects of concurrent and simultaneous use of alcohol and cocaine in different psychiatric populations and settings.

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DIRK DHOSSCHE, M.D.
Genk, Belgium
JOAN RUBINSTEIN, M.D.
Stony Brook, N.Y.

Dr. Salloum and Colleagues Reply

TO THE EDITOR: We appreciate the comments of Drs. Dhossche and Rubinstein on our findings of greater homicidal behavior among psychiatric patients with concomitant alcohol and cocaine dependence. These results differed from their findings of less aggressive behavior among emergency room patients whose urine toxicology results indicated alcohol and cocaine use. We agree with Drs. Dhossche and Rubinstein that methodology may account for the different results obtained.

A major difference between the two studies is the definition of aggressive or homicidal behavior. In Drs. Dhossche and Rubinstein's study, aggressiveness was recorded if there was

a chart entry for assaultive or threatening behavior. No specific inquiries were reported regarding the intent of the aggressive act or regarding specific homicidal behavior. This measure differs substantially from our measure of homicidal behavior. In our study, clinicians inquired specifically about the presence of homicidal ideation, plans, or acts. Clinicians were required to record this information in a specific section of the evaluation form dedicated to collect data that targeted this key area of the clinical evaluation.

Urine toxicology screens were generally performed as part of the analysis before admission to the inpatient psychiatric units. However, there were also limitations concerning these screenings, such as delays or patient refusal to take the test, which limited their usefulness and reduced their appropriateness for inclusion in our report. Also, we agree that our study could have been stronger if urine toxicology results were available for all patients. However, it is also true that self-reported cocaine (crack) use is usually associated with positive drug screen results (1).

Severity of substance use is a particularly important dimension that may influence the clinical presentation. Therefore, we reanalyzed our data by comparing the three groups (alcohol and cocaine dependence, alcohol dependence only, cocaine dependence only) on current levels of adaptive functioning (Global Assessment of Functioning Scale) and on the duration of alcohol use and cocaine use. Scores on the Global Assessment of Functioning Scale indicated slightly more impairment for the alcohol- and cocaine-dependent group (mean=36.4 [SD=13.4]) than the other two groups (alcohol only: mean=43.0 [SD=14.4]; cocaine only: mean=44.0 [SD=14.0]) ($F=2.94$, $df=2, 101$, $p<0.07$). However, the two groups with alcohol dependence were very similar in the duration of alcohol use (mean=12.5 years [SD=7.0] for the alcohol and cocaine group versus mean=13.0 years [SD=8.0] for the alcohol only group), whereas the alcohol and cocaine group differed from the cocaine only group in the duration of cocaine use (mean=7.0 years [SD=6.7] versus mean=4.0 years [SD=2.5], respectively) ($t=2.30$, $df=36$, $p<0.04$). We repeated the logistic regression analysis by comparing the two cocaine-dependent groups (with and without alcohol dependence) and controlled for duration of cocaine use. Our main statistically significant finding reported in our article regarding the presence of homicidal plans was still significant (odds ratio=5.92, $p<0.04$). Although inferences are limited by our retrospective study design, we suspect that cocaine is an active factor in the expression of homicidality, rather than a simple epiphenomenon of severe alcoholism.

Given that concurrent alcohol and cocaine abuse is highly prevalent and results in more pronounced subjective euphoric and toxic effects than cocaine use alone (as reported in the 1993 study of Farre et al. that Drs. Dhossche and Rubinstein cite), we agree that further study of this devastating combined addiction is clearly needed.

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IHSAN M. SALLLOUM, M.D., M.P.H.
DENNIS C. DALEY, M.S.W.
JACK R. CORNELIUS, M.D., M.P.H.
LEVENT KIRISCI, PH.D.
MICHAEL E. THASE, M.D.
Pittsburgh, Pa.