

LETTERS

Letters from readers are welcomed. They will be published at the discretion of the editor as space permits and will be subject to editing. They should be a maximum of 500 words with no more than five references and should be submitted in duplicate in a double-spaced format. Address letters to John A. Talbott, M.D., Editor, *Psychiatric Services*, American Psychiatric Association, 1400 K Street, N.W., Washington, D.C. 20005; e-mail, psjournal@psych.org.

Violent Behavior

To the Editor: In their article in the September 1997 issue, Grant Harris and Marnie Rice (1) presented a succinct review of current methods to evaluate, manage, and treat violent individuals in psychiatric facilities. However, their review left me with some troubling questions.

First, they assert that "no evidence has been found that clinicians' unaided judgments are better than those of lay persons in assessing violence risk." Some of the studies reviewed in their paper (McNiel and Binder, reference 30; Lidz and associates, reference 23; and Gardner and associates, reference 25) provide evidence that clinicians can, on the basis of their clinical observations and judgments, predict violent acts by their patients with 59 percent to 69 percent accuracy, and can predict the seriousness of the violent acts with the same accuracy as researchers using actuarial tools. But where is the evidence to support their claim that laypersons can predict violence with equal accuracy?

Second, when Harris and Rice state that "major mental disorder and psychiatric disturbance are poor predictors of violence," they appear to be arguing that accurate predictions about the behavior of potentially violent patients cannot be based on psychopathological data. Yet studies they cite by Swanson and associates (reference 63) and by Link and Stueve (reference 48) indicate that symptoms of psychosis and major mental disorder

are strong predictors of violence, even after taking into account comorbid substance use disorder and various demographic factors.

The findings of Swanson and Link are consistent with reviews (2,3) indicating that actuarial methods like those advocated by Harris and Rice may be relevant to large populations but that predictive statements about individual risk must take into account individual psychopathology. As Mulvey (2) explained, "An overall coefficient of association describes only the linear trends in a total data set. It does not describe the strength of the association for every individual in the sample, nor does it allow for an adequate representation of the prevalence of individuals in a sample for whom the relationship is strong enough to warrant intervention or policy concern." Furthermore, individual psychopathology may not always lend itself to quantification, and for that reason actuarial methods may discount important information relevant to individual risk (4). Monahan has observed that "denying that mental disorder and violence may be in any way associated is disingenuous and ultimately counterproductive. . . . the flat denial that any relationship exists between disorder and violence can no longer credibly be prefaced by 'research shows'" (5).

Third, Harris and Rice make several contradictory statements. They assert that mental symptoms are poor predictors of violence (page 1171) after having earlier stated that command hallucinations increase the risk of violence (page 1169). To support their statement about this increased risk, they cite a study by Zisook and associates (reference 50) that found command hallucinations did *not* increase risk. They declare that violent patients can be managed effectively with medications (page 1171), but later, citing a study by Allan and associates (reference 90), they state that little evidence exists that violence can be managed with medications (page 1172), although the Allan study actually suggested that medications *can* be useful in managing violence.

And finally, Harris and Rice contend that "treatments aimed specifically at increasing self-esteem are contraindicated because good evidence exists that self-esteem is positively related to violence." This statement contradicts their earlier observation elsewhere that feelings of worthlessness, among other depressive symptoms, can lead to violent action (6). In their article, they deplore the expenditure of resources for the treatment of anything except personality and lifestyle deviations that correlate highly with violence, stating that it is impossible to justify spending scarce resources on other targets, including mental disorders associated with violence. Surely more balanced judgment would accept that mental disorders and personality-lifestyle factors both contribute to violent actions and deserve measured and appropriate responses.

Despite these problems and contradictions, Harris and Rice demonstrate convincingly that individuals at risk for violent behavior can be evaluated accurately and managed effectively by experienced clinicians who take into account both individual-psychopathological and population-actuarial risk factors. We should feel grateful to them for that lesson.

Morton N. Menuck, M.D.

Dr. Menuck is director of medical education and consulting psychiatrist at the Mental Health Centre in Penetanguishene, Ontario.

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5. Monahan J: Mental disorder and violent behavior: perceptions and evidence. *American Psychologist* 47:511-521, 1992

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6. Rice ME, Harris GT, Varney GW, et al: Violence in Institutions: Understanding, Prevention, and Control. Seattle, Hogrefe & Huber, 1989

In Reply: Dr. Menuck fires a salvo at the messengers; we address his four darts in order:

First, he takes issue with our statement that there is in fact "no evidence . . . that clinicians' unaided judgments are better than those of laypersons in assessing violence risk." Better-than-chance accuracy of clinical judgment does not contradict that. Predictions by psychiatrists and laypersons are indistinguishable in basis, substance, and reliability (1). Forensic clinicians' predictions are no different from those of clinicians without forensic training and experience (2). Because clinicians and laypersons alike base their predictions on history of violence, it is unsurprising that accuracies sometimes exceed chance. Dr. Menuck's assertion that clinicians predict violence with the same accuracy as actuarial tools is false. The references he cited do not support his contention, and the one by Gardner and associates demonstrated yet again the practically universal finding from hundreds of studies that actuarial methods are superior.

Second, our review explicitly concerned violence among psychiatric patients, forensic patients, and released prisoners. Among such populations, psychosis is unrelated or inversely related to subsequent violence (3). Psychoses (or more accurately, some symptoms) might be related to violence in the general population, but that was clearly not our topic. The size of the relationship in the general population, if it exists, is small compared with, for example, the relationships for substance abuse and psychopathy. Menuck's comments about the nonapplicability to individuals of results derived from large samples and the unquantifiable nature of individual psychopathology illustrate but two of many well-documented errors to which human judgment is prone (4-6).

Third, our point was that a few symptoms *might* be related to vio-

lence or treatment for them *might* help control it. Zisook and associates found two of 46 patients with violent command hallucinations (albeit toward themselves) committed lethal violence, leading us to suggest that command hallucinations might increase risk. Nowhere did we state that violent patients can be effectively managed with drugs. Allan and associates found that nadolol was unrelated to improvement in aggression, though related to other improvements, leading them to conclude, "Aggressiveness and psychosis can be orthogonal."

Fourth, consider, by analogy, the difference between a treatment target and a beneficial collateral effect for a fractured tibia. Proper treatment (splinting, immobilization with a cast, and so on) inevitably brings pain relief—a collateral benefit. However, treatment targeted only at pain relief obviously brings harm rather than benefit. Research indicates that in treating violent individuals, harm results from therapy aimed at raising self-esteem even though increased self-esteem might be a collateral benefit of appropriate treatment.

We appreciate that these scientific facts might be unwelcome news to practitioners who rely on unaided clinical assessment of dangerousness, drugs to treat psychotic symptoms (and thereby violence), and insight-oriented, emotionally based psychotherapy for violent offenders. Nevertheless, our positive message was that improvements in the prediction and management of violent recidivism among high-risk offenders can be attained when clinicians practice along the lines we suggested.

Grant T. Harris, B.Sc., Ph.D.

Marnie E. Rice, M.A., Ph.D.

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ADHD in Adults

To the Editor: The article in the October 1997 issue by Drs. Lomas and Gartside (1) about the prevalence of symptoms of attention-deficit-hyperactivity disorder (ADHD) among homeless veterans cites several studies (2-5) that, like the study they describe, purport to demonstrate that ADHD persists into adulthood. Yet the shortcomings of both the cited studies and the authors' own study call the assumptions of ADHD in these populations into serious question.

The authors failed to adequately address questions of likely organic brain damage or dysfunction secondary to substance use. Relying on self-reports alone in screening for and assessing ADHD among those with highly probable substance use disorder seems quite inadequate. The finding that these ADHD veterans were 18 times more likely to suffer from an anxiety disorder than their non-ADHD counterparts suggests a confounding of the anxiety diathesis with the ADHD-like behavioral and cognitive correlates. It also suggests that the authors did not attempt to distinguish between these variables. Indeed, the entire matter of how other comorbid disorders were added to, or distinguished from, ADHD was not addressed.

The concepts of adult residual attention-deficit disorder or attention-deficit-hyperactivity disorder are undergoing repeated exploration, theorizing, and attempted validation. This thoughtful inquiry should not be abridged by superficial evaluations of homeless persons who abuse substances and who, through a variety of

organic and environmental influences, appear to evidence symptoms of ADHD that have no definable continuity with a relatively well-defined childhood disorder. Adults with this constellation of symptoms merit closer scrutiny as to the likelihood of more typical adult disorders—anxiety, depression, or personality disorders—and better attention to their treatment needs.

Jeffrey P. Holmgren, M.D.

Dr. Holmgren is medical director of Northwoods Guidance Center in North Branch, Wisconsin.

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In Reply: I thank Dr. Holmgren for the opportunity to clarify our study of ADHD in homeless veterans. For the sake of accuracy, we did not cite the studies by Susser and associates and Gomez and associates as purporting to demonstrate the persistence of ADHD symptoms into adulthood. In my view, the articles that were cited, along with others not cited, clearly establish the persistence of ADHD symptoms in many adults with childhood-onset ADHD.

Contrary to Dr. Holmgren's impression, we excluded patients with organic brain dysfunction due to any cause. We used a thorough physical and comprehensive laboratory examination along with at least three independently structured mental status

examinations. Furthermore, we did not rely on self-reports alone to make the diagnosis of ADHD but scored all patients by *DSM-III-R* behavioral criteria, which we had the luxury of doing over a four- to six-month period. We had positive childhood documentation in 40 percent of the cases.

Once a diagnosis of ADHD was made, all other diagnoses became comorbid. Dr. Holmgren should know that the literature is clear that children, adolescents, and adults with ADHD are at greater risk for anxiety and substance use disorders.

Finally, Dr. Holmgren should read my earlier letter (1) and search his own experiences before describing our study as "superficial," and homeless persons who abuse substances as unsuitable for the study of ADHD.

Ben Lomas, M.D.

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Alzheimer's Disease and Depression

To the Editor: Before the introduction of acetylcholinesterase inhibitors, patients with Alzheimer's disease would slowly lose their cognitive abilities and at some point also lose insight into their decline. Although depression is common in the course of Alzheimer's disease, it usually occurs early. When a patient has lost enough insight, the depression disappears (1,2).

The introduction of medication that can improve both cognition and insight thus raises the question of whether treatment can induce depression as insight improves and the patient becomes aware of his or her cognitive decline. The following case illustrates this situation.

Mr. B is a 78-year-old white male who was hospitalized for management of severely depressed mood, crying spells, and withdrawn behavior. He had been diagnosed as having probable Alzheimer's disease three years earlier and was started on tacrine at that time. In spite of this medication, during the last several

months his Mini Mental State Examination had begun to show a marked decrease in cognitive functioning.

Three months before his admission, Mr. B's medication was changed to 5 mg donepezil daily. His wife reported that after about eight weeks of the new medication, his cognition had improved but he had become depressed. He would complain of feeling distressed over not being able to remember things or not being able to help his wife with their business affairs. He withdrew and would cry frequently about his current condition. His wife stated that before taking donepezil, he was oblivious to his cognitive deficiency and had shown no signs of depression.

Mr. B met *DSM-IV* criteria for major depression and was started on venlafaxine. His mood improved, and he was discharged to the care of his family.

This case brought to our attention the possibility that the treatment of Alzheimer's disease may unintentionally induce a depression that is secondary to regaining insight. This situation led us to question whether we are doing our patients a disservice by offering them a treatment that may induce another disorder. In light of the problems of treating depression in older patients, who usually have multiple medical conditions and take many drugs, the addition of yet another drug is always of concern.

This case is presented to stimulate debate about the use of medication that may offer hope but that also may create more problems for patients and clinicians.

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