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 include the writer's telephone and fax numbers and e-mail address. Letters related to material published in *Psychiatric Services* will be sent to authors for possible reply. Address letters to John A. Talbott, M.D., Editor, *Psychiatric Services*, APA, 1400 K Street, N.W., Washington D.C. 20005; fax, 202-682-6189; e-mail, psjournal@psych.org.

A Misleading Review?

To the Editor: Dr. Harold Carmel's review of my book *Understanding Violence* (1) in the December 1998 issue was a fair representation of the response I have generally received from medical psychiatry. In that sense, it was a very appropriate review for a psychiatric journal. However, I feel compelled to point out some shortcomings in the review that might mislead potential readers.

The purpose of the book is not to provide a comprehensive, in-depth review of the literature in the area of violent behavior. Rather, it is to provide a summary of that literature. One of the characteristics of the literature on violence is its enormous variety; many researchers are working in very different fields. To criticize the book because it does not spend a great deal of space on any one area is to misunderstand the book's mission. As the author, I am responsible for clarifying that mission, and Dr. Carmel's misunderstanding of it is at least partly due to my lack of clarification.

Specifically, Dr. Carmel criticizes the book for "trivializing" the diagnosis of antisocial personality disorder and for "dismissing" the link between serious mental illness and violence. The psychiatric community has traditionally seen these two areas as being of great importance as causes of violence. However, one of the major thrusts of the book is that when the literature is taken as a whole, antisocial personality disorder and mental illness are far from the most important causes of violence. In my opinion, this is an important fact. It may be that the mental illness model of violence is much less tenable than other, more modern theories. It was, and is, my purpose to educate my readers about this shift in the emphasis of most research. Of course, others will have different opinions. However, I stand by mine.

Elizabeth Kandel Englander, Ph.D.

Dr. Englander is assistant professor of psychology at Bridgewater (Mass.) State College.

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1. Carmel H: Understanding violence (book review). Psychiatric Services 49:1633, 1998

In Reply: I do not have a particular quarrel with Dr. Englander when she states that "antisocial personality disorder and mental illness are far from the most important causes of violence." That is a legitimate point of view, which can be debated scientifically. That was not why I criticized her book. My criticisms (most of which her letter does not mention) were broader, and addressed whether the book would be useful to *Psychiatric Services* readers. I did not and do not think so.

It is not clear what Dr. Englander means by "the mental illness model of violence." If she means that she believes most psychiatrists believe mental illness is the sole explanation for violence, I think her view would be inaccurate. If she means that a mental-illness-oriented assessment of violence has little to offer, a look at the recent literature would be instructive (1). There is an active and expanding literature on the relationship between psychiatric diseases and violent behavior, a literature that is very relevant to the readers of this journal, and it is essentially ignored in Dr. Englander's book.

Dr. Englander seems to argue that it is proper for her book to trivialize the diagnosis of antisocial personality disorder (and indeed not mention any other personality disorder) and to dismiss the relationship between serious mental illness and violence in three paragraphs. The readers of this journal can judge whether a text on violent crime that does that would be useful to them.

Harold Carmel, M.D.

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1. Tardiff K: Violence, in American Psychiatric Press Textbook of Psychiatry, 3rd ed. Edited by Hales RE, Yudofsky SC, Talbott JA. Washington, DC, American Psychiatric Press, 1999

A Mischaracterization

To the Editor: In their review article on treatment of sex offenders in the March 1999 issue, Grossman and her coauthors (1) badly mischaracterized my article in *Science* (2) when they stated that "Zonana has suggested, however, that the consequences of recidivism in sex offenders are so detrimental to society that a recidivism rate of zero is the only acceptable risk level."

The entire thrust of my article was to oppose the sexual predator statutes following the *Kansas v. Hendricks* decision in which the U.S. Supreme Court found them to be constitutional. I argued that the popularity of the statutes was due to the fact that *the public* expected a recidivism rate of zero and seemed to tolerate nothing less. The public could not care less where such offenders are housed– in prisons or in mental hospitals. To me, it makes an enormous difference. This mischaracterization of my views places me in the untenable position of appearing to make a recommendation that I find quite objectionable.

Howard V. Zonana, M.D.

Dr. Zonana is director of the law and psychiatry division of the Connecticut Mental Health Center in New Haven.

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1. Grossman LS, Martis B, Fichtner CG: Are sex offenders treatable? A research overview. Psychiatric Services 50:349–361, 1999

2 Zonana H: The civil commitment of sex offenders. Science 278:1248–1249, 1997

In Reply: We thank Dr. Zonana for calling attention to our incomplete representation of his statement that "from the public viewpoint, only a relapse rate of zero is acceptable." As we inferred, such a view might lead to the conclusion that the only solution for sex offenders would be indefinite confinement, with or without treatment. Zonana makes no such recommendation. Rather, he acknowledges the growing evidence of treatability for some sex offenders. He develops several lines of argument against the civil commitment statutes, one of which involves his representation of the public viewpoint. Since our review of the treatment research literature yielded evidence that treatment makes a difference, we meant to point out that treatment would be expected to have some beneficial impact whether it is prison based or in the context of civil commitment.

This evidence of some beneficial impact leaves unanswered important questions of differential responsiveness of subpopulations of sex offenders and of where treatment should be provided (prisons, mental hospitals, "hybrid" institutions, or outpatient settings). We noted evidence that treatment in institutional settings and an extensive criminal record are in general associated with poorer outcome. We also mentioned several concerns of mental health professionals and public administrators, including risks associated with mixing populations and the drain on mental health services resources. Reid (1) has pointed out that questions of treatability, where such patients are housed, and what resources are available for their care are conceptually separable.

We believe the main policy implication of our article is that, given the prospect of effective treatments and the major uncertainties surrounding verification of just how effective these treatments may be, new treatment programs cannot be responsibly developed without clinical research components.

Linda S. Grossman, Ph.D.
Christopher G. Fichtner, M.D.

Reference

1. Reid WH: Myths about violent sexual predators and all that pesky legislation. Journal of Practical Psychiatry and Behavioral Health 4:246–248, 1998

Olanzapine and NMS

To the Editor: Neuroleptic malignant syndrome (NMS) is a serious adverse reaction to neuroleptic drugs. It is characterized by muscle rigidity and elevated temperature with at least two of the following findings: tachycardia, elevated or labile blood pressure, diaphoresis, dysphagia, tremor, incontinence, altered level of consciousness, mutism, leukocytosis, and elevated creatine phosphokinase (CPK) (1).

The frequency of NMS in patients treated with neuroleptics ranges from .02 to 1.9 percent (2). We present a case of thioridazine-induced NMS, possibly exacerbated later by the new atypical antipsychotic medication olanzapine.

A 37-year-old African-American man with schizophrenia was hospitalized for possible acute renal failure. Treatment had previously included thioridazine 300 mg per day, and valproic acid 750 mg per day. At entry into the hospital the patient had a fever of 101 degrees F, hypertension (166/95 mm Hg), and altered mental status with prominent confusion. Rigidity was found on physical examination. Laboratory studies revealed elevated levels of blood urea nitrogen (BUN) (43 mg/dL), creatinine (2.6 mg/dL), sodium (155 mmol/L), and CPK (3294 U/L). The patient was dehydrated.

Neuroleptic malignant syndrome was the primary diagnosis. Thioridazine and valproic acid were discontinued. Seven days later, after hydration, the patient was physiologically stable and was transferred to the psychiatric service with no sign of NMS. Rigidity had disappeared. Laboratory results showed normal levels of BUN at 12 mg/dL, creatinine at 1.2 mg/dL, sodium at 142 mmol/L, and CPK at 174 U/L. A mental status examination evidenced only psychosis.

The morning after the patient's transfer, olanzapine 5 mg per day was prescribed because of its relative lack of association with NMS. On the second day in the psychiatric unit, as a result of newly documented hypertension (148/94 mm Hg), the olanzapine dosage was decreased to 2.5 mg per day. The patient's blood pressure then stabilized, and olanzapine was titrated over a three-day period up to 7.5 mg daily. On the fifth day after beginning olanzapine treatment, he became tachycardic (pulse, 114), hypertensive (blood pressure,139/90 mm Hg), and diaphoretic. His temperature remained normal. Serum CPK increased to 704 and BUN and creatinine levels again became elevated slightly to 28 mg/dL and 1.5 mg/dL, respectively.

Olanzapine was discontinued, and the patient recovered the following day, with normal vital signs. Because he was clinically improved but still psychotic, olanzapine was then restarted at 2.5 mg and more slowly titrated to 5 mg daily over five days with monitoring of vital signs, physical observations, and serum chemistries. No further complications occurred, and the psychosis diminished. The patient was discharged on olanzapine 5 mg per day with improved mental status, without NMS, well hydrated, and with a blood chemistry profile within normal ranges.

The patient's initial hospitalization was precipitated by neuroleptic malignant syndrome induced by thioridazine and complicated by dehydration. One week later, after the resolution of NMS and pre-renal azotemia, olanzapine was prescribed to treat the patient's psychosis because of the drug's relatively low affinity for dopamine D_2 striatal receptors and its low risk for causing NMS (3). However, administration of olanzapine at a 7.5 mg dose re-created a clinical picture compatible with very early NMS. By beginning olanzapine one week after recovery from NMS, the patient may have been at a higher risk for its return.

On the second olanzapine trial, at two weeks post-NMS and with a more slowly escalating dosage, the patient was stabilized on 5 mg per day

without evidence of NMS. Good hydration, the lower dosage, and the longer wait after recovery from NMS may have provided additional safety. An antipsychotic response occurred without problems.

Whether olanzapine could result in NMS or similar adverse side effects remains an unanswered question. Caution, close observation, and hydration are warranted whenever antipsychotic pharmaceuticals, including olanzapine, are given to people at risk for NMS.

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Steven Lippmann, M.D.

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3. Zyprexa (olanzapine) package insert. Indianapolis, Ind, Eli Lilly, May 1, 1997

Pimozide in the Treatment of Litigious Delusions

To the Editor: In subtyping delusional disorder, the authors of *DSM-IV* did not include a litigious variant, although the syndrome had been previously described (1). It is thought to be both rare and resistant to treatment, especially when related to an organic condition, in which case a diagnosis of psychotic disorder due to a general medical condition would be appropriate. This report describes the successful treatment of a litigious delusional patient with pimozide.

Mr. A, a 52-year-old Caucasian male, began exhibiting litigious delusions at age 51, about a year after his father's death. He falsely believed that his sister, attorneys, and judges conspired to deny him his share of his father's estate, and he had hired and fired nine attorneys in his efforts to claim imaginary missing funds. He wrote numerous letters to state and federal authorities charging unfair treatment by the courts and made death threats toward his sister that resulted in his arrest. He was found incompetent to stand trial and subsequently was hospitalized.

Mr. A had a premature and difficult birth. He was deaf since age 2, secondary to streptomycin treatment for an infection; developed conduct disorder at age 13; and abused alcohol from age 20 to age 40. He developed a grand mal seizure disorder at age 39. When he was 40, during the course of the work-up after his first seizure, a 5 cm left frontal lobe meningioma was diagnosed and resected. He had been maintained on anticonvulsant therapy and remained free of seizures since his surgery. His most recent CT scan, at age 50, showed no regrowth of the meningioma or other abnormalities.

On his initial mental status examination after hospitalization, Mr. A was clear and logical in his thinking and oriented to person, time, and place. However, his affect was constricted, and he had prominent paranoid litigious delusions. An MRI revealed a 3 cm venous angioma in the right posterior temporal lobe.

Mr. A was initially given prolixin, but it was discontinued after four weeks due to intolerance. He then was started on pimozide 2 mg per day, gradually titrated to 6 mg per day.

After five weeks Mr. A's symptoms substantially resolved. He no longer considered pursuing litigation or writing letters to various state and federal authorities, although he still felt that he was cheated of his share of the estate. He no longer threatened his sister and was discharged. He was subsequently linked to aftercare treatment and, six months later, had not been readmitted to our facility.

The meningioma and the anginoma may have contributed to Mr. A's illness. To our knowledge, this is the first reported case in which a patient with litigious delusions that appear to be associated with medical conditions has responded well to pimozide. Ungvari and Hollokoi (2) reported using pimozide to successfully treat an 85-year-old litigious patient who suffered with the illness for 40 years without any medical conditions.

Numerous publications show that pimozide successfully treats various types of delusional disorders, including litigious delusions. Its unique mechanism of action is largely devoid of noradrenergic impact; it does not block postsynaptic D_2 receptors, while having relatively little impact on D_2 autoreceptors. Further studies to determine the usefulness of pimozide in delusional disorders associated with other organic conditions are desirable in view of the unique features in pimozide's neuropharmacological profile (3).

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