this area” (emphasis added). Thus, it was (and remains) our intent to stimulate further observation, discussion, and research in this area.

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The Locus Ceruleus and Anxiety Disorders in Demented and Nondemented Familial Parkinsonism

To the Editor: Luis M. Iruela, M.D., Ph.D., and colleagues (1) queried whether anxiety disorders occur selectively in nondemented Parkinson’s disease patients due to ceruleal dopamine/norepinephrine imbalance, resolving at dementia onset. We analyzed data from our 38 patients with familial parkinsonism (2) and defined cognitive impairment by a score ≤ 48/57 on the Mayeux et al. modified (3) Mini-Mental State exam (4). We studied anxiety conditions (rather than DSM-III diagnoses) to achieve the best sample sizes and maintain biologic construct validity. We compared cognitively impaired familial parkinsonism subjects (N=11) with (“+”) and without (“−”) anxiety conditions to nonimpaired subjects (N=27) with and without anxiety conditions. Results indicate: 1+/10− versus 1+/26− (generalized anxiety); 1+/10− versus 8+/19− (panic attacks); 1+/10− versus 0+/27− (agoraphobia without panic); 1+/10− versus 5+/22− (obsessions or compulsions); 0+/11− versus 1+/26− (social phobia); 2+/9− versus 2+/25− (nonphobic social embarrassment over familial parkinsonism); 2+/9− versus 11+/16− (simple phobia); 1+/10− versus 3+/24− (insomnia); 5+/6− versus 19+8− (any anxiety condition excluding nonphobic embarrassment).

We further considered dementia as clinically diagnosed DSM-III probable or definite dementia. One demented patient scored 52 while three nondemented patients scored 48, 43, and 47 on the modified mental state exam. Comparison of demented subjects (N=9) with and without anxiety conditions to nondemented subjects (N=29) with and without anxiety conditions indicates: 1+/8− versus 1+/28− (generalized anxiety); 1+/8− versus 8+/21− (panic attacks); 1+/8− versus 0+/29− (agoraphobia without panic); 1+/8− versus 5+/24− (obsessions or compulsions); 0+/9− versus 1+/28− (social phobia); 2+/7− versus 2+/27− (nonphobic social embarrassment over familial parkinsonism); 1+/8− versus 12+/17− (simple phobia); 0+/9− versus 4+/25− (insomnia); 4+/5− versus 20+/9− (any anxiety condition excluding nonphobic embarrassment).

None of the comparisons achieved statistical significance by Fisher’s exact test. Perhaps a larger sample size might support the hypothesis of Dr. Iruela and colleagues. In cognitive impairment, significance may be easiest achieved for panic attacks (Fisher’s p=0.18), simple phobias (p=0.17), and any anxiety condition (p=0.14), while DSM-III diagnostic dementia criteria suggest this for phobias (p=0.16) and any anxiety condition (p=0.17).

In accord with parkinsonian ceruleal and nigroceruleal degeneration relating to anxiety disorders, we previously noted attenuation of panic attack phenomena with the onset of parkinsonian freezing (2), a disorder perhaps involving norepinephrine and the locus ceruleus (5). We also noted that anxiety disorders often precede the onset of familial parkinsonism (2). Further, classical panic disorder typically precedes familial parkinsonism onset (2), while atypical panic phenomena (with fewer symptoms and fewer attacks than DSM-III panic disorder) have an onset after familial parkinsonism onset. These relationships in our patients with this disorder suggested to us that early ceruleal or nigroceruleal dopaminergic pathway degeneration may dis inhibit the locus ceruleus before parkinsonian motor signs develop and may eventuate in anxiety conditions, particularly panic attacks. These conditions possibly attenuate when further ceruleal degeneration ensues.

REFERENCES

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Refine Bizarre Delusion Criterion

To the Editor: The article by Dodi Goldman, M.A., M.M.H., and colleagues (1) is disconcerting because the results could have been interpreted quite differently. The authors advocate discarding the bizarre delusion criterion in schizophrenia on the basis of low specificity and lack of validation by cross-sectional measures. An alternative interpretation of their data is that the definition of bizarre delusions should be refined, perhaps as does the ICD-10 proposal (2).

The study has significant methodological flaws:
1. From the methods section we know only that the authors attained reliability about whether a delusion is bizarre, but we are not informed if they focused upon delusions that Schneider described as characteristic of schizophrenia such as delusions of passivity/influence or delusional perception. Their method would seem to include mood congruent delusions in affective disorders as bizarre if they were sufficiently implausible.
2. Their data are cross-sectional and therefore lack important longitudinal validity measures.
3. They do not compare other DSM-III-R active symptom criteria for specificity, sensitivity, and validity, although these would seem available in their data set. I would expect thought disorder, catatonic symptoms, and hallucinations to perform more poorly than bizarre delusions in differentiating schizophrenia from psychotic affective disorders.

The authors seem to assume bizarre delusions would predict worse disease, but data to the contrary are well known. Most clinicians know bizarre delusions predispose to the paranoid type of schizophrenia, which seems to have better premorbid characteristics and outcome on some measures than other subtypes (3). The authors’ own neuroimaging and cognitive data show that the patients who have schizophrenia with bizarre delusions have higher IQs, better card sorting, and more normal computerized tomography scans. These results actually lend validity to the bizarre delusion concept.

It appears that careful phenomenologic description (4) is of more interest to European psychiatrists than we Americans. I am concerned that deletion rather than refinement of the bi-
zarre delusion criterion will discourage research and teaching of these important concepts and widen the transatlantic conceptual rift.

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Dr. Haas and Associates Reply

TO THE EDITOR: Validation of diagnostic criteria is a topic which tends to evoke strong opinion. As evidenced by Dr. Weiler's letter, our article on the bizarre delusion criterion in DSM-III-R schizophrenia is no exception to the rule. The controversial nature of proposals to define or modify taxonomy in psychiatry is not surprising in that we still essentially rely on expert opinion, rather than laboratory test, as the "gold standard" in our definition of clinical syndromes. Until we have a better-developed knowledge base regarding the nature of schizophrenia, we are dependent on empirically guided evaluation of clinical indicators. Research such as ours aims to provide an empirical basis to help guide decisions regarding diagnostic criteria and classification schema. To the extent that such research challenges conventional wisdom or regional principles of clinical practice, it is likely to pique the intellectual interest, if not the emotions, of professionals in our field.

We appreciated Dr. Weiler's comments but found them to reflect goals for our study more ambitious than our own. With regard to his first question, our goal of actuarial assessment of one of the still controversial DSM-III-R criteria did not extend to consideration of various other definitions of "bizarre" delusions (e.g., with a specific focus on Schneiderian delusions). In fact, the DSM-III-R definition of bizarre delusions was itself not restricted to the Schneiderian type—largely because substantial empirical data indicate that these particular features are not specific, nor even sensitive, indices of schizophrenia (1-3). Turning to the question of a longitudinal design, this study was retrospective, reviewing lifetime history of psychotic and affective symptoms. Because we did not follow patients beyond the hospital phase of study, estimates of the stability of the diagnoses were, unfortunately, not available.

We recently conducted analyses of the comparative efficiency of bizarre delusions and several other psychotic symptoms. Our findings indicate that only Schneiderian delusions of mind control showed poorer diagnostic efficiency than did the bizarre delusions criterion. Each of the other DSM-III-R psychotic symptoms (i.e., prominent hallucinations, loose associations, incoherence, catatonia, flat affect, inappropriate affect, hallucinations, and other delusions) exceeded bizarre delusions in terms of their efficiency in discriminating DSM-III-R schizophrenia from other psychotic disorders.

Finally, critics of an empirical approach to diagnostic validation often mistake the emphasis on parsimony and the use of quantitative methods as a devaluation of careful phenomenologic study of clinical syndromes. Such misconception harkens back to the heated debates of the 1950s over clinical versus actuarial prediction in psychodiagnostic testing (4). It is our hope that actuarial studies such as our own may help to better improve understanding of the clinical phenomenology of schizophrenia.

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PTSD and Substance Abuse

TO THE EDITOR: We are writing in response to the article on the chronological sequencing of traumatic events, PTSD symptoms, and substance abuse symptoms in the general population by Linda B. Cottler, Ph.D., and her associates (1). Although we believe that etiologic research studies using community samples are important to understanding the true course of disorders, we have some concerns about the procedures and conclusions in their report.

Our first concern relates to the article's suggestion that "illicit substance use predisposes the individual to exposure to traumatic events." Although substance abuse symptoms were found to be likely to occur prior to posttraumatic stress disorder (PTSD) symptoms in this sample, we feel that this conclusion goes beyond the data because the temporal relationship between the occurrence of traumatic events and the onset of PTSD and substance abuse symptoms was not assessed. Further, recent research suggests that a variety of psychiatric disorders may be sequelae to traumatic events, and diagnostic profiles may change over time (2). The findings in this report may represent cases in which postexposure substance abuse evolved into PTSD due to the failure of substance abuse as a coping mechanism and/or because, over time, additional stressors were introduced in the individuals' lives.

Our second concern relates to the validity of the study's PTSD case identification procedure. Dr. Cottler and associates identified cases using the PTSD module of the Diagnostic Interview Schedule (DIS). We are aware of no studies that have tested the module's validity by comparing the DIS/PTSD diagnosis to one based on any other method (e.g., a structured clinical interview or a multisource, multimethod procedure). The only psychometric information cited in the article is a Breslau and Davis report (3) in which the DIS was administered twice and the two diagnoses compared for concordance as a test of the module's reliability. Further, analysis of data