trast and revealed decreased attenuation consistent with chronic ischemic changes of the white matter.

Five days after the admission she developed negativism, mutism, and akinesia, made no eye contact with others, and had perioral dyskinetic movements, waxy flexibility, and catalepsy (Table 1). After clinical evaluation at 5 days after the admission, lorazepam, 2 mg i.m., and memantine, 5 mg p.o., were given. The patient responded quickly and gave coherent responses to what was asked, walked and interacted with other patients, and was able to take care of herself. Risperidone and zolpidem were discontinued at this point. However, this improvement lasted for 24 hours but once again catatonia returned. The dose of lorazepam was changed to 2 mg i.m. b.i.d. and memantine to 7.5 mg p.o. b.i.d.

Gradual improvement in catatonic signs was seen with reduced severity of some signs and a 50% reduction in catatonic signs by the 5th day of treatment (Table 1).

Two weeks after admission the patient was free of most catatonic signs and symptoms. In addition, she was alert, fully oriented, and without significant evidence of cognitive dysfunction on mental status exam. Lorazepam was converted to 3.75 mg p.o. b.i.d. and memantine to 7.5 mg p.o. b.i.d. The patient remained on this medication regimen for 1 year without the return of significant catatonic signs or symptoms.

This patient's presentation for major depression was complicated by catatonia. She had cerebrovascular disease which may have contributed.[3] However, the treatment of major depression with catatonia in this case is interesting because of the combined use of lorazepam, a $GABA_A$ promoter, and memantine, an NMDA antagonist.[2] We feel that this case may suggest a useful treatment approach for major depression with catatonic features.[4]

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Ethnicity and Cognition: Dangers of Biological Determinism

To the Editor: I read with interest the article in the August issue of this journal[1] which reported a negative association between Mexican American ethnicity and cognitive function in late life, but I believe that the findings should be considered within a certain context.

These findings may come to form the basis of improved screening programs enabling early detection and intervention as well as improved allocation of resources, but it would be remiss not to balance such optimism with a cautionary note.

Claims that ethnic or racial groups possess certain biological or cognitive characteristics have a long history within the behavioral sciences and have attracted considerable controversy. The negative social implications may not be readily apparent but such claims may have undesirable social effects.

TABLE 1. Catatonic Signs Rated by Day after Onset of Catatonia (day 5 of admission)

Treatment Day	1	2	3	4	5	6	7	8	9
Waxy flexibility/catalepsy	+	+	+	+	+	+	+	0	0
Immobility/stupor	+	+	+	0	0	0	0	0	0
Staring	+ +	+ +	+ +	+ +	+ +	+ +	0	0	0
Refusal to drink or eat	+	+	+	0	0	0	0	0	0
Negativism/negative symptoms	+	+	+	0	0	0	+	0	0
Mutism	+	+	+	+	0	0	0	0	0
Rigidity	+ +	+ +	+ +	+ +	+ +	+	+	+	+
Incontinence	+ + +	+ + +	+ +	+ +	+ +	+	+	+	0
Grimacing	+ +	+ + +	+ + +	0	0	0	0	0	0
Posturing	+ +	+ +	+ +	+ +	+ +	+ +	+	+	+
Total # Signs	10	10	10	6	5	5	4	3	2

Prejudice along ethnic lines is often predicated on fallacious notions of the existence of fundamental difference between groups and of uniformity within them. Be it IQ or other biological or cognitive profiles, such markers carry the danger of concretizing perceptions of difference between groups and of sameness within them. In so doing they serve to justify the perception of difference between ethnic groups where such difference does not exist. In the area of race or ethnicity, this can have particular unwanted effects of validating social inequalities.

The most masterful exponent of such ideas was the late Stephen Jay Gould who articulated his ideas most lucidly in *The Mismeasure of Man*.[2] Gould enunciated the dangers of biological determinism in the context of race.

One of Gould's criticisms of *The Bell Curve*[3] was that, in championing the idea that inequality is subserved by innate differences, it understates the role of socio-political factors in its perpetuation. Gould asked, "What argument against social change could be more effective than the claim that established orders exist as an accurate reflection of innate intellectual capacities?" [1]

Heller et al.'s[1] findings need to be considered in this light. The authors strike an encouraging note of receptivity to possible explanations when they suggest that "as yet unmeasured variables" mediate the reported association and socioeconomic disadvantage may be a potent "etiological factor." Neuropsychiatry is biased toward locating the explanation for particular cognitive findings "in the brain." In the absence of an integrative model of cognition incorporating social factors, there is a tendency to misattribute etiology in a manner which minimizes sociogenic factors. When the message is that social inequalities between ethnic groups are underpinned by innate differences, the implications may be particularly invidious.

Sociologists are at pains to stress the complexities and ambiguities surrounding notions of ethnicity.[4] A common error is to assume homogeneity within an ethnic group. The authors fail to address these points to a depth that their complexity warrants. With no guarantee that successive generations are immune to repeating the mistakes of previous ones, it is incumbent upon new generations of neuroscientists and psychiatrists to be cognizant of the issues raised by Gould.

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Lamotrigine for the Treatment of Impulsive Aggression and Affective Symptoms in a Patient with Borderline Personality Disorder Comorbid with Body Dysmorphic Disorder

To the Editor: Many psychiatric disorders are characterized by impulsive auto-hetero-aggression and violence which are manifested as suicide attempts, self-injurious behavior, or physician-directed violence or domestic violence. The consequences of these behaviors significantly increase costs as they often lead to the patient's hospitalization and/or property destruction. Borderline personality disorder and body dysmorphic disorder share some similarities such as impulsive aggression, anger, hostility, and irritability associated with moderate to severe depressive symptoms.

Pharmacological approaches to borderline personality disorder have focused mostly on the mood and impulsive aggressive domains, thus leading to the frequent use of combination treatments, mainly selective serotonin reuptake inhibitors (SSRIs), atypical antipsychotics, and mood stabilizers such as carbamazepine or valproate.[1] As for body dysmorphic disorder, SSRIs like fluvoxamine have proven to be beneficial in reducing depressive symptoms, anxiety symptoms, and anger outbursts.[2]

Lamotrigine is a mood stabilizer with antidepressant properties which has been approved for acute treatment maintenance of bipolar depression; as an anticonvulsant it might possess an anti-aggressive effect.

Our patient was a 26-year-old man diagnosed with borderline personality disorder disorder at the age of 19. He was twice hospitalized mainly due to frequent episodes of inappropriate anger or rage, aggression toward parents, and self-injurious behavior. During our interview we also diagnosed presence of body dysmorphic disorder. His major complaints were depressed mood, loss of interest, irritability, impulsive behavior, and anger outbursts. His depressive symptoms were rated moderately severe—18 points on Hamilton Depression Rating Scale (HAM-D)—while his impulsive aggression was