on DSM-IV criteria. Pharmacological treatment was the primary mode of management. If records indicated that a patient had returned to "baseline," or had improvement in more than half the number of presenting symptoms, that patient was considered a "treatment responder." A patient who had absent or minimal improvement in symptoms was considered a "nonresponder."

About a third of the sample (n = 17, 32%) was diagnosed with personality changes.

The mean age of the sample was 41.1 years (SD = 12.26; range = 18–69). Most patients were male (N = 37, 68.5%), and 81.5% were Caucasians. The mean level of education was 13.4 years (range = 8–20). Forty-four percent of the patients had suffered mild traumatic brain injury, 20.4% moderate injury, and 35.2% had a severe injury, based on the Glasgow Coma Scale (GCS) scores at the time of the injury. Those with personality change were compared to those without personality change. The two groups did not differ in number of injuries, type of trauma, age, education, pre-traumatic brain injury marital, employment and legal status, substance abuse, and/or pre-traumatic brain injury history of mood disorder. However, those with personality change were disproportionately non-Caucasians, had lower scores on the Mini-Mental State Exam (MMSE), had more severe traumatic brain injury, were less likely to be employed post-TBI, and were more frequently judged to have failed pharmacologic treatment.

Discussion

In this study personality changes are associated primarily with severe traumatic brain injury, as was the case in an earlier study.[3] Association between personality change and mild traumatic brain injury has also been reported.[4] We are not surprised that our patients who had personality change did not benefit from pharmacological treatment since it is well established that multimodal regimens that include medications and psychotherapy are the best.[5] Our patients with personality change had worse cognitive function and were less successful in finding employment after traumatic brain injury but did not have a higher rate of substance abuse, legal problems, or mood disorder as has been reported by others.[4,6]

Conclusion

Our investigation relied on a highly selected sample and was limited in scope by the source of the data and the size of the sample. However, these observations are still helpful for guiding the development of future prospective studies.

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Memantine in Major Depression with Catatonic Features

To the Editor: Catatonia is a movement disorder defined primarily by a cluster of signs including immobility, mutism, and withdrawal or refusal of food and water,[1] and its presence in the elderly patient can lead to rapid medical decompensation necessitating rapid and effective treatment for this condition. Catatonia may arise from GABA-$_A$ hypoactivity, dopamine (D_2) hypoactivity and possibly glutamate *N*-methyl-D-aspartic acid (NMDA) hyperactivity.[2]

Ms. A, an 80-year-old female, was admitted to a geriatric psychiatry residential unit presenting with depressive symptoms. She had been a widow for 3 years and was brought to the hospital at the request of her four sons because they felt that she could no longer care for herself. The patient had no prior episodes of depression or any other psychiatric disorder.

Her medical history was significant for hypertension, pacemaker placement, chronic renal failure, hiatal hernia, and gastritis. She was being treated for cognitive dysfunction. Medications prior to admission were enalapril, 20 mg/day, memantine, 10 mg/day, zolpidem, 10 mg/day, risperidone, 0.5 mg/day, and omeprazole, 20 mg/day.

A clinical and neurological exam showed no additional findings. Laboratory studies did not reveal significant abnormalities, creatinine was 1.2 on admission and remained at 1.1, and BUN was 41. CT of the brain was performed without con-

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