focusing on youth-specific issues, such as negative effect on normal growth that appears to be associated with these medications (1). To our knowledge, no cases of hemorrhagic stroke have been described in association with mixed amphetamine salts or other stimulants used for treatment of ADHD. However, amphetamines, cocaine, and ecstasy have all been reported to cause spontaneous intracerebral hemorrhages (3). We do not intend to suggest direct causality between the present patient's treatment with mixed amphetamine salts and hemorrhagic stroke. The intent of this report is to increase awareness among psychiatrists that, with increased prescribing of stimulants to adults with ADHD, a new level of vigilance is needed to identify safety risks salient to adults.


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## ECT for Catatonia After Mitral Valve Replacement

TO THE EDITOR: Malignant (lethal) catatonia may present in the postoperative period (1). It is a medical emergency, with high morbidity and mortality. We report the case of a 45-year-old patient with schizoaffective disorder who developed malignant catatonia 5 days after mitral valve replacement. His catatonia was successfully treated with ECT, starting on postoperative day 7. To our knowledge, this is the shortest reported interval between cardiac surgery and ECT.

**"Mr. A" was admitted to our cardiothoracic surgery service with mitral regurgitation, pulmonary hypertension, and class III heart failure from a paravalvular leak. Eight months earlier, he had endocarditis and required mitral valve replacement, using a mechanical valve. He had three psychiatric admissions in his history (the last admission was 13 years earlier) for catatonia, all of which were successfully treated with ECT. The patient had been stable with olanzapine treatment (10 mg/day). He again underwent mitral valve replacement, but with a bioprosthetic valve. He did well in the perioperative period. Psychiatry was consulted on postoperative day 2 for anxiety. The patient was fully oriented, with normal attention and concentration, but thought blocking, paranoia, and disorganized thinking was observed. Olanzapine was increased to 15 mg/day. On postoperative day 5, he became mute and immobile, with staring, negativism, and waxy flexibility. He refused oral intake and was intermittently combative. Catatonia was diagnosed, and olanzapine was discontinued. Intravenous lorazepam, up to 12 mg daily for 3 days, was ineffective. Medical evaluation, including head computed tomography, chest X-ray, and blood/urine cultures, was unrevealing for a nonpsychiatric etiology of catatonia. Catatonia was presumed to be due to psychotic disorder.**

**ECT was initiated on postoperative day 7. A total of nine bilateral (bitemporal) ECT sessions (stimulus dose: 302 mC–505 mC; pulse width: 1.0 msec) were given between postoperative days 7 and 21. The initial five ECT sessions involved two seizure inductions each (in order to speed response); only one seizure induction was used in each of the subsequent sessions. Anesthesia was carried out with methohexital and succinylcholine. Esmolol and nitroglycerin were administered to control tachycardia and hypertension. There were no cardiac complications. After the initial five ECT sessions, the patient was less catatonic and more verbal, but still psychotic. By postoperative day 10, he was febrile (40°C), with a white blood cell count of 23,000 and creatine phosphokinase level of 8,000 U/l. Infectious disease evaluation was negative. Despite antibiotic therapy, these abnormalities persisted until postoperative day 15, when the patient's fever spontaneously resolved and his white blood cell count and creatine phosphokinase level were normalized. His fever and elevated white blood cell count and creatine phosphokinase level were presumed to be due to the catatonia itself. ECT was continued. By postoperative day 21, the patient was alert, speaking, and moving normally and not psychotic. Olanzapine at 10 mg/day was resumed. On postoperative day 32, the patient was discharged home well.**

There are case reports of the safe use of ECT in depressed patients after cardiac surgery, including as early as 2 weeks after coronary artery bypass graft (2) and 27 days after aortic and mitral valve replacement surgery (3). Our patient had no cardiac complications from ECT 1 week after mitral valve replacement. Definitive treatment of malignant catatonia with ECT should be considered, even in the early postoperative period after major cardiac surgery.


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## Naltrexone Effects in Patients With Dementia

TO THE EDITOR: In their recent articles published in the *Journal*, Blazer and Wu (1) and Mathews and Oslin (2) have drawn attention to alcohol use among the elderly. Furthermore, a recent Treatment in Psychiatry article, by Johnson (3), highlighted pharmacologic interventions for alcoholism, where

naltrexone was suggested in two out of three cases, including for a 66-year-old patient. To add to the discussion of naltrexone use in the elderly, three cases are presented suggesting potential benefits of naltrexone for the treatment of alcoholism complicating dementia.

**"Mr. S" was an 84-year-old man with long-standing bipolar I disorder and alcoholism. He had developed dementia as a result of Alzheimer's disease. His persistent alcoholism included consumption of up to one-fifth of whiskey daily, which led to bizarre and dangerous behaviors. He began naltrexone (50 mg daily), which resulted in reduced interest in alcohol within the first week. This improvement was sustained even after treatment was discontinued 6 months later.**

**"Ms. A" was an 86-year-old woman with frontotemporal dementia who repeatedly drank scotch to intoxication, resulting in frequent falls. Bottles of liquor were stashed throughout her house, and she became acutely agitated if she was prevented from buying more. While receiving treatment with naltrexone (50 mg daily), the forcefulness of her alcohol-seeking behavior abated, and after 3 weeks she discontinued drinking altogether. Naltrexone was continued for a full year, with continued sobriety.**

**"Mr. C" was a 68-year-old man with Wernicke-Korsakoff syndrome. He was able to live independently with a supportive landlord, but placement in a secure facility was considered after several hospitalizations resulted from his drinking to unconsciousness. While receiving treatment with naltrexone (50 mg), he discontinued frequenting local bars and was able to continue living in his familiar neighborhood.**

Published data on the use of naltrexone in the elderly is not available, but it is of note that alcohol abuse ended promptly in these three cases. Clinical benefit was significant given that these patients continued to live in their communities with a reduction in caregiver stress and reduced harmful events such as falls. The beneficial response observed in these cases is unlikely the result of a placebo response, and the prompt reduction in alcohol craving probably represents a true neuropharmacologic effect of reducing alcohol-related euphoria and undermining alcohol-seeking behavior. While progression of dementia may have contributed to reduced addictive behaviors, the prompt reduction in drinking does suggest that anticraving medications may have a role for elderly patients.

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## A Proper Name for Chronic Tic Disorder

To the Editor: When Georges Albert Édouard Brutus Gilles de la Tourette (1857–1904) described, at age 28, the disease of convulsive tics, he could not anticipate the taxonomic destiny of that clinical picture. As known, he gave the pioneer descriptions of patients with chronic tics, echolalia, and coprolalia (1). His writings on the clinical concept of hysteria are also well documented, with empirical observations as well as historical studies, for instance, the work on the famous Loudon case (2), which gave rise to the fine essay by Aldous Huxley ("The Devils of Loudon").

Chronic tics disorder is known as *Tourettschen Krankheit* in German, *Maladie de Gilles de la Tourette* in French, and Tourette's disorder in English (3). It is one of the very few eponyms that remain in DSM, which has eliminated most, following the example of the International Classification of Diseases.

Why was the last name of the physician, Gilles de la Tourette, abbreviated as Tourette in the 3rd and 4th editions of DSM? The name of the disorder appears in DSM-IV-TR as F95.2 Tourette Disorder (307.23). The first part of the famous physician's name is Georges Albert Edward Brutus, and his last name is Gilles de la Tourette (4). Abbreviating the last name contradicts the honor implicit in a medical eponym and is inexact in terms of linguistic science and tradition. While DSM editions have significantly improved the reliability of psychiatric diagnosis, the use of eponyms may have the quality of being neutral in terms of social stigma (compared with historical terms such as hysteria or, nowadays, schizophrenia), and thus chronic, vocal, and motor tics, which generally appear during childhood and adolescence and are still idiopathic, could be maintained for taxonomic purposes in DSM-5 with the use of an eponym: Gilles de la Tourette disorder.

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