

“Silent” Strokes and Dementia

TO THE EDITOR: Vermeer et al. (March 27 issue)¹ demonstrate associations between “silent” (asymptomatic) stroke and recurrent stroke and dementia. The risk of large strokes was the highest, with non-significant trends toward an increased risk of small-vessel or white-matter lesions. Whether incident silent infarcts and incident symptomatic infarcts confer similar risks of recurrent symptomatic stroke and dementia is unclear. If an analogy to carotid disease holds, there may be different risks for silent strokes and for symptomatic strokes.

Although the accompanying editorial by Blass and Ratan² raises important issues about risk-factor modification in patients with a silent stroke, information is limited on combination antiplatelet therapy for the reduction of the risk of dementia. There are data showing decreases in overall mortality among patients with vascular dementia who are treated with any antithrombotic agent.³ However, there is no absolute benefit of antiplatelet drugs (including aspirin) in the prevention of a first symptomatic stroke.⁴ Sustained-release dipyridamole and aspirin are quite reasonable treatments for patients with symptomatic stroke, but clinical trials are needed in order to show whether specific antiplatelet drugs are cost effective in decreasing the risk of dementia after a silent infarction.⁵ Pending future investigations, aspirin alone might still be the initial choice for patients who are at high risk for cerebrovascular disease.^{4,5}

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Editor's note: Dr. Schneck reports having received speaker's fees from Boehringer Ingelheim and Bristol-Myers Squibb/Sanofi.

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DRS. BLASS AND RATAN REPLY: Dr. Schneck's point about the need for further studies of silent stroke is well taken. We tried to make the same point in our editorial. Further studies will, we hope, provide clear information on whether clinically silent stroke should be treated more as a previous stroke or more as a marker of cardiovascular disease.

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MDMA and Parkinsonism

TO THE EDITOR: Recreational use of 3,4-methylenedioxymethamphetamine (MDMA), or “ecstasy,” has been increasing. MDMA has classically been associated with serotonergic neurotoxicity in humans and in animal models. Recent investigations in non-human primates, at doses modeled after those used by humans, revealed severe dopaminergic neurotoxicity with less serotonergic neurotoxicity.¹ We report a case of juvenile Parkinson's disease after repeated use of MDMA.

A 19-year-old man was observed to have a resting tremor in his right hand on going to sleep and

waking from a nap in March 2002. The tremor progressed to affect the right leg. Magnetic resonance imaging of the head was normal. He was given a diagnosis of Parkinson's disease, and selegiline was prescribed, but since his symptoms did not improve, he discontinued treatment. Trihexyphenidyl was then prescribed; it reduced his tremor, but because of nausea the patient takes it only once a week. Within the past four months, he has reported progressive difficulty in rising from bed. In the morning, he says, his “muscles don't want to move.” He has also noticed that his typing proficiency has deteriorated.

The patient's father, who is 62 years old, has a 13-year history of rest tremor that was diagnosed as Parkinson's disease 10 years ago. The Parkinson's symptoms have improved with levodopa-carbidopa, but he has motor fluctuations. The father's half brother, who is 55 years old, also has Parkinson's disease and has had symptoms including bradykinesia and asymmetric rest tremor for four to five years. There is no other family history of Parkinson's disease or other movement disorders.

The patient had ingested MDMA twice a month for six months before the onset of symptoms of Parkinson's disease. His last ingestion was in January 2002. He had trismus, diaphoresis, and a sense of euphoria after taking MDMA. He said he did not use other illicit drugs. He drinks six or seven beers per week and has a smoking history of one to two pack-years. There is no history of use of neuroleptic drugs.

There has been controversy surrounding the case of MDMA-induced parkinsonism that was reported in 1999.²⁻⁵ In the case we report here, juvenile Parkinson's disease occurred in a man with a family history of Parkinson's disease. Ricaurte et al.¹

have speculated that cases of MDMA-induced parkinsonism are not commonly reported because Parkinson's disease is not clinically apparent until 70 to 80 percent of striatal-nigral dopamine is depleted. This patient may have had a genetic predisposition to Parkinson's disease, and symptoms may have developed earlier than they otherwise would have because of this environmental insult to his dopaminergic system.

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