Cessation of Paroxysmal Atrial Fibrillation during Acute Intravenous Propofol Administration

To the Editor—Paroxysmal atrial fibrillation is a reentrant arrhythmia for which prompt pharmacologic or electrical cardioversion is warranted.1 This treatment strategy has a number of benefits, including immediate alleviation of patient symptoms, avoidance of antiarrhythmic and anticoagulative therapy, and prevention of electrophysiologic remodeling, which is thought to contribute to the perpetuation of the arrhythmia.1 Initial pharmacologic cardioversion is frequently attempted, and electrical cardioversion is reserved for patients with hemodynamic symptoms or in whom pharmacologic cardioversion has been unsuccessful. For the latter cases, pretreatment with ibutilide given immediately before transthoracic electrical cardioversion has been reported to enhance the rate of conversion to sinus rhythm.2 We suggest that the use of propofol for sedation before electrical cardioversion can also improve the rate of successful cardioversion.

A 35-yr-old woman with a history of hyperthyroidism caused by Grave’s disease previously treated with carbimazole and currently controlled with propranolol (10 mg/8 h) was examined in the emergency department because of the acute onset of palpitations. Electrocardiogram showed atrial fibrillation with ventricular response of 140 beats/min, which was not present in previous electrocardiograms. No hemodynamic symptoms were present. Because atrial fibrillation persisted after intravenous infusion of amiodarone (1 g/24 h), this antiarrhythmic was stopped, and it was then decided to use electrical cardioversion. Propofol was elected for sedation. We administered a first bolus dose of propofol (1 mg/kg; total dose, 50 mg) through an antecubital vein, and after 90 s, ventricular response to atrial fibrillation slowed to 96 beats/min, which was immediately followed by a return to sinus rhythm. The patient only complained of transitory face flushing. No further arrhythmias were recorded, and she was discharged on sinus rhythm with a normal electrocardiogram.

Since then, we are aware of two additional cases (a 70-yr-old man and a 58-yr-old woman) observed in our institution in whom atrial fibrillation reverted in close temporal relationship with the sedative pretreatment of propofol (50 and 75 mg, respectively) while patients were waiting to receive electrical shock. In both patients, intravenous amiodarone (1 g) for 24 h failed to reverse the arrhythmia, and this drug had been tapered approximately 1 h before start of sedation with propofol.

Propofol is an anesthetic that causes a rapid induction of hypnosis and provides effective anesthesia. Because of its short half-life, it is frequently used for brief invasive procedures, including cardioversion. It causes occasional, often mild, hemodynamic effects that include hypotension and bradycardia.3 Based on animal experiences, proposed mechanisms for such negative dromotropism and chronotropism are slowing atrial rate and nodal conduction time, depressing Hiss-Purkinje system functions, and prolonging Wenckebach cycle length and effective refractory period.4,5 Accordingly, some investigators have suggested that propofol may impart antiarrhythmic protection to patients who are susceptible to supraventricular tachycardias,6,7 but no extensive clinical data reinforcing such hypothesis are currently available. In fact, we have only found two reports in which propofol reverted a severe supraventricular tachycardia and multiple premature atrial beats,8 respectively. Our cases represent the first reported in whom propofol seems to convert atrial fibrillation to sinus rhythm. If this association is further supported by other case reports and/or proved by clinical trials, propofol (at dose from 1 to 3 mg/kg) should be consequently considered as the first-choice sedative drug for electrical cardioversion to improve rates of success in treatment of patients with atrial fibrillation.

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(Accepted for publication October 28, 1999.)