Nicorandil Successfully Abolished Intraoperative Torsade de Pointes

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TORSADE DE POINTES (TdP), polymorphic ventricular tachycardia with QT interval prolongation, is one of the life-threatening arrhythmias and may occur in association with intracranial disorders.1-6 Nicorandil, a potassium-channel opener, has been used as a coronary vasodilator.7 Recently antiarrhythmic therapy using nicorandil has been attempted. Chinushi et al.8 reported that nicorandil suppressed TdP in patients with idiopathic long QT syndrome. We experienced a case in which TdP occurred during surgery and was clearly abolished by nicorandil.

Case Report

A 70-year-old woman weighing 50 kg was admitted for evaluation of Parkinson’s disease. She had ataxia for 1 year without other neurologic signs. Examination revealed a cerebellar syndrome with mild dysmetria and saccadic pursuit eye movements. Laboratory tests were within normal limits. The patient was premedicated with 0.5 mg atropine and 25 mg hydromorphone administered intramuscularly. She had an uneventful anesthesia with propofol and was intubated after the administration of vecuronium. Continuous electrocardiogram (ECG) (fig. 1A), 

Received from the Department of Anesthesiology, Jichi Medical School, and the Department of Anesthesiology, Juntendo University School of Medicine, Tokyo, Japan. Submitted for publication October 6, 1997. Accepted for publication January 30, 1998.

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Key words: Neurosurgical anesthesia; QT prolongation.

Anesthesiology, V 88, No 6, Jun 1998
**CASE REPORTS**

![Electrocardiogram Tracings](image)

**Discussion**

In this patient, nicorandil clearly abolished TdP during surgery. Nicorandil has potent vasodilating action and has been used as a coronary vasodilator. Nicorandil increases the outward potassium current of not only vascular smooth muscle but also of cardiac muscle. In recent studies suppression of this outward potassium current has been shown to produce early after depolarizations and TdP concomitantly with QT interval prolongation. Triggered activity originating from early after depolarizations has been proposed as a mechanism responsible for TdP. In one experimental model, potassium channel openers including nicorandil have been shown to suppress early after depolarizations and ventricular arrhythmias. In the clinical setting, nicorandil suppresses early after depolarizations and TdP in patients with idiopathic long QT syndrome. To our knowledge, however, the current report is the first to describe the usefulness of nicorandil to treat ventricular tachycardia or TdP during anesthesia.

There are several principles of treatment for ventricular tachycardia. Electrolyte abnormalities and hypothermia should be looked for and appropriately corrected. Although magnesium concentrations were not measured, other electrolyte imbalances were not present, and magnesium did not successfully treat her dysrhythmia. The patient remained normothermic. Another approach is to decrease adrenergic tone. Isoflurane is vasodilatory and can increase sympathetic tone. Sevoflurane is not associated with sympathetic activation. Therefore, in the current patient, isoflurane was discontinued and sevofoflurane was administered. Again this was unsuccessful. Although antiarrhythmic therapy for TdP begins with lidocaine or magnesium, these agents were ineffective in the current patient. β-Blockers have been used to treat TdP in congenital long QT syndrome. Conversely, β-stimulation may be effective and β-blockers contraindicated with acquired long QT syndrome. Therefore, β-blocker therapy was not attempted in our patient.

In controlling TdP in this patient, marked QT prolongation received attention. We postulated that this phenomenon reflected prolongation of the action potential duration. Therefore, we thought potassium channel activation may shorten action potential duration and be effective for this arrhythmia, and we decided to use nicorandil. The antiarrhythmic action of nicorandil is considered to be attributable not only to shortening action potential duration but also to hyperpolarization of the resting potential and suppression of automaticity. Therefore, nicorandil may be effective for treating arrhythmias originating from enhanced automaticity and reentry. In the current patient, nicorandil successfully terminated TdP without obvious hemodynamic change, providing additional evidence that potassium channel agonists may have a place in the treatment of TdP.

We postulate that the neurosurgical procedure may have caused QT prolongation in this patient. The patient had no history of arrhythmias or syncope attack. Preoperative electrocardiogram did not show obvious QT prolongation either. The patient did show marked...
QT prolongation during operation, however. The influence of preexisting hypertension was unknown. Because blood pressure and heart rate were unchanged, we did not consider that the QT prolongation was secondary to cardiac ischemia. Instead surgery may have locally distorted various autonomic nuclei of the brain stem and perhaps hypothalamus. Stimulation of these areas can cause a variety of sympathetic and parasympathetic responses. Intracranial mass lesions often are associated with changes in the electrocardiogram, including QT prolongation and TdP, and volatile anesthetic agents may prolong the QT interval. At least some contribution of volatile anesthetic agents to the prolongation of the QT interval interval can not be excluded in this patient.

Ventricular tachycardia or TdP is an infrequent complication during anesthesia. Early treatment is of paramount importance. Nicorandil may, at least in part, play an important role in the treatment of TdP with long QT interval during anesthesia, especially when lidocaine and magnesium are not effective.

References