Conversion of Paroxysmal Supraventricular Tachycardia due to a Concealed Extranodal Pathway with Intravenous Bolus of Lidocaine*

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In 1969, in two cases of paroxysmal supraventricular tachycardia complicating the Wolff-Parkinson-White syndrome, Dye1 reported conversion of the tachycardia to sinus rhythm with intravenous administration of lidocaine. In a subsequent study, Rosen and co-workers2 provided a pharmacologic basis for this conversion, by demonstrating that lidocaine depressed conduction in anomalous pathways. This demonstration suggested that the mechanism of conversion of paroxysmal supraventricular tachycardia was an increase in the refractoriness of an anomalous pathway, resulting in interruption of circus movement. In a recent report, Josephson and co-workers3 demonstrated marked slowing of ventricular response with administration of lidocaine in a patient with paroxysmal atrial fibrillation and pre-excitation. This slowing reflected an increase in the refractoriness of an anomalous pathway (total failure of anomalous pathway), so that antegrade conduction during atrial fibrillation occurred only via the atrioventricular node.

In the present study, we report conversion of paroxysmal supraventricular tachycardia in a patient with a known concealed Kent’s bundle (unidirectionally conducting extranodal anomalous pathway).4-9 The significance of this conversion is discussed.

Case Report

The patient was a 12-year-old boy admitted on May 15, 1976 with an attack of paroxysmal supraventricular tachycardia. He had a history of recurrent paroxysmal supraventricular tachycardia. Intervening electrocardiograms between attacks revealed normal sinus rhythm, normal P-R intervals, and narrow QRS complexes of normal morphology. Electrophysiologic study in April 1976 had revealed the presence of a retrogradely conducting (concealed) left posterior extranodal pathway.4-9 Paroxysmal supraventricular tachycardia reflected easily inducible circus movements utilizing antegrade conduction via the normal pathway and retrograde conduction via the left concealed extranodal pathway. Following electrophysiologic studies, the patient has been maintained on therapy with digoxin (0.25 mg/day) and propranolol (120 mg/day).

The attack of paroxysmal supraventricular tachycardia necessitating this admission began at approximately 8:00 PM on May 14, 1976 and did not respond to vagal maneuvers. After being at home for six hours without conversion, the patient was admitted to the University of Illinois Hospital at 2:00 AM on May 15. At the time of admission, the patient was noted to have narrow QRS paroxysmal supraventricular tachycardia at a rate of 150 beats per minute. Vagal maneuvers, including eliciting the diving reflex, did not result in conversion of paroxysmal supraventricular tachycardia. Because the arrhythmia was well tolerated, the patient was put to bed in hopes that spontaneous conversion would occur during the night. By noon the next day (May 15), it was apparent that spontaneous conversion would not occur. The patient was then given a 75-mg bolus of lidocaine intravenously. Approximately one minute after injection of the bolus, the tachycardia converted to normal sinus rhythm (Fig 1).

Discussion

The circus movement in most cases of paroxysmal supraventricular tachycardia complicating the Wolff-Parkinson-White syndrome consists of the normal pathway (usually antegrade conduction), anoma-
luous pathway (usually retrograde conduction), and final common pathways (atrium and ventricle). The acute attack of paroxysmal supraventricular tachycardia may be converted by increasing the refractoriness of either the atrioventricular node or anomalous pathways, so that the circus movement is not maintained. For example, atrioventricular nodal refractoriness can be increased with vagal maneuvers or with administration of cardiac glycosides or propranolol. These methods of therapy can be used singly or in combination. The refractoriness of the anomalous pathway can be increased by administration of quinidine, procaine amide, and lidocaine.5,10

The present case is an example of the use of lidocaine for management of acute paroxysmal supraventricular tachycardia complicating preexcitation. In our patient, circus movement tachycardias involved the normal pathway (antegrade) and a concealed extranodal anomalous pathway (retrograde). The latter pathway had ability only for retrograde conduction and was, thus, not detectable on the surface ECG.4,9 The delineation of this pathway was dependent upon electrophysiologic studies.4,9 Apparently the concealed pathway had similar pharmacologic response to anomalous pathways that are antegrade manifest, in that it was depressed with administration of lidocaine, allowing conversion of paroxysmal supraventricular tachycardia.

The present report has direct clinical implications. We believe that lidocaine is a suitable drug for treatment of circus movement paroxysmal supraventricular tachycardia complicating the Wolff-Parkinson-White syndrome and for paroxysmal supraventricular tachycardia involving concealed extranodal anomalous pathways. In the immediate management of paroxysmal supraventricular tachycardia in these patients, lidocaine has several advantages in comparison to other drugs, these being minimal toxicity, rapid onset of action, and rapid clearance after intravenous administration. If paroxysmal supraventricular tachycardia fails to convert with administration of lidocaine, other drugs can be used almost immediately.

In most clinically encountered cases of paroxysmal supraventricular tachycardia, the mechanism of arrhythmia is not readily apparent, the delineation of mechanism being dependent upon electrophysiologic study.11 Intravenous administration of lidocaine could be utilized in such cases without delineation of mechanism. In those cases due to atrioventricular nodal reentrance, lidocaine would probably be ineffective. In those cases utilizing retrograde conduction over an anomalous pathway, administration of lidocaine could result in conversion to sinus rhythm.

References


ANNOUNCEMENTS

Thrombosis: Diagnosis, Prevention and Treatment

Mount Sinai Medical Center of Greater Miami will present the postgraduate course, Thrombosis: Diagnosis, Prevention and Treatment, February 7-9 at the Sonesta Beach Hotel, Key Biscayne. Co-sponsors are the American Heart Association, Florida Heart Association and Heart Association of Greater Miami. For information, contact Ms. Miniver S. Reed, CME Coordinator, Mount Sinai Medical Center, 4300 Alton Road, Miami Beach 33140.

Advances in Cardiology

The Foundation for Cardiovascular Research and the Hospital of the Good Samaritan will present a three-day symposium on Advances in Cardiology in Palm Springs, March 14-16. For information, contact the Foundation, Ten Congress Street, Suite 203, Pasadena 91105.

Fifth Annual Taos Lung Disease Symposium

The Fifth Annual Taos Lung Disease Symposium will be held February 25-27 at the Kachina Lodge, Taos, New Mexico. Sponsor is the New Mexico Chapter, American Thoracic Society. For information, write the New Mexico Thoracic Society, 216 Truman Avenue NE, Albuquerque 87108.