mented as causes of angina in patients who had normal coronary arteriograms.[7]

Unfortunately, coronary vasodilatory flow reserve before or during this study was not measured. One would anticipate significant abnormality of the vasodilatory response. Syndrome X, a constellation of findings of chest pain, myocardial ischemia, ECG abnormalities, and normal coronary arteries, is the most likely diagnosis in this particular patient. This case emphasizes that cardiologists who perform ergonovine tests should be aware that ECG alterations, particularly those of an atypical response, can occur in a patient with normal coronary arteries. Prolonged ischemia may be difficult to manage, and, although absent in this patient, adverse ischemic events may occur.

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Effect of verapamil on infranodal conduction in patients with baseline His-Purkinje conduction delay

Shimon Rosenheck, MD, Joao Sousa, MD, Hugh Calkins, MD, Alan H. Kadish, MD, and Fred Morady, MD. *Ann Arbor, Mich.*

Several experimental and clinical studies have demonstrated that verapamil does not depress infranodal conduction when baseline conduction is normal.[1,2] The absence of a depressive effect of a calcium channel blocking agent on infranodal conduction is consistent with the fact that depolarization of normal His-Purkinje fibers depends on sodium channels.[3] However, in damaged His-Purkinje fibers, as in fibers suspended in sodium-free solution, the action potential has a lower resting potential, slower depolarization velocity, and a longer duration, suggesting the use of slow calcium channels instead of fast sodium channels.[4] Therefore it is possible that verapamil might depress conduction in the His-Purkinje system when baseline conduction is abnormal. Although a recent study[5] demonstrated that verapamil has no effect on rate-dependent bundle branch block, no studies have investigated the direct effect of verapamil on the diseased His-Purkinje system. Therefore the purpose of this study was to evaluate the effect of verapamil on infranodal conduction in patients with baseline conduction delay in the His-Purkinje system.

Five patients with prolonged infranodal conduction were studied. There were four men and one woman, and their mean age was 69 ± 8 years (±standard deviation). Four patients had no structural heart disease and one had coronary artery disease; their mean left ventricular ejection fraction was 0.52 ± 0.13. Two patients had a left bundle branch block, two had a right bundle branch block and left anterior hemiblock, and one had a right bundle branch block and left posterior hemiblock. The baseline HV interval was 70 to 170 msec (mean 98 ± 42 msec). After informed consent was obtained, each patient underwent an electrophysiology test for evaluation of syncope or ventricular tachycardia. Three quadripolar electrode catheters were positioned in the right atrium, the right ventricular apex, and across the tricuspid valve to record the His bundle electrogram. Leads I, III, V_1 and the intracardiac electrograms were recorded with a Siemens-Elema Mingograf 7 recorder (Siemens Elema AB, Solna, Sweden) at a paper speed of 100 mm/sec. The AH and HV intervals and the duration of the His bundle depolarization were measured at atrial pacing cycle lengths of 700, 600, and 500 msec. The atrioventricular (AV) block cycle length was determined by incremental atrial pacing in steps of 10 msec. At each pacing cycle length, measurements were made from five consecutive paced beats after at least 20 seconds of pacing, and the means of the measurements were used for analysis. Measurements were made in the baseline state and 3 minutes after the infusion of consecutive doses of 10, 5, and 5 mg of verapamil. These doses of verapamil were infused over 3 to 5 minutes and were separated by 5-minute intervals. Mixed model analysis of variance was used for statistical analysis.

The effects of verapamil are described in Table I. The 10, 15, and 20 mg cumulative dosages of verapamil significantly prolonged the AV node block cycle length ($p < 0.01$) and AH interval ($p < 0.05$), but had no effect on the HV interval or His duration. A decrease in the atrial pacing cycle length resulted in a significant increase in the AH interval ($p < 0.01$) and had no effect on the HV interval and His duration. Three patients had infranodal block in the baseline state. After verapamil infusion, these three patients either developed AV nodal Wenckebach block at a

From the Department of Internal Medicine, Division of Cardiology, University of Michigan Medical Center.

Reprint requests: Fred Morady, MD, Division of Cardiology, University of Michigan Medical Center, 1500 E. Medical Center Dr., UB B1 F245 Box 0022, Ann Arbor, MI 48109-0022.

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Table I. The effect of 10, 15, and 20 mg of verapamil injected intravenously on the sinus cycle length, AH and HV intervals, His deflection duration, and AV nodal block cycle lengths (in milliseconds)

			Verapamil		
	PCL	*Baseline*	*10 mg*	*15 mg*	*20 mg*
SCL		786 ± 101	782 ± 88	772 ± 65	792 ± 60
AH	Sinus	112 ± 31	130 ± 55*	140 ± 70*	125 ± 63*
	700	123 ± 26	136 ± 46*	149 ± 52*	153 ± 46*
HV	Sinus	98 ± 42	87 ± 21	88 ± 13	85 ± 10
	700	87 ± 12	103 ± 22	108 ± 43	107 ± 31
His	Sinus	19 ± 2	23 ± 6	23 ± 6	23 ± 5
	700	21 ± 6	25 ± 6	23 ± 5	25 ± 6
AVN BCL I		464 ± 136	500 ± 89*	530 ± 110*	530 ± 79*
AVN BCL II		402 ± 116	436 ± 94	454 ± 95	468 ± 68

PCL, Pacing cycle length; SCL, sinus cycle length; AVN BCL I, AV node Wenkebach block cycle length; AVN BCL II, AV node 2:1 block cycle length.
*$p < 0.05$, compared with baseline.

cycle length longer than the infranodal block cycle length, or had no change in the pacing cycle length at which the infranodal block occurred.

The present study demonstrates that verapamil has no effect on the severely impaired His-Purkinje system in human subjects. In contrast to these results, experimental studies have demonstrate a depressant effect of verapamil on conduction in the damaged His-Purkinje system.[6] However, these experimental studies were performed using in vitro preparations that may not have accurately simulated in vivo conditions. For example, a Na^+-free tissue bath was used.[6] The present study demonstrates that the use of verapamil in patients with a prolonged HV interval is safe, even when the HV interval prolongation is marked.

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Fatal verapamil toxicity and hypokalemia

Ricci A. Minella, MD, and Douglas S. Schulman, MD
Pittsburgh, Pa.

Verapamil is a commonly prescribed calcium channel blocking drug. The predominant mechanism of its actions and side effects is mediated through slow calcium channel blockade. Although not common, severe verapamil overdose has been reported.[1] We report a case of fatal verapamil overdose that demonstrates the salient features of acute verapamil toxicity.

The patient was a previously healthy 33-year-old white man who presented 20 minutes after ingesting 52 80 mg verapamil tablets (4.16 gm) in a suicide attempt. The medicine was prescribed for a family member. Initially, he was alert with a pulse of 103 beats/min and a blood pressure of 110/70 mm Hg. Initial laboratory values of note seen within 20 minutes of presentation included a potassium level of 2.8 mEq/dl, a glucose level of 252 mg/dl, a calcium level of 9.9 mEq/dl, a magnesium level of 1.4 mEq/dl, a verapamil level of 2060 ng/ml (50 to 200 ng/ml therapeutic), and a norverapamil level of 523 ng/ml (50 to 200 ng/ml). He was treated with gastric lavage, syrup of ipecac, and activated charcoal. His heart rate and blood pressure began to decrease and he developed first-degree atrioventricular (AV) block (Fig. 1). Within 25 minutes, his blood pressure had decreased to 97/50 mm Hg and his heart rate had slowed to 61 beats/min. The patient was given 3 ampules of (1 gm/amp) calcium chloride, 1 mg atropine, 0.4 mg naloxone hydrochloride and

From the Department of Medicine, Division of Cardiology, University of Pittsburgh School of Medicine.

Reprint Requests: Douglas S. Schulman, MD, University of Pittsburgh, School of Medicine, 376N Scaife Hall, Pittsburgh, PA 15261.

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