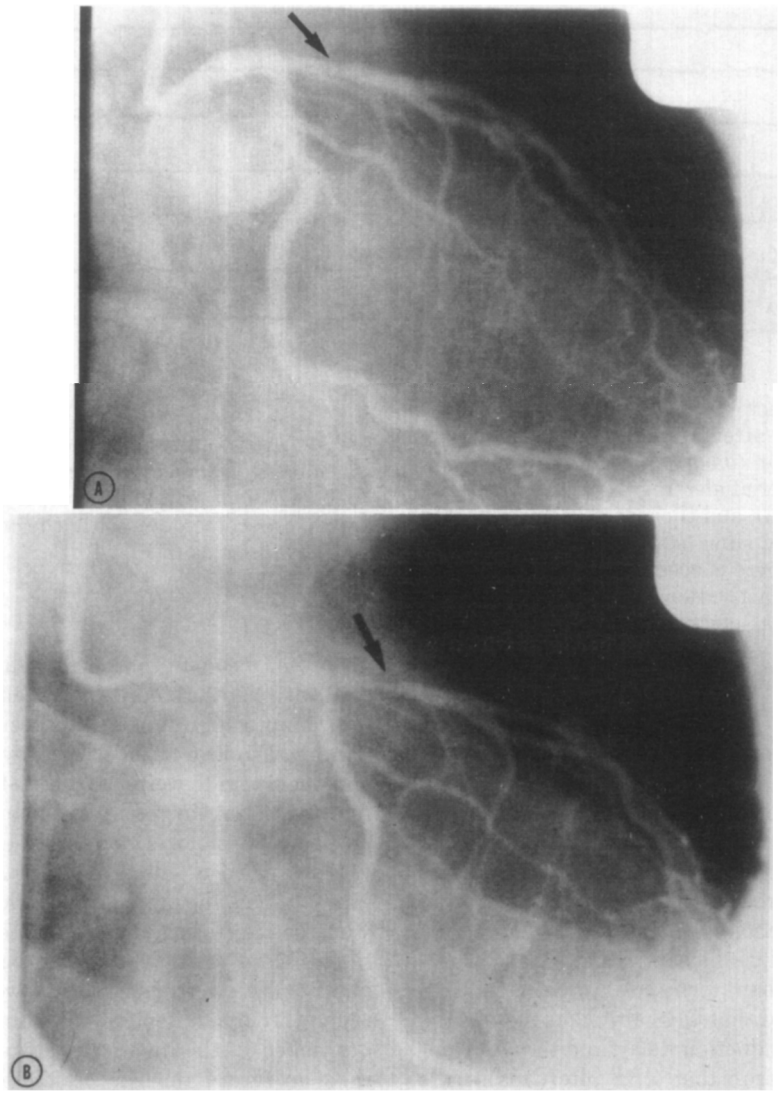

Fig. 2. *A*, Cineangiogram of the left coronary artery under basic circumstances. *Arrow* indicates slight spindle-shaped narrowing in the proximal part of the left anterior descending artery. *B*, Cineangiogram of the left coronary artery after induced hyperventilation. Arrow indicates 70% stenosis in the proximal part of the left anterior descending artery.

this patient we learned that apparently transient hyperventilation-induced coronary artery spasm, complicated by potentially serious arrhythmia, can occur in otherwise asymptomatic persons.

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Observations on QRS alternans in a patient with two types of narrow QRS tachycardia

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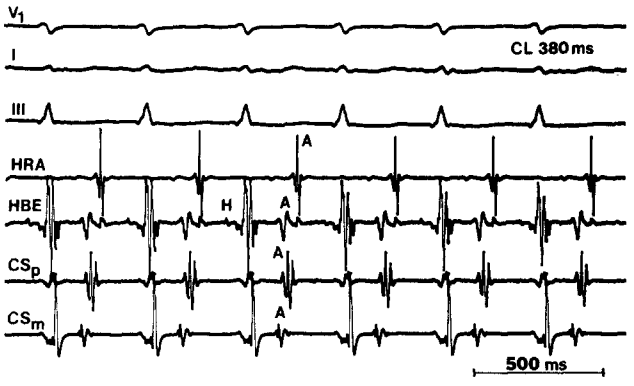

Fig. 1. Orthodromic reciprocating tachycardia, cycle length 380 msec, induced after the administration of 1 gm of procainamide. *From top to bottom* are ECG leads V_1, I, and III, the high right atrial electrogram *(HRA)*, the His bundle electrogram *(HBE)*, and the electrograms reported at the proximal coronary sinus *(CS_p)* and mid coronary sinus *(CS_m)*. Note that there is eccentric atrial activation, with the earliest atrial activation recorded at the mid coronary sinus. *A* = atrial electrogram; *H* = His bundle depolarization.

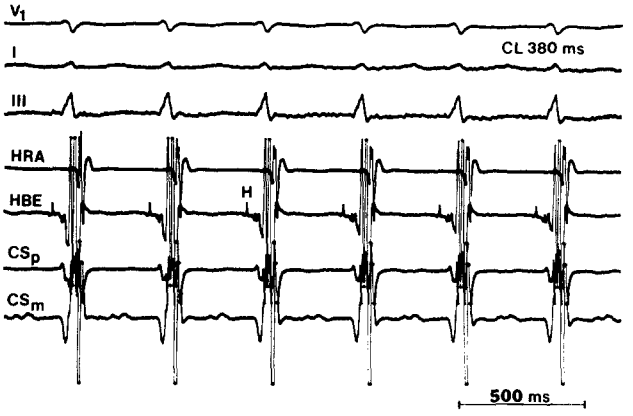

Fig. 2. Atrioventricular nodal reentrant tachycardia, cycle length 380 msec, induced after the administration of 1000 mg of procainamide. Note that the atrial electrograms are coincident with the ventricular electrogram. Abbreviations as in Fig. 1.

QRS alternans during narrow complex tachycardias has been demonstrated to have a high degree of specificity (96%) for orthodromic reciprocating tachycardia utilizing an accessory atrioventricular connection.[1] The presence of QRS alternans has been found to be helpful in differentiating orthodromic reciprocating tachycardia from other types of narrow complex tachycardia.[2] Green et al.[1] reported that tachycardias that displayed QRS alternans generally had faster rates than tachycardias without QRS alternans. They suggested that QRS alternans may be the result of a functional conduction delay in alternate complexes. However, the reason that QRS alternans occurs more commonly in orthodromic reciprocating tachycardia than in other types of tachycardia is unclear. Green et al.[1] postulated that patients with orthodromic reciprocating tachycardia may have anatomically or functionally different conduction systems than patients with other types of tachycardia, and that these differences may predispose patients with orthodromic reciprocating tachycardia to changes in conduction at faster heart rates. However, in their study the different types of tachycardia occurred in different patients, and it was therefore not possible to assess the importance of tachycardia mechanism independent of differences in the atrioventricular conduction system or heart rate. In this case report we describe a patient who had orthodromic reciprocating tachycardia and atrioventricular nodal reentrant tachycardia, both of which had the same rate. This patient provided the unique opportunity to assess the importance of the tachycardia mechanism in the generation of QRS alternans, independent of specific characteristics of the atrioventricular conduction system and heart rate.

The patient was a 35-year-old woman with a 1-year history of frequent episodes of rapid palpitations associated with lightheadedness. The episodes generally were 1 to 2 minutes in duration and occurred up to three times per day. Empiric trials of therapy with nadolol, atenolol, and verapamil were unsuccessful. The patient was referred to the University of Michigan Medical Center for an electrophysiologic study. Physical examination and the ECG revealed no abnormalities. After informed consent was obtained, the patient underwent an electrophysiologic study in the fasting, unsedated state, 3 days after discontinuation of therapy with antiarrhythmic drugs. Four quadripolar electrode catheters were inserted percutaneously through a femoral or subclavian vein and were positioned in the high right atrium, across the tricuspid valve to record the His bundle electrogram, within the coronary sinus, and against the apex of the right ventricle. Leads V_1, I, and III, the high right atrial electrogram, the His bundle electrogram, and the electrograms recorded in the coronary sinus were displayed on an oscilloscope and were recorded on photographic paper with an Electronics for Medicine VR-16 recorder at a paper speed of 100 to 150 mm/sec. In addition, a 12-lead ECG recording of all induced tachycardias was obtained. Program stimulation was performed with a programmable stimulator (Bloom Associates, Ltd., Narberth, Pa.) with stimuli 2 msec in duration and twice diastolic threshold in intensity.

The baseline atrioventricular nodal conduction time was 70 msec and the baseline infranodal conduction time was 35 msec. During incremental right and left atrial (coronary sinus) pacing, there was no ventricular preexcitation and atrioventricular nodal Wenckebach block occurred at a paced cycle length of 300 msec. Programmed atrial stimulation with a single atrial extrastimulus during a basic drive cycle length of 500 msec demonstrated the presence of dual atrioventricular nodal pathways. At a coupling interval of 280 msec, orthodromic reciprocating

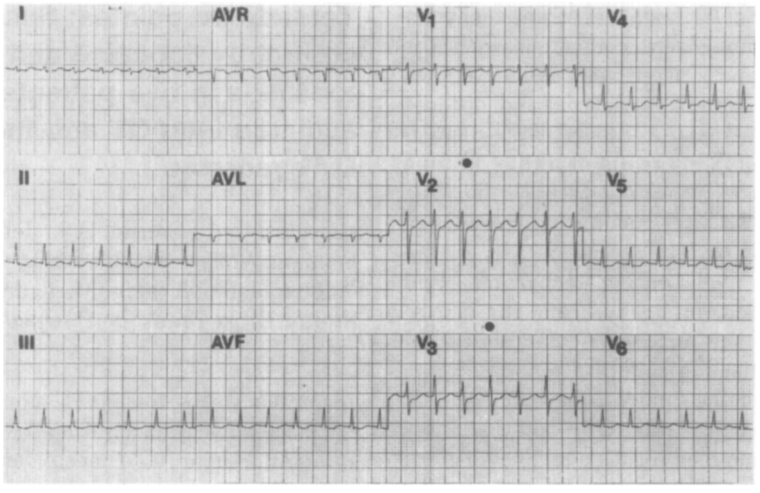

Fig. 3. A 12-lead ECG recording during the same episode of orthodromic reciprocating tachycardia shown in Fig. 1. Note the presence of QRS alternans in lead V_3.

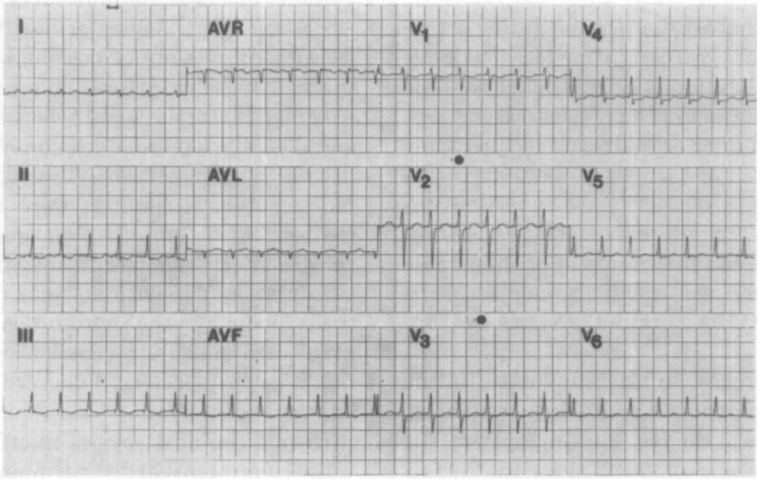

Fig. 4. A 12-lead ECG recording during the same episode of atrioventricular nodal reentrant tachycardia shown in Fig. 2. QRS alternans is not present.

tachycardia was induced; mapping demonstrated that a left posterolateral accessory atrioventricular connection was the retrograde limb of the reentry circuit. The cycle length of the tachycardia was 310 msec and QRS alternans was not present. During the tachycardia, a paced ventricular complex coincident with the His bundle depolarization resulted in advancement of the atrial electrograms without a change in the sequence of atrial activation. During sinus rhythm, ventricular pacing resulted in the same pattern of eccentric atrial activation, with the earliest atrial electrogram being recorded at the mid coronary sinus. After the intravenous administration of 1 gm of procainamide (serum procainamide concentration 6.8 mg/L), the atrioventricular nodal conduction time was 80 msec and the infranodal conduction time was 40 msec. Neither incremental atrial pacing nor programmed stimulation with a single atrial extrastimulus resulted in the induction of a tachycardia. Ventricular pacing at a cycle length of 350 msec resulted on two occasions in the induction of orthodromic reciprocating tachycardia (Fig. 1), and on another two occasions in the induction of atrioventricular nodal reentrant tachycardia (Fig. 2). Both tachycardias had a cycle length of 380 msec. The interval between consecutive His bundle depolarizations was constant at 380 msec during both types of tachycardia. During orthodromic reciprocating tachycardia there was eccentric atrial activation, with the shortest ventriculoatrial interval (160 msec) recorded at the mid coronary sinus; a paced ventricular complex coincident with the His bundle depolarization resulted in advancement of the atrial electro-

grams. During atrioventricular nodal reentrant tachycardia, the atrial electrograms were coincident with the ventricular electrogram, and a paced ventricular complex coincident with the His bundle depolarization did not advance the atrial electrograms. A 12-lead ECG demonstrated the presence of QRS alternans in lead V_3 during episodes of orthodromic reciprocating tachycardia (Fig. 3), and the absence of QRS alternans during episodes of atrioventricular nodal reentrant tachycardia (Fig. 4).

In both orthodromic reciprocating tachycardia and atrioventricular nodal reentrant tachycardia, the ventricles are depolarized via the atrioventricular node–His-Purkinje axis. In the patient in this case report, despite ventricular activation over the same atrioventricular conduction system at an identical rate, QRS alternans was present during orthodromic reciprocating tachycardia but not during atrioventricular nodal reentrant tachycardia. This finding demonstrates that the specificity of QRS alternans for orthodromic reciprocating tachycardia may be independent of specific characteristics of the atrioventricular conduction system and also independent of the tachycardia rate.

Green et al.[1] reported that QRS alternans during orthodromic reciprocating tachycardia persisted in some patients after the administration of drugs that slowed the tachycardia rate. In the patient in the present case report, QRS alterans appeared during orthodromic reciprocating tachycardia *only* after the cycle length of the tachycardia had been lengthened from 310 to 380 msec by procainamide. The appearance of QRS alternans despite to slower tachycardia rate after the administration of procainamide suggests that QRS alternans was related to depression in conduction through the His-Purkinje system by procainamide.

In the absence of differences in rate, differences in the pathway of anterograde ventricular activation, or intrinsic conduction characteristics of the His-Purkinje system, the explanation for the presence of QRS alternans during orthodromic reciprocating tachycardia but not during atrioventricular nodal reentrant tachycardia is unclear. However, it should be noted that both tachycardias were induced by ventricular pacing. Therefore, a possible explanation is that there was a difference in the degree of retrograde penetration of the His-Purkinje system by the ventricular depolarizations that induced the tachycardias. Whether the same specificity of QRS alterans for orthodromic reciprocating tachycardia would have been observed had the tachycardias been inducible by atrial stimulation remains unknown.

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Torsade de pointes: Successful acute control by lidocaine and chronic control by tocainide in two patients—one each with acquired long QT and the congenital long QT syndrome

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In patients with torsade de pointes (polymorphous ventricular tachycardia associated with a preexisting long QT interval), there is a need for treatment alternatives because some treated patients have recurrent symptoms and/or torsade de pointes (TDP).[1-3] We present two patients who had TDP, one with acquired and one with congenital long QT syndrome (LQTS). Each patient successfully responded to lidocaine and subsequently to chronic tocainide, an oral lidocaine-derivative drug.

Patient No. 1. A 14-year-old, 90 kg white male presented with complaints of fatigue, headache, and chest pain during football practice. A paternal uncle also had an irregular rhythm at age 15; he was treated with quinidine and died a few days later in an automobile accident while driving by himself. On physical examination of patient No. 1, positive findings were limited to the irregular rhythm. ECG and 24-hour ambulatory ECG (Holter) showed alternating morphology (bidirectional) ventricular tachycardia (VT) with no sinus rhythm present. Between the episodic VT, the rhythm was predominantly low right atrial with multiform ventricular premature beats (VPB) in a bigeminy pattern. Echocardiogram showed a dilated left ventricle (LV).

During the next several months, ineffective control of ventricular tachycardia was found during trials at therapeutic serum concentrations of phenytoin, digoxin, verapamil, digoxin plus verapamil, digoxin plus propranolol, digoxin plus mexiletine, and digoxin plus tocainide. Therefore, the patient was hospitalized to begin oral amiodarone therapy (loading dose, 1200 mg/dl). After 2 days, the first nonbigeminal sinus rhythm was noted and after 4 days, only occasional multiform VPBs occurred (Fig. 1). However, the QT_c (Bazett) was prolonged to 0.52 second. After 7 days, the patient had sudden cardiac arrest due to TDP (Fig. 1). Successful control was accomplished after multiple DC cardioversions and by adding continuous lidocaine infusion because atrial pacing and isoproterenol infusion were unsuccessful. Serum potassium was normal. Because of the lidocaine success, tocainide was started. Two days later, amiodarone also was

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