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# Paroxysmal Fascicular Tachycardia and Ventricular Tachycardia Due to Mechanical Stimulation by a Mitral Valve Prosthesis

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**Electrophysiologic studies were performed in a woman who had two varieties of paroxysmal wide QRS tachycardia after mitral valve replacement with a Starr-Edwards prosthesis. One tachycardia originated in the left anterior fascicle; QRS complexes were 100 ms wide and resembled right bundle branch block with left posterior fascicular block, and a His bundle potential preceded each QRS by an interval of 20 ms (compared with 50 ms during sinus rhythm). The other tachycardia originated in the left ventricle. Clinical and echocardiographic observations suggested that the tachycardias were caused by mechanical stimulation of the interventricular septum by the mitral prosthesis.**

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Wide QRS rhythms induced by digitalis in canines frequently originate in the left bundle branch (1). These rhythms are characterized by QRS complexes that resemble right bundle branch block, and by almost simultaneous onset of His bundle and ventricular depolarizations. The His bundle studies performed in human patients have suggested that wide QRS escape rhythms that occur in the setting of acute myocardial infarction (2,3), or accelerated wide QRS rhythms related to digitalis (4), occasionally arise in one of the divisions of the left bundle branch (fascicular rhythms).

We describe a patient who had rapid, paroxysmal left anterior fascicular tachycardia and ventricular tachycardia after mitral valve replacement with a Starr-Edwards prosthesis. Clinical and echocardiographic observations suggested that the tachycardias were caused by mechanical stimulation of the interventricular septum by the mitral prosthesis.

## Case Report

A 34 year old, medium-sized (167 cm, 50 kg) woman was admitted to the University of Illinois Hospital for mitral valve replacement. She had had acute rheumatic fever at age 20 years, and had had progressive dyspnea on exertion for 4 years. Diagnostic cardiac catheterization revealed severe mitral regurgitation, a dilated and hyperdynamic left ventricle, a dilated left atrium and normal coronary arteries. Treadmill exercise (Naughton-Balke protocol) was terminated after 14 minutes (peak heart rate 165 beats/min) because of fatigue; there were no arrhythmias. On January 6, 1983, the patient underwent mitral valve replacement with a 32 mm Starr-Edwards valve (model 6120). The early postoperative course was complicated only by a transient episode of paroxysmal atrial fibrillation.

Beginning on the 10th postoperative day, the patient experienced brief attacks of palpitation, and paroxysmal tachycardia was documented (see later). Continuous electrocardiographic monitoring revealed that the attacks occurred in association with abrupt standing or walking in the hallway. During each of two treadmill exercise tests, repetitive bursts of nonsustained (duration 1 to 8 seconds) tachycardia occurred at 7 minutes (peak sinus heart rate 120 beats/min). The bursts ceased within 2 minutes after the termination of exercise. Tachycardia could also be reproduced by having the patient inspire deeply. The occurrence of paroxysmal tachycardia during inspiration was not affected by performance of carotid sinus massage or intravenous administration of 2.0 mg of atropine.

**Review of electrocardiograms.** Electrocardiograms during sinus rhythm were normal: QRS duration was 80 ms and frontal plane axis was +30° (Fig. 1A). Two distinct varieties of paroxysmal tachycardia were documented. The more common tachycardia (tachycardia 1) was characterized by ventricular rates of 150 to 210 beats/min, QRS complexes

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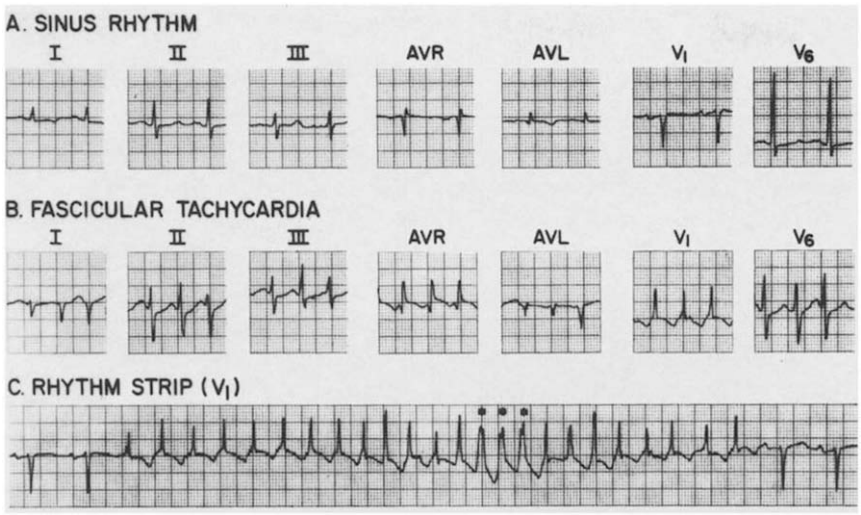


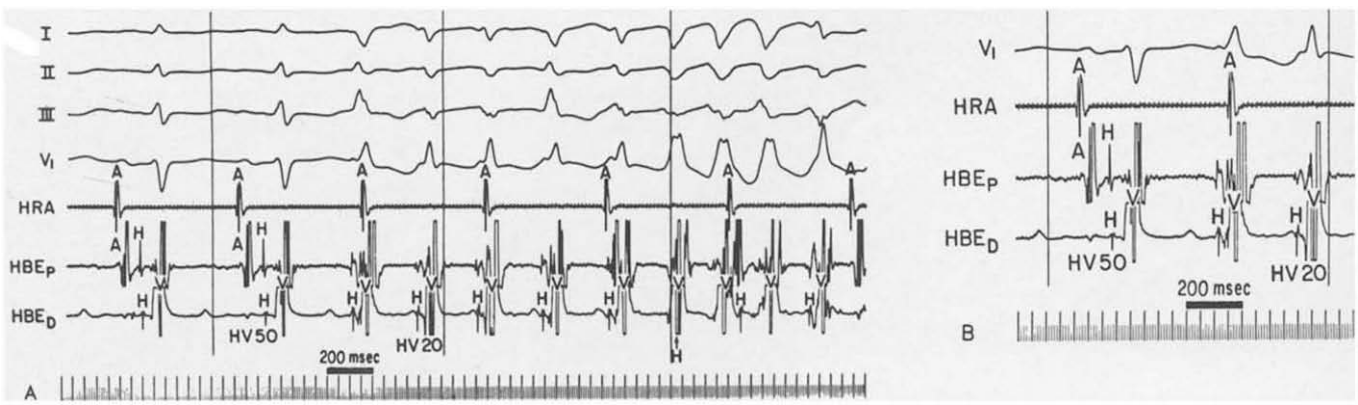

**Figure 1.** Surface electrocardiograms. **A,** Sinus rhythm; **B,** fascicular tachycardia; **C,** rhythm strip recorded during deep inspiration. There are three beats of ventricular tachycardia (**asterisks**) in the midst of an 8 second episode of fascicular tachycardia.

that resembled incomplete right bundle branch block with left posterior fascicular block (QRS duration 100 ms, frontal plane axis 100 to 180°) and atrioventricular dissociation (Fig. 1B). These features suggested that this tachycardia originated in the left anterior fascicle. In the midst of some episodes of tachycardia 1, there were short bursts (as many as seven beats) of a different tachycardia (tachycardia 2), which was characterized by ventricular rates of 210 to 240 beats/min and QRS complexes resembling complete right bundle branch block (QRS duration 140 ms)(Fig. 1C). These features suggested a diagnosis of ventricular tachycardia.

**Electrophysiologic studies.** After informed written consent was obtained, and with the patient in the postabsorptive, unmedicated state, electrophysiologic studies were performed using standard techniques. During sinus rhythm (rate 80 beats/min), basic intervals were: PA 30 ms, AH 164 ms and HV 53 ms. During programmed atrial stimulation, QRS complexes were narrow (no aberrancy). Ventricular pacing at rates slightly faster than sinus rate revealed that ventriculoatrial conduction was absent. Programmed ventricular stimulation was performed in an attempt to induce paroxysmal tachycardia. However, with single and double extrastimulus testing, and burst pacing through a rate of 250 beats/min from the right ventricular apex and then the outflow tract, there was a maximum of two ventricular echo beats that did not resemble the configuration of either tachycardia.

*Infusion of isoproterenol, 2 μg/min, replicated both tachycardias (Fig. 2A).* During tachycardia 1, there was atrioventricular dissociation. However, a His bundle potential

**Figure 2.** Recordings during infusion of isoproterenol. Shown are surface leads I, II, III and $V_1$, as well as high right atrial (HRA), proximal His bundle ($HBE_P$) and distal His bundle ($HBE_D$) electrograms. Time lines are at bottom. **A,** Eleven ventricular beats (V) are shown. The first two beats are conducted sinus rhythm; the HV interval is 50 ms. Beats 3 to 7 are fascicular tachycardia; there is dissociation between the atria (A) and the His bundle (H), and the HV interval is 20 ms. Beats 8 to 10 are ventricular tachycardia; there is dissociation between the His bundle and the ventricles, and between the His bundle and the atria (double dissociation). The last beat is of fascicular origin. **B,** Ventricular beats 2 to 4 have been magnified to increase detail. During fascicular tachycardia (beats 3 and 4) His bundle depolarization is inscribed at $HBE_P$ before $HBE_D$. Thus, it is likely that depolarization at $HBE_D$ reflects antegrade conduction in fascicular tissue (distal His bundle or proximal right bundle branch) that is insulated from the left anterior fascicle.



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preceded each QRS complex by 20 ms (compared with 50 ms during sinus rhythm) (Fig. 2B). Tachycardia 2 occurred in the midst of tachycardia 1. During tachycardia 2, there was dissociation between QRS complexes and His bundle potentials, as well as between His bundle potentials and atrial depolarizations, that is, double dissociation (Fig. 2A).

**Other observations.** *Two-dimensional echocardiography* revealed that there was contact between the cage of the prosthetic valve and the base of the interventricular septum during ventricular systole (Fig. 3). During deep inspiration, the left ventricular chamber size was decreased in the apical two-chamber view, and contact between the struts of the prosthesis and the basal septum was increased.

*Serial treadmill exercise tests* were performed to assess the effectiveness of antiarrhythmic drugs. However, both of the tachycardias occurred despite the oral administration of 1 g of procainamide every 4 hours, 80 mg of propranolol every 6 hours or 80 mg of verapamil every 6 hours.

The patient was discharged from the hospital without medication. Over the subsequent year she had progressively fewer attacks of palpitation. At present she can mop the floor or dance without symptoms.

## Discussion

**Fascicular tachycardias.** Damato et al. (1) studied digitalis-induced wide QRS rhythms in dogs by recording bipolar electrograms from the His bundle, the right bundle branch and left bundle branch, and found that these rhythms frequently originated in the left bundle branch. From this origin, there was anterograde conduction to the left ventricle, and retrograde conduction to the His bundle followed by anterograde conduction over the right bundle branch to the right ventricle. On the basis of these observations, a fascicular origin can be suspected when a wide QRS rhythm resembles incomplete bundle branch block (2). The diagnosis can be confirmed by demonstration that the relation between the onset of His bundle depolarization and the onset of ventricular depolarization is intermediate between the HV interval of normally conducted beats and the VH interval expected with ventricular beats (2).

His bundle recordings have suggested that occasional clinical rhythms originate in one of the divisions of the left bundle branch. Castillo et al. (2) and Massumi et al. (3) studied patients with acute myocardial infarction who had escape rhythms resembling incomplete right bundle branch block with right or left axis deviation; HV intervals ranged from −5 to +5 ms, suggesting that the rhythms originated in the left anterior fascicle or the left posterior fascicle. Cohen et al. (4) reported on a patient who had an accelerated wide QRS rhythm (rate 88 beats/min) while receiving digitalis in the setting of hypokalemia. The QRS complexes (duration 100 ms) resembled incomplete right bundle branch block with left axis deviation and the HV interval was 10 ms, suggesting an origin in the left posterior fascicle. Goolsby and Oliva (5) reported on a patient who had recurrent, paroxysmal, sustained tachycardia (rate 150 beats/min) after an inferior infarction. Although they suggested that the tachycardia originated in the left anterior fascicle, the QRS complexes were very wide (180 ms in duration), and the

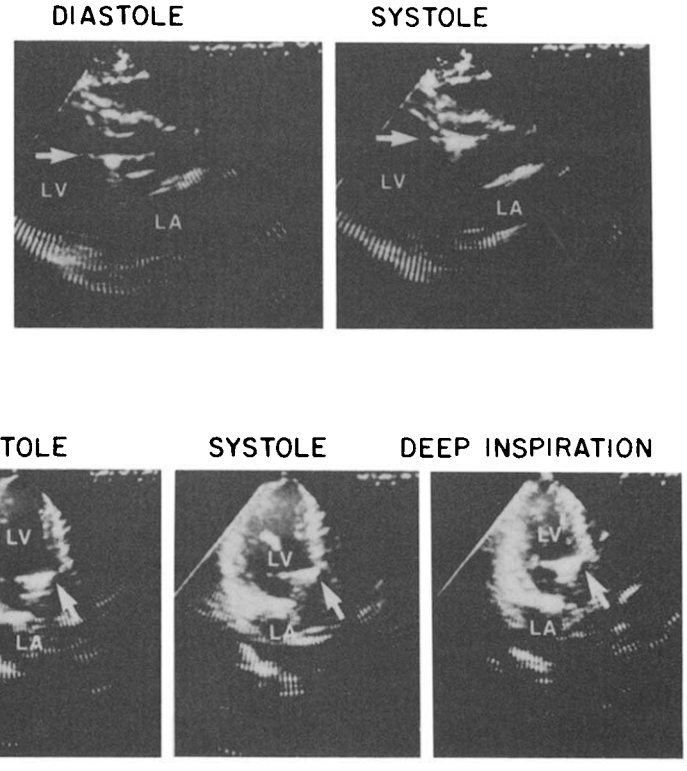


**Figure 3.** Two-dimensional echocardiographic still frames. **Top,** Long-axis view; **bottom,** apical two chamber view. During systole there was contact between the cage of the prosthesis (**arrows**) and the base of the interventricular septum. During deep inspiration (**lower right**) the left ventricular (LV) chamber size was decreased, and contact between the cage and the septum was increased. LA = left atrium.

VH interval was 55 ms (the VH interval during pacing from the right ventricular apex was only 50 ms). Thus, it is likely that this tachycardia originated in the Purkinje network, perhaps in the distribution of the left anterior fascicle.

The patient described in this report is unique in having a rapid, paroxysmal tachycardia (rate 150 to 210 beats/min) of fascicular origin. This tachycardia (tachycardia 1) was characterized by dissociated QRS complexes that resembled incomplete right bundle branch block and left posterior fascicular block. A His bundle recording revealed that the HV interval during this tachycardia was 20 ms. The diagnosis of fascicular tachycardia was further supported by the occurrence of ventricular tachycardia (tachycardia 2) in the midst of tachycardia 1; during tachycardia 2 there was dissociation between the His bundle and the ventricles as well as between the His bundle and the atria (double dissociation).

**Mechanical stimulation by mitral prosthesis.** We believe that our patient's tachycardias were caused by mechanical stimulation of the interventricular septum by the mitral valve prosthesis. Rosenzweig and Nanda (6) recently performed two-dimensional echocardiography in 30 patients with the Starr-Edwards mitral prosthesis and were able to demonstrate impaction of a ventricular wall (usually the septum) by the prosthesis in eight of the patients. Four of the eight patients with impaction, versus none of the 22 patients without impaction, had ventricular arrhythmias. In two of the patients, ventricular arrhythmias (ventricular tachycardia or ventricular premature beats) were precipitated by assumption of an upright posture.

*Evidence that our patient's tachycardias were caused by contact between the mitral prosthesis and the interventricular septum can be summarized as follows:* 1) Preoperatively, the patient did not have paroxysmal palpitation, and did not have ventricular arrhythmias during treadmill exercise or cardiac catheterization (deep inspirations). 2) Contact between the prosthesis and the interventricular septum (impaction) was demonstrated by two-dimensional echocardiography. 3) The tachycardias were precipitated by standing, deep inspiration, exercise or isoproterenol, that is, by interventions that can decrease left ventricular chamber size or increase the force of ventricular contraction. 4) There were no other apparent causes for the tachycardias.

*We speculate that the following occurred in our patient:* With contact between the mitral valve prosthesis and the interventricular septum there was stimulation of the left anterior fascicle, causing fascicular tachycardia. Variability of the frontal plane axis (100 to 180°) during fascicular tachycardia could have reflected stimulation of different portions of the left anterior fascicle, or intermittent antegrade block in portions of this fascicle (due to impaction). With increased contact with the interventricular septum there was stimulation of left ventricular myocardium, causing ventricular tachycardia in the midst of fascicular tachycardia. The coexistence of (slower) fascicular tachycardia and (faster) ventricular tachycardia suggests that the fascicular focus was "protected," because of retrograde block in the trifascicular conduction system. It is not clear whether this block was constant or only intermittent (due to impaction). Attacks of paroxysmal tachycardia eventually subsided. It is possible that repeated trauma to the interventricular septum by the mitral prosthesis caused localized fibrosis with ablation of the arrhythmogenic foci (6).

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