
Alternate Patterns of Premature Ventricular Excitation during Spontaneous Atrial Bigeminy*

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This report documents two instances of spontaneous alternate patterns of premature ventricular excitation during atrial bigeminy (alternating premature ventricular excitation). Previous reports of "alternating premature ventricular excitation" have required control of electrophysiologic parameters by atrial pacing methods. Illustration of this arrhythmia and a discussion of its mechanism constitute the substance of this report.

The clinical presentation of two instances of spontaneously occurring alternate patterns of premature ventricular excitation during atrial bigeminy (alternating premature ventricular excitation) and a discussion of its mechanism constitute the substance of this report. The previously reported instances of such "alternating premature ventricular excitation" have required the controlled conditions provided by atrial pacing methods.1

Case Reports

Case 1

The patient was a 58-year-old white man with a history of arteriosclerotic heart disease and angina pectoris who complained of prolonged chest pain. Physical examination revealed an irregular pulse. The patient was referred to the Beth Israel Hospital with a diagnosis of possible myocardial infarction. The arrhythmia was diagnosed as atrial bigeminy with alternate patterns of premature ventricular excitation (Fig 1 and 2). Administration of oral quinidine sulfate 400 mg each six hours eliminated the ectopia. The patient was discharged from the hospital on the fourth day without evidence of myocardial damage.

Case 2

The patient was a 69-year-old woman who was referred as an ambulatory patient because of palpitation of two weeks' duration. She had a prolonged episode of rapid heart action one year before which responded to mild sedation. A brief syncopal episode occurred six months ago which was preceded by palpitation and dizziness. She was being treated with hydrochlorothiazide, 50 mg a day, for hypertension. Physical examination revealed blood pressure of 170/100 and an irregular pulse. The arrhythmia was diagnosed as intermittent atrial bigeminy with alternate patterns of premature ventricular excitation (Fig 3). Serum electrolytes were normal. Oral administration of quinidine gluconate, 330 mg every eight hours, resulted in normal sinus rhythm within two days.

Discussion

Atrial premature beats commonly excite the ventricles abnormally through an altered pathway.2 During such aberrant conduction, normal intraventricular impulse propagation is altered because of a refractory state in part of the usual conduction pathway.3 Thus, aberrant conduction occurs whenever an atrial premature beat traverses the atioventricular node and spreads to a portion of refractory distal specialized conduction tissue. During any given cycle, the length of the refractory period of the branches of the specialized conducting system is directly related to the length of the preceding cycle;1,4-6 a longer preceding cycle results in a longer refractory period than does a shorter cycle. Accordingly, atrial premature beats which follow a long cycle are more likely to excite the ventricles abnormally than do those which follow a shorter cycle. The first atrial premature beat in Figure 1 followed a short cycle and was conducted normally; it was followed by a pause which prolonged the refractory period after the next regular beat so that the subsequent atrial premature beat was conducted abnormally.

The alternate patterns of ventricular excitation may take several forms.1 The premature beats may alternate between a normal and an abnormal configuration (Fig 3C) or between two abnormal configurations (Fig 2). Alternating patterns of right and left bundle branch block may result from differences in refractoriness of the right and left bundle branches due to changes in preced-

Figure 1 (top). After four consecutive sinus beats, there is a normally conducted atrial premature beat which is followed by a pause before the next sinus beat. The following atrial premature beat is aberrantly conducted through the ventricles. The bottom panel demonstrates an alternating pattern of premature ventricular excitation for the atrial premature beats.

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ing cycle lengths. In Figure 2, for example, the cycles from right bundle-branch block pattern premature beats to normal sinus beats are 50-60 milliseconds longer than those from left bundle-branch block pattern premature beats to normal sinus beats. This alteration in preceding cycle length might explain the alteration in aberrant conduction. Thus, a longer cycle before the bigeminal pair results in right bundle-branch block and a short cycle before the bigeminal pair resulted in left bundle-branch block. Alternating premature ventricular excitation may also occur where there is no apparent change in lengths of the preceding cycle (premature beats to sinus beat), or in the atrioventricular conduction time or atrial coupling interval (Fig 3C). In these cases, it is believed that alternating premature ventricular excitation occurred because delayed retrograde activation of the blocked bundle branch caused a shorter diastolic recovery period for that branch when the next normal beat occurs. Accordingly, the next atrial premature beat would find this previously blocked branch less refractory than the alternate one. For example, when the right bundle-branch is refractory to antegrade conduction, the depolarization wave proceeds down the left bundle-branch and excites the left ventricle. The right ventricle is activated late after the excitatory wave traverses the septum, and the right bundle-branch is excited in a retrograde fashion. The recovery period included in the cycle between the premature beat of right bundle-branch block configuration and the next normal sinus beat is characterized by a longer interval between electrical excitation for the left than for the tardily excited right bundle-branch. Therefore, the refractory period of the left bundle-branch becomes longer than that of the right, and the next premature atrial beat propagates to a receptive right bundle-branch and a refractory left bundle-branch. The result is an aberrant beat of left bundle-branch block pattern characterized by early excitation of the right bundle branch system. Longer recovery and refractory periods then occur in the earlier excited right bundle-branch system, and the next atrial premature beat again finds the right bundle-branch refractory.

REFERENCES