NONPAROXYSMAL BIDIRECTIONAL RHYTHM

A bidirectional slow ventricular rhythm was observed in a 61-year-old man with acute myocardial infarction. The ventricular origin of this arrhythmia was suggested by the presence of fusion beats. Moreover, it was different from other types of bidirectional tachycardias in that the rate was below 100/min and because digitalis could not be incriminated as a causal factor. It was possible that this bidirectional tachycardia could have been due to the presence of two ventricular foci—one in the superior and the other in the inferior divisions of the left branch respectively.

Bidirectional tachycardia is a well-known, but relatively uncommon rhythm. Most previous case reports have noted ventricular rates of approximately 150 per minute, as well as a high mortality. The following case report documents a previously undescribed variant of this rhythm.

CASE REPORT

A 61-year-old man with a history of four previous hospitalizations for myocardial infarction and recurrent angina pectoris, was first seen at the Miami Veterans Administration Hospital with substernal chest pain in March, 1969. At this time, he had not been taking any medication. He presented with a regular pulse rate of 84/min, blood pressure of 150/85 and was in congestive heart failure. Electrocardiograms showed normal sinus rhythm with evidence of old inferior and anterior wall myocardial infarction, but without acute changes. His hospital course was uneventful except for multiple episodes of angina decubitus with transient ischemic ECG changes (deepening inverted T waves in mid-to-lateral precordium). At the time of discharge he was taking digitalis, isosorbide dinitrate and nitroglycerin.

Between March and September of 1969, the patient discontinued all of the medications except the nitroglycerin. On September 9, 1969, he noted a sharp, anterior chest pain associated with dyspnea and diaphoresis. The pain radiated to the left arm and was unrelieved by repeated doses of nitroglycerin. On admission his pulse rate was 92/min and regular, blood pressure 115/75, and respirations 22/min. Serial electrocardiograms and enzyme studies were compatible with acute myocardial infarction.

The initial tracing (Fig 1) revealed a regular rhythm at a rate of 93/min. No definite P waves were seen except possibly on the down-slope of the QRS in lead II, making it difficult to assess the exact ventricular duration. Thus, it was questionable whether the rhythm was a junctional or a non-
paroxysmal ventricular tachycardia since the rate was compatible with either possibility.

A rhythm strip (Fig 2) taken immediately after Figure 1 revealed both a premature ventricular contraction (PVC) and a fusion beat (FB), i.e., a fusion beat between the PVC and the complex, which during sinus rhythm (Fig 5) was identified as the basic supraventricular complex in the same lead (II). No treatment was given at this time.

Within an hour of admission, the next tracing (lead II, Fig 3) revealed a bidirectional rhythm at a rate of 87/min. One of the complexes was negative and resembled the PVC in Figure 2. The other complex was positive and was very similar to the ones shown in Figure 1 and 2. The R-R intervals were equal at a distance of 0.68 seconds. This rhythm was present for approximately 15 minutes. It was unaccompanied by any change in the patient’s status and spontaneously converted to a ventricular bigeminal rhythm (Fig 4). In this tracing, there was normal sinus rhythm and a coupled beat. With a morphology similar to that in lead II in the original tracing (Fig 1) the coupling to the sinus beat was still 0.68 seconds. There were premature P waves (P, Fig 4) before some of the beats which could have been atrial premature contractions or a marked sinus arrhythmia. It is postulated that these P waves probably did not wholly contribute to ventricular depolarization since similar coupled beats with an identical coupling time were present even when not immediately preceded by a P wave. However, it is possible that some of these coupled beats could have been fusion beats (e.g., beat 4 may be a fusion beat from the sinus beat 5 plus beat 6). Since the patient remained stable, no treatment was given. Approximately two hours after admission, the ECG (Fig 5) showed marked sinus arrhythmia or a sinus rhythm with premature atrial contractions and an acute anterior wall myocardial infarction pattern. No abnormal beats were noted.

Three days after admission, another significant episode of chest pain occurred with ECG evidence of inversion of T waves in leads II, III, and AVF suggestive of inferior wall damage. No arrhythmia was noted during this episode.

The patient’s further hospital stay was unremarkable and he was discharged to the outpatient clinic.

**DISCUSSION**

This case documents the presentation of a bidirectional alternating rhythm in the setting of an acute myocardial infarction. This is different from previously described cases of bidirectional tachycardia for the following reasons.

**Etiologic factors:** In prior cases,1,2 digitalis toxicity superimposed on a chronically diseased myocardium were conditions necessary for the production of the bidirectional tachycardia. This patient had an acute myocardial infarction, as well as prior disease of the myocardium, but had not taken digitalis for a number of months.

**Rate:** The usual range of ventricular response in bidirectional tachycardia is 130-180/min.3 In this case, the rate was 87/min.

**Prognosis:** This rhythm usually augurs a grave prognosis. In a study of 35 cases,2 26 died within hours to days after discovery of the arrhythmia. In this case, the arrhythmia spontaneously reverted to normal sinus rhythm and the patient recovered uneventfully.

**Pathophysiology:** The most common theories of origin of bidirectional tachycardia have been: (a) two ventricular foci firing in alternation, (b) a junctional rhythm with a coupled ventricular focus, or (c) a junctional rhythm with alternate conduction down the bundle branches. Rosenbaum4 recently discussed this rhythm in light of the concept of trifascicular conduction and concluded that “bidirectional tachycardia is a supraventricular tachycardia with fixed aberrant conduction in the right bundle branch and alternating aberrant conduction in the two divisions of the left. This may be the situation when the ventricular response is rapid (i.e., over 130), but a number of questions persist in this case and may suggest ventricular origin(s).”
one would expect a more rapid rate than that seen here. In addition, the presence of fusion beats (beat FB in Fig 2 and Fig 4) are also indicative of a ventricular origin.

This rhythm is perhaps a variant of that described by Rothfeld et al, who noted an idioventricular rhythm (or non-paroxysmal ventricular tachycardia) occurring in the setting of an acute myocardial infarction. The arrhythmia described by these authors occurred at a rate of 60-100/min, showed fusion beats, lasted for a short time, and spontaneously disappeared. It was associated with a favorable prognosis. Much of our patient’s course was similar except that his apparently benign arrhythmia can be expected that a focus located in the inferior division should produce the pattern of a block in the superior division. This would result in abnormal left axis deviation with a predominant negative deflection (rS complex) in lead II (Fig 2 and 3). On the other hand impulse formation in the superior division should produce the pattern of block in the inferior division. The corresponding QRS complex should be tall and positive in lead II as shown in Figures 1-3. His bundle potential studies could possibly have answered the question of the origin of this arrhythmia.

Treatment: There has been no accepted method for the treatment of bidirectional tachycardia. Some have reported variable success with potassium or magnesium infusions, myocardial depressant drugs, and occasionally vagal stimulation. This patient’s arrhythmia converted spontaneously.

REFERENCES

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Mechanically Induced Cardiac Arrhythmia following Open Heart Surgery*

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Since the advent of cellular electrophysiology, a mechanical etiology for cardiac arrhythmias is rarely proposed. We observed a 23-year-old man, three months following mitral valve replacement surgery, who complained of palpitations that occurred only on deep inspiration in recumbency. Following physiologic and pharmacologic evaluation, we concluded that the mechanism for the arrhythmia causing the palpitations (junctional or ventricular tachycardia) is most likely direct stimulation of the irritable focus by the prosthetic valve struts. The mechanical etiology for the arrhythmia in this case suggests that patients with arrhythmias occurring under unusual circumstances should be pharmacologically and physiologically evaluated to determine the mechanism of their arrhythmias. A more accurate prognostic evaluation and therapeutic regimen would then result.

The cellular electrophysiologic studies of impulse formation and impulse conduction have uncovered considerable information regarding the genesis of cardiac arrhythmias. The seemingly simplest, yet least often suggested mechanism for an arrhythmia, is mechanical. We had the opportunity to observe an arrhythmia in a patient with a prothetic mitral valve replacement, who