Reciprocal Beating from Artificial Ventricular Pacemaker

Report of a Case

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SUMMARY
A case is reported of reciprocal beating induced by a transvenous ventricular pacemaker inserted because of permanent sinus arrest. The mechanism of this finding is discussed.

Additional Indexing Words:
A-V junctional (nodal) rhythm Retrograde conduction Sinus arrest

In 1963, Burchell predicted that reciprocal beating could result from the use of a ventricular pacemaker in the presence of normal atrioventricular (A-V) conduction.1 The following case report documents this finding in a patient whose pacemaker was inserted because of a slow heart rate resulting from permanent sinus arrest.

Report of A Case

J.B. (MGH #93-39-60), a 65-year-old man, entered the Massachusetts General Hospital on June 14, 1966, for implantation of a cardiac pacemaker. Ten years previously, without any history of prior cardiac disease, he began to have angina pectoris. Shortly thereafter, he experienced episodes of flushing and sweating associated with a Bradycardia of between 30 and 50 beats per minute. An electrocardiogram at that time showed an A-V junctional (nodal) rhythm at a rate of 37 (fig. 1).

Over the next 10 years, he gradually developed a permanent nodal rhythm with a rate that varied between 32 and 40. He had frequent episodes of supraventricular arrhythmias consisting of atrial flutter, atrial fibrillation, and paroxysmal atrial tachycardia with block which were controlled with variable success by digitalis, quinidine, and procainamide.

On May 30, 1966, he entered another hospital because of abdominal pain. An exploratory laparotomy was performed, but no precise cause of the pain was found. Postoperatively he developed paroxysms of atrial flutter-fibrillation. He was given quinidine, which terminated the arrhythmia, but he began to have prolonged asymptomatic intervals, particularly immediately after cessation of each paroxysm of tachycardia. Finally, a sustained period of cardiac arrest led to a severe syncopal attack from which he was successfully resuscitated. The post-resuscitation tracing showed a nodal rhythm at a rate of 22. He was given isoproterenol, which accelerated the nodal rate to 50 to 60, but produced ventricular irritability. Because of increasing difficulty in the management of his arrhythmias, he was transferred to the Massachusetts General Hospital for implantation of a pacemaker.*

On June 16, 1966, a permanent transvenous endocardial pacemaker was inserted uneventfully.† Irregularity of the heart beat was evident after pacemaker installation, and the mechanism is illustrated and discussed in figure 2. Retrograde activation of the atria by the pacemaker-induced ventricular beats with reciprocal capture of the ventricles was evident.

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†Chardack-Greatbatch Pulse Generator, Model 5870-C; Chardack Cardiac Catheter Electrode Model 5816; manufactured by Medtronic Inc., Minneapolis, Minn.


**Discussion**

It has been well established that impulses from ventricular premature systoles and ventricular tachycardia are sometimes conducted in a retrograde fashion to the atria. The frequency of this pattern is greater than is usually appreciated because the retrograde P wave is often buried in the T wave of the previous ventricular beat. Esophageal electrodes have been especially helpful in demonstrating the retrograde conduction of P waves more clearly. The R-P' interval has been found to vary, a phenomenon which may be due either to the presence of differing degrees of retrograde A-V block or to retrograde conduction over separate pathways with different speeds of conduction.

Demonstration of retrograde atrial activation in the presence of A-V block was first reported clinically in 1914 and this finding has been repeatedly confirmed. The most widely accepted explanation for retrograde conduction in such cases is that the A-V junctional tissues have the capacity to conduct in either direction, and at any particular time the degree of forward A-V block may be greater than the degree of retrograde block. However, it has recently been suggested that this type of unidirectional block may, at least in some cases, develop in the peripheral bundle branches rather than in the A-V junctional tissue itself.

Reciprocal or “echo” beats most commonly arise from nodal pacemakers, but their origin from ventricular foci has also been shown. Echo beating does not appear in complete A-V block despite the occasional presence of retrograde conduction from the idioventricular or artificial ventricular pacemaker. However, when A-V block is ab-
Reciprocal beating from ventricular pacemaker. Electrocardiographic lead aVp and ladder diagram. The pacemaker stimuli (E on ECG, small black circle on ladder diagram) drive the ventricles (QRS') at a fixed rate of 75 per minute (an interval of 0.80 second). These impulses are transmitted forward into the ventricles (V on ladder diagram) and retrograde (dotted line) into the atrioventricular junctional tissues (A-V) and atria (A). The inverted P waves (P' on the ECG) identify the retrograde atrial depolarization. Each second paced ventricular beat is followed by a reciprocal ("echo") beat conducted from A-V tissues into the ventricles (QRS) and associated with a P'-R interval of 0.18 second. In the first sequence the E-P' interval is 0.46 second following the first paced beat and 0.48 second following the second paced beat. Since the reciprocal beat only occurs after the longer delay, it is presumed that the forward A-V pathway is still refractory following the first paced beat. The pacemaker stimuli following the reciprocal beats are blocked from transmission in either direction because the ventricles or the His-Purkinje pathways or both are refractory at that time.

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