Recurrent Tachyarrhythmia Associated with a Bifocal Demand Pacemaker

Donald R. Ricci, M.D.; ** Alan K. Rider, M.D.; and Jay W. Mason, M.D.

A recurrent tachyarrhythmia complicated the course of a patient with a permanent atrioventricular sequential (bifocal) demand pacemaker. Investigation by intracardiac electrocardiographic studies revealed that the arrhythmia was associated with a normally functioning bifocal pacemaker, whose atrioventricular sequential interval approximated the patient's conduction time from atrium to ventricle. The mechanism of initiation and conversion of the arrhythmia was elucidated. Appreciation of this arrhythmia is necessary for appropriate clinical use of a bifocal demand pacemaker.

*From the Cardiology Division, Stanford University School of Medicine, Stanford, Calif. Supported in part by grant HL-5860 and program project grant 1-F01-150833 from the National Institutes of Health.

**Fellow, Canadian Heart Foundation.

Reprint requests: Ms. Dorothy McCain, Cardiology Division, 701 Welch Road, Stanford 94305

Atrioventricular sequential pacemakers are indicated in patients with disease of the sinus node or the atrioventricular conduction system, in whom the preservation of atrial systole is desirable. This report describes an unusual arrhythmia complicating the use of an atrioventricular sequential pacemaker.

The separate atrial and ventricular demand units of the atrioventricular sequential (bifocal) demand pacemaker are QRS-inhibited. Whereas the atrial electrode paces only, the ventricular electrode provides both pacing and sensing activity. By monitoring ventricular activity the ventricular sensing circuit programs both atrial and ventricular stimuli. The delay between atrial and ventricular pacing stimuli is adjusted before implantation of the pacemaker, and when possible, a delay time is chosen to allow antegrade atrioventricular conduction, avoiding ventricular pacemaker firing, and thereby prolonging the life of the pulse generator.

Problems associated with the bifocal demand pacemaker have limited its use; they include instability of the atrial electrode, a short life span of the pulse generator, asystole, and the precipitation of arrhythmias.

The purpose of the present report is to analyze in detail a tachyarrhythmia associated with a normally functioning atrioventricular sequential demand pacemaker. Fields et al,1 Furman et al,6 and Santos et al7
have described similar arrhythmias whose etiology and pathogenesis were not clear. Our elucidation of the pathogenesis of such tachyarrhythmias allows certain recommendations regarding the use of this pacemaker.

**CASE REPORT**

A 63-year-old man was hospitalized in August 1975 for evaluation of recurrent tachyarrhythmias. In 1968 and 1971, he had suffered myocardial infarction. In 1972, recurrent supraventricular tachycardia alternating with sinus bradycardia occurred, and a permanent ventricular demand pacemaker was implanted. In the subsequent 12 months, progressive cardiomegaly and congestive failure developed. A bifocal (atrioventricular sequential ventricular-inhibited demand) pacemaker (American Optical Corp.) was inserted in December 1973, in an attempt to regain the atrial contribution to the cardiac output. The congestive failure improved moderately, but a different recurrent tachyarrhythmia emerged, which was refractory to therapy with quinidine, procainamide, hydrochloride, and lidocaine and to carotid sinus pressure. The arrhythmia appeared to be ventricular in origin, with atrial stimulus artifacts coincidentally preceding each QRS complex. Direct-current cardioversion at low energy was usually successful in reestablishing bifocal demand pacing, and often the arrhythmias converted spontaneously.

In order to further characterize the relationship between the pacemaker's function and the tachyarrhythmia, a quadrupolar pacing catheter was positioned high in the right atrium, a tripolar His-recording catheter was positioned across the tricuspid valve, and base-line recordings were made with the patient in a normal bifocal pacing mode (Fig 1). Tachyarrhythmia occurred spontaneously during the procedure (Fig 2) and was also induced by atrial pacing at a cycle length of 540 msec (Fig 3). Atrial pacing at a cycle length of 300 msec interrupted the tachyarrhythmia (Fig 4).

**DISCUSSION**

During normal bifocal pacemaker rhythm (Fig 1), the atrial (Sₐ) and ventricular (Sᵥ) stimulus artifacts are separated by an atrioventricular sequential interval of 250 msec. Atrial depolarization is recorded in the atrial electrogram 25 msec after Sₐ, and ventricular depolarization, seen in the surface and ventricular electrograms, immediately follows Sᵥ. The atrio-His (A-H) interval is prolonged (255 msec), confirming the presence of conduction disease of the atrioventricular node. Since the atrioventricular sequential interval is less than the sum of the SₐA and A-H intervals, Sᵥ intervenes to cause ventricular depolarization. The next Sₐ occurs at the atrial escape interval 480 msec from Sᵥ, and the rhythm perpetuates itself.

When the tachyarrhythmia was recorded via intracardiac techniques (Fig 2), it became clear that the rhythm was, in fact, a supraventricular pacemaker tachycardia. The R-R interval is 530 msec (113 beats per minute). The A-H interval is even more prolonged (390 msec) at this rate, and the His-ventricle (H-V) interval is also prolonged (80 msec). In concert with the normal sensing function of the ventricular electrode, the atrial escape interval of 480 msec is measured from the large-amplitude portion of the QRS complex, some 70 msec following the onset of ventricular depolarization. As seen on the surface electrocardiogram and demonstrated in the ladder diagram, Sₐ is buried in the initial portion of the previous QRS complex, and, via slowed transmission through diseased conduction tissues, results in a ventricular depolarization 495 msec later (Sₐ-V interval). The atrial escape and Sₐ-V intervals are similar, so that Sₐ occurs before the large-amplitude portion of the QRS complex and, therefore, is not inhibited; hence, the tachyarrhythmia is self-sustaining.

Figure 3 demonstrates initiation of the tachyarrhythmia by atrial pacing at an arbitrary cycle length of 540 msec. The pacing stimulus (Fig 3, P₁) causes atrial depolarization and conduction through the atrioventricular nodal and His-Purkinje tissues. The next Sₐ (Sₐ* in Fig 3) is not inhibited by the resultant ventricular depolarization because it occurs prior to sensing of the large-amplitude portion of the ventricu-
Cardiac depolarization by the ventricular electrode. The $S_A$ (Fig 3, $S_A^*$) conducts with increased atrioventricular nodal delay (A-H interval, 400 msec), producing a ventricular depolarization with its major deflection occurring after the next $S_A$, which occurs at the appropriate atrial escape interval (400 msec). The sequence is repeated and the tachyarrhythmia established.

We have identified three requirements for the initiation of this peculiar tachyarrhythmia. First, a premature atrial depolarization must conduct with an A-H interval more prolonged than usual (or a premature ventricular depolarization must occur in a setting of more prolonged A-H conduction). Second, the resultant sensed ventricular depolarization must closely follow the next $S_A$, so that $S_A$ is not inhibited. Third, total time of antegrade transmission through the conduction system must be similar to the atrial escape interval to produce the temporal relationships just described.

Figure 4 demonstrates conversion of the tachyarrhythmia to the normal bifocal rhythm during atrial pacing at an arbitrary cycle length of 300 msec. An appropriately timed premature stimulus (Fig 4, $P_4$) renders the atrium refractory to $S_A$ (Fig 4, $S_A^*$) and itself is blocked in the atrioventricular node. Another $S_A$ (Fig 4, $S_A'$) is not conducted because of atrial refractoriness induced by $P_4$. Therefore, ventricular depolarization (Fig 4, $V^{**}$) results from $S_V$ at the ventricular escape interval. The subsequent atrial escape after $S_V$ occurs in the usual fashion, and bifocal rhythm is reestablished.

The key factor in breaking the tachyarrhythmia, therefore, is a premature stimulus which prevents conduction of $S_A$ and, hence, interrupts the temporal relationship between $S_A$ and the preceding ventricular depolarization. This may be a premature atrial stimulus leaving the atrium refractory to $S_A$, or a premature ventricular stimulus (or direct-current countershock) which occurs early enough to cause ventricular inhibition of the next $S_A$.

The tachyarrhythmia described herein is unique to the atrioventricular sequential type of pacemaker. The coincidental similarity of the atrial escape interval and the intrinsic atrioventricular conduction time in this patient allowed for occurrence of the tachyarrhythmia by permitting $S_A$ and ventricular depolarization to occur in phase. Although the patient in this case report had atrioventricular conduction disease, a similar tachyarrhythmia may be expected in any patient in whom the atrial escape interval approximates the sum of the A-H and H-V intervals. Since prolongation of the A-H interval at rapid heart rates might allow this approximation to occur, the degree of prolongation at fast rates
Figure 4. Pacing-induced conversion of tachyarrhythmia to bifocal rhythm. Atrial pacing artifacts (P) occur at 300-msec intervals. P₁, P₂, and P₃ do not conduct, due to atrial refractoriness. P₄ and P₅ conduct through atrium, leaving it refractory to S₄* and S₅†, but are blocked at atrioventricular node (AVN) due to refractoriness. Because conduction of S₄* and S₅† is interrupted, ventricular escape allows S₅ to cause ventricular depolarization (V***) in temporal isolation from any S₆; and, hence, atrial escape after V** allows S₅ to occur in temporal isolation from ventricular depolarization. Even if P₅ had not occurred, bifocal rhythm would ensue, because S₅† would have conducted in usual fashion, with prolonged A-H and H-V intervals, and S₅ would intervene to cause V** in temporal isolation from S₆. Following V***, there is retrograde His spike which is blocked and is irrelevant to reestablishment of rhythm. AE, Right atrial electrogram; HBE, His bundle electrogram; A, atrium; H, His bundle depolarization; HPS, His-Purkinje system; and V, ventricle.

should be measured by atrial pacing in any patient who is a candidate for a bipolar pacemaker. Alternatively, before implantation, the atrial escape interval can be lengthened by shortening the atrioventricular sequential interval, as described by Fields et al, 1 to prevent these arrhythmias in two patients. If a bifocal demand pacemaker is to be utilized, the physician must be aware of this tachyarrhythmia, in order to avoid unnecessary pharmacologic therapy and to take measures before implantation to prevent the occurrence of the tachyarrhythmia.

ACKNOWLEDGMENTS: We wish to thank Dr. Walter S. Stullman, who referred the patient; Ms. Geraldine Derby, R.N., for her assistance; and Ms. Mary Parrish for assistance in manuscript preparation.

REFERENCES

Spontaneous Rupture of a Coronary Artery with False Aneurysm Formation*

Successful Surgical Repair

John K. Frischknecht, M.D.; David Shander, M.D., F.C.C.P.; Thomas L. Kurt, M.D.; Phillip S. Wolf, M.D., F.C.C.P.; and Lane D. Craddock, M.D.

A patient with diffuse atherosclerotic coronary arterial disease was demonstrated to have a spontaneous rupture of the proximal right coronary artery, with formation of

*From General Rose Memorial Hospital and the University of Colorado Medical Center, Denver. Reprint requests: Dr. Shander, 800 Clermont, Denver 80220

CHEST, 72: 1, JULY, 1977

SPONTANEOUS RUPTURE OF CORONARY ARTERY 123