Reciprocal Beating Induced by Ventricular Pacing


SUMMARY

Ventricular pacing is frequently associated with retrograde ventriculo-atrial conduction. In the absence of atroventricular (A-V) block, the relatively intact antegrade A-V conduction system supplies the necessary pathway for a re-entry mechanism that may become operative in the presence of delayed retrograde conduction.

Two patients with bradycardia and unimpaired antegrade A-V conduction exhibited reciprocal (echo) beating following nodal beats before ventricular pacing was instituted. After the introduction of an artificial ventricular pacemaker, reciprocal beats followed the paced ventricular beats in both patients.

These observations suggest that a similar mechanism is involved in the production of reciprocal beats linked to impulses originating from the A-V node and the ventricles.

When ventricular pacing is indicated for the treatment of bradycardia in the presence of intact antegrade A-V conduction, a situation which may produce echo beats, demand pacing is considered preferable to fixed-rate pacing as the latter may deliver a stimulus into the vulnerable phase following an echo beat.

Additional Indexing Words:

Atrial electrogram A-V junctional (nodal) rhythm Demand pacemaker
Fusion beat Retrograde conduction Sinus arrest Wenckebach’s phenomenon

Patients with permanent or prolonged sinus arrest with a slow atroventricular (A-V) junctional (nodal) rhythm and patients with complete heart block frequently present with similar physiological and clinical aspects. Both conditions often necessitate the use of a temporary or permanent cardiac pacemaker. In contrast to complete heart block, antegrade conduction is usually intact in patients with sinus arrest and an A-V nodal rhythm. Intact antegrade A-V conduction combined with delayed retrograde ventriculo-atrial conduction provides the setting for the establishment of spontaneous or ventricular pacemaker induced reciprocal (echo) beats.

Rosenbleuth1 and Moe and associates2 demonstrated experimentally the phenomenon of reciprocal beating by the electrical stimulation of the ventricles of dogs. Burdell3 in 1963 recognized that in a subject with bradycardia related to sinus arrest and a slow nodal mechanism, ventricular pacing would establish the physiological circumstances favorable to the production of reciprocal beats in the presence of delayed retrograde conduction. Indeed, he predicted this occurrence in man. The first case of reciprocal beating from an artificial ventricular pacemaker was reported by Kastor and DeSanctis in 1967.4

The purpose of this communication is to document the same mechanisms in two additional patients. One patient had bradycardia due to permanent sinus arrest and a slow nodal rhythm, and the other exhibited recurrent episodes of prolonged sinus arrest. Interestingly, both patients exhibited reciprocal beating following nodal beats before

From the Division of Cardiology, Department of Medicine, Mount Sinai Hospital, Miami Beach, and the Department of Medicine, University of Miami School of Medicine, Coral Gables, Florida.

Supported by U. S. Public Health Service Grant HE-09782-03.
ventricular pacing was instituted. After the introduction of the artificial ventricular pacemaker, reciprocal beats followed the paced ventricular beats.

**Case Histories**

Case 1, M.P. (no. 213289), a 61-year-old woman, was admitted to Mount Sinai Hospital on April 6, 1967, for the evaluation of bradycardia. Ten months previously she developed sudden blurring of vision and sweating and entered another hospital with a suspected myocardial infarction. During that hospitalization, bradycardia was noted and she had several attacks of blurring of vision. Since that time, she was aware of having a slow pulse and continued to experience periodic blurring of vision followed by a flushing sensation and sometimes accompanied by palpitations. She was involved in a car accident 1 week prior to her admission to this hospital. She sustained fractures of both clavicles and of some ribs, but no head injury. Twenty-four hours after the accident, when she was at home, she experienced a syncopal episode for the first time and was unconscious for several seconds. She denied chest pain, dyspnea, and ankle edema.

On physical examination, the pulse rate was regular at 48 to 50/min. The blood pressure was 110/80. Apart from the traumatic lesions, the physical examination was unremarkable. The electrocardiogram on admission showed an A-V junctional (nodal) rhythm at a rate of 48/min. There was T-wave inversion in leads II, III, and aVF, and leads V1 to V4 (fig. 1). The chest x-ray demonstrated a normal-sized heart and the healing fractures. On fluoroscopy, slight left atrial enlargement was detected.

On April 8, 1967, right and retrograde left heart catheterization with selective coronary arteriography was performed. Bipolar electrode catheters* were passed to the right atrium and the outflow tract of the right ventricle, respectively. The functional integrity of the A-V conduction system was demonstrated by pacing the right atrium at rates of 69 and 90/min (fig. 2). The right atrial unipolar electrogram was recorded simultaneously with lead II during ventricular pacing. The progressive lengthening of retrograde ventriculo-atrial (V-A) conduction leading to the formation of reciprocal beats was

---

Upper strip: Some of the fixed-rate pacemaker stimuli are depicted by E. Lead II recorded simultaneously with the right atrial unipolar electrogram (RA) during ventricular pacing at 72/min at cardiac catheterization. Retrograde P waves (P') are clearly delineated by the atrial electrogram. The progressive lengthening of retrograde V-A conduction is evident. Each paced ventricular beat is followed by a retrograde P wave. The E-P' for the first paced ventricular beat (V₁) in the sequence is 0.20 sec. The E-P' for the second paced beat (V₂) is 0.37 sec. Retrograde conduction following the third paced beat (V₃) is sufficiently delayed (E-P' = 0.42 sec) to permit re-entry causing a reciprocal beat (Re) with a P'-R interval of 0.17 sec. The interval from E to the onset of the QRS complex of the reciprocal beat is 0.59 sec. E₄ is ineffective as it falls in the absolute refractory period of the reciprocal beat. The sequence then repeats itself. The progressive delay in V-A conduction is the manifestation of a Weckebach phenomenon. Lower strip: The pacing rate is identical but the sequence consists of only two paced ventricular beats followed by a reciprocal beat.

clearly demonstrated during ventricular pacing (fig. 3). The pressures on the right and left sides of the heart were normal. The cardiac index was moderately reduced during nodal rhythm and increased with ventricular pacing. The left ventricular cineangiogram was normal. The right and left coronary arteries were the site of mild nonobstructive disease.

A bipolar catheter was left in the outflow tract of the right ventricle and was connected to
VENTRICULAR PACING

Figure 4

Lead II recorded on 4/17/67, 7 days after the insertion of a temporary pacemaker catheter into the outflow tract of the right ventricle. When the pacemaker is turned off, the underlying nodal rhythm at a rate of 41/min is evident. Inverted P waves (P’) follow immediately after each QRS complex. The R-P’ interval is 0.20 sec. There is a slight variation in the rate of the A-V nodal discharge.

Figure 5

Tracing of lead II taken on 4/22/67, with pacemaker turned off, shows repetitive grouping of the ventricular complexes due to reciprocal beating. The basic rhythm is nodal with delayed retrograde conduction to the atria. Each ventricular complex is followed by an inverted P wave (P’) with a prolonged R-P’ interval of 0.36 sec, indicative of retrograde V-A block. The retrograde P wave is succeeded by a reciprocal beat with a configuration similar to the dominant nodal beat. The P’-R interval of the reciprocal beat is 0.16 sec, and the interval separating the QRS complexes in each group is 0.53 sec. The QRS complex of the reciprocal beat in the second pair of beats is also followed by a retrograde P wave (P”).

A fixed-rate external Medtronic pacemaker,* and over the next few days the responses to varying rates of ventricular pacing were observed. When the pacemaker was turned off, sinus activity was consistently absent and an A-V junctional (nodal) rhythm emerged with intermittent retrograde conduction with an R-P interval of 0.20 second (fig. 4). At another time, bigeminy due to regularly occurring reciprocal beating was evident (fig. 5). Reciprocal beating was also noticed following paced ventricular beats. As ventricular capture subsequently became irregular, the patient was taken back to the cardiac catheterization laboratory on April 18, 1967, and the catheter was pulled back a short distance in the outflow tract of the right ventricle. Regular capture was again obtained.

Over the next few days, pacemaker induced reciprocal beating was repeatedly observed. Representative electrocardiograms are illustrated in figure 6. It became evident that fixed rate pacing was undesirable and potentially dangerous, as some pacemaker stimuli were falling close to the apex of the T wave of the reciprocal beats (fig. 6B). Consequently, a Cordis Ventricor III (Ectocor)‡ demand pacemaker was implanted.

---

*Medtronic Inc., Minneapolis, Minnesota.

‡Cordis Corporation, Miami, Florida.

Circulation, Volume XXXVIII, August 1968
Figure 6

Strips of lead II recorded on 4/22/67. Some of the fixed rate pacemaker stimuli are depicted by E. Strip A: Fixed rate ventricular activation occurs at an R-R interval of 1.20 sec. Each paced ventricular beat is followed by delayed retrograde V-A conduction, the E-P' interval being 0.40 sec. A reciprocal beat with a P'-R interval of 0.18 sec follows each paced beat producing an alternating rhythm of paced and reciprocal beats. The configuration of the reciprocal beat is similar to the ones in figure 5. The interval from E to the onset of the QRS of the reciprocal beat is 0.58 sec. Strip B: As the pacing rate is increased (R-R = 0.88 sec) a different sequence is produced. The first paced ventricular beat (E1) does not engender a visible retrograde atrial depolarization. The second paced beat (E2) is followed by a relatively early retrograde P wave which deforms the S-T segment. The third paced beat (E3) is succeeded by a clear-cut inverted P wave (P'). The E-P' interval for the third cycle is 0.40 sec. A reciprocal beat with a P'-R interval of 0.16 sec and a supraventricular configuration follows the third paced beat. The next paced beat (E4) falls on the descending limb of the T wave of the preceding reciprocal beat. The paced beat gives rise to a retrograde P wave with an even longer E-P' interval of 0.44 sec, which also initiates a reciprocal mechanism. The fifth paced beat behaves similarly. The sixth pacemaker stimulus (E6) is ineffective and breaks the sequence. The progressive lengthening of the retrograde transmission time is explicable on the basis of a retrograde Wenckebach phenomenon. Strip C: This strip was obtained at a faster pacing rate (R-R = 0.62 sec). Each couplet consists of a paced ventricular beat followed by a reciprocal beat. The E-P' interval is 0.40 sec and the P'-R interval is 0.18 sec. Every alternate pacemaker stimulus falls during the inscription of the R wave of the reciprocal beats and is thus ineffective as it occurs in the absolute refractory period. The supraventricular QRS complex of the recipro-
with pervenous catheter pacing. On discharge, the patient was asymptomatic and the electrocardiogram (fig. 7) disclosed regular ventricular capture without apparent atrial depolarization. She has remained well since.

Case 2. M.B. (no. 206602), a 57-year-old man, was admitted to Mount Sinai Hospital on June 30, 1966, because of a short-lived attack of left anterior chest pain. Three months previously he began to experience recurrent brief spells of sudden weakness associated with a sensation of faintness. No frank syncope occurred. On occasions he noticed transient blurring of vision and diplopia without the other symptoms. Physical examination was unremarkable. Blood pressure was 140/80. The initial electrocardiogram (fig. 8) revealed an A-V junctional (nodal) rhythm at a rate of 50/min. Prominent U waves were present across the precordial leads. He was connected to an Avionics unit for constant monitoring of the electrocardiogram, and 10-hour recordings were obtained on several days. The recordings disclosed sinus rhythm interrupted by long pauses of sinus arrest, nodal rhythm with and without retrograde atrial conduction, reciprocal beats, wandering pacemaker, and occasional atrial premature beats. Some of these rhythms are illustrated in figure 9. The episodes of sinus arrest with total cardiac standstill lasted for periods up to 3.66 seconds (fig. 9A). A nodal escape beat usually terminated these pauses. The patient was able to correlate his symptoms with the episodes of sinus arrest. Drug therapy with sympathomimetic drugs and atropine was largely unsuccessful. Atropine in doses of 3 mg every 6 hours reduced the frequency of the sinus pauses, but induced side effects, such as marked dryness of the mouth, agitation, insomnia, and interference with bladder sphincter action. The visual symptoms were considered to be diabetic in origin. Myasthenia gravis was excluded by pharmacological means.

On July 15, 1966, a Cordis Ventricor II* demand pacemaker was implanted with pervenous catheter pacing. The day following the implantation he developed epigastric pain accompanied by T-wave inversion in leads II, III, aVF, and V4 to V6 of the electrocardiogram. These abnormalities were only visible when the pacemaker was inoperative. The serum glutamic oxaloacetic acid transaminase levels did not rise. The picture was consistent with, but not diagnostic of, acute coro-

---

* Cordis Corporation, Miami, Florida. The Ventricor II demand pacemaker is no longer manufactured.
nary insufficiency. Subsequent electrocardiograms showed periods of normal sinus rhythm with the demand pacemaker essentially inactive (fig. 10, upper strip). On occasions, when the sinus rate approximated the preset firing rate of the pacemaker, competition with the production of fusion beats was evident (fig. 10, lower strip). At other times there were runs of ventricular pacing with reciprocal beating (fig. 11). The rest of his hospital course was uneventful. He was asymptomatic on discharge and also several months later.

Discussion

The entire subject of retrograde ventriculo-atrial (V-A) conduction of ventricular impulses has been recently enlightened by numerous investigators using intra-esophageal,
VENTRICULAR PACING

Lead II recorded on 7/18/66, following the implantation of the Ventricor II demand pacemaker. The pacemaker is inactive as the sinus rate is above the demand rate of the pacemaker. When the sinus rate is slowed by carotid sinus pressure, the pacemaker becomes operative.

Figure 10

Lead II recorded on 7/20/66. The demand function of the Ventricor II pacemaker is evident. The first, sixth, and ninth beats are paced, and each is followed by delayed retrograde V-A conduction which induces reciprocal beating. The E-P' intervals vary from 0.46 to 0.48 sec and the P'-R intervals are 0.12 sec. The reciprocal beats are sensed by the pacemaker which re-cycles itself and discharges after its preset interval of 0.86 sec. The fourth QRS complex, which is widened, may be a ventricular extrasystole. Although no visible retrograde P wave precedes it, the relationship to the previous paced beat suggests a reciprocal mechanism with aberrant conduction.

Many of the pacemaker stimuli in the illustrations have been retouched for the purpose of reproduction.

Intra-atrial, and epicardial electrocardiography. Retrograde conduction of a ventricular impulse with retrograde activation of the atria is not only a well-documented phenomenon, but is considered a frequent accompaniment of ventricular rhythms which may be spontaneous or electrically induced.

Retrograde V-A conduction in man was considered infrequent until 1951, when Kistin and Landowne, using esophageal electrocardiography, demonstrated its existence in 15 of 33 subjects with ventricular premature beats. Kistin also established the relative frequency of V-A conduction during ventricular tachycardia. When ventricular tachycardia is induced by a catheter during diagnostic cardiac catheterization, V-A conduction with varying degrees of block also occurs frequently. Lister and associates utilized epicardial electrocardiograms to delineate atrial activity at open-heart surgery and demonstrated the common occurrence of V-A conduction in ventricular rhythms. In the presence of varying degrees of antegrade A-V
block, including complete heart block, retrograde V-A conduction has been reported by many investigators.12–20 Electrically induced ventricular rhythms and tachycardias are associated with a high incidence of V-A conduction.9,10 Using intracardiac electrocardiography, with a bipolar electrode catheter in the right atrium and another in the right ventricle, Samet and associates10 detected retrograde P waves in 29 of 31 individuals with normal antegrade A-V conduction in whom a ventricular rhythm was induced by ventricular pacing. In complete heart block, retrograde V-A conduction during ventricular pacing is not a rare phenomenon, particularly when the ventricular rate exceeds the sinus rate.11,18,21,22

Reciprocal (echo) beats represent a particular form of re-entry dependent upon delayed retrograde V-A conduction of an impulse originating either from the A-V node or the ventricles.26 When the delay in the retrograde transmission reaches a critical limit, an impulse may return across the A-V node to reactivate the ventricles. Reciprocal beats originate more commonly from nodal than from ventricular rhythms.24 Reciprocal beating triggered by ventricular extrasystoles, although considered less common, is well documented in the literature.25,26 Both types are observed uncommonly in clinical practice.

The two patients reported here exhibited reciprocal beats prior to the insertion of the pacemaker. In both instances, an A-V nodal impulse was the initiating mechanism. When the ventricle was electrically paced, reciprocal beats occurred following pacemaker-triggered ventricular beats. This observation suggests that a similar mechanism is involved in the production of reciprocal beats linked to impulses originating from the A-V node and the ventricles. Only in one previous report were reciprocal beats precipitated by retrograde V-A conduction from A-V nodal and ventricular extrasystoles in the one patient.27

One case of reciprocal beating from a fixed-rate permanent ventricular pacemaker has been described by Kastor and DeSanctis.4 Their patient had an A-V nodal rhythm at a rate varying from 32 to 40/min for 10 years and frequent episodes of supraventricular arrhythmias. A nodal rhythm of 22/min following a cardiac arrest prompted the insertion of a fixed-rate pacemaker. In that patient, a reciprocal beat was produced when the interval from the pacemaker stimulus to the retrograde P wave was 0.48 second, but failed to appear when the interval was 0.46 second. These measurements are essentially similar in our patients.

Nathan and associates28 described a re-entry mechanism that they called pacemaker reciprocal beating in a patient with complete heart block and a synchronous pacemaker. In the published illustration, when a retrograde P wave was sufficiently delayed so as to fall outside the refractory period of the atrial sensing electrode, a ventricular stimulus was delivered consequent to the detection of atrial depolarization. As the reciprocal circuit was via the pacemaker, which in effect bypasses the A-V node, this interesting observation does not represent a true reciprocal mechanism.

The concept of longitudinal dissociation of the A-V conduction system proposed by Scherf in 194120 still remains the most widely accepted explanation for the re-entry of excitation. He postulated two functionally separate pathways in the A-V node. An impulse traveling up the A-V node finding one pathway refractory is propagated to the atrium along the unblocked pathway. If the ascending impulse is sufficiently delayed, it may, after having reached a certain level, spill over and descend into the previously refractory alternate pathway, which by then has had time to recover. The return into the antegrade nonrefractory pathway reactivates the ventricles and produces a reciprocal beat. Since its original formulation, evidence for this explanation has been repeatedly accumulated experimentally.1,2,30–32

Mendez and Moe32 in their brilliant studies demonstrated a dual A-V nodal conduction system in the isolated rabbit heart. The intranodal propagation of ventricular echoes in an in vitro preparation of the rabbit heart was
traced from microelectrode records at many puncture sites. They concluded that the upper part of the node can be functionally and spatially dissociated into two pathways (α and β) which communicate with a final common pathway about halfway between the atrium and the bundle of His. Ventricular echoes induced by premature stimulation of the bundle of His passed from the final common pathway to the atrium over the α route and back to the final common pathway over the β route.

There is evidence suggesting that the atrium is an essential link in the echo circuit in the in vitro preparation of the rabbit heart and the denervated perfused dog heart.31, 32 However, ventricular echoes persist when atrial activity is abolished by high potassium levels33 and when the atrium is rendered refractory,34 showing that re-entry may occur within the region of the A-V node. The creation of a new impulse initiated in some way by the retrograde one, as the basic mechanism of echo beating, has not been ruled out.

With the continuing improvement of the performance of pacemakers and the accumulated experience in their clinical use, pacemaker implantation is now considered in selected cases of bradycardia not due to A-V block. The common occurrence of retrograde conduction across the A-V node when the ventricle is paced, and the relatively intact antegrade A-V pathway available in the absence of A-V block supply the necessary circuit for a re-entry mechanism which may become operative if retrograde conduction is delayed. This interesting physiological phenomenon and seemingly benign mechanism may give rise to a potentially dangerous situation if a fixed-rate pacemaker delivers a stimulus on the apex of the T wave of a reciprocally beat induced by the preceding paced ventricular beat. Since demand pacing provides a more rational method of treatment in this situation, it was utilized in our two patients. In one patient the re-entry mechanism was eventually abolished. In the other patient, when a reciprocal beat occurred, it was sensed by the pacemaker and the next stimulus was blocked, obviating its delivery on the apex of the T wave.

References


