Apparent Malfunction of Demand Pacemaker Caused by Nonpropagated (Concealed) Ventricular Extrasystoles*

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An example of apparent malfunction of an implanted R-wave-inhibited demand pacemaker is described. The pacer arrhythmia was characterized by slowing, with lengthening of spike-to-spike cycles from the preset value of 0.86 sec (heart rate 70) to as long as 1.34 sec (heart rate 43). Spike-to-spike intervals containing visible PVB's were uniformly longer than 1.34 sec. At times, the pacer was completely inactivated and the heart was controlled entirely by conducted impulses from the fibrillating atria. Both the sensing and the pulse generating circuits of the pacemaker were found to be free of malfunction. The slowing was shown to be the result of concealed ventricular extrasystoles occurring within 0.48 sec of the preceding pacer discharge. Though they failed to propagate to the entirety of the ventricular myocardium and produce visible QRS complexes, they were able to generate sufficient electrical energy, perhaps within the Purkinje fibers and the myocardium immediately surrounding the electrode, to recycle the pacemaker. The pacemaker in this case proved instrumental in documenting the existence of the otherwise elusive entity of concealed ventricular extrasystoles. It is suggested that concealed ventricular extrasystoles should be considered in cases of pacemaker slowing and appropriate measures aimed at suppressing premature ventricular beats instituted before decision for replacing the pulse generator is reached.

The mechanisms underlying malfunction of demand or standby pacemakers resulting in arrhythmic pacing of the heart are numerous and can be generally classified into three categories. These are: a) failure of the generator-lead assembly to deliver stimuli of adequate amplitude commensurate with the myocardial threshold, b) failure of the sensing circuit to perceive each QRS complex either because of a loss of adequate responsiveness, or because of insufficient QRS amplitude. The third important malfunction is also related to the sensing circuit. In this, extraneous electrical potentials, or endogenus non-QRS potentials such as large T waves, are mistaken for QRS, and the pacemaker accordingly recycles or ceases to function altogether.1 This communication reports a case of striking pacer arrhythmia with periods of slow discharge and occasional lapses of pacing caused by nonpropagated premature ventricular beats (PVB). The concealed PVB's were first recognized by their effects on the pacer cycles. Subsequently, the pacer malfunction was duplicated by external subthreshold stimuli delivered to the right ventricle through an electrode catheter placed immediately adjacent to the permanent pacemaker catheter. It is suggested that the concealed PVB's were confined to the Purkinje network and the adjacent subendocardial myocardium, but were not propagated to the entire ventricle, and hence were unable to produce visible QRS complexes, probably because of incomplete recovery or diminished excitability of the surrounding myocardium. To our knowledge, this case presents the most direct evidence thus far obtained for the existence of concealed PVB's, and furthermore, is the first example of pacer arrhythmia caused by such PVB's. Awareness of this phenomenon may uncover other similar cases and obviate the necessity of replacing pacemaker generators which are free of intrinsic malfunction.

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Description of the Electrocardiograms

Figure 1: The top strip shows leads I, II, V1 and V6 taken two weeks before insertion of an implanted pacemaker in this 84-year-old man. The atrial fibrillation, the slow ventricular rate of 40 to 54 beats/min, together with normal QRS complexes are to be noted. Strip B dated February 20, 1971 was taken shortly after percutaneous insertion of an R-wave inhibited, demand-type Medtronic pacemaker (model 5842). Pacer spikes are very small with sharp deflections at the beginning of the QRS complexes. The lead was bipolar and was inserted in the apex of the right ventricle. Regular uninterrupted pacing at the rate of 70/min representing pacer function in escape mode is present. When examined on April 12, 1971, the pacer discharges were noted to be quite irregular and frequent PVB's were noted. The patient was admitted to the hospital for observation, but during three days of monitoring the rhythm was regular and he was therefore discharged. On a subsequent visit on May 3, 1971, the arrhythmia was again observed together with PVB's and patient was once more hospitalized for possible replacement of the pulse generator. The amplitude and spatial direction of the pacer spikes did not indicate battery failure and the catheter tip appeared to be properly wedged in the apex of the right ventricle. Strips C, D and E in this illustration show several long cycles, the longest seen in strip C measures 1.34 sec as compared with the intrinsic escape cycle length of 0.88 sec. No abnormally short cycles were found at any time. It was clear that some electrical energy transients were recycling the pacer.

In Figure 2, five strips from the monitor recorder are illustrated. The same pacer arrhythmia was again observed (strips A to C). The striking feature here was the frequency of visible PVB's which interrupted the basic tracing and caused recycling of the pacer. It should be noted that the long spike-to-spike intervals containing PVB's are 1.48 sec as

Figure 2. Rhythm strips taken during the three days of the second hospitalization showing the coexistence of pacer arrhythmia with PVB's in strips A, B and C, ventricular tachycardia in strip D and complete disappearance of both PVB's and pacer arrhythmia after administration of lidocaine.

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compared with other long spike-to-spike intervals not containing PVB's all of which were 1.34 sec or shorter.

In some areas, such as the one in the middle of strip C, slowing of the pacer results in a complete take-over by several conducted beats from the fibrillating atrium. These QRS complexes are irregular and narrow, identical with those observed before insertion of the pacer. Two days after admission and while being monitored, the patient developed a paroxysm of tachycardia which was thought to be ventricular in view of the abundance of fusion forms (strip D). An intravenous injection of lidocaine promptly stopped the tachycardia, and with it, the pacer arrhythmia disappeared. Three more days of monitoring showed clearly that pacer arrhythmia was present only when visible PVB's were also present and it was absent when no PVB's were found in long rhythm strips. Pacer discharges were quite regular when PVB's were absent either spontaneously or during infusion of a lidocaine drip. Administration of quinidine by mouth had a similar protective effect and was found sufficient for eliminating the pacer arrhythmia altogether. It was concluded that the pacer arrhythmia was the result of recycling by invisible, "concealed" PVB's which were confined to the area near the tip of the catheter, remained nonpropagated, but created sufficient electrical energy from depolarization of the Purkinje fibers and the surrounding myocardium to be perceived by the sensing circuit of the pacemaker. The fact that spike-to-spike intervals containing nonpropagated PVB's were always shorter than those containing visible PVB's indicates that the concealed PVB's occurred earlier in the cycle, before the ventricular myocardium was completely recovered. Subtracting the intrinsic pacer escape interval of 0.86 sec from the spike-to-spike interval of 1.34 sec, (1.34 - 0.86 = .48) one may conclude that PVB's occurring within 0.48 sec after the pacer discharge were able to recycle the pacer but could not propagate to the myocardium of the ventricles because of the incomplete recovery of the latter.

In order to test the hypothesis regarding the concealed PVB's, a temporary pacemaker catheter was inserted percutaneously and its tip positioned fluoroscopically in the immediate vicinity of the permanent catheter for the purpose of duplicating the pacer arrhythmia through externally generated subthreshold stimuli. Figure 3 containing strips A and

![Figure 3](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21537/) Pacer arrhythmia (A) and PVB's (B) reproduced by external stimuli delivered to the right ventricle near the tip of the permanent pacer catheter.

![Figure 4](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21537/) External stimuli (S₀) are delivered after four regular pacer beats 1 to 4. Stimuli delivered after beats 4, 5 and 6 recycle the pacemaker, while those delivered a few hundredths of a second later after beats 7 and 8, and for many beats thereafter not shown here, deactivated the pacer altogether and caused a complete return to conduction from the fibrillating atria.
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FIGURE 5. With a magnet superimposed on the pacemaker generator, the sensing circuit is completely inactivated. External stimuli $S_m$ now do not recycle the pacemaker.

B shows a typical example of records so obtained. Each strip, lead II on top, and $V_1$ in the middle, is recorded simultaneously with the input from the poles at the tip of the temporary pacemaker catheter. Beats 1, 2 and 3 are pacemaker beats produced by implanted pacemaker stimuli marked $S_a$. After beats 3, 4 and 5, test stimuli marked $S_b$ were delivered to the right ventricle through the tip of the test catheter. The test stimuli were coupled to the preceding QRS complexes at predetermined intervals of 225 msec by means of a Medtronic pulse generator (model 5837). Stimuli of 0.10 to 0.20 mA intensity were able to recycle the pacemaker even though they were not propagated to the ventricular myocardium. Note the recycling of the pacemaker and the absence of QRS after stimuli marked $S_a$. In strip B, the amplitude of the test stimuli was increased to 0.30 mA. These are followed by PVB's numbered 8, 10, 12 and 14 with the expected recycling of the permanent pacemaker, identical with the recycling noted in strip A.

Figure 4 shows that nonpropagated PVB's delivered in a strategic area of the cycle could completely suppress the permanent pacemaker and allow the fibrillating atria to control the ventricles as in beats 8 and 9. In this situation, complete return to conducted beats could be achieved for long periods of time.

In order to test the sensing circuit and also the pacemaker function in a fixed-rate mode, a magnet was applied to the battery pack and the pacemaker was converted to a fixed-rate unit. Figure 5 shows recordings made with the magnet applied continuously. Pacemaker discharges at the rate of 71 per min continued so long as the magnet was in place. It was possible at this time to deliver both subthreshold and suprathreshold stimuli ($S_m$) within consecutive pacemaker discharges without recycling the pacemaker. Stimuli $S_m$ in the top strip are subthreshold. They do not produce QRS and do not change the basic pacemaker cycles. Stimuli $S_m$ after beats 9, 11, 13 and 15 are above threshold and are followed by PVB's numbered 10, 12, 14 and 16. The pacemaker, however, cannot "see" these PVB's because of inactivation of the sensing circuit by the overlying magnet and therefore continues to discharge at its fixed rate. This results in interpolation of the PVB's between pacemaker-induced beats.

DISCUSSION

The most important cause of slow pacing with demand pacemakers is the non-QRS electrical po-

FIGURE 6. Apparent malfunction of a demand pacemaker caused by PVB's originating from the left ventricle and therefore "seen" by the right ventricular pacemaker catheter 0.07 sec later.

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tentials which are misinterpreted by the sensing circuit of the pacemaker assembly as QRS, and thus, cause recycling or deactivating of the pulse-generating circuit. Currents from such electrical apparatuses as shavers, kitchen appliances, automobile ignition system are well-known examples. Less well known are large P and T waves in some individuals, break currents, loose connections at the lead-battery junction, and occasionally slipping of the electrode or delayed capacitance discharges.\textsuperscript{1,2} Minor pacer arrhythmias causing rate fluctuations of up to three or four beats per minute may be expected in demand pacemakers because of the occasional shifts of the pacing mode from one of demand to one of fixed rate. According to Kas-tor and co-workers\textsuperscript{3} the intrinsic pacer rate in fixed-rate mode always exceeds the rate in demand or escape mode. When a R-wave inhibited demand pacemaker is functioning in demand mode, it fires only if it fails to sense an endogenous QRS within a predetermined constant escape interval. Since the endogenous QRS complexes may be of different origins, and hence, variable in configuration and in duration, the sensing circuit of the pacemaker is triggered by the initial portion of one type of endogenous QRS, its midpoint in another type and by its terminal portion if the QRS originates from the contralateral ventricle. Although the pacemaker irregularity caused by this phenomenon is of considerable practical importance, the alterations of rhythm are not great and spike-to-spike cycles may vary by no more than 0.04 to 0.08 sec which is the interval required for a PVB from the left ventricle to reach the endocardium of the right ventricle and be sensed by the demand pacemaker. Differences in spike-to-spike intervals of almost 0.50 sec as seen in the present case certainly cannot be explained on this basis. In Figure 6, an example of an apparent malfunction of an R-wave-inhibited demand pacemaker is illustrated. The first five and last five beats in both strips A and B are pacer-induced beats with cycles of 0.62 sec. The premature ventricular beats marked by X show features resembling right bundle branch block (RBBB) in lead V\textsubscript{1} and therefore originate from the left ventricle. They are sensed by the pacemaker and cause it to recycle. However, the interval from the onset of the PVB's to the next pacemaker discharge is 0.69 sec; a value longer than the preset spike-to-spike interval of 0.62 sec. The difference of 0.07 sec suggested malfunction. Actually, however, this is to be expected because the PVB's, originating from the left ventricle are not “seen” by the pacemaker lead lying in the right ventricle until after they have traversed the interventricular septum and reached the endomyocar-dium of the right ventricle. The interval of 0.07 sec is consistent with this thesis.

In the case presented here, pacer arrhythmia occurred in the absence of any change in QRS configuration (Fig 1) and the cycle variations were too large to be explained on the basis of a shift from demand to fixed rate mode. The recurrent association between the pacer arrhythmia and the emergence of PVB's and its complete disappearance after treatment with lidocaine and subsequently with quinidine, together with the ease of reproducing an identical arrhythmia by exogenous, subthreshold stimuli leave little doubt as to the mechanism of pacer arrhythmia in this case. It is indeed remarkable that similar cases have not been observed in the past. The case of Lasseter and associates\textsuperscript{5} was similar to ours in many respects. Their published tracings show numerous PVB's and also areas of pacer arrhythmia not related to visible PVB's. While the authors concluded that pacer arrhythmia was related to changes in resistance secondary to intermittent fracture of the lead system, no special studies were carried out to exclude concealed PVB's as the responsible mechanism.

That premature beats may fire but fail to propagate to the myocardium, thus remaining invisible or concealed was suggested by Langendorf and Mehlan in 1947.\textsuperscript{4} In their case, nonconducted A-V nodal premature systoles were presumed to occur and account for the otherwise unexplained first and second degree A-V block. Similar observations were made by Need and Fisch\textsuperscript{6} in 1962. It was not until almost a quarter of a century after the original observation that this mechanism was documented by His bundle electrography in a patient by Rosen et al.\textsuperscript{6} In this case, unexpected prolongation of the P-R interval or complete block of the P waves was shown clearly to be related to the appearance of isolated His spikes, i.e., nonpropagated His bundle extrasystoles. The existence of concealed PVB's was first suggested by Schamroth and Marriott in 1961.\textsuperscript{7} In two cases of ventricular bigeminy, the authors noted intermittent disappearance of the PVB's. However, the long intervals separating the visible PVB's always contained an odd number of normal sinus beats such as 3, 5, 7, 9 and 11. The authors concluded that firing of the ectopic ventricular beats had persisted throughout even in the PVB-free intervals. Two years later, the same authors published new examples of concealed bigeminy and trigeminy.\textsuperscript{8} Friedberg\textsuperscript{8} recently published a convincing account of concealed PVB's which occurred in company with manifest PVB's and altered the basic rhythm of 2-1 A-V block in a similar manner.

A distinction should be made at this point be-
tween concealment and exit block. In exit block, an impulse-generating focus fires, but the discharges fail to exit from the generating focus. Grouping of sinus P waves following Wenckebach cycles or sudden doubling or halving of the rate in junctional rhythms are consistent with exit block of otherwise regularly discharging impulses. A ventricular extrasystole may remain hidden from view because it fails entirely to exit from its focus, or the impulse does exit from its focus and even propagates to the Purkinje system or the His bundle-branch system, but fails to depolarize the myocardium because of the refractoriness of the latter. In this example, the impulses are concealed. In the case presented here, it must be assumed that the PVB's originated near or at the electrode tip possibly because of the mechanical contact. Why the excitation wave did not propagate to the entire ventricular myocardium is not known. It may be speculated that the Purkinje fibers and the myocardium surrounding the electrode tip were more excitable than the remaining portions because of the presence of some injury caused by contact with the catheter tip.

**Editorial Expression**

It has been known that an ineffective stimulus of a failing electronic pacemaker can prevent cardiac stimulation by a well functioning demand pacemaker if the rate of the former exceeds that of the latter. A related phenomenon is described for the first time by Massumi and associates—postponement of the demand pacemaker stimulus by an ineffective spontaneous ventricular impulse, a ventricular premature impulse which does not yield a QRS complex, a "concealed ventricular premature systole." The interest of their observation goes far beyond its practical significance (avoidance of an erroneous diagnosis of pacemaker malfunction) because it lends strong support to Schamroth and Marriott's hypothetical concept of "concealed ventricular extrasystoles." Finally, this report recalls the concept of concealed ventricular escape beats, "embryonic escapes," whose phase-4 depolarization is held responsible for conduction delay or block of supraventricular impulses (Singer et al: Circ Res 21:537, 1967) and has been postulated to account for bradycardia-dependent bundle branch block (Massumi: Circulation 38:1066, 1968) and bradycardia-dependent atrioventricular block (Coumel et al: J Electrocardiol 4:168, 1971).

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