A Mechanism for "False" Inhibition of Demand Pacemakers

By KENNETH C. LASSETER, M.D., JACK W. BUCHANAN, JR., M.S.E.E.,
and KARL F. YOSHONIS, M.D.

SUMMARY
Certain variations in discharge rate of demand pacemakers may be associated with intermittent fractures and resulting resistance changes in the electrode-lead system. These variations in rate are related to inhibition of the demand circuit of the pacemaker. Most pacemakers produce a biphasic output pulse which results in a low amplitude current during the interval between stimuli. Therefore, sudden changes of resistance in the electrode-lead system will produce a potential change at the pacemaker terminals which is of sufficient magnitude to be interpreted as spontaneous ventricular depolarization and inhibit the demand pacemaker. Such inhibition may occur in the absence of other signs of lead fracture and present a difficult differential diagnostic problem of pacemaker malfunction.

Additional Indexing Words:
Atrioventricular conduction disturbances
Pacemaker failure
Lead fracture
Ventricular inhibited pacemaker
Biphasic stimulation

The use of ventricular inhibited (demand) pacemakers in the treatment of atrioventricular conduction disturbances has introduced problems of sensing in addition to those of adequate stimulation. Variations in discharge rate are known to occur if the pacemaker recognizes other ECG deflections or extraneous electrical interference in addition to the QRS complex, or if there is circuit malfunction. Recently Kastor and associates reported that benign variations in the discharge rate of demand pacemakers may be due to variations in intraventricular conduction and the time of sensing. We have observed marked variations in the discharge rate of ventricular inhibited pacemakers in the absence of detectable extraneous interference or circuit malfunction in several patients. In one of these patients, such variations were found to be related to inhibition of the pacemaker by intermittent changes in lead resistance. This "false" inhibition of demand pacemakers has been reproduced in a laboratory model and the underlying mechanisms investigated.

Report of Case
A 46-year-old white female presented with a 3-day history of syncopal episodes. Physical examination revealed a heart rate of 30/min and the electrocardiogram revealed complete atrioventricular block. A temporary transvenous bipolar pacing catheter was placed in the apex of the right ventricle and connected by an extension cable to the terminals of a Medtronic model 5840 External Demand Pacemaker. The patient improved clinically, and the ECG demonstrated normal function of the demand pacemaker in response to occasional extrasystoles (fig. 1A). The patient was monitored continuously in the coronary intensive care unit where all equipment is appropriately grounded. Six hours after inser-

From the Cardiovascular Division, Department of Medicine, University of Kentucky College of Medicine, Lexington, Kentucky.

Supported in part by Training Grant HE 05598 from the National Institutes of Health, U. S. Public Health Service.

Address for reprints: Department of Pharmacology, University of Miami School of Medicine, Miami, Florida 33152.

Received June 11, 1970; revision accepted for publication August 10, 1970.
Figure 1

Serial strips of ECG lead II recorded during temporary transvenous pacing with Medtronic model 5840 demand pacemaker. Pacer rate remains at 82/min throughout the tracings. (A) Normal function shortly after insertion demonstrating inhibition of discharge by spontaneous beats. (B and C) Marked variations in pacemaker escape interval subsequently related to movement of extension cable. (D) Normal function of pacemaker in fixed rate mode. Note the pacemaker artifact after spontaneous beat.

Experimental Results

Resistance measurements of the bipolar pacing catheters used in this institution revealed that several such catheters exhibited this same phenomenon of motion-related intermittent changes of resistance. Figure 2 illustrates an experiment with one such catheter. The output of the Medtronic 5840 Demand Pacemaker was recorded at the catheter tip using a 500Ω (ohms) resistor to simulate myocardial resistance. The intermittent changes in resistance did not interrupt conduction since no beats were lost during fixed rate pacing and since one beat was conducted with decreased amplitude during the high resistance phase in panel C (fourth from last beat).

The circuit diagrammed in figure 3 was constructed as a model of this type of demand pacemaker failure. Resistor R₁ had a constant
value of 500Ω and resistor R2 varied in value. Figure 4 illustrates representative experiments with this model using a typical value of 10,000 Ω for R2. In figure 4A, the eighth beat is delayed until 750 msec after switching resistor R2 into the circuit. This interval is the escape interval of the pacemaker, indicating that the pacemaker was “reset” by this event. If R2 is constantly left in the circuit, no change in rate is seen although there is diminution of the stimulus strength. It should be noted that if R2 is switched into the circuit during the refractory period of the pacemaker, no delay is seen. In Figure 4B, R2 is intermittently and randomly switched in and out of the circuit to simulate intermittent fracture of a lead. Each delayed beat occurs 750 msec after a change of resistance, either switching R2 into or out of

Figure 2
Recordings of pacemaker output at catheter tip with a Tektronix 564B oscilloscope with a 500Ω load. Pacemaker rate remains at 60/min throughout recording. (A) Normal pacing in demand mode. (B and C) Variations in discharge rate produced by manipulation of catheter. (D) No variation of rate during catheter manipulation in fixed rate mode.

Figure 3
Circuit diagram of model to simulate changes in lead resistance. CRO = cathode ray oscilloscope. R1 = 500Ω load simulating myocardial resistance at electrodes. R2 = variable resistance of lead fracture. Rapid opening and closing of switch simulates intermittent fracture.

Figure 4
Recordings of pacemaker output with the model diagrammed in figure 3. The upper trace in each panel records amplitude and rate of pacemaker output. In the lower trace in each panel the positive steps indicate that resistor R2 is in the circuit. R2 had constant value of 10,000Ω in these experiments. Pacemaker rate = 80/min.
the circuit. Various combinations of switching \( R_2 \) in and out of the circuit can result in wide variations in pacemaker discharge rate. Figure 4C demonstrates that if such sudden changes in resistance occur at the proper frequency, long periods of pacemaker silence can be induced. All values of \( R_2 \) greater than 1,000\( \Omega \) were found to inhibit the demand pacemaker. Similar inhibition has been demonstrated with the Medtronic Implantable Demand Pacemaker model 5841 and with the General Electric Implantable Demand Pacemaker model A2072A.

**Discussion**

Most commonly used cardiac pacemakers produce a biphasic output pulse which results in no net transfer of energy during each cycle. The large amplitude, short duration output pulse is delivered by a capacitor which is then recharged by a low amplitude, long duration current during the interval between stimuli. This current is shown in figure 5 as the voltage drop across the 500\( \Omega \) load. The instantaneous magnitude of this current is dependent on the output stimulus setting and decreases exponentially with time.

At a pacemaker setting of 3 ma, which might be used clinically, and at 500 msec, a time clearly after the refractory period of these pacemakers, the magnitude of this current is about 4 microamperes (\( \mu \)A) (fig. 5). In the circuit diagram in figure 3, the potential across the pacemaker terminals (\( E_p \)) at any given time is dependent on the current (\( I \)) in the circuit and the series resistance. When the switch is closed (representing no lead fracture),

\[
E_p = IR_1
\]

When the switch is open (representing lead fracture),

\[
E_p = I (R_1 + R_2)
\]

The potential change produced by opening the switch would be

\[
\Delta E_p = IR_2
\]

Assuming a current of 4 \( \mu \)A, a sudden resistance change of 1,000\( \Omega \) (the smallest value found to inhibit the pacemaker) would produce a potential difference of the order of 4 mv at the pacemaker terminals.

The magnitude of the ventricular depolarization potential, recorded with an endocardial electrode, is of the order of 8 to 10 mv. To provide a margin of safety, the magnitude of the signal required to inhibit most demand pacemakers is less than this value and is of the order of 1.5 to 2.0 mv. Any sudden potential change of this magnitude could be interpreted as a spontaneous ventricular depolarization and could inhibit the pacemaker.

**Figure 5**

Recordings of current between stimuli of Medtronic 5840 Demand Pacemaker as the voltage drop across 500\( \Omega \) load. At this sensitivity only the negative component of the biphasic stimulus pulse is seen, the positive spike being far off the screen. (A) Pacemaker stimulus setting of 3 ma. (B) Pacemaker setting of 25 ma. Vertical scale: 5 mv/division which corresponds to 10 \( \mu \)a/division. Horizontal scale: 500 msec/division. The solid line in each panel represents the zero potential.
The potential difference produced by a sudden resistance change in the electrode system can, therefore, inhibit the pacemaker.

In the case reported herein, such sudden resistance changes did inhibit the pacemaker. The basic escape interval of this pacemaker is 730 msec (fig. 1). The irregular beats exhibit R-R intervals from 900 to 3,000 msec, none of which were even multiples of the escape rate. The demonstration of the temporal relationship between these intervals and maneuvers which produced sudden changes in the resistance of the electrode-lead system, strongly supports this as the mechanism of pacemaker inhibition in this case. Zuckerman and associates have reported a case, in which intermittent variable prolongation of the pacer escape interval developed 1 year after transthoracic implantation of a ventricular inhibited (demand) pacemaker. This was attributed to an intermittent electrode connection "which emitted a signal and produced false cancelling." This problem was overcome by removing the faulty lead from the circuit and pacing unipolarly. The same mechanism would seem to explain the pacemaker inhibition in each of these cases.

Although lead fracture is a well-recognized complication of cardiac pacing, it would not generally be expected to change the escape interval of the pacemaker except by failure to pace. It has been postulated that body fluids may enter the fracture of an intracorporeal lead allowing adequate conduction of the stimulus at all times. In this situation the conductor could have two resistance states, either of which would conduct the stimulus. A change between these states might produce a potential change adequate to inhibit the pacemaker. Variation in the pacemaker discharge rate due to sudden resistance changes in the electrode system is a potential problem in all pacers using biphasic stimulation and common electrode systems for stimulation and sensing. This includes not only the most ventricular inhibited (demand) pacemakers but ventricular-triggered pacemakers as well. This is not a problem with continuous asynchronous (fixed rate) pacemakers because of the absence of a sensing circuit, nor in the atrial-triggered pacemakers because of the separation of the sensing and stimulating electrode systems.

When triggered pacemakers are used, the physician should be aware of the additional problems introduced and should be able to identify and correct the source of these problems. We have shown that at least one type of triggered pacemaker, the ventricular inhibited (demand) pacemaker is sensitive to defects in its lead system which would not be apparent with asynchronous pacing. This problem can be overcome by identifying and replacing the defective component of the electrode lead system.

References

A Mechanism for "False" Inhibition of Demand Pacemakers
KENNETH C. LASSETTER, JACK W. BUCHANAN, JR. and KARL F. YOSHONIS

Circulation. 1970;42:1093-1097
doi: 10.1161/01.CIR.42.6.1093
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1970 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/42/6/1093

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/