Measurement of pacemaker performance

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This report concerns a simple method of assessing pacemaker function by externally analyzing its signal. The method has proved to be of practical value, not only in monitoring pacemaker performance after implantation, but also in identifying the cause of "failure" when and if it occurs. Satisfactory treatment of complete heart block with electronic pacemakers has been complicated by an inability to measure pacemaker function after implantation. The duration of artificial pacing is limited by the "life" of the individual unit, and when it fails, reimplantation of a new pacemaker is usually mandatory. Routine replacement of each pacemaker, after 15 months of service, has been recommended as one method of assuring a high probability of trouble-free performance.[1] Such prophylactic replacement is reasonable in the absence of adequate knowledge of pacemaker function after implantation, since the only alternative is for the physician to await failure before recommending replacement, thus incurring the risk of untreated complete heart block during the interim period.

Two major problems can be identified in the long-term management of pacemaker patients: first, uncertainty of when a pacemaker will fail; and second, accurate information about the cause for pacemaker failure if it occurs unexpectedly. The former is not only medically important for the protection of the individual patient, but economically important for assurance of full use of the pacemaker prior to replacement. The latter is also vital, since the management of different types of pacemaker "failures" varies, depending on the cause.[2-6] The method to be described enables the physician to answer both of these vital questions. Although this method is applicable to only one of the commercially available units† at the present time, the problems of management are common to all artificial pacemakers.

Materials and methods

This study consisted of three phases of investigation. During the first phase, in order to analyze the exact shape and magnitude of the pacemaker waveform,

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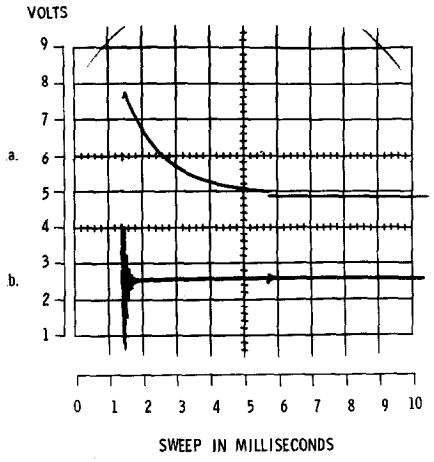

Fig. *1a*, Oscilloscopic picture of pacemaker pulse waveform, showing sudden onset, RC decay, and abrupt termination. From onset to termination is the pulse width. *b*, Detector-coil stimulus deflections, coinciding with beginning and end of pacemaker pulse. Note that the onset deflection is greater than the termination deflection, due to greater change in current.

measurements of voltage between the myocardial electrodes were made from a series of 4 dogs with external pacemakers. This was done by using a calibrated, cathode-ray oscilloscope (Tektronic, type 502), capable of pulse synchronization and having a sweep of 1 millisecond per centimeter. Fig. 1,*a* shows a photograph of the voltage of a pacemaker pulse as seen on the oscilloscope. Multiple measurements were made on each animal, and were recorded by means of a Polaroid camera. To measure current, a 1-ohm resistance was inserted in series with the heart impedance, and by the same method the voltage across this resistance was measured. The voltage value thus obtained is equivalent to current. Total pulse energy was then calculated from these two curves by multiplying average voltage times average current for each millisecond throughout the pulse, and integrating the millisecond decrements of energy for the duration of the pulse.

The second phase of the investigation was the construction of a laboratory model with a simulated interelectrode impedance identical to the "biologic" impedance as calculated from the dog studies. This model allowed precise variation of load impedance and battery voltage so as to simulate all possible clinical conditions, with measurement of pacemaker voltage and current using the method described above. It was found that, for any biologic impedance, an equivalent electrical resistance and capacitance could be substituted.

Investigation showed that the pulse duration, or pulse width, can be exactly measured by placing a detector coil (7,000 turns of No. 38 magnet wire) on the surface of the skin over the pacemaker unit. Fig. 1,*b* shows oscilloscopic measurement of the pulse width. It is the distance between the two deflections, due to voltages induced in the detector coil by the rapidly changing currents during the upstroke and downstroke of the pacemaker pulse. The upstroke of the pulse represents a greater rate of change in current than does the downstroke, and therefore induces in the detector coil a greater voltage. The two deflections correspond exactly to the onset and termination of the pacemaker pulse.

The third phase of the investigation was the application of these methods to the human heart. The first step was to make direct measurements of voltage and current from the external pacemaker electrodes of one patient, in order to be sure that there were no significant differences between canine and human cardiac impedances. Then, two pacemakers were carefully precalibrated (rate and pulse width determined for a range of biologic impedances), after which they were implanted into patients. Serial measurements were made of exact pacemaker rate and pulse width, using the detector method. This was done externally, by placing the detector coil over the implanted pacemaker. Finally, we applied these methods to a clinical series of 42 patients whose pacemakers were not specially calibrated for all possible impedances.

Results

The studies in dogs showed that an average interelectrode impedance was about 300 ohms resistance and 47 microfarads capacitance. Interelectrode impedance measured from the human heart was not significantly different. Total output of pacemaker pulse energy in dogs averaged 60

microjoules. In the one patient in whom it was measured, the pulse energy was 67 microjoules.

The pacemaker used in this study has a resistance-capacitance (RC) discharge waveform as shown in Fig. 1,*a*. Any biologic load can be replaced by an indistinguishable simulated load of resistance (R_L) in series with capacitance (C_L), as shown in Fig. 2. Using variable simulated interelectrode impedances (R_L and C_L), and varying battery voltage, we found that changes in any of these three parameters were reflected by measurable changes in one or both of two clinically obtainable variables, pacemaker rate and pulse width. Especially noteworthy was the finding that a change in R_L caused a measurable change in pulse width, as shown in Fig. 4. Varying R_L did not change pulse rate until R_L exceeded 5,000 ohms. In all animal and human measurements, R_L was in the range of 200 to 500 ohms, provided that the pacemaker was functioning normally. We never observed it to exceed 500 ohms. Therefore, it could be concluded that biologic increases in R_L do not affect the pacemaker rate.

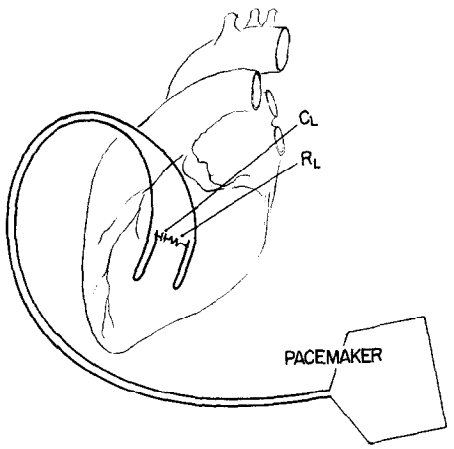

Fig. 2. Schematic representation of pacemaker interelectrode, or myocardial impedance. R_L (load resistance) and C_L (load capacitance) are in series with the pacemaker electrodes.

Changes in C_L, on the other hand, caused a proportionate change in pulse width, and an inverse change in pacemaker rate. Fortunately, changes in pulse width were minimal and within the limit of error of the method (1/10 millisecond). Changes in rate, however, were very significant when C_L varied within the observed biologic range. A variation of 20 microfarads produced a detectable variation in rate of 3 pulses per minute in the range of the average biologic value of C_L (47 microfarads). See Fig. 3.

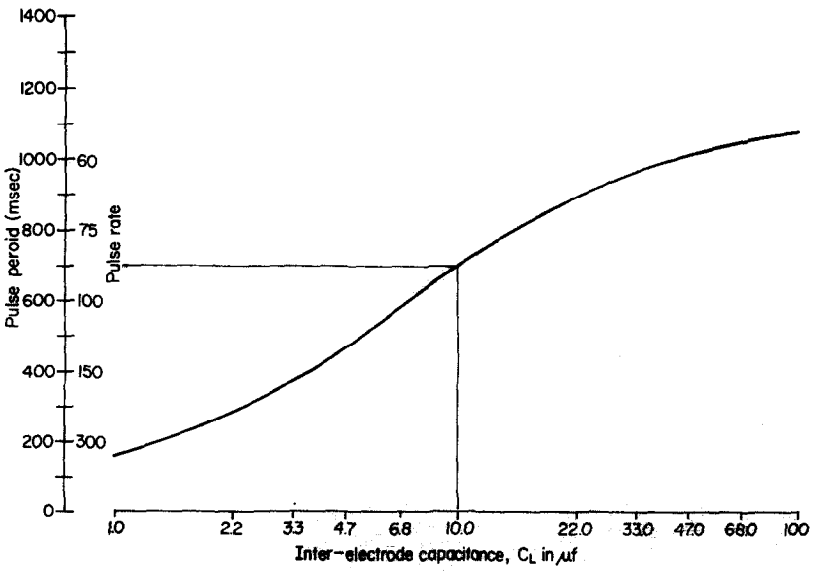

Fig. 3. C_L versus pulse period in milliseconds. The pulse period is the time between pulses. Approximate rates are shown in parentheses. In the example shown, a rate of 86 gives a C_L of 10 microfarads.

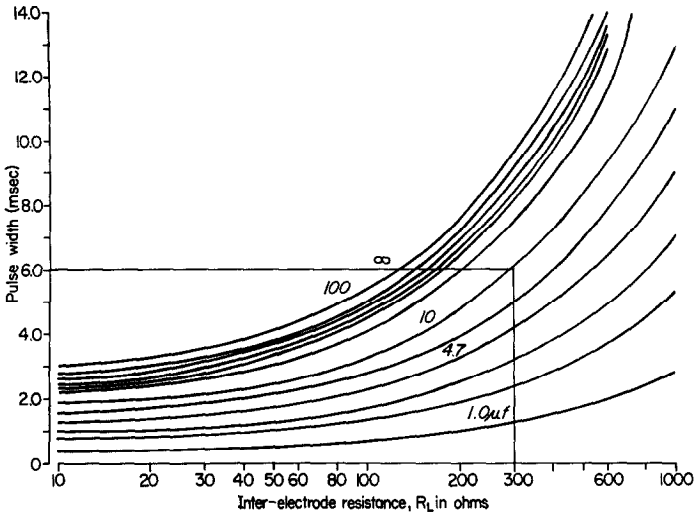

Fig. 4. R_L versus pulse width. In the example shown, for $C_L = 10$, a pulse width of 6.0 gives $R_L = 300$ ohms.

Changes in battery voltage also affected both rate and pulse width, but to an equal degree. A decrease of 7 per cent (0.5 volt) in the voltage of the main battery resulted in a 3.5 per cent increase in rate (2 pulses per minute), and at the same time caused a 4 per cent increase in pulse width (0.3 millisecond). These changes pertain only if the bias battery remains constant. A decrease in the voltage of the bias battery results in a decrease in rate and no change in pulse width.

Application of these observations to the practical problem of monitoring pacemaker function resulted in the following deductions: (1) An isolated increase in pulse width, with no change in rate, denotes a predictable increase in interelectrode resistance. (2) An isolated decrease in rate with no change in pulse width denotes a predictable increase in interelectrode capacitance. (3) Progressive, simultaneous increases in both pulse width and rate most likely represent a decreasing voltage of the main battery. Although this combination could be simulated by an *increase* in R_L with a concomitant *decrease* in C_L, subsequent clinical observations proved that this possibility was unlikely. (4) Abrupt, sizable increases in rate and pulse width, with or without variations from one pulse to the next, cannot be accounted for by biologic variations in R_L and C_L, and therefore can only be produced by a lead fracture, which would suddenly introduce a marked change in both R_L and C_L. Subsequent experience showed that the open circuit produced by an electrode break is frequently bridged by an impedance which may be variable, causing changes in R_L and C_L from one pulse to the next.

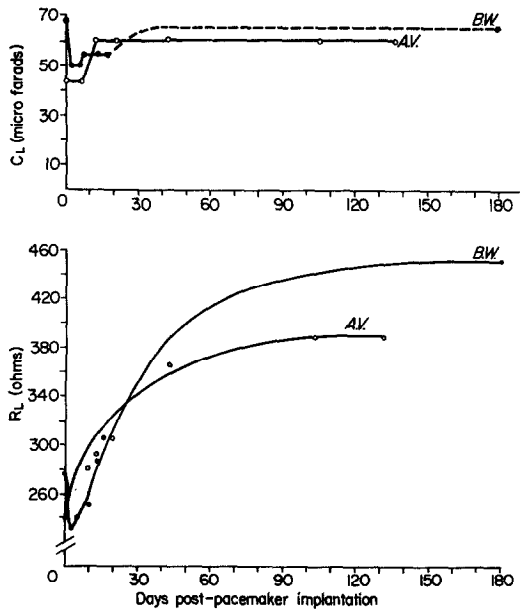

Fig. 5. R_L and C_L versus time for 2 patients with precalibrated pacemakers. Note the rise in R_L, while C_L becomes constant after the postoperative period.

Application of these principles to the human problem began by our making serial measurements of rate and pulse width on 2 patients whose implanted pacemakers had been precalibrated by determining rate and pulse width for various values of R_L and C_L within biologic limits. The values of these two variables were determined over a period of 6 months, and are shown in Fig. 5. Note that capacitance varies insignificantly after implantation, whereas resistance tends to plateau after an initial rise.

Measurements were also made on a total of 42 patients whose pacemakers were not precalibrated. In 19 of these, serial measurements were made from the day of implantation; in 10, serial measurements were made beginning 1 month or more after implantation; and in 13, only one measurement was made. See Table I.

Discussion

There is general concern that the electrochemical effect of implanted cardiac pacemakers on the tissue surrounding the electrodes, and on the myocardium between them, may ultimately alter pacemaker performance. After direct implantation of electrodes, the interelectrode impedance, with its resultant effect on myocardial threshold, has been shown to be altered by at least three factors: (1) the type of electrodes, (2) the pulse waveform, and (3) foreign-body reaction around the electrodes.[7-10] The results of this study show that interelectrode impedance does vary with time, in a predictable manner.

Other investigators have made analyses of electrical signals coming from implanted pacemakers, but these methods have not given definite information of diagnostic value about interelectrode impedance or changes in pacemaker performance.[11,12] The key to this method of analysis lies in the circuit design of this particular pacemaker, which has an output pulse that is dependent on the load. The entire electrical analysis can be performed externally in less than 1 minute, without directly touching the skin of the patient, and absolutely without harm. For this reason it should have practical, clinical value.

In determining interelectrode impedance by this method, we assume that battery voltage remains constant throughout the period of observation. We think that this is valid for three reasons. (1) The pacemaker power supply has been shown to reach a constant mercury voltage level, after which there is insignificant variation until the battery energy is near depletion. For the pacemakers used in this series, quality control requires constant output voltage for at least 3 months prior to implantation. (2) Serial measurements on five pacemakers incubated at 37°C. over a period of 3 years demonstrated no significant changes until one of the component batteries had reached the end of its life. (3) The rates of implanted pacemakers have been observed to be unexpectedly constant after the immediate recovery period. This means that voltage must also be constant, since the two are interdependent.

For a calibrated pacemaker, impedance is determined as follows: First, the rate is measured accurately, and from this rate the interelectrode capacitance is obtained from a plotted graph of C_L versus rate (see Fig. 3). Once C_L is known, R_L can then be determined from a graph of R_L versus pulse width for various values of C_L (see Fig. 4). The results show that pacemaker rate becomes constant within the first 6 weeks; therefore, interelectrode capacitance must become constant. The serial measurements after implantation, in 19 patients with uncalibrated pacemakers, showed a uniformly rising pulse width with constant rate. This indicates that the interelectrode resistance in all of these patients (in addition to the 2 patients with calibrated pacemakers) increased during the first 3 months after implantation. However, serial measurements in 10 cases, beginning at least 3 months after implantation, showed no further increases in pulse width. This leads to the conclusion that after 3 months R_L remains constant, or nearly so. The near-linear increase in R_L during the first 3 months after implantation can be attributed to changes at the electrode surfaces (electrolysis), in addition to fibrosis in the area surrounding the electrodes.

Changes in pacemaker performance result in definite and distinct changes in the

Table I. *Measurements of pulse width in 19 patients with pacemakers not calibrated for* R_L *and* C_L*

	Pulse width vs. days after implantation for 19 patients																		
	H.M.	*H.H.*	*R.B.*	*M.R.*	*M.Y.*	*E.D.*	*B.W.*	*F.H.*	*L.H.*	*A.V.*	*G.H.*	*F.L.*	*O.B.*	*B.N.*	*E.L.*	*W.D.*	*H.H.*	*M.G.*	*X.S.*
0	5.8	6.7	5.8	5.7	—	7.0	8.1	8.2	9.0	6.8	5.6	—	6.1	6.5	6.2	—	8.9	6.0	—
1	.0	6.2	—	6.1	—	—	—	—	—	—	—	6.3	—	6.1	6.6	5.3	8.5	—	6.1
2	—	—	5.6	6.1	6.1	6.7	7.3	7.7	8.7	—	5.8	—	6.5	6.0	6.9	—	—	6.0	—
3	6.5	6.5	—	—	6.2	—	7.2	—	—	7.2	—	6.2	—	—	6.9	5.4	8.8	—	5.9
4	6.8	—	5 5	6.3	—	6.8	—	—	8.5	7.3	5.9	—	—	—	7.1	—	—	6.0	—
5	6.9	6.6	—	—	6.2	—	7.3	—	—	—	—	6.4	6.4	—	7 3	5.6	8.9	—	6.1
6	—	—	5.6	—	—	—	7.4	8.4	8.5	7.5	6.0	—	—	6.0	—	—	—	6.0	—
7	—	7.0	—	6.4	—	7.0	7.5	—	—	7.6		6.4	6.6	—	—	5.6	—	—	6.2
8	7.4	7.0	—	—	6.4	—	7.6	—	8.7	—		—	—	5.9	—	—	9.0	—	—
9	—	7.1	—	—	—	—	—	—	—	—		6.6	—	5.9	7.4	5.7	—	—	6.4
10	7.6	—	—	6.5	6.6	7.2	—	—	8.9	—		—	—	—	—	—	—	—	—
11	—	—	—	—	—	—	7.9	—	—	—		—	—	—	—	5.7	—	—	6.4
12	—	—	—	—	—	—	—	—	9.1	—		—	—	—	—	—	—	—	—
13	—	—	—	6.6	—	—	8.1	—		—		—	—	—	—	—	—	—	—
14	7.9	—	—		—	—	—	—		—		—	—	—	—	—	—	—	—
15	—	7.7	—		—	—	—	—		8.0		—	—	—	—	—	—	—	6.4
16	8.1	—	—		—	—	—	—		—		—	—	—	—	—	—	—	—
17		7.8	—		—	—	—	—		—		—	—	—	—	—	—	—	—
18		8.0	—		—	—	8.7	—		—		—	—	—	—	—	—	—	—
19-21		8.3	—		—	—	—	—		—		—	—	—	—	—	—	—	—
22-30		8.3	6.5		—	—	—	—		8.2		7.3	—	7.0	—	—	—	—	6.8
31-40		9.1			—	—	—	—		—		—	—	7.4	—	—	—	—	
41-50					—	—	—	—		9.1		—	—	—	—	—	10.6	6.9	
51-60					—	—	—	—		—		—	7.7	—	—	—		—	
61-70					—	—	—	—		—		—	—	—	9.1	—		—	
71-80					—	—	—	—		—		—	—	—	—	7.7			
81-90					—	—	—	—		—		—	—	—	—	7.6		7.0	
91-100					—	—	—	—		—		8.2	—	8.3	—				
101-110					8.1	—	—	—		9.8		—	7.7		9.6				
111-120					—	—	—	—		—		8.2							
121-130					—	—	—	—		—									
131-140					—	—	—	11.4		9.8									
141-150					—	—	—												
151-180					—	—	10.8												
181-210					—	10.0	—												
211-240					8.1														

*In all cases, rate either remained constant or decreased slightly; increasing pulse width is due, therefore, to increasing interelectrode resistance. (See results.)

two pacemaker variables, pulse width and rate. The immediate value of these findings is in the differential diagnosis of "pacemaker failures," and in identifying incipient failures. Fortunately, with the pacemaker used there have been no component failures. Therefore, our clinical experience has been concerned solely with differentiating between battery failure, electrode fracture, and critical elevation of myocardial threshold.

Battery failure was correctly diagnosed by this method in all of 8 cases. Of particular importance is the fact that failure in 5 cases was detected while the myocardium was still responding to the stimulus. The other 3 cases presented after the stimulus level had fallen below threshold, and the heart was no longer responding to the pacemaker. In 4 of these cases the main batteries were involved, and a progressive increase in both pacemaker rate and pulse width was manifest. (See Table II.) The other 4 cases involved failure of the bias battery, which resulted in decreasing rate without a change in pulse width. Each pacemaker has one bias battery which controls switching, and deterioration of this battery has the above-mentioned effect, which is easy to identify.

Electrode breaks were correctly diagnosed in all of a total of 5 cases, and in an additional unit the diagnosis of short circuit of the electrodes was made. A short circuit is manifested by a very short pulse width and a constant rate. The 5 instances of wire breakage resulted in sudden increases in pacemaker rate, and measurements showed very long and variable pulse widths, in addition to variation in rate from one pulse to the next. These changes are caused by an open circuit, giving a greatly increased and variable interelectrode resistance (greater than 5,000 ohms).

Table II. *Differential diagnosis of "pacemaker failures," based on changes in pacemaker rate and pulse width*

	Pulse width	*Pacemaker rate*
Wire break	Sudden increase	Sudden increase
Exit block	Rapid increase	No change
Main battery	Slow increase	Slow increase
Bias battery	No change	Slow decrease

Increased myocardial threshold, to a level above the constant-energy pacemaker stimulus, was diagnosed in 11 cases. In 7 of the cases the diagnosis was confirmed by testing the removed pacemaker unit and finding it to be functioning within normal limits, and in 4 cases it has not been necessary to replace the pacemaker. We have applied the term "exit block" to this situation. The definition given for exit block is "inability of an impulse to leave its point of origin, the mechanism for which is conceived as an encircling zone of refractory tissue denying passage to the emerging impulse."[13] We think that this term characterizes the clinical situation in which the myocardium becomes unresponsive to a normally functioning pacemaker. In our experience, the diagnosis can be anticipated by finding a rising pulse width with a constant pacemaker rate. This indicates a rapidly rising interelectrode resistance. There was no correlation of exit block with high absolute values of interelectrode resistance, but there was a definite correlation with rapidly increasing R_L for any given patient.

We think that, by serial measurements after implantation, pacemaker performance can be adequately evaluated so as to make prophylactic replacement of the unit unnecessary. Although electrode breaks cannot be identified until after a complete fracture occurs, and may always be an unpredictable factor, there is hope that improvements in the design of electrodes will drastically reduce the incidence of this type of failure. Each of our patients is now asked to record daily his pacemaker rate, using a transistor radio held over the unit, and he is instructed to report immediately any change in rate greater than 3 pulses per minute. Each patient is also asked to attend a pacemaker clinic which is held every 3 months,

for the purpose of taking serial measurements. The method of analysis described requires only the instruments mentioned, the type pacemaker used in this study, and an adequate knowledge of the pacemaker circuit.[14]

Summary

Entirely satisfactory treatment of complete heart block has been prevented by an inability to measure pacemaker function after implantation. Two major problems can be identified in the long-term management of pacemaker patients: first, adequate prediction of when a pacemaker will fail; and second, accurate diagnosis of the type of pacemaker failure if it occurs.

Observations on dogs gave precise measurements of interelectrode impedances. A method is described of externally measuring the interelectrode resistance and capacitance of implanted pacemakers. In studies on both dogs and human beings it was found that all changes in interelectrode impedance could be identified by changes in pacemaker rate and pulse width. Serial measurements of these two parameters allowed accurate diagnosis of the different types of "pacemaker failures." The method also enabled anticipation of failures, other than wire breakage, before they were clinically evident. We think that pacemaker performance can be adequately evaluated by this method, making unnecessary prophylactic replacement of the unit.

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