Electrocardiographic Recognition of the Various Pacemaker Types and Dysfunction*

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This discussion deals with electrocardiographic interpretations of the various types of pacemakers as well as with their dysfunction which may be due to pulse generator failure, wire breakage, change in threshold, or polarization of the ventricle. Hemodynamic problems may also arise concerning placement of the pacemaker leads particularly in those patients who lack atrial contribution.

Figure 1 demonstrates competition resulting from a fixed-rate pacemaker. Here are four different types of beats showing the various responses that may occur. It is important to point out that if a T wave is not observed after a pacemaker spike then there is no propagation of the impulse through the ventricle. I prefer not to use this type of pacing because of competition and the possibility of sudden death, but as yet we do not have any cases that verify such a situation. Figure 2 demonstrates the most serious form of competition in which stimulation of the T wave had resulted in ventricular fibrillation. I have observed a number of cases in which the spike did occur on the T wave but repetitive rhythms such as ventricular tachycardia did not occur. However, with those patients who die suddenly one can only surmise that T-wave stimulation and ventricular fibrillation could have been related. I have no proof, but I am worried about this problem as is everyone else. I believe that the sicker the heart the more vulnerable it is and the greater the chance of these complications.

One of the major concerns I have with fixed-rate pacing is the problem of premature ventricular beats other than those associated with ventricular fibrillation. Figure 3, strip b, shows the development of premature ventricular contractions after each paced beat which result in a form of ventricular tachycardia that is hazardous.

Noncompetitive pacemakers such as the R-wave ventricular-triggered pacemaker (Fig. 3, strip c) have a built-in refractory period, but as will be seen in the last portion of the strip, they do not sense all beats. Figure 3, strip a, is an example of coupled rhythm with an R-wave triggered pacemaker that does not produce the form of ventricular tachycardia associated with the fixed-rate unit.

The atrial-triggered pacemakers have given us a host of headaches. There are not many of these around anymore, but when the bifocal type becomes more widely utilized we may run into similar difficulties again. This patient (Fig. 4) developed atrial flutter with an atrial-triggered pacemaker. Digitalization will not work under these circumstances, and there is not much you can do except shock the patient (strip d). I attempted to use quinidine, but the rate went up to 140.

Figure 5 shows a premature ventricular contraction which occurred during use of an atrial-triggered pacemaker. There is no refractory period built into the unit, therefore, the ectopic beat is not sensed. This could create a dangerous situation, and at least the bifocal pacemaker, as Mr. Berkovits pointed out, does not have this problem. In addition to runaway pacemakers, atrial-triggered units include such pitfalls as this instance of a patient in complete A-V block and intermittent function (Fig. 6). Here the "demand," or escape feature of the pacemaker was working, but the atrial threshold increased tremendously, and the atrial electrode was not sensing except in the supernormal period. You can occasionally see atrial-triggered beats coming through and evidence of bigeminy which subsequently developed for some reason. There is nothing wrong with this pacemaker unit. It was just that the atrial circuit was working only intermittently because of build-up of the threshold at the atrial lead. One of the problems with atrial epicardial leads is that after a period of time they may cease to function, and you are again faced with fixed-rate pacing in a patient with high-degree A-V block.

Rate hysteresis is another concern in using demand pacemakers. In order to permit spontaneous sinus activity, the escape rate is set at a slower driving frequency of 60 beats per minute while the driving rate is set at 70 beats per minute. This allows the heart to be paced in the presence of asystole. Following a

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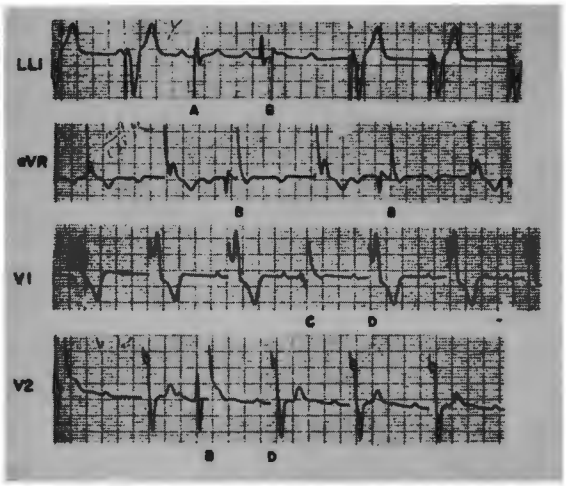

Fig 1—Competition resulting from a fixed-rate pacemaker. A, B, C, and D represent four types of beats, showing the various responses that may occur.

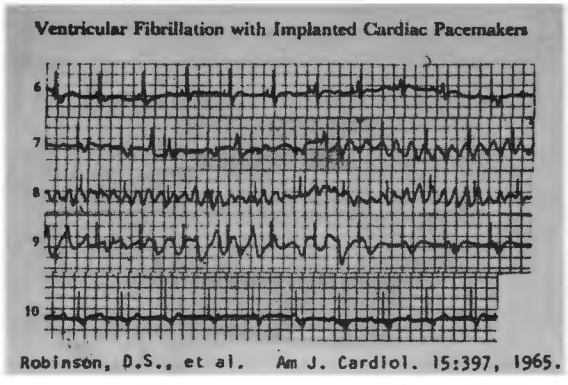

Fig 2—The most serious form of competition resulting from a fixed-rate pacemaker. Strip 7 shows stimulation of the T wave which results in ventricular fibrillation (strip 8).

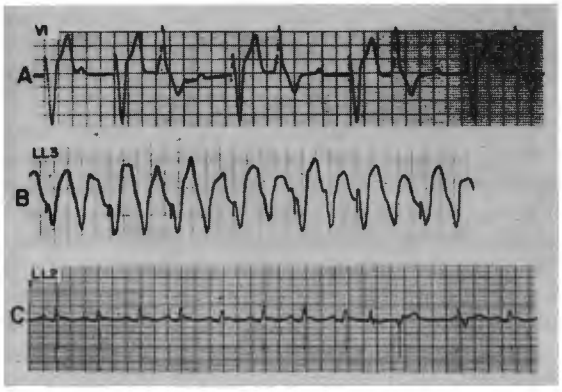

Fig 3—(Strip a) coupled rhythm with an R-wave triggered pacemaker; (strip b) development of premature ventricular contractions after each paced beat with a fixed-rate pacemaker; (strip c) an R-wave ventricular-triggered pacemaker that is not sensing all beats.

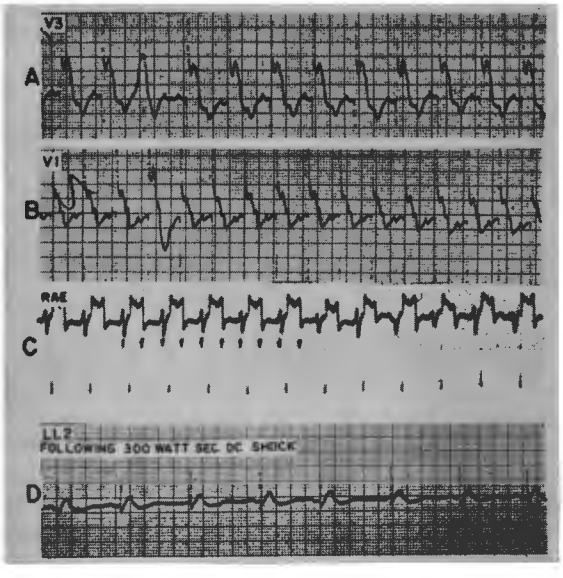

Fig 4—Development of atrial flutter with an atrial-triggered pacemaker. Strips a and b represent an EKG recording of atrial flutter with an atrial-triggered pacemaker. Strip c, documents the presence of flutter with a right atrial electrogram. (Strip d) EKG recording after applied shock.

period of sinus rhythm the pacemaker will not return its pacing cycle for at least one second in order to permit slower sinus rates to emerge and maintain optimum cardiac output.

Other problems with demand pacemakers are sometimes encountered. If the tip of the electrode catheter is too close to the atria or the electrodes of the epicardial leads are too close to atrial tissue, P-wave sensing may occur and cause a double reset of the pacemaker. Also, large T waves occasionally can be confused with QRS complexes, and these too can reset pacemakers and produce long diastolic intervals. Sometimes the electrogram does not quite reach two millivolts, and incomplete sensing may occur. If the pacing capacitors are not fully discharged they may recycle at an earlier time and thus, the pacing cycle is shorter than the expected ordinary driving cycle. But in most instances the pacemaker corporations have attempted to correct these problems.

In coronary sinus pacing, the pacemaker may be located above or below the A-V junction. As Dr. Samet pointed out, you have varying delays of propagation to the ventricle from His bundle recordings. However, the coronary sinus is an unreliable place from which to pace. I recently lost a patient completely dependent upon coronary sinus pacing when the catheter flipped out.

The runaway pacemaker (Fig. 7) demonstrates a situation which should no longer occur. For early detection of this problem so that pulse generators can be replaced, it is entirely possible that ten-hour electrocardiographic taping may be of value.

I would now like to deal with some of the interesting aspects of cardiac pacemaking that relate to electrophysiology. In the past we did not know what the supernormal period of the human heart was. We discovered it in the laboratory when we stimulated the heart at half threshold. The subsequent development of pulse generator failure shown in Fig. 8 reveals pacemaker spikes not followed by QRS complexes. However, the supernormal phase is identified at the end of the T wave because of the propagated response. Thus, a defunct pacemaker with less than threshold stimulus remaining is able to propagate an impulse during the supernormal period.

Another example of the supernormal period during pacemaker failure is incomplete A-V dissociation in the presence of 2-1 conduction block (Fig. 9). Why do we get sudden conduction? And sudden conduction occurring only after a paced spike? This is one of the varieties of the so-called supernormal period of conduction. In some way this pacemaker spike is influencing subsequent transmission of facilitation of the next P wave, and this offers an interesting insight into electrocardiographic analysis. The major problem in this situation is to determine whether the failure is due to a defunct battery, broken wire, threshold increase, or

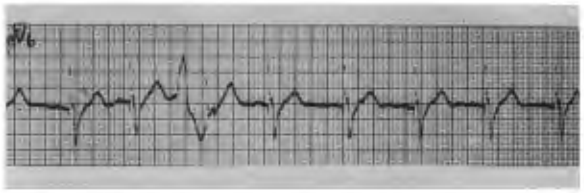

Fig 5—A premature ventricular contraction which occurred during use of an atrial-triggered pacemaker.

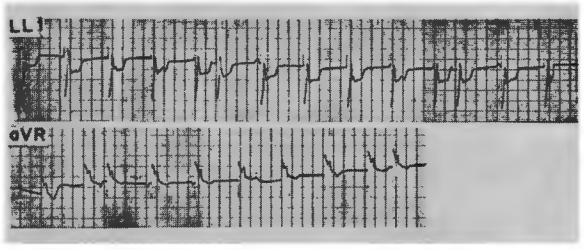

Fig 6—An EKG recording of a patient in complete A-V block and intermittent function using an atrial-triggered pacemaker. The strips show that the atrial electrode is not sensing except in the supernormal period. There is also evidence of bigeminy.

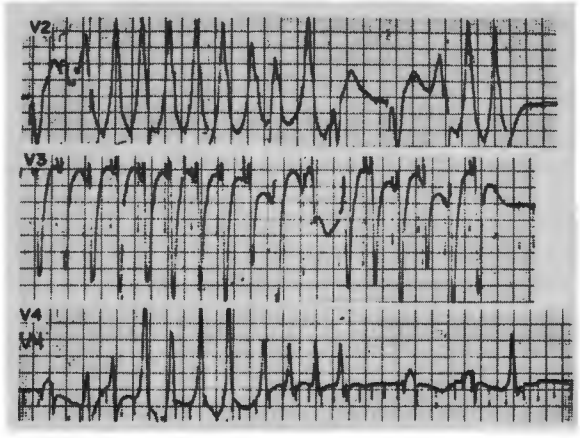

Fig 7—Electrocardiographic recordings of a runaway pacemaker.

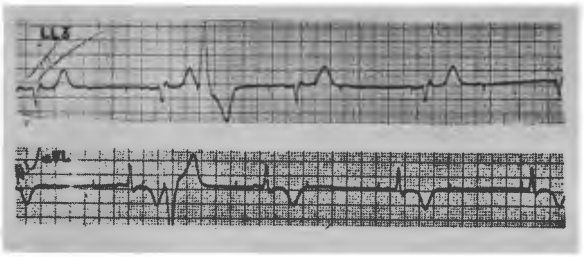

Fig 8—Development of pulse generator failure. Pacemaker spikes are not followed by QRS complexes, but the supernormal phase is identified at the end of the T wave because of the propagated response.

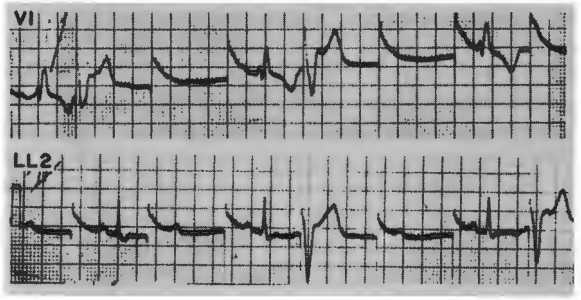

Fig 9—Electrocardiographic recording of the supernormal period during pacemaker failure in an instance of incomplete A-V dissociation in the presence of 2-1 conduction block.

to actual myocardial contractions.

However, to avoid pacemaker failure, we now rely on an early detection approach of rate counting and recording the arterial pulse. Pulse information can be recorded transtelephonically. Thus, if wire breakage should occur you would see spike, pulse, spike, pulse, spike and no pulse. This means that there was no contraction of the ventricle and no peripheral pulse. The rate is recorded by an interval counter, and if there is a change of 15-20 milliseconds in an R-R interval of 830 milliseconds, you can detect it and recommend a pulse generator change. It is interesting to note that 12 percent of our pacemakers are failing before the indicated battery life span of 18 months. This is a very serious problem, and it is our hope that with the present technique of interval counting we will be able to extend the life of the pulse generator to 23 months.

PANEL DISCUSSION

Dr. Richardson: Regarding the use of pacing to prevent ventricular tachycardia. Does this work by eliminating the VPC's which precipitate ventricular tachycardia or is there another mechanism?

Dr. Dreifus: When you have VPC's falling on the T wave this can set up repetitive phenomena, and it takes only one beat falling at the apex of T wave to produce ventricular fibrillation. Now VPC's may be due to reëntry or to increased automaticity. By speeding up the ventricular rate you prevent asynchronous diastolic depolarization and conduction abnormality. When pacing assumes control of the heart it depresses other pacemakers. Therefore, depression of the ectopic pacemaker by the electric one will usually prevent rhythm of development from occurring so that diastolic depolarization does not have a chance to take hold. So there are two basic principles when you use overdrive: you cut down the tendency for inhomogeneous depolarization and reëntry, and at the same time you keep the pacemaker depressed.

Questioner: Are you implying parasystolic focus is also depolarized, practically speaking?

Dr. Dreifus: Parasystole implies that already there is undirectional block. The undirectional block is built into the pacemaker. It is true that you may not be able to depolarize the parasystolic pacemaker, but then the chances of its emerging to the rest of the heart may then be inhibited. The most perfect example of parasystole is the electrical parasystole. Obviously it can't get into a fixed-rate pacemaker and so it is going to emerge anyhow, and that is a problem. Fortunately, most ventricular trachycardias with acute infarction are not parasystolic.