LETTERS TO THE EDITOR

Pacemaker Failure following External Defibrillation

To the Editor:

The "Brief Communication" by Dr. Giedwown (Circulation 44: 293, 1971) requires additional comment.

Knowledge of "normal" function of a pacemaker is essential in order to realize that the abnormalities depicted in the electrocardiograms might easily represent normal pacemaker behavior. In strip B, for example, where the author states that the "pacemaker rate was faster than the set rate," one can easily conceive of the phenomenon of partial recycling of the pacemaker by a relatively weak intracardiac signal. The rest of that strip revealed persistent ventricular fibrillation which undoubtedly activated the pacemaker-sensing circuit and suppressed any pacemaker activity. Similarly, in strip C, with the rate apparently slower, there may have been intracardiac activity not detected on the surface electrogram. (The author also suggested this possibility.) To further corroborate normal pacemaker function, strips D and E revealed the return to the normal fixed-rate interval.

Obviously, one must be aware of the possibility of damaging a pacemaker with external countershock, but these days most pacemakers are well protected from this eventuality. The author should have suggested a better emergency action, namely the application of an external magnet to the skin overlying the pacemaker to convert it to its fixed-rate mode. In the case presented, however, failure to interrupt ventricular fibrillation by external countershock was a more significant observation than the erroneous conclusion that the pacemaker had been damaged. There are several excellent recent papers on unusual modes of function of noncompetitive pacemakers.1

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**Figure 1**

Schematic diagram of an alternate possible mechanism in the Lown-Ganong-Levine syndrome. The impulse from the sinoatrial node (SAN) arrives either via the posterior internodal tract (PIT) or the normal tract to the atrioventricular node (AVN) at a His-Purkinje junction (LAMBDA) distal to the recording site of the His electrode. Retrograde depolarization of the proximal, and antegrade depolarization of the distal His-Purkinje system may therefore occur, producing an unusually shortened H-V interval.

The authors reply:

To the Editor:

Dr. Douglas presents a thoughtful analysis of how a bypass fiber entering the His bundle distal to the recording electrode can result in apparent shortening of the H-V interval due to simultaneous antegrade and retrograde conduction. Depending upon the relative proximity of the bypass fiber to the proximal His bundle, the distal His bundle, and antegrade versus retrograde conduction times, it would also be possible to explain simultaneous occurrence of His and ventricular electrograms, or His potential following the V potential.

In order to explain the variation in A-H time observed in two of the patients during rapid atrial pacing, Dr. Douglas essentially postulates a bypass fiber between the upper A-V node and mid-His bundle. Although theoretically possible, such an anatomic variant has as yet, to our knowledge, not been demonstrated.

The possibility of accelerated A-V conduction cannot be ruled out. Fibers of greater diameter or hyperpolarized fibers could account for such a phenomenon.

Based upon the recent work of Drs. Moore and Spear1 demonstrating absence of delta waves in X, Y, and Z ECGs with preexcitation of the base of septum along the course of the His bundle, we would have to consider a combination of James and Mahaim fibers as a more likely explanation of the low normal A-H and short H-V times.

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LETTERS TO THE EDITOR

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Reference


The author replies:

To the Editor:

Following external DC defibrillation the recorded electrocardiogram showed: irregular pacemaker activity, rate faster than the set rate, absence of pacemaker activity, slowing of the pacemaker rate, and return to the set rate.

It is possible to explain these changes by the presence of ventricular fibrillation not recorded by surface electrocardiogram, by recycling, by over-sensing of a potential generated by the heart, by a current leak from the equipment used, or from the influence of an external electromagnetic field. The source of an electromagnetic field could be within or even outside the hospital. Increase from a slow pacemaker rate to the set rate could be explained by Gaskell’s rhythm of development.3

To explain as a normal phenomenon all the changes observed we would have to assume several different mechanisms altering pacemaker function within a short period of time.

Broken electrode wire may mimic interference by alternating current. If this explanation is accepted, the correct diagnosis is missed.4 In many cases of true but transient or incomplete pacemaker failure an alternate explanation can be suggested. Total pacemaker failure may occur only a few days or few months later.1, 4

Failure of implanted pacemakers following external DC shock was demonstrated.5 Also damage of external demand pacemakers following external DC shock was reported.6

In the case presented, pacemaker failure was not proved but remains the most likely possibility.

Conversion of a demand pacemaker to fixed rate should always be considered when a failure or undesirable function of a demand unit is suspected. In the case reported, a Medtronic Demand Pacemaker model 5841 was implanted. This model could be converted to a fixed rate only with a special rate-control transmitter model 5855. Unfortunately this device is not available in the majority of well-equipped hospitals. Demand Pacemaker model 5841 was replaced by model 5842. This model is supplied with a magnet that reverts it to fixed-rate pacing.

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References


Mid-systolic Clicks in Arteriosclerotic Heart Disease

To the Editor:

We have read with great interest the article by Steelman et al. (Circulation 44: 503, 1971). We have been interested in the syndrome associated with mid-systolic click1, 2 and have already observed 75 patients with this syndrome (in 32 of the patients, mitral prolapse was demonstrated angiographically, and in 18 of the 32 a normal selective coronary arteriogram was obtained). This series attests to the prevalence of this syndrome.

Despite our interest in this syndrome we have observed only one case of proven coronary artery disease associated with a mid-systolic click in a coronary care unit where 3,250 patients have been treated in the past 6 years and in an active cardiac clinic and hospital cardiac practice. On the basis of our experience, we disagree with the authors’ interpretation of their findings. In all likelihood, they have observed a coincidental association of two common diseases, i.e., myxomatous degeneration of the mitral valve as evidenced by a mid-systolic click and coronary heart disease.

The authors report that the symptoms of coronary artery disease preceded the discovery of the click or murmur in all their patients and they use this chronologic finding as proof of an
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