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(Accepted for publication December 3, 1993.)

Anesthesiology
80:717–718, 1994

J. B. Lippincott Company, Philadelphia

Pacemaker Interactions with Transcutaneous Cardiac Pacing

To the Editor:—Kemnitz and Peters[1] describe interactions of pacemakers with transcutaneous cardiac pacing. We would like to offer alternative explanations to some of their conclusions.

The first case reported[1] involved a patient with sick sinus syndrome who had a permanent VVI pacemaker. Sinus rhythm prevailed for 20 min after induction of anesthesia, at which point the heart rate decreased progressively below the programmed rate to initiate VVI pacing. As the authors point out, this pacing mode was associated with a decrease in blood pressure due to loss of the atrial contribution to ventricular filling. To improve hemodynamics, the anesthesiologist intended to increase the heart rate using a previously applied transcutaneous pacemaker, but before this could be accomplished he noticed that low currents delivered by the transcutaneous pacemaker inhibited the permanent pacemaker and restored sinus rhythm with improved hemodynamics.

We question the very decision to use the transcutaneous cardiac pacemaker because it is a temporary VVI pacemaker and would offer no advantage to this patient with congestive heart failure, who was dependent on atrioventricular synchrony and the atrial contribution. Fortunately, incremental increases in the current were therapeutic to restore sinus rhythm by inadvertently inhibiting the permanent pacemaker. As discussed by the authors, other measures to inhibit permanent pacemakers include applied stimuli *via* a cutaneously applied nerve stimulator or perhaps by another temporary pacemaker; careful titration of isoproterenol; and overdrive transesophageal atrial pacing. In addition, temporary transvenous atrial pacing may be used in the acute setting while the option of a permanent dual chamber pacemaker is considered with the patient's cardiologist.

The second patient described by Kemnitz and Peters[1] was pacemaker-dependent after cardiac surgery. Pacing was achieved by a temporary VVI pacemaker at a rate of 90 beats/min. To test the feasibility of transcutaneous cardiac pacing, the noninvasive pacemaker was set at 95 beats/min with the threshold current progressively increased to 40 mA. The authors state that this maneuver failed to produce ventricular pacing and inhibited the temporary pacing, resulting in temporary asystole.

Here again, intentional delivery of external current transcutaneously should not be interpreted as an interference but as an expected interaction with the temporary VVI pacemaker. The failure for pacing capture with the transcutaneous pacemaker may be due to the relatively low current output used, as acknowledged by the authors, especially in a patient recovering from cardiac surgery, because of the presence of air or fluid in the chest, which may mitigate pacing

Table 1. Rate Settings of the Noninvasive Transcutaneous Pacemaker

Pacing Rate Desired (pulses/min)	Actual Heart Rate (beats/min)
60	55
70	63
80	71
90	78
100	86
110	93
120	100
130	107
140	114
150	120
160	126

Comparison of actual paced heart rate and displayed pacing rate. This assumes that every paced beat is sensed.

$$\text{Actual heart rate} = \frac{60{,}000\ (\text{ms})}{\text{pacing rate (ms)} + 100\ \text{ms}}$$

Example: desired pacing rate = 60 (pulses/min)

$$\text{Actual rate} = \frac{60{,}000\ \text{ms}}{1000\ \text{ms} + 100\ \text{ms}} = 55\ \text{beats/min}$$

Data provided as a technical communication by Zoll Medical Company (Woburn, MA).

capture.[2] In addition, the transcutaneous pacemaker rate was set at 95 pulses/min with the intention of overriding the temporary pacemaker, which was set at 90 beats/min. With commercially available transcutaneous cardiac pacemakers in the United States, the displayed pacing rate actually corresponds to that time interval, plus an additional 100-ms interval used to enhance sensing of intrinsic rhythms. The "hidden" 100-ms interval translates into a much lower actual pacing rate than would be anticipated, as shown in table 1. Whether the same delay exists in the external pacemaker used by the authors is uncertain. This discrepancy, though reported previously,[2] is little appreciated by anesthesiologists and needs to be emphasized to avoid the false impression of pacing failure when the feasibility of external pacing is sought to override intrinsic rhythms, paced or not.[3]

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Anesthesiology
80:718, 1994

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(Accepted for publication December 8, 1993.)

In Reply:—With regard to the first case we did not intend in our article[1] to discuss the most appropriate treatment options for a decreased arterial pressure due to loss of sinus rhythm and ventricular pacing. Rather, we drew attention to an interesting pattern of interaction between two pacemakers that, unexpected or not, to our knowledge has not previously been described.

Amar and Gross are obviously correct in pointing out that one treatment option includes temporary transvenous atrial or atrioventricular sequential pacing. This likely would have involved flotation of a multipurpose pulmonary artery catheter. In the case presented, however, this choice appears quite invasive, and, in our opinion, would have represented therapeutic "overkill." Indeed, experience suggests that many cases of bradycardia respond rapidly to a decrease in volatile anesthetic concentration.

With regard to the second case, figure 2 of our paper clearly shows that transcutaneous pacing (with a current of 40 mA) inhibited epimyocardial pacing over several seconds and, by doing so, evoked temporary asystole. Accordingly, it remains unclear to what extent the "delay" referred to by Amar and Gross should provide an alternative explanation in relation to the case presented.

Furthermore, as measured from the original tracing, the transcutaneous pacing (VOO) rate was 93 pulses/min, *i.e.,* very close to the rate set on the pacer dial (95 pulses/min) and much greater than expected on the basis of the table provided by Amar and Gross. In addition, in response to the comments made, we again tested in the demand mode and at different rates the transcutaneous pacer used in our study and did not note any discrepancy between the desired and actual pacing rates. Thus, it appears that the type of transcutaneous pacer we used does not share with other brands the delay to which Amar and Gross refer.

In conclusion, therefore, transcutaneous pacing can interact with implanted transvenous as well as temporary epimyocardial pacemakers, potentially resulting in cessation of pacing.

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(Accepted for publication December 8, 1993.)