

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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In Reply:—With regard to the first case we did not intend in our article¹ 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

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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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