Knocking the Chest as a “Bridge to Pacemaker”: Treatment of Bradyasystole by Percussion Pacing — Reply —

We thank Dr. Drinhaus et al for reporting their case demonstrating the efficacy of precordial percussion pacing (PPP) on a patient with profound bradycardia most likely caused by third-degree atrioventricular block. In their video, PPP effectively triggered ventricular contractions to generate blood pressure and cardiac output in the near normal physiological range. We appreciate their demonstration of the clinical utility of PPP.

As Drinhaus et al note in their letter, we have previously shown the efficacy of PPP in a cardiac standstill microminipig model. In that study, we performed PPP by quickly percussing the chest wall of the microminipig with the palm from a height of approximately 20–30 cm above the body, at a rate of 60 beats/min, and with a mechanical energy of approximately 3 J. Electrical and pharmacological analyses indicated that PPP could activate the non-selective, stretch-activated ion channels, inducing ventricular electrical depolarization, which resulted in ventricular contraction. Importantly, PPP can maintain effective circulation together with adequate blood pressure without inducing any neurological deficit. Although systolic/diastolic arterial pressure generated by PPP was comparable to those by standard chest compression (S-CPR) and ventricular electrical pacing, the duration of developed arterial pressure by PPP as well as ventricular electrical pacing was >4-fold greater than that with S-CPR, indicating that PPP can trigger physiologically comparable ventricular contractions. Dr. Drinhaus et al confirmed these findings by percussing the chest wall with a closed fist at a frequency of approximately 90 beats/min in a patient with third-degree atrioventricular block in a clinical emergency situation. They successfully generated arterial blood pressure and cardiac output in the upper left corner, cardiac output on the small PiCCO monitor in the lower left corner.

Please find supplementary file(s);

AUTHOR’S REPLY

Figure 1. Representative traces showing the lead I ECG, aortic pressure and left ventricular (LV) pressure of a microminipig during sinus bradycardia followed by precordial percussion pacing (PPP) at 30 min after the intravenous administration of 0.3 mg/kg of bepridil. Note that PPP triggers ventricular electrical activity, resulting in the development of LV pressure. *Ventricular electrical activity; **percussion artifact; HR, heart rate.
output, leading to effective systemic circulation. Their results are in accordance with our animal study.

Recently, we assessed the efficacy as well as the safety of PPP using a bepridil-induced sinus bradycardia model of halothane-anesthetized microminipigs. At 30 min after the administration of a therapeutically relevant intravenous dose of 0.3 mg/kg of bepridil, PPP delivered at the electrical diastolic period triggered ventricular electrical activity, resulting in the development of left ventricular (LV) and aortic pressures as in the cardiac standstill model of microminipigs (Figure 1). Meanwhile at 30 min after the additional administration of a QT-interval prolonging i.v. dose of 3 mg/kg of bepridil, PPP applied just after the QRS complex did not induce ventricular electrical activity but left a percussion artifact, whereas upstroke of LV pressure was confirmed following the start of the preceding QRS complex (Figure 2). Importantly, the next PPP, which was delivered at the end of the T wave to mimic R-on-T phenomenon, induced ventricular electrical activity resulting in the development of LV pressure, but did not cause any lethal arrhythmias such as commotio cordis despite the presence of excessive QT-interval prolongation. Thus, the proarrhythmic risk of PPP was not confirmed, at least in the heart of the halothane-anesthetized microminipig with drug-induced excessive QT-interval prolongation.

Taken together, PPP can help as a clinically important bridge to definitive pacing therapy when ECG monitoring, invasive blood pressure measurement and/or reliable pulse palpation are available. The efficacy and safety of PPP, as first described in pigs and now shown in a patient, may have significant utility in patients with life-threatening bradycardia until more definitive therapies are available.

**Disclosures**

The authors declare that there are no conflicts of interest.

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**References**


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