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

To the Editor:
The investigation by Wada et al published in the Journal compared standard CPR, precordial percussion pacing (PPP) and electrical pacing in microminipigs and has provided important new mechanistic insights as it showed the probable importance of stretch-activated ion channels. With reference to their study, we would like to present a video of PPP which, to the best of our knowledge, shows for the first time in the human cardiac output (CO) measurement during percussion pacing. We can demonstrate that it was able to generate a normal CO. PPP (i.e., repetitive taps to the chest) has potential to function as a manual transcutaneous pacemaker. Even though the technique is mentioned briefly in the guidelines of the European Resuscitation Council, it is seldom used in clinical practice. A 76-year-old man developed bradyasystole after resection of a renal tumor that extended into the inferior caval vein. Luckily, the patient was under continuous hemodynamic monitoring using a PICCO-device, permitting us to measure CO during the event. As bradyasystole occurred, most likely caused by third-degree atrioventricular block (AVB), circulation stopped immediately as indicated by the flat lines on the tracings of pulse oximetry (SpO2) and invasive blood pressure (IBP). While waiting for an external pacemaker, one physician commenced to knock on the chest with one fist at a frequency of approximately 90 beats/min and with a force far less than required for chest compressions as in cardiopulmonary resuscitation (CPR) (Figure A, Movie S1). Each beat was followed by a ventricular complex on ECG and amplitudes on IBP and SpO2 of usual appearance. This tapping of the chest was interrupted four times. Each time ECG, IBP and SpO2 tracings went flat and returned after resumption of the taps. BP generated by this manual pacing was 140/60 mmHg, and a cardiac index of 3.0 L/min/m² was measured by PICCO (Figure B, Movie S1). Pacing was continued electrically, first via a transcutaneous, later a transvenous pacemaker. Despite initially successful resuscitation, the patient died 3 days later from persistent multi-organ failure.

This is, to the best of our knowledge, the first reported case of percussion pacing on video and the first documentation of PPP, including online BP and CO measurement in a human. It demonstrates that PPP, which is based on the conversion of mechanical to electrical energy, can stimulate ventricular contractions sufficient to generate normal BP and CO. Case series have reported its successful application for asystole or total AVB in both adult and pediatric patient populations. However, case reports or human studies directly measuring CO achieved by PPP have not been available. Chan et al extrapolated a presumed CO of 3.3 L/min generated by PPP, based on measurements from a pulmonary artery catheter inserted after stabilization of the patient. In dogs, percussion pacing restored a virtually normal CO and was much more effective than chest compressions in a study from 1987. Our video illustrates that PPP might be worth a try in case of unstable bradycardia or bradyasystole as a potential “bridge to pacemaker” to ensure sufficient CO. As animal studies have shown a higher efficiency compared with CPR, PPP might be preferable to CPR and the treatment approach of choice if monitoring of its efficiency, such as ECG, IBP measurement or reliable pulse palpation, is available. We deem it prudent, however, to perform PPP only when its efficiency can be monitored and to perform conventional CPR in other circumstances.

Conflicts of Interest / Funding Sources
None.

References
3. Eich C, Bleckmann A, Schwarz SK. Percussion pacing—an almost forgotten procedure for haemodynamically unstable bradycardias?: A report of three case studies and review of the
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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 20–30cm 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);

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**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 0.3mg/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.