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Pressure-Volume Analysis Illustrating the Mechanisms of Short-Term Hemodynamic Effects Produced by Premature Ventricular Contractions

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UNDER PRESSURE

Pressure-Volume Analysis Illustrating the Mechanisms of Short-Term Hemodynamic Effects Produced by Premature Ventricular Contractions

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An 84-year-old man with obstructive coronary artery disease underwent high-risk percutaneous coronary intervention with a high-fidelity conductance catheter seated in the left ventricular apex to assess procedural changes in ventricular contractility via pressure-volume analysis. In the midst of the intervention, the patient developed quadrigeminy (ie, a premature ventricular contraction [PVC] every fourth beat) as shown by the intraventricular electrogram in Figure [A]. Figure [B] shows the corresponding ventricular pressure tracing and illustrates a phenomenon referred to as postextrasystolic (PES) potentiation. Relative to the last sinus beat (labeled beat no. 1), pressure generated on the PVC (beat no. 2) is significantly reduced while it is significantly increased on the PES beat (beat no. 3). The simultaneously recorded pressure-volume loops (Figure [C]) show reduced ventricular filling on the PVC due to the short time interval between the PVC and the prior beat, and the increased ventricular filling on the PES beat due to the longer than normal interval between the PVC and PES beats (referred to as a compensatory pause). More striking than changes in ventricular filling, however, are the marked beat-to-beat changes in contractility evidenced by changes of the slope (referred to as end-systolic elastance) of the respective end-systolic pressure-volume relationships.

Differences in ventricular pressure generation accompanying changes in the beat-to-beat interval are generally attributed to changes in preload caused by variable filling times. Although it is evident that changes in preload contribute to this effect, ventricular contractility is by far the more substantial factor in determining differences in pressure generation. Interval-dependent changes of contractility are the global manifestation of the myocardial force-interval relationship, which relates the force of myocyte contraction to the beat-to-beat time interval.[1,2] The force-interval relationship, in turn, results from the time interval-dependent availability of calcium for release from the sarcoplasmic reticulum to the myofilaments in a process referred to as calcium restitution.[3] Calcium restitution starts at the end of phase 3 of the action potential and follows an exponential time course (with time constant ≈150–200 ms) to a plateau by ≈1 second. On a PVC, only a small amount of the total sarcoplasmic reticulum calcium pool is available for release, resulting in reduced contractility. The enhanced contractility on the PES beat results from greater sarcoplasmic reticulum calcium release, reflecting the sum of calcium normally released during a given beat plus calcium leftover from the prior premature beat in combination with the long time for calcium restitution due to the compensatory pause. Here, we describe the hemodynamic effects of a single PVC and PES beat, but these mechanisms also explain the pulse pressure variations observed in other irregular arrhythmias like atrial fibrillation. Although these concepts are well known to students of muscle physiology and cardiac excitation-contraction coupling,[4] they are not typically part of modern medical school curricula.

Key Words: calcium ■ coronary artery disease ■ hemodynamics ■ percutaneous coronary intervention ■ sarcoplasmic reticulum

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ARTICLE INFORMATION

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Disclosures

None.

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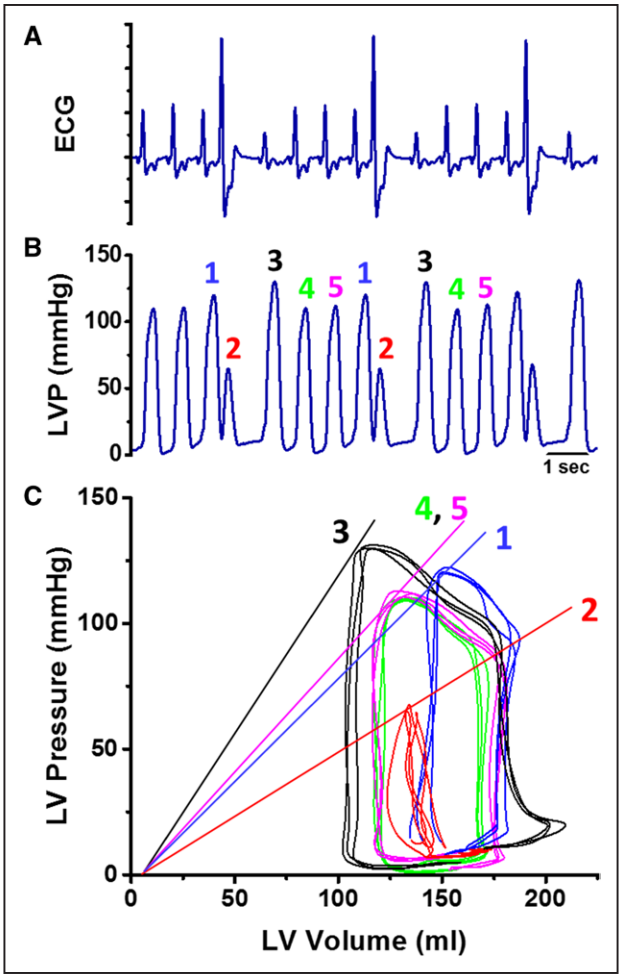

Figure. Pressure-volume analysis illustrating the short-term hemodynamic effects of a premature ventricular contraction and the following compensatory pause.
A, ECG. **B**, Left ventricular pressure (LVP). **C**, Pressure-volume loops corresponding to beats 1, 2, 3, 4 and 5 as labeled in **B**.