

A closer look at electrocardiographic P waves before and during spontaneous cardioinhibitory syncope

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Statement of authorship:

Both authors take responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation.

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Spontaneous cardioinhibitory syncope has rarely been documented by high-precision ECG measurements. Although the extended monitoring of ECG with implantable loop recorders (ILRs) in the past two decades has increased the knowledge of the mechanisms related to cardioinhibitory syncope, the P waves in these recordings are weak or even absent [1]. A further limitation of the ILR is its single-channel recording, which prevents any study of atrial events during spontaneous syncope. The profound influence of the autonomic nervous system (ANS) on the sinoatrial (SA) node was fortuitously recorded during a syncope episode by a 31-channel body-surface ECG measurement. The authors of this manuscript have certified that they comply with the Principles of Ethical Publishing in the International Journal of Cardiology [2].

We illustrate the changes in the ECG of a 19-year-old volunteer during routine performance measurements with a 31-channel ECG system. Three channels measured the standard bipolar leads (I, II, III), while the electrodes of the remaining 28 channels were placed in a regular mesh around the thorax [3]. Approximately 10 seconds after the start of the measurements in the seating position, the volunteer reported feeling bad and, a few seconds later, lost consciousness. The assistant supported him and brought him into the lying position on the floor. While supported, the volunteer's body jerked and some of the electrodes were torn away, interrupting the measurement. About 10 seconds later the volunteer responded, reporting intense "dreams". After a few minutes of rest he asserted that he was well and was transported home. Later he reported no consequences and said that he had experienced such events on previous occasions.

An analysis of the volunteer's medical record indicated that this syncope was probably a psychologically triggered, neurally mediated (vasovagal) syncope. Careful analysis of the measurement revealed an abrupt change in the morphology of the P wave in beat 12 (Figure A, B). Further analysis of the signals in the P-Q interval confirmed that not only did the morphology of the P waves change, but so did the timing relative to the QRS complex. The P-Q intervals of beats 1 to 11 (group A) are similar in duration (142 ± 2 ms), while those of beats 12-15, 17 (group B) are also similar but significantly shorter (121 ± 2.5 ms). Thus, a sudden reduction, by 21 ms, in the P-Q interval occurred after beat 11, accompanied by a change in the P wave's morphology (Figure C). To assess the spatial changes of the P waves, body-surface potential maps were calculated from the averaged beats of both groups, 10 ms after the beginning of the P wave (Figure D). Note the difference between the shape and the position of the zero-potential (green) contours on the thorax. The potentials of most electrodes on the front part of the thorax after the change in the P wave are positive (Figure C, right),

whereas, before the change, the potentials were positive and negative (Figure C, left). This confirms that the change in the position of the leading pacemaker modified the P wave just after the start of the depolarization.

It is plausible to say that after the SA node was blocked by a high vagal drive, the extranodal pacemaker took control of the heart for a few heart beats, with its own response to the ANS drive, but not long enough to avoid syncope. With a further change of ANS drive the extranodal pacemaker was knocked out too and no third region anywhere in the heart could meet the actual conditions of the ANS for the remaining 13.2 seconds, when the extranodal pacemaker took control of the heart once again, with an immediate transition of the control back to the SA node. The heart rate increased rapidly to 100 beats/min, the arterial blood pressure rose, and the brain returned to the conscious state.

To the best of our knowledge this is the first body-surface, multichannel ECG measurement of spontaneous cardioinhibitory syncope that confirms a sudden change of the P-wave morphology, together with a shortening of the P-Q interval. Assuming the conduction velocity in the right atrial wall is 88 cm/s, the shortening of the P-Q interval is probably the result of an approximately 2-cm shift of the leading pacemaker site closer to the atrioventricular node [4]. Careful examination of the P waves provides further evidence for the physiological operation of extranodal pacemaker sites of a widely distributed atrial pacemaker complex in the human heart [5], uncovered by a profound influence of the ANS on the pacemaking structures during the syncope.

The described case could encourage the manufacturers of ILRs to improve their recorders for greater sensitivity to P waves, which often hold information about the cause of the syncope.

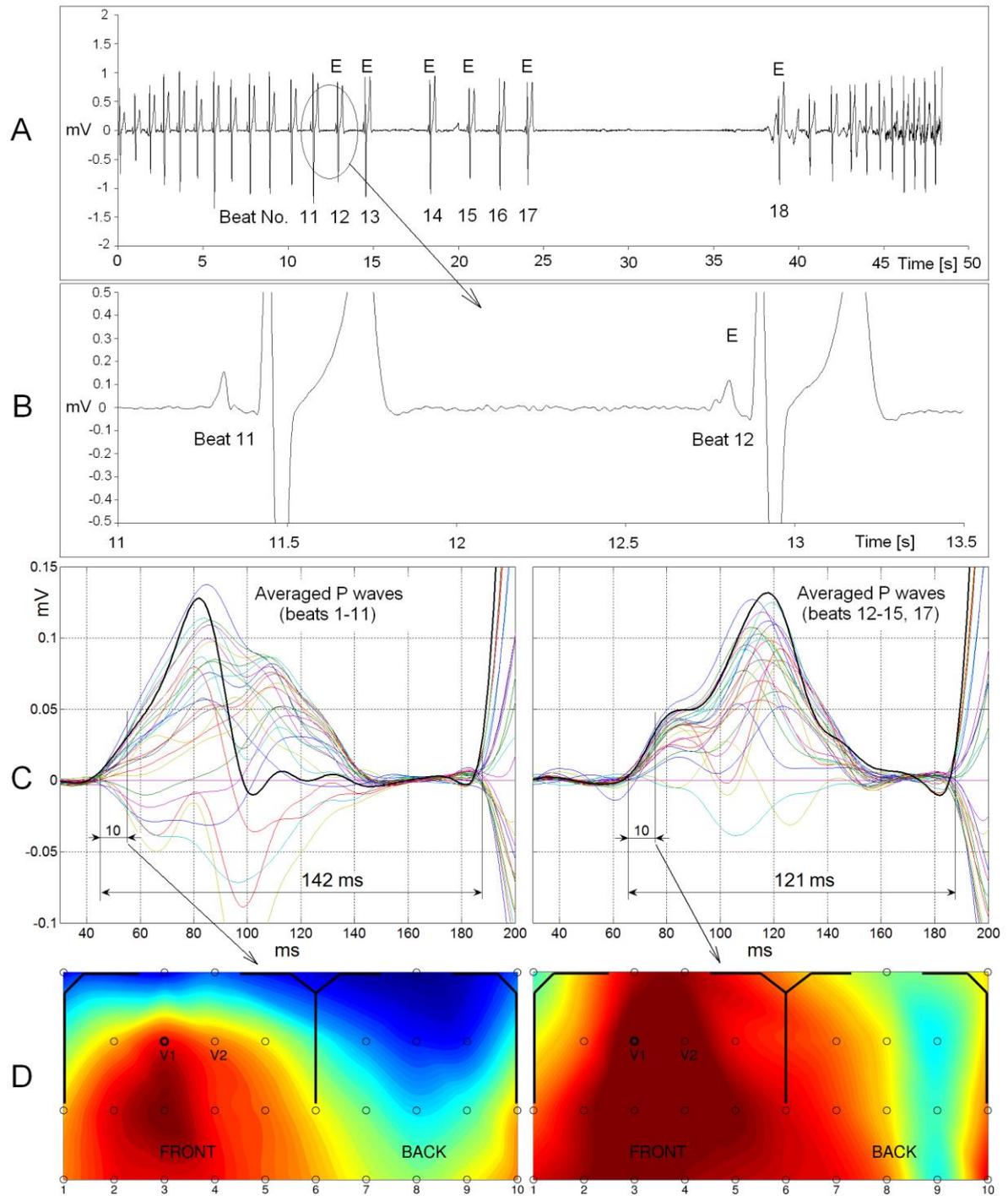


Figure 1. A, The electrocardiogram of channel 10 (equivalent to V1 of the standard ECG) during a spontaneous syncope. The heart rate slowed down until beat 12, when a sudden change in the P-wave morphology occurred. After a 3.5-second pause, the heart rate increased, with a modified P wave, until beat 17, when the heart entered a 13.2-second pause. The beats 12-15, 17 and 18 are denoted by E, indicating the extranodal pacemaker origin of the beat. The circle denotes beats 11 and 12, where the abrupt change of the P-wave morphology occurred, as shown magnified in panel B. C, Averaged P waves obtained from beats 1 to 11 (C - left, group A) and from beats 12 to 15 and 17 (C - right, group B) for all chest electrodes. The P-Q intervals are 142 ms before the change of the P-wave morphology and 121 ms after the change. D, Body-surface maps 10 ms after the beginning of the P wave before the change in its morphology (D - left) and after the change in morphology (D - right). Small circles represent the body-surface electrodes on the front (1-6) and on the back (6-10) part of the thorax. The electrode of channel 10 (V1) is marked with a bold circle. Positive potentials are shown in red and negative potentials in blue.

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