Unusual Modulated Ventricular Parasystole Due to Premature Beats

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Introduction
Typical ventricular parasystole (VP) is characterized by a regular rhythm with wide QRS complexes that competes with, and is not influenced by, the dominant rhythm. As a consequence, the parasystolic rhythm gives rise to premature ventricular contractions (PVCs) with highly variable coupling intervals and the longest interectopic intervals are exact multiples of a common minimum denominator (the “pure” interectopic interval).1 However, most VPs widely depart from this “perfect” form, due to incomplete entrance block, diverse types of exit blocks, and electrotonic modulation by non-parasystolic beats.2–4

In this report we describe a peculiar example of a modulated VP due to PVCs detected in a 24-hour Holter monitoring.

Case and ECG description
A 45-year-old man with history of chronic Chagas’ cardiomyopathy, heart failure functional class II (NYHA), and severely depressed left ventricular systolic function underwent a radiofrequency ablation of a typical atrial flutter. Two weeks later, a 24-hour electrocardiogram (ECG) Holter was recorded. Figure 1 depicts a peculiar phenomenon. A VP is easily identified, with the characteristic varying coupling intervals of the manifest parasystolic beats (marked with X, while the “concealed” parasystolic discharges are marked with asterisks) and a “pure” parasystolic interval of 1150 ms.

Also it is apparent that the parasystolic cycle length is modified when premature beats occur (marked with R) as after the atrial extrasystole with ventricular aberrancy (fourth beat in the strip) or the ventricular extrasystoles (11th and 14th beats in the strip). The coupling interval measures 460 ms for the first premature non-parasystolic contraction, 530 ms for the second one and 550 ms for the third ventricular premature QRS complex. The parasystolic intervals for these beats are prolonged to 1320 ms, 1180 ms and 1270 ms, respectively.

Points to ponder
In this case of “atypical” VP, the fortuitous occurrence of premature beats led to the diagnosis of electrotonic modulation of automaticity of the parasystolic focus. The example depicts a peculiar characteristic: the “pure” parasystolic cycle length that measured 1150 ms was prolonged by 30 ms to 170 ms by the effect of intervening non-parasystolic premature contractions.

Several clinical and experimental studies have shown that the automatic activity of a “protected” ventricular automatic focus may be modulated by non-parasystolic beats with a clear relationship between the coupling interval of these beats and the resulting effect on
parasystolic automaticity. In fact, non-parasystolic beats falling during the first half of the parasystolic interval induce a prolongation of this interval. Conversely, those non-parasystolic beats occurring during the second half of the parasystolic cycle length causes an abbreviation of this interval. The electrotonic modulation (causing either prolongation or shortening of the parasystolic cycle length) is maximal at the middle point of the parasystolic interval.

In our case this occurred in a peculiar way, as the prolongation of the parasystolic interval was clearly greater when the intervening non-parasystolic premature beat showed the shortest coupling interval, then was slightly prolonged as the coupling interval of the non-parasystolic interval approached the middle point of the parasystolic interval, and, finally, it was intermediate as the coupling interval of the non-parasystolic ventricular interval was even close to the turning point of the electrotonic modulation. It may be hypothesized that this occurred as a consequence of differences in the magnitude of the electrotonic influences of the myocardium surrounding the parasystolic focus depending on the spatial direction of the premature ventricular depolarization.

Careful interpretation of the surface ECG helps in identifying complex electrophysiologic mechanisms.

References