CASE REPORTS

Postextrasystolic Changes in the Complexity of the QRS Complex and T Wave

Velislav N. Batchvarov, M.D. and A. John Camm, M.D., F.R.S.C., F.E.S.C., F.A.C.C., F.A.H.A.

From the Division of Cardiac and Vascular Sciences, St. George's University of London, Cranmer Terrace, London, United Kingdom

We tested the hypothesis that ventricular repolarization of the first sinus beat following a ventricular premature beat (VPB) can be modulated in the absence of clearly discernible T-wave changes. We applied principal component analysis (PCA) to assess QRS and T-wave complexity of sinus beats preceding and following VPBs in multiple 10-second resting 12-lead electrocardiograms of two subjects with frequent VPBs and no apparent heart disease. In both subjects, T-wave complexity of the first post-VPB beat was significantly increased compared to the beats preceding the VPB.

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T wave; postextrasystolic T wave changes; ventricular repolarization

Changes in the T-wave amplitude, shape, or polarity of sinus beats immediately following a ventricular premature beat (VPB) are sometimes observed in both healthy subjects as well as cardiac patients.^{1,2} It is conceivable, however, that ventricular repolarization of the post-VPB sinus complex may be modulated even in the absence of visible T-wave changes. In order to test this hypothesis, we used principal component analysis (PCA)^{3,4} to assess the complexity of the QRS complex and T wave of digital 12-lead electrocardiograms (ECGs) acquired in two healthy subjects with frequent VPBs and no clearly discernible postextrasystolic T-wave changes.

CASE REPORT

We analyzed digital 10-second 12-lead ECGs (MAC 5000, GE Medical Milwaukee, WI, resolution 4.88 μ V, 500 Hz), which were previously recorded in the supine resting position in a 38-year-old woman (23 ECGs) (subject 1) and a 23-year-old man (32 ECGs) (subject 2) as part of two different research projects. In each subject, all ECGs were recorded immediately one after the other over

a period of approximately 5 minutes in subject 1 and 7 minutes in subject 2. Both subjects were asymptomatic, except for occasional palpitations, had negative medical history, normal physical examination, and were not taking any medications. All ECGs were normal except for the presence of frequent monomorphic VPBs (mean 1.3, range 1–2 VPB per ECG in subject 1, and mean 0.8, range 0–3 VPB per ECG in subject 2) with left bundle branch block morphology and long coupling interval (i.e., beginning after the T-wave end of the preceding sinus beat) (Figs. 1 and 2). There were no visibly discernible changes in the T-U wave or the QT interval of the first post-VPB sinus beat compared to the pre-VPB sinus beats on any ECG. In subject 2, small beat-to-beat changes in the T-wave shape were observed but they were not specifically confined to the postextrasystolic sinus beat (see notching of the T wave of the fourth and sixth pre-VPB sinus complexes and the first and third post-VPB complexes in lead V₄ in Figure 2).

All ECGs with at least four sinus beats preceding and two sinus beats following a VPB were selected ($n = 13$ in subject 1 and $n = 14$ in subject 2) and were converted into text files (ECG Research

Address for reprints: Dr. Velislav N. Batchvarov, M.D., Division of Cardiac and Vascular Sciences, St. George's University of London, Cranmer Terrace, London SW17 0RE, United Kingdom. Fax: +44(0)208-767-7147; E-mail: vbatchva@sgul.ac.uk

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Figure 1. Resting 12-lead ECG in subject 1. In this one and in the next figure, the leads are arranged from top as follows: I, II, III, AVR, AVL, AVF, V₁–V₆.

Workstation Software, GE Medical). In each ECG text file, eight independent leads (I, II, ..., V₁–V₆) were selected and were transferred into a statistical package (Statistica 5.1, StatSoft Inc., Tulsa, OK). Following that, each ECG was displayed with all eight leads superimposed at the beginning and end of the QRS complex and T wave (common for all eight leads) of each sinus and ectopic complex were manually identified. The T wave was taken from the J point to the T-wave end, the latter defined as the point of return of the signal to the baseline. PCA

was performed separately on each QRS or T-wave data array (Statistica 5.1). The complexity of the QRS complex or T wave was defined as the ratio of the second to first eigenvalue (%).^{3,4} (QRS-PCA ratio and T-PCA ratio, respectively). In each subject, the QRS-PCA ratio and T-PCA ratio of the six sinus complexes surrounding a VPB (4 preceding and 2 following) were compared using repeated measures analysis of variance (ANOVA) with Scheffe post hoc tests. All data are presented as mean $\pm$ standard deviation.

Figure 2. Resting 12-lead ECG in subject 2.

The main results are presented in Figure 3. In both subjects, T-PCA ratio of the first post-VPB sinus complex was significantly increased compared to the T-PCA ratio of each of the four sinus complexes preceding and the second sinus complex following a VPB (subject 1: $P = 0.029, 0.014, 0.002, 0.006$, and 0.001 , respectively; subject 2: $P < 0.00001$ for all five comparisons).

Figure 3 also demonstrates that T-PCA ratio of the second post-VPB complex was decreased com-

pared to the four pre-VPB complexes. However, the differences were statistically significant ($P < 0.05$) only in subject 2 (not shown in Figure 3).

In both subjects, there were no statistically significant differences between the T-PCA ratios of the pre-VPB sinus complexes.

Interestingly, in subject 1 the QRS-PCA ratio of the first post-VPB complex was highly significantly decreased ($P < 0.0001$) compared to each of the five surrounding sinus complexes. In subject 2, the

Figure 3. QRS-PCA ratio (open squares) and T-PCA ratio (closed diamonds) of the 4 sinus complexes preceding and the 2 sinus complexes following a VPB in the 2 subjects. Data are presented as mean $\pm$ standard deviation of all ECGs ($n = 13$ in subject 1, $n = 14$ in subject 2). QRS-PCA ratio and T-PCA ratio of the premature beats are not shown. * $P < 0.05$ for comparison with the first post-VPB sinus complex.

QRS-PCA ratio was significantly decreased compared only to the second post-VPB complex (Fig. 3).

The QRS-PCA and the T-PCA ratio of the first post-VPB complex were significantly correlated to the duration of the postextrasystolic pause in subject 2 (T-PCA ratio: $r^2 = 0.87$, $P = 10^{-6}$; QRS-PCA ratio: $r^2 = 0.46$, $P = 0.008$), but not in subject 1 (T-PCA ratio: $r^2 = 0.009$, $P = 0.76$; QRS-PCA ratio: $r^2 = 0.10$, $P = 0.29$).

DISCUSSION

Our observations demonstrate that ventricular repolarization of the first post-VPB sinus complex can be modulated even in the absence of clearly discernible T- or U-wave changes. Therefore, this phenomenon is probably more common than previously appreciated. The premature beats (or the post-VPB pauses) seem to exert a biphasic effect on T-wave complexity of the subsequent sinus beats, with increase in T-wave complexity in the first and

decrease in the second post-VPB sinus beat. Increased PCA ratio signifies increased heterogeneity of repolarization; it has previously been reported in cardiac patients and has been shown to predict increased cardiovascular mortality.^{3,4} It is therefore possible, at least theoretically, that the post-VPB increase in T-wave complexity can pose an additional arrhythmic risk under certain conditions.

To the best of our knowledge, postextrasystolic changes in the QRS complex have not been reported so far. At this stage, we cannot speculate on the mechanism or significance of the decrease in QRS complexity of the first post-VPB sinus beat that we observed in subject 1. It seems unlikely that this phenomenon is linked to phase-4 (bradycardia-dependent) aberrancy⁵ because there was no correlation between QRS-PCA ratio and the duration of the post-VPB pause in this subject and, conversely, such correlation was present in subject 2 in whom there was no significant change in QRS complexity of the first post-VPB complex.

Postextrasystolic changes of the T wave were first reported by P.D. White in 1915⁶ and repeatedly by other authors thereafter^{7,8} (for a modern review see reference 2). Unlike postextrasystolic augmentation of the U wave, which is linked to the arrhythmic risk in the long QT syndrome⁹ and other conditions,¹ changes limited to the T wave have not been shown to be a marker of arrhythmic risk.^{1,10} Their mechanism is still not fully understood. It has been suggested that when the vector of the post-VPB T wave mirrors the general direction of the ectopic QRS complex (the so-called "obedient" T-wave changes), the mechanism likely represents a form of short-term cardiac memory.¹¹

Our limited observations have substantial limitations. Although the quality of all ECGs was visually satisfactory, the noise level was not objectively assessed. PCA ratio, however, is known to be relatively insensitive to the noise level of the ECG.¹² The reproducibility of PCA ratio assessed with the method that we used is not known. The modulatory effect of the VPB possibly extends beyond the second postextrasystolic sinus complex and therefore needs to be assessed from longer ECG recordings.

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