Cyclic Bursts of Ventricular Premature Contractions of More than One Minute Intervals

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SUMMARY

Ventricular premature contractions (VPCs) occasionally appear successively in the form of bigeminy, trigeminy or quadrigeminy associated with quiescent periods. However, details of these rhythmic VPC bursts have not been well documented. We analyzed the incidence, periodicity and interval of VPC bursts exhibiting bigeminy or trigeminy using ambulatory ECG monitoring and computer analysis. We defined VPC bursts as more than 5 successive groups of VPCs each containing more than 20 VPCs in the form of bigeminy or trigeminy that were interrupted by normal sinus rhythm lasting for more than 60 seconds. Bursts thus defined were observed transiently or continuously in 78 out of 500 consecutive patients showing > 3000 VPCs a day. Their age ranged from 14 to 76 years (mean 48). Forty patients were men and 38 were women. We could discriminate between two types of bursts on the instantaneous heart rate tachograms. Dome type bursts (n = 48) showed gradual shortening of the VPC coupling intervals whereas horizontal type bursts (n = 30) demonstrated fixed coupling intervals during the bursts. Cycle length of the dome type burst was 185 ± 40 seconds and regular, whereas it was 210 ± 63 seconds and irregular in the horizontal type (NS). Duration of the VPC bursts was 101 ± 31 seconds in the dome type and 98 ± 41 seconds in the horizontal type. Both burst types were associated with transient increases in sinus rate and abbreviated VPC-VPC intervals. We suspect ventricular parasystole to be the mechanism of these bursts especially in the dome type. Recognition of these two burst types from heart rate tachograms may be of value in the suppression of VPCs. (Jpn Heart J 1999; 40: 135–144)

Key words: Ventricular premature contractions, Burst, Cyclicity, Ambulatory ECG, Antiarrhythmic agent

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VENTRICULAR premature contractions (VPCs) occasionally appear in groups in the form of bigeminy, trigeminy or quadrigeminy. In addition, bigeminy or trigeminy tend to cluster for a long term and at specific periods during the day. The details of these periodic and repetitive VPC bursts have not been well documented. Using the instantaneous heart rate tachogram obtained from ambulatory electrocardiographic (ECG) monitoring, we discovered two distinct types of VPC bursts exhibited on the tachograms. One type showed gradual shortening of the VPC coupling interval, and the other showed fixed VPC coupling.

The purpose of this study was to evaluate the clinical features of these two types of repetitive VPC bursts, focusing on the incidence, periodicity, interval and relation to the mean heart rate. We attempted to document the features of these bursts by computer analysis of successive VPC-VPC intervals. The mechanism and clinical significance of these two burst types are discussed.

 Patients and Methods

Patients and heart rate tachograms: From 9000 consecutive records of routine ambulatory ECG monitoring performed from January 1986 to May 1998 using a Fukuda Denshi SR-29 or SR-30 recorder (Tokyo, Japan), we chose 500 patients experiencing more than 3000 VPCs during the 24-hour recording period. Original tapes of these patients were played back on a Fukuda Denshi SCM 270 or SCM-3000 analyzer, which could record instant heart rate tachograms at a paper speed of 3.5 mm/minute and real-time ECG at a paper speed of 180 mm/minute (Figure 1A).

Using the tachogram, we could discriminate normal sinus rhythm from VPCs, as well as the VPC mode. Succeeding bigeminy could be seen as two separated clusters of dots as shown in Figure 1B, and trigeminy or quadrigeminy could be observed as three dot zones on the tachogram. Full 24-hour ECG recordings and the tachograms were printed out.

Definition of periodic VPC bursts: Bursts of VPCs were searched for in the heart rate tachograms. We defined VPC bursts as more than 5 successive groups of VPCs each containing more than 20 VPCs in the form of bigeminy or trigeminy that were interrupted by normal sinus rhythm lasting for more than 60 seconds. (Figure 1B). Duration, interval and cycle length of the VPC bursts were measured as indicated on the heart rate tachogram. A representative series of four bursts (a to d) on the ECG and corresponding tachogram is shown in Figure 1A and B.

Two types of bursts: Depending on the pattern of VPC dot distribution on the tachograms, we discriminated between two types of VPC bursts on the
tachogram. Dome type bursts showed a gradual ascending series of VPC dots followed by a descending portion as shown in Figure 1 B. They showed initial shortening of the VPC coupling interval. Horizontal type bursts revealed a flat dot distribution pattern during the bursts, which indicated a fixed VPC coupling interval, as shown in Figure 3 (top panel).

**Computer analysis of the VPC bursts:** In 20 patients (10 with each burst type) with typical VPC bursts, R-R interval measurements including the VPC coupling interval were performed on a Fukuda Denshi computer system SCM 3000 using a minimum time resolution of 8 msec. VPCs were discriminated from normal sinus beats based on QRS configuration and R-R interval that was shorter than the preceding interval. Full 24-hour R-R interval data were digitized and transferred to a personal computer. An expanded tachogram was produced from these data, which allowed magnification of specific portions of the tachogram by arbitrary time and heart rate scales.

The resulting data were displayed simultaneously: (1) instantaneous heart rate tachogram, (2) mean heart rate tachogram (calculated from the moving average of the preceding and following 8 R-R intervals including both VPCs and subsequent pauses), (3) VPC-VPC interval tachogram and (4) VPC coupling interval tachogram.
RESULTS

Bursts were transiently or continuously observed in 78 out of 500 patients (15.8%) with VPCs > 3000/day. Their age ranged from 14 to 76 years (mean 48). Forty patients were men and 38 were women. VPC bursts appeared sporadically in 93% of patients and appeared continuously throughout the day in 7%. In both groups, bursts were associated with 1 to 10 minute normal sinus rhythm intervals with no VPCs. During the VPC bursts, significant ST-segment elevation or chest pain was not documented.

Two types of bursts: Dome type bursts were seen in 48 patients (25 men and 23 women) with a mean age of 53 ± 14 years. Horizontal type bursts were seen in 30 patients (15 men and 15 women) with a mean age of 40 ± 20 years ($p = 0.01$). Underlying heart disease was observed in 20 patients with dome type and 12 patients with horizontal type bursts (NS). None of these patients suffered from heart failure.

Cycle length of the bursts was $185 ± 40$ seconds in the dome type and $210 ± 63$ seconds in the horizontal type (NS). Duration of the VPC bursts was $101 ± 31$ seconds in the dome type and $98 ± 41$ seconds in the horizontal type.

![Figure 2](image.png)

Figure 2. Heart rate tachogram from the same patient as shown in Figure 1 with typical VPC bursts (open circles; dome type) is shown together with an additional three simultaneous rows. The 17-minute record was obtained at 5 PM. Mean heart rate tachogram in the second row indicates transient rate increase following the start of burst. VPC-VPC interval tachogram in the third row shows sharp decrease during the initial portion of the burst. R-R interval tachogram in the bottom indicates gradual shortening of the VPC coupling interval (open circles).
Lown grade > 4 was observed in 27 and 10 patients, respectively (NS).

Antiarrhythmic agents were administered in 33 patients, (22 dome type and 11 horizontal type; NS) during the recordings. Reproducibility of the bursts was observed in 15 patients. There was no transition between the two types of the bursts in the same patient at a full day recording or during the repeated recordings. Further aggravation of the arrhythmia into ventricular tachycardia or sudden death was not observed during the recordings in either of the two types.

Figure 2 shows a typical dome type VPC tachogram. Dots corresponding to each VPC gradually shift upward for the initial 15 seconds and thereafter shift downward until the disappearance of the VPCs, indicating the initial shortening of VPC coupling intervals followed by gradual prolongation as shown in the bottom panel. Mean heart rate abruptly increased during the burst as shown in the second panel. The VPC-VPC interval during bigeminy was transiently abbreviated in the third panel. VPC to the next sinus beat interval also decreased and thereafter increased as in the VPC coupling intervals in the bottom panel. VPC-VPC interval during the bigeminy and VPC to the next sinus beat interval (upper dots during the burst in the bottom panel) changed in parallel direction.
The horizontal type bursts displayed almost consistent location of VPC dots on the instantaneous heart rate tachograms as shown in Figure 3 Top. VPC coupling intervals were fixed as shown in the bottom panel. Mean heart rate increased from the beginning of the bursts as shown in the second panel. VPC-VPC interval during the burst in the third panel decreased slightly.

**Coupling interval in both groups by computer analysis:** Mean coupling interval in the dome type bursts throughout the day (551 ± 45 msec) was significantly longer than in the horizontal type (457 ± 30 msec, p = 0.003). Horizontal type coupling interval was constant as can be seen in the tachogram. The difference between the maximum and minimum coupling interval was 128 ± 31 msec in the dome type showing gradual shortening.

**Time course of the bursts:** Bursts were observed throughout the day in 7% of patients, during the day only in 37%, during the night only in 40% and transiently in 16%. Bursts were most frequently observed at mean heart rate of 60 to 80 bpm in both burst types. Bursts became infrequent at mean heart rates above 100 bpm or below 50 bpm.

In Figure 4, two burst types from the same patients as in Figure 2 and 3 are
Figure 5. Time course of VPC bursts with bar-code style display. Blank areas between the lines indicate quiescent periods without VPCs. A: Dome type VPC bursts during successive 12 hours from the same patient as shown in Figures 2 and 4-A. B: Horizontal type VPC bursts during successive 12 hours from the same patient as shown in Figures 3 and 4-B.

Figure 6. Burst interval time course during successive 24 hours for the two VPC burst types from the two representative patients shown in Figures 2 to 5. A: Dome type VPC bursts from the same patient as shown in Figures 2, 4A and 5A. B: Horizontal type VPC bursts from the same patient as shown in Figures 3, 4B and 5B.
shown to express their time courses. Dome type bursts tended to repeat more cyclically, while horizontal bursts appeared transiently. In Figure 5, two burst types from the same patients are shown in three successive rows with black bars to stress VPCs for a half-day. The blank areas represent normal sinus rhythms. The dome type bursts repeated more cyclically. Horizontal bursts appear at limited times and less frequently. Full-day time courses of the burst intervals from these two types of patients are displayed in Figure 6. In panel A, burst intervals are generally stable in dome type bursts. These stable bursts were not observed in the horizontal type as shown in panel B.

DISCUSSION

We demonstrated the two hitherto undescribed types of periodic VPC bursts in approximately 16% of patients evaluated for frequent VPCs. On the heart rate tachograms, the height of the VPC dots was inversely proportional to the VPC coupling interval. Depending on the time course of the VPC dots, we could discriminate between two types of bursts. The two burst types were observed independent of organic heart disease, appeared in almost all ages and both sexes, and exhibited several specific features. Dome type bursts displayed decreasing coupling intervals. The burst began with a relatively long coupling interval and subsequently shortened. After maximum shortening, the coupling interval prolonged again until the end of the burst. In contrast, horizontal type bursts showed almost constant coupling intervals through the entire bursts. This type was mainly observed in younger patients and was not stable compared with the dome type bursts.

Mechanisms of the cyclic bursts: In experimental studies, the burst mode of excitation, including gating, has been regarded as an intrinsic property of native channels. Bursts have been observed in sodium channels from a number of tissues including heart, skeletal muscle, brain and the rat pituitary cell lines. Kline et al. observed periodic bursts in a transgenic mouse cultured atrial cell line while recording spontaneous activity. They suggested that the bursts were due to the essential feature of the slightly reduced maximum diastolic potential. Chay et al. proposed that bursts in excitable cardiac cells could be explained by two functionally distinct current inactivations. Our two types of bursts might be introduced by these essential properties of the sodium channel.

Parasystole can be proposed as another mechanism of VPC bursts, especially in the dome type bursts. Parasystole is strongly suggested since each VPC-VPC interval during the bigeminy seemed to be modulated quickly by the normal sinus beat between the two VPCs. In our horizontal type bursts with fixed VPC coupling, the contribution of modulation seemed to be limited since the
VPC-VPC interval showed slight change during the burst.

Recently, Kinoshita et al. paid special attention to the termination pattern of VPC bigeminy with fixed coupling in 14 patients. They observed tachycardia-dependent termination in 12 patients and bradycardia-dependent termination in 10. As a mechanism of those termination modes, they proposed longitudinal dissociation and atypical Wenckebach periodicity of conduction in the reentrant pathway of VPC, similar to the delay seen in the periodic variation in atrioventricular conduction time. Their patients may correspond to our horizontal type patients with fixed VPC coupling interval. In our horizontal type, termination of bigeminy was associated with slight increases in mean heart rate (Figure 3). The bradycardia-dependent termination of the bigeminy they reported was not observed in our two type of bursts. Since the duration of the bigeminy was not precisely documented in their report, the commitment of bursts could not be fully determined.

Finally, the possibility of triggered activity could not be excluded in either type of bursts in our study. Direct proof of the triggered activity in humans is difficult except with the proper use of the intracardiac pacing or injection of adenosine. However, the contribution of triggered activity seemed to be limited since VPC burst could not introduce further malignant arrhythmias, including sustained or non-sustained ventricular tachycardias.

**Clinical significance of the cyclic bursts:** The major problem associated with the cyclic bursts is temporal and complete remission of the VPCs for brief periods. Exact diagnosis of the VPCs may be difficult when the arrhythmia is not observed on ECG at all. Furthermore, short time evaluation of antiarrhythmic agents administered intravenously may be affected by the cyclic disappearance of the VPCs just prior to or during the injection.

The observed bursts of several minutes interval themselves have not been well explained in the cardiovascular system. As a representative rhythmic phenomenon, cyclic coronary artery spasm of 3–15 minutes interval in variant angina pectoris is well documented. The relation of VPC burst to coronary spasm seems limited, since we could not find significant ST-segment elevation or chest pain during the VPC bursts. Another well-known specific rhythm is the Cheyne-Stokes respiration of 1 to 2 minutes cycle length observed in congestive heart failure. This could not explain the VPC bursts because none of our patients suffered from heart failure.

**Limitations of this study:** The number of patients studied was small and the effect of antiarrhythmic agents could not be systematically analyzed. Reproducibility of the bursts was pursued in a small number of patients. However, as far as we know, few studies describe VPC bursts and their stability even after application of sophisticated ambulatory ECG monitoring. Since the mechanism of these
cyclic bursts can not be fully clarified, further study using blood pressure and respiratory monitoring is required. Our findings may be of value in the clinical management and suppression of VPCs.

REFERENCES


