Concealed ventricular extrasystoles. A family of premature ventricular complexes with high spontaneous variability

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Background. **The term concealed ventricular extrasystoles defines a phenomenon in which premature beats have a cyclical distribution and manifest after a predictable number of intervening sinus beats. The extent of its spontaneous variability as well as the stability of its orderly distribution, however, have not been defined yet. The aim of this study was to assess whether there is any difference between the variability of concealed ventricular extrasystoles and their allorhythmic patterns.**

Methods. **The distribution of premature ventricular complexes (PVCs) was evaluated in 39 patients with frequent monomorphic PVCs (> 1000/die) during a baseline 24-hour ambulatory monitoring electrocardiogram. Patients were divided into two groups: group A had evidence of concealed ventricular extrasystoles, while in group B PVCs were randomly distributed. All patients underwent a second ambulatory monitoring electrocardiogram within 30-360 days.**

Results. **The overall number of PVCs did not differ between the groups. Patients of group A showed a very high spontaneous variability (p = 0.006) between the first and the second ambulatory monitoring electrocardiogram, whereas significant differences were not observed among patients of group B.**

Conclusions. **Concealed ventricular extrasystoles are not casual and transient, but should be regarded as a marker of a "family" of PVCs that have the tendency either to maintain their orderly distribution for long periods or to disappear suddenly.**

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Introduction

The number of isolated premature ventricular complexes (PVCs) observed during a 24-hour ambulatory monitoring electrocardiogram has a very wide spontaneous variability, and is related to the baseline number of PVCs detected[1], to the interval between the recordings[2,3], to the underlying disease[4,5], to the use of drugs such as beta-blockers[6,7], and to specific environmental conditions[8].

Sometimes PVCs show a cyclical distribution and manifest after a predictable number of intervening sinus beats. The phenomenon, described for the first time by Satoh et al.[9], was termed "concealed ventricular extrasystoles" (CVEs) by Schamroth and Marriott[10], and, despite the fact that several patterns have been reported[10-13], is still regarded as an uncommon finding. Experimental and clinical studies[14-18] have demonstrated that the underlying mechanism is the presence of a protected focus[19].

The aim of this study was to assess whether there is any difference between the variability of CVEs and their allorhythmic patterns, measured serially over an extended period of time, with respect to the spontaneous variability of random PVCs.

Serial ambulatory monitoring electrocardiograms, widely used to assess the spontaneous variations of cardiac arrhythmias, have been employed in the present study.

Methods

The patients included in the study fulfilled both the following criteria: a) a baseline 2-channel 24-hour ambulatory monitoring electrocardiogram showing ≥ 23 hours of analyzable tracing, and b) the presence of ≥ 1000 monomorphic PVCs.

Ambulatory monitoring electrocardiograms manifesting > 5% of PVCs with different morphologies were excluded.

CVEs were defined as sequences consisting of at least 10 consecutive isolated monomorphic PVCs that fulfilled one or more of the known criteria[20,21], i.e., having manifested after a number of predictable

intervening sinus beats, according to specific CVE formulas (Table I). As previously reported[20], only baseline ambulatory monitoring electrocardiograms in which at least 20% of the overall PVCs were included in the CVE sequences were accepted.

The study population consisted of 39 patients divided into two groups on the basis of the baseline findings:
- group A consisted of 19 consecutive patients (10 males, 9 females, mean age 61.21 ± 13.36 years, range 36-86 years) presenting with evidence of CVEs;
- group B consisted of 20 consecutive patients (14 males, 6 females, mean age 61.6 ± 15.4 years, range 17-76 years) presenting with PVCs which were randomly distributed.

All 39 patients underwent a second ambulatory monitoring electrocardiogram after 30-360 days.

Two patients of group A (cases no. 10 and 19) and 8 patients of group B (cases no. 3, 5, 7, 8, 11, 13, 14, and 15) occasionally manifested ventricular couplets (not included in the analysis).

Patients were not allowed to take any antiarrhythmic therapy with the exception of beta-blockers that were assumed without dose modifications by 2 patients of group A (cases no. 8 and 12) and by 3 patients of group B (cases no. 3, 14 and 20). Patients taking other cardioactive drugs did not modify their therapeutic regimen between the baseline and the final ambulatory monitoring electrocardiogram.

Ambulatory electrocardiographic recordings. Each patient underwent two 2-channel 24-hour recordings. Ambulatory monitoring electrocardiograms were performed using SpaceLabs tape recorders model 90205. Tapes were analyzed on a SpaceLabs FT2000 Medical Analysis and Review Station, a computerized system configured to provide automatic processing of the tapes and the possibility of reviewing/editing the data by the operator.

Detection of concealed ventricular extrasystoles. CVEs were detected by analyzing the whole 24-hour tracings[21]. Sequences containing supraventricular arrhythmias were excluded since interruption of the sinus rhythm could influence the distribution of the PVCs[22].

The basic patterns of CVEs (including some variants), the formulas, and the related number of intervening sinus beats, are listed in table I. For example, the "classical" concealed ventricular bigeminy is defined by the formula *2n-1* (where *n* is any integer), namely, by the presence of PVCs always intervening after an odd number of sinus beats. Concealed ventricular trigeminy is revealed by the presence of PVCs fulfilling the formula *3n - 1*, i.e. manifesting after 2 (*3×1-1*), 5 (*3×2-1*), 8 (*3×3-1*), 11, 14, 17, etc., intervening sinus beats (Fig. 1).

Only sequences consisting of at least 10 consecutive PVCs that fulfilled one of the CVE formulas were taken into account[20,21]. Thus, the possibility that 10 consecutive PVCs randomly manifest for example as concealed ventricular bigeminy, i.e., always after an odd number of intervening sinus beats, is 1/1024 (binomial probability), and it is 1/39 366 in case of concealed ventricular trigeminy.

Assessment of premature ventricular complex variability. The natural logarithm of the ratio PVCs/day, *ln*(PVCs/day + 1), was used to analyze the PVC variability. This transformation reduces variability among patients and potentially eliminates the skewness in the distribution of PVCs/day associated with the large differences among patients. One was added to the PVC rate so that in the event of a zero response rate, the logarithm is defined[3]. The mean and SD of the logarithms were calculated for both groups and both recordings. A second measure of variability was performed to assess the day to day variability among patients and was pooled over all patients in the study. This measure has been used to define the day to day variance for individual patients and is calculated using the formula $\Sigma (X - X)^2$ where the number of observations is always two, and the number of degrees of freedom is one.

Statistical analysis. Unless otherwise specified, data are presented as mean ± SD. The Student's t-test has been used to compare parametric data.

Table I. Basic patterns of concealed ventricular extrasystoles.

	Formula	No. intervening sinus beats
Concealed ventricular bigeminy	2n-1	1, 3, 5, 7, 9, 11, 13, etc.
	2n	2, 4, 6, 8, 10, 12, 14, etc.
	1 + 2n	1, 2, 4, 6, 8, 10, 12, 14, etc.
Concealed ventricular trigeminy	3n-1	2, 5, 8, 11, 14, 17, 20, etc.
	3n	3, 6, 9, 12, 15, 18, 21, etc.
Concealed ventricular quadrigeminy	4n-1	3, 7, 11, 15, 19, 23, 27, etc.
	4n	4, 8, 12, 16, 20, 24, 28, etc.
Concealed ventricular quintageminy	5n-1	4, 9, 14, 19, 24, 29, 34, etc.
Concealed ventricular hexageminy	6n-1	5, 11, 17, 23, 29, 35, 41, etc.

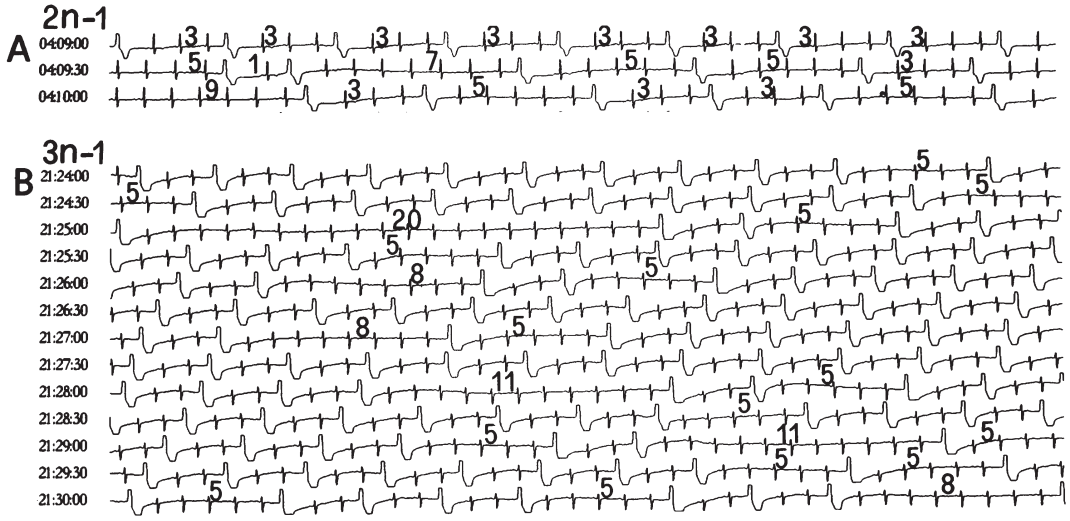

Figure 1. A: *case no. 3, lead CM5, continuous recordings. Sequences of concealed ventricular bigeminy, i.e., premature ventricular complexes fulfilling the formula 2n-1.* B: *case no. 8, lead CM5, continuous recordings. Sequences of concealed ventricular trigeminy, i.e., premature ventricular complexes fulfilling the formula 3n-1.*

Results

Tables II and III summarize the clinical findings of the patients. The patients' age (61.21 ± 13.36 vs 61.60 ± 15.41 years, p = NS) and the number of PVCs (349.68 ± 453.52 vs 225.75 ± 315.91, p = NS) did not significantly differ between the groups despite the fact that the total number of PVCs was higher in group A.

Spontaneous premature ventricular complex variability. Group A demonstrated a high variability in the number of PVCs. In this group, the natural logarithm of the ratio PVCs/day shifted from 5.262 ± 1.091 to 3.776 ± 2.548 (p = 0.006). In 6 patients presenting with a reduction ≥ 95%, the PVCs disappeared almost completely.

In group B patients, on the contrary, the variations in the number of PVCs were not significant (4.913 ± 0.927 vs 4.734 ± 1.294, p = NS). An abrupt reduction in the frequency of PVCs (≥ 95%) was never observed in this group.

The difference in the day to day variability between groups was statistically significant (1.6401 ± 2.341 vs 0.325 ± 0.505, p = 0.019).

Table II. Electrocardiographic data and clinical findings of Group A patients.

Case	Age (years)	Sex	Clinical data	Baseline		Control		Interval (days)
				PVCs/hour	Distribution	PVCs/hour	Distribution	
1	35	F	N	68	CVB	0	–	118
2	86	M	IHD (MI)	139	CVB	31	CVT	355
3	79	F	IHD (Hy)	62	CVB	3	–	350
4	65	M	IHD (MI)	1660	CVB	529	CVB	32
5	35	F	N	235	CVB	104	CVB	91
6	62	F	N	146	CVB	208	CVB	183
7	48	M	N	348	CVQ	557	CVQ-CVB	30
8	62	F	Hy	1420	CVT	371	CVT	360
9	67	M	HCM	704	CVB	606	CVB	237
10	57	F	N	382	CVB	0.21	–	45
11	56	M	IHD	285	CVB	105	CVB	31
12	76	M	HHD	42	CVT	380	CVB	58
13	49	F	N	384	CVB	470	CVB	35
14	60	F	HHD	88	CVB	0	–	325
15	60	M	IHD (MI)	147	CVB	2	–	274
16	74	M	HHD	51	CVB	12	CVB	32
17	65	M	IHD	97	CVB	52	CVB	31
18	70	F	IHD	51	CVB	0	–	58
19	57	M	IHD	335	CVB	85	CVB	85

CVB = concealed ventricular bigeminy; CVQ = concealed ventricular quadrigeminy; CVT = concealed ventricular trigeminy; DCM = dilated cardiomyopathy; HCM = hypertrophic cardiomyopathy; HHD = hypertensive heart disease; Hy = hypertension; IHD = ischemic heart disease; MI = myocardial infarction; N = normal; PVCs = premature ventricular complexes.

Table III. Electrocardiographic data and clinical findings of Group B patients.

Case	Age (years)	Sex	Clinical data	PVCs/hour		Interval (days)
				Baseline	Control	
1	67	M	IHD	62	65	32
2	64	M	N	315	510	31
3	67	M	IHD (MI)	75	60	205
4	71	M	DCM	73	100	35
5	78	M	HHD	202	161	40
6	17	M	N	1417	1220	360
7	60	F	Hy	52	435	45
8	67	M	Hy	508	196	278
9	46	F	N	180	77	64
10	63	M	Hy	534	261	60
11	44	F	N	54	171	32
12	76	M	HHD	110	13	353
13	72	F	Hy	56	289	361
14	68	F	IHD (MI)	304	501	98
15	66	M	Hy	67	14	343
16	77	M	IHD (MI)	125	7	218
17	76	F	HHD	170	128	60
18	36	M	IHD	72	116	88
19	62	M	IHD	71	38	153
20	55	M	IHD (MI)	68	72	122

Abbreviations as in table II.

Spontaneous variability of concealed ventricular extrasystoles. CVEs were persistent, i.e. were present in both the ambulatory monitoring electrocardiograms, in 13 out of 19 patients.

During the reassessment, group A patients manifested only PVCs fulfilling the CVE criteria, while in group B significant sequences of CVEs never occurred. CVEs disappeared in 3 group A patients and decreased by almost 95% in 3 other patients.

In 1 patient (no. 16), despite the fact that only 290 PVCs were observed during the second recording, the detection of CVEs was still possible since nearly all the PVCs occurred within 1 hour, manifesting the pattern of a concealed ventricular bigeminy variant (*2n*, i.e., PVCs occurring after an even number of intervening sinus beats).

Sixteen patients mostly showed concealed ventricular bigeminy (typical or variants) during the first recording. Two patients presented with concealed ventricular trigeminy. In 1 case, the PVC distribution fulfilled the criteria of concealed ventricular quadrigeminy.

Several patients showed different types of CVEs throughout the same ambulatory monitoring electrocardiogram; for example, the recording of patient no. 12, despite the prevalence of concealed ventricular trigeminy, sometimes revealed sequences of concealed ventricular bigeminy or its variants.

The prevalent pattern of CVEs detected during the first recording was maintained in the second ambulatory monitoring electrocardiogram in nearly all patients (Table II). Only in 3 patients was the prevalent pattern different, shifting from concealed ventricular bigeminy to trigeminy (patient no. 2), from concealed ventricular trigeminy to bigeminy (patient no. 12), and from concealed ventricular quadrigeminy to the even variant of concealed ventricular bigeminy (patient no. 7).

Discussion

The existence of a wide spontaneous variability of PVCs is a well-known phenomenon[1-5,23-25]. The extent of variability has been related to several factors: the number of PVCs[1], the interval between the baseline and the second ambulatory monitoring electrocardiograms[2,3,26], the duration of monitoring[1,5,6], the baseline clinical conditions such as the presence of coronary artery disease[4] or congestive heart failure[5], pharmacological treatment (for example, beta-blockers)[6,7], and specific environmental situations[8].

Our data indicate that the spontaneous variability of PVCs was significantly higher in group A, i.e., in the patients with CVEs. Other known factors that could potentially exert an influence on variability were homogeneously represented in both the groups (Tables II and III). The mean number of PVCs during the baseline ambulatory monitoring electrocardiogram was slightly higher in group A than in group B (349.68 ± 453.52 vs 225.75 ± 315.91/hour; p = NS). For this reason, on the basis of the literature[1], a lower variability in group A is to be expected.

The wide spontaneous variability of CVEs is somewhat predictable, since their manifestation, assuming a

focal origin, is governed by all the interactions between the ectopic activity and the dominant rhythm.

CVEs were described by Satoh et al.[9] and by Schamroth and Marriott[10]. The first pattern reported was concealed ventricular bigeminy[10,11]. Several other patterns[12,13] as well as different variants[21,27,28] were then observed. Schamroth and Marriott[10] postulated a focal genesis for these arrhythmias. This hypothesis was later confirmed by Jalife and Moe[29] who demonstrated that the impulses of the dominant rhythm can exert an electrotonic influence upon a protected (parasystolic) focus. Under experimental conditions, in the presence of the phenomenon of electrotonic modulation, PVCs often manifest according to the formulas of CVEs after a predictable number of intervening sinus beats[13,14,19,30,31]. In such situations, the parasystolic impulses may generate PVCs with an almost fixed coupling interval, often without fusion beats, and characterized by an orderly distribution. These studies demonstrated that several different patterns of CVEs may be observed during the same recording, depending on the reciprocal relationship between the cycle length variations of both the dominant rhythm and the ectopic pacemaker, and, moreover, on the variations of the effect exerted by the electrotonic modulation[20,21,31-33]. In fact, an individual patient does not present with a pure concealed ventricular bigeminy or trigeminy, but with CVEs that can frequently shift from one pattern to another[20,21,34,35], and, sometimes, can even shift to a typical parasystolic pattern (mathematically related interectopic intervals, variable coupling intervals, presence of fusion beats)[36]. This behavior has been confirmed by several clinical observations, and every CVE pattern has been explained on the basis of the electrotonic influence exerted by the dominant impulses upon a parasystolic focus[13,14,21,22,31,34,35,37-41]. Occasionally, CVEs have been interpreted as a manifestation of reentry[34,42-46]. The phenomenon of reflection, an unusual

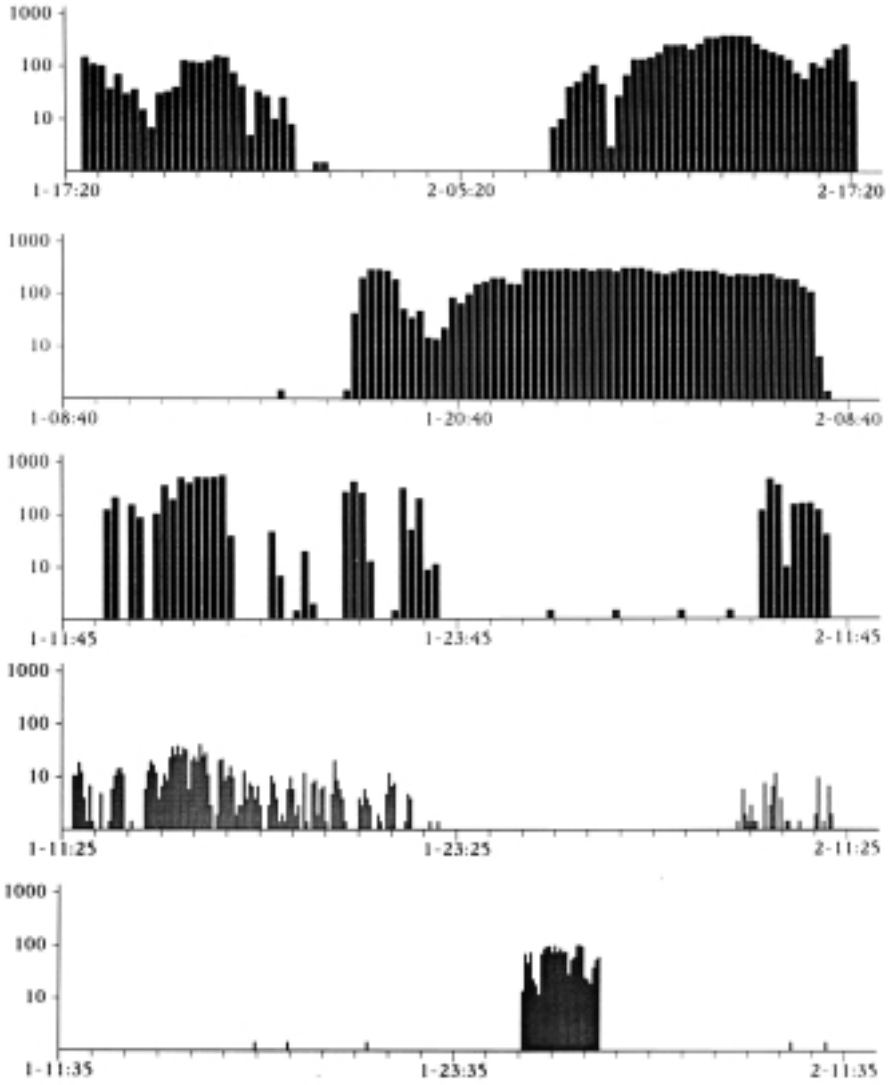

Figure 2. *Histograms reflecting the number of premature ventricular complexes during the 24-hour recordings, as detected in 5 cases of group A (cases no. 8, 7, 11, 14, and 15). Concealed ventricular extrasystoles are evident for several hours and then suddenly disappear, or, less frequently, are concentrated in a few hours* (bottom).

pattern of reentry based on the presence of a focus and thus closely related to parasystole[19,47], has been proposed in these cases.

CVEs can disappear for relatively long periods. This phenomenon is well known and could depend either on the possible irregularity of the dominant rhythm, such as sinus arrhythmia, or on modifications of the relationships between the cycle length and the characteristics of modulation[32,36,41,48-53].

CVEs can either manifest intermittently for a few minutes or a few hours (Fig. 2), or completely disappear within a few days. However, they cannot be regarded as a casual phenomenon since they identify a well-defined population of patients: none of the group A patients presented with random PVCs during the second ambulatory monitoring electrocardiogram, while no patient in group B showed PVCs with an orderly distribution.

Reassessment of the patients after a relatively long period of time furthermore indicated that CVEs are not transient. Thirteen out of 19 patients still had a regular distribution of the PVCs in the second ambulatory monitoring electrocardiogram. In 3 cases PVCs totally disappeared, and in the remaining 3 patients the reduction was ≥ 95%. When the PVCs were sporadic, it was not possible, obviously, to identify any pattern of distribution. Sometimes, however, sequences of CVEs were detected even if the mean number of PVCs/hour was < 50. In these cases (case no. 12, first ambulatory monitoring electrocardiogram; cases no. 2 and no. 16, second ambulatory monitoring electrocardiogram), about 50% of the PVCs were concentrated in less than 1 hour and it was thus possible to evaluate their distribution (Fig. 2).

In conclusion, CVEs are not casual and transient, but should be regarded as a marker of a "family" of PVCs that tend to either maintain their orderly distribution for long periods or to disappear suddenly. Their identification is not meaningless since a) it provides information about the electrogenesis, mostly focal or less commonly due to reentry mechanisms closely linked to parasystole, and b) it indicates that a very wide spontaneous variability should be expected. In these patients, all the criteria currently acknowledged for the evaluation of the efficacy of antiarrhythmic drugs, even the more restrictive, may be ineffective.

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