Premature Ventricular Complex Morphology

A Marker for Left Ventricular Structure and Function

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The shape of a premature ventricular complex (PVC) might reflect the presence or absence of myocardial disease. To test this, 100 patients with a PVC on a 12-lead electrocardiogram at cardiac catheterization or nuclear angiography were classified according to PVC morphology. Group 1 (n=50) had PVC QRS complexes with either smooth and uninterrupted contour or with narrow (<40 msec) notching. Group 2 (n=50) demonstrated PVC with broad (≥40 msec) notching or shelves. Clinical, electrocardiographic and angiographic variables were assessed to define group differences. All patients had one or more etiological forms of heart disease none of which distinguished either group. Groups 1 and 2 differed with respect to a history of congestive heart failure (12% vs. 66%, p=0.0004), dilated cardiomyopathy (2% vs. 38%, p=0.0005), and the presence of mitral regurgitation (13% vs. 58%, p=0.001), respectively. In group 1, 45 of 50 (90%) patients with a PVC had no notching. Patients in group 2 had greater PVC QRS duration as compared with patients in group 1 (181±6 vs. 134±3 msec, p=0.0001). End-diastolic volume index (EDVI) (78±3 vs. 139±11 ml/m^2, p=0.0000) and ejection fraction (EF) (0.59±0.02 vs. 0.34±0.03, p=0.0000) significantly discriminated between group 1 and 2, respectively. By defining left ventricular structure and function as EDVI less than or equal to 90 ml/m^2 or greater than 90 ml/m^2 and EF equal to or greater than 0.50 or less than 0.50, sensitivity was 92% and specificity was 80% for PVC morphology. We conclude that a broadly notched PVC of long duration (≥160 msec) is a simple and reliable 12-lead electrocardiographic marker for a dilated and globally hypokinetic left ventricle in a nonspecifically diseased heart while a PVC with smooth contour or narrow notching with short duration (<160 msec) reflects a normal size heart with normal or near-normal systolic function despite the presence of underlying disease. (*Circulation* 1990;81:1245–1251)

The morphological features of the premature ventricular complex (PVC) have been cited as a clue to the presence or absence of underlying cardiac disease.[1–4] Scherf and Schott[1] recognized that PVCs with exceptionally wide QRS complexes frequently occurred in diseased hearts but the degree of widening permitting identification of such a condition was not speculated on. Soloff[4] found that PVCs with a bizarre and distorted configuration were highly suggestive of underlying myocardial disease in contrast to those with the "classic" smooth pattern. Approximately half of the patients with the classic PVC, however, had clinically evident cardiac disease. The etiological or pathophysiological basis for this apparent relation was not examined, partly because these studies were performed at a time when the more sophisticated diagnostic techniques currently in widespread use were not available.

The purpose of the present study was to better characterize the nature of the relation between PVC morphology and myocardial disease state, and to determine if structural or functional information can be reliably inferred from PVC morphology on the 12-lead electrocardiogram (ECG).

Methods

Twelve-lead ECGs (Marquette Electronics, Milwaukee, Wisconsin, or Hewlett-Packard Co., Palo Alto, California) were reviewed both retrospectively (n=30) and prospectively (n=70) in 100 consecutive patients undergoing cardiac evaluation by elective cardiac catheterization, echocardiography, or nuclear ventriculography. Electrocardiographic tracings were obtained within 3 months of evaluation for patients with no new events and at the time of angiography for those admitted with acute events. In each patient, at least one PVC was present, enabling classification into one of two groups based on PVC morphology. Group 1 consisted of patients having classic PVC

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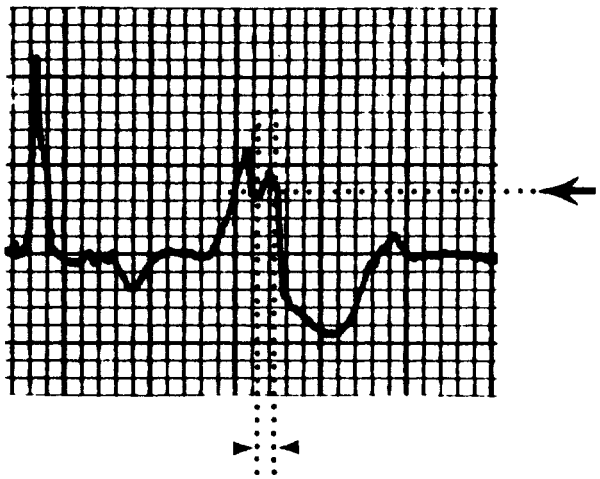

FIGURE 1. *Graph of method of PVC QRS notch duration measurement. To establish onset and end point, horizontal line is drawn at level of notch nadir. In example shown, onset of notch corresponds to vertical line through nadir and ends where QRS intersects horizontal line.*

QRS complexes with either smooth and uninterrupted contour or with narrow (<40 msec) notches (type I). Group 2 was comprised of patients with PVC QRS complexes containing wide (≥40 msec) notches or shelves (type II). The notch-width measurement is shown in Figure 1. When notching occurred near the summit, the nondominant peak was measured. A shelf was defined as a horizontal interruption in the inscription of the PVC QRS complex. Because electrocardiographs record three simultaneous leads, a minimum of three and a maximum of 12 lead configurations of a single PVC are provided. A patient was classified as having a type II configuration if the PVC from at least one of the 12 leads met the morphological criterion. In group 1, an average of four leads per tracing contained a PVC, whereas an average of five leads per tracing displayed a PVC in group 2. In group 2, the type II morphological criterion was clearly met in approximately three of four (72%) leads. Care was taken to exclude fusion, aberrant supraventricular complexes, and p wave superimposition on the PVC. Figure 2 provides representative examples of type I and type II PVC.

In all patients, a complete history and a recent chest radiograph were available. Data was available from echocardiography in 42 patients (42%) and from cardiac catheterization or nuclear ventriculography in 98 (98%). Clinical variables analyzed included the presence or absence of the following: a history of congestive heart failure by New York Heart Association (NYHA) functional class; dilated, ischemic, or hypertrophic cardiomyopathy; hypertension; myocardial infarction; valvular heart disease; and ventricular tachycardia. Other rhythm disturbances were not categorized. These diagnoses were either previously made by conventional means, as indicated in the medical record, or established from the cardiac catheterization. Because many patients suffering acute events such as myocardial infarction

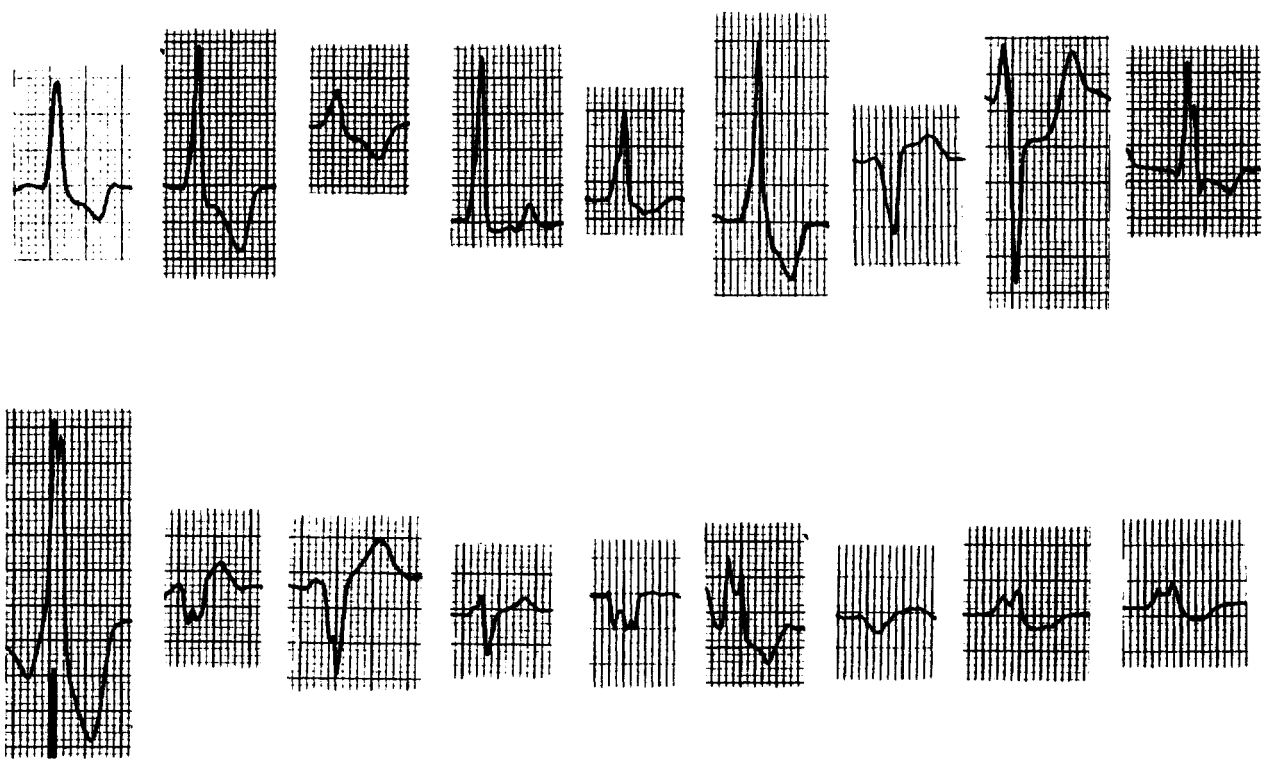

FIGURE 2. *Representative tracings of type I* (upper panels) *and type II* (lower panels) *premature ventricular complexes. Contrast smooth and uninterrupted contour of narrow type I QRS to notched or shelved, wide type II QRS.*

or heart failure had preexisting chronic heart disease, no attempt was made to relate PVC morphology to disease acuity. Electrocardiographic variables included sinus conducted and PVC QRS duration, mean sinus-conducted QRS axis, evidence (Estes criteria) for left ventricular hypertrophy (LVH) with or without "strain" pattern, myocardial infarction, and nonspecific intraventricular conduction defect (NIVCD). NIVCD was diagnosed if the sinus QRS duration was greater than 0.10 seconds and could not be explained by patterns of LVH, myocardial infarction, or fascicular block.

Echocardiographic variables included interventricular septal and posterior wall thicknesses.

Descriptions of global and regional left ventricular wall motion and values for end-diastolic volume indices (EDVIs), end-systolic volume indices (ESVIs), and ejection fraction (EF) were obtained from either contrast or nuclear ventriculography. Regional wall motion pertained to five segments (i.e., anterior, apical, inferior, septal, and posterobasal) and was graded as hypokinetic, akinetic, or dyskinetic. Additional data obtained from cardiac catheterization included the presence of obstructive (>70%) involvement of the three major coronary vessels.

Patients receiving Vaughan-Williams class I and III antiarrhythmic drugs were excluded from the study.

Statistical analysis of all discrete variables was performed with a 2×2 χ^2 test and expressed as a raw score or as a percentage of the total number of patients in either group. For continuous variables, an unpaired *t* test was used with data expressed as a mean±SEM.

Results

Clinical Characteristics

Of the 100 patients evaluated, 50 were classified into group 1 (type I PVC) and 50 were classified into group 2 (type II PVC). Ages were in the range of 34–72 years (mean, 60±2 years) in group 1 and in the range of 37–77 years (mean, 60±2 years) in group 2. Body weights were in the range of 56–128 kg (mean, 86±3 kg) in group 1 as compared with 57–130 kg (mean, 82±3 kg) in group 2. These differences were not statistically different. Table 1 lists select characteristics of the initial 30 patients evaluated retrospectively to demonstrate that this series of patients did not differ from the total number of patients with respect to important variables.

As shown in Table 2, only six (12%) of the patients with type I PVC had a history of congestive heart failure as compared with 33 (66%) of the patients with type II PVC (p=0.0004). In group 2, there was an even distribution between NYHA class I–II and class III–IV functional status, that is, 32% and 34%, respectively. One (2%) patient from group 1 and 19 patients (38%) from group 2 carried the diagnosis of dilated cardiomyopathy (p=0.0005). Ischemic cardiomyopathy was not present in group 1 but was diagnosed in 16 (32%) patients from group 2 (p=0.0004). Hypertrophic cardiomyopathy was not present in group 1 but was diagnosed in three patients from group 2. Although mitral regurgitation was significantly more common in group 2 than in group 1 (58% vs. 13%, p=0.001), aortic valvular disease did not discriminate between groups. The prevalence of hypertension, myocardial infarction, or ventricular tachycardia by history was also similar in the two groups. Importantly, no patient from either group was free of ischemic, hypertensive, valvular, or idiopathic forms of heart disease.

Table 1. Selected Characteristics of Patients Evaluated Retrospectively

Characteristic	Group 1	Group 2	*p*
n	15	15	
Age (yr±SEM)	62±2	63±2	NS
Weight (kg±SEM)	86±5	80±6	NS
Dilated cardiomyopathy	3/15	13/15	0.01
Sinus QRS duration (msec±SEM)	91±5	113±6	0.008
PVC QRS duration (msec±SEM)	137±6	180±8	0.001
Normal left ventriculogram	7/15	3/15	0.27
Global hypokinesis	1/15	11/15	0.01
EDVI (m/m^2±SEM)	85±5	109±15	0.09
Ejection fraction	0.57	0.34	0.002

PVC, premature ventricular complex; EDVI, end-diastolic volume index.

Electrocardiographic Characteristics

As shown in Table 3, no significant differences were observed between the two groups regarding the

Table 2. Clinical Characteristics Associated With Types I and II Premature Ventricular Complex

Characteristic	Type I PVC	Type II PVC	*p*
n	50	50	
Mean age (yr±SEM)	60±2	60±2	NS
Mean weight (kg±SEM)	86±3	82±3	NS
Congestive heart failure (%)	6 (12)	33 (66)	0.0004*
NYHA class I–II	4 (8)	16 (32)	0.013*
NYHA class III–IV	2 (4)	17 (34)	0.002*
Hypertension (%)	27 (54)	15 (30)	0.12
Myocardial infarction (%)	16 (32)	29 (58)	0.10
Cardiomyopathy (%)			
Dilated	1 (2)	19 (38)	0.0005*
Hypertrophic	0 (0)	3 (6)	0.08
Ischemic	0 (0)	16 (32)	0.0004*
Valvular disease (%)			
Mitral regurgitation	6 (13)	28 (58)	0.001*
Aortic stenosis	4 (8)	6 (12)	0.55
Aortic regurgitation	5 (10)	3 (6)	0.50
Ventricular tachycardia (%)	11 (22)	15 (30)	0.49
Diabetes mellitus (%)	6 (12)	4 (8)	0.55
COPD (%)	6 (12)	11 (22)	0.26

PVC, premature ventricular complex; COPD, chronic obstructive pulmonary disease.
*Significant difference comparing group 1 with group 2.

TABLE 3. Electrocardiographic Characteristics Associated With Types I and II Premature Ventricular Complex

Characteristic	Type I PVC	Type II PVC	*p*
Normal ECG (%)	12 (24)	5 (10)	0.27
LVH (%)	6 (12)	5 (10)	0.77
LVH with strain (%)	7 (14)	9 (18)	0.65
Anterior infarct (%)	5 (10)	13 (26)	0.79
Inferior infarct (%)	8 (16)	11 (22)	0.54
Bundle branch block (%)	4 (8)	7 (14)	0.40
Nonspecific intraventricular conduction defect (%)	3 (6)	15 (30)	0.009*
Sinus QRS duration (msec±SEM)	90±3	113±4	0.0005*
PVC QRS duration (msec±SEM)	134±3	181±6	0.0001*
Mean QRS axis (°±SEM)	13±9	19±13	NS

PVC, premature ventricular complex; ECG, electrocardiogram; LVH, left ventricular hypertrophy.
*Significant difference comparing group 1 vs. 2.

electrocardiographic diagnoses of a normal ECG, LVH, or LVH with strain or myocardial infarction. Right bundle branch block was present in four patients from group 1 and six patients from group 2. The one patient with left bundle branch block was from group 2. A nonspecific intraventricular conduction defect was encountered in three of 50 (6%) patients from group 1, whereas 15 of 50 (30%) patients in group 2 ($p=0.009$) showed this finding. Consistent with this latter observation, sinus QRS duration was significantly greater in group 2 as compared with group 1, that is, 113±4 versus 90±3 msec, respectively ($p=0.001$).

In group 1, 45 of 50 (90%) patients exhibited smooth and uninterrupted PVC QRS morphology without notching. Five patients had PVC with narrow notching. As shown in Figure 3, group differences in PVC QRS duration were found to be significant; the mean value of the type I PVC was 134±3 msec as compared with the type II mean of 181±6 msec ($p=0.0001$).

The direction of the mean sinus QRS axis measured +13±9° in group 1 as compared with +19±13° in group 2 and was not significantly different.

Echocardiographic Characteristics

Echocardiographic data was available in 16 of the 50 patients in group 1 and in 24 of the 50 patients in group 2. Three patients (19%) in group 1 had interventricular septal thickness exceeding 11 mm as compared with nine patients (33%) in group 2 ($p=0.13$). Posterior wall thickness was increased in five patients (27%) in group 2 and in three patients in group 1. Neither variable achieved statistical significance, and the numbers might be too small for meaningful comparisons.

Radiological and Angiographic Characteristics

Table 4 lists the radiographic, global, and regional wall motion; left ventricular volume index; and ejection fraction data. Cardiomegaly was evident in only five patients (10%) in group 1 as compared with 33 patients (66%) in group 2 ($p=0.0003$). Pronounced group differences were noted in global left ventricular wall motion determined by either contrast or nuclear angiography. Three of 49 patients (6%) in group 1 were judged globally hypokinetic as compared with 37 of 49 patients (76%) in group 2 ($p=0.0001$). Conversely, 36 of 49 patients (73%) in group 1 had a normal left ventriculogram as compared with eight of 49 patients (16%) in group 2 ($p=0.0007$). Ten patients (20%) in each group had one or more hypokinetic segments. One (2%) patient from group 1 had one or more akinetic segments as compared with 18 (38%) patients from group 2 ($p=0.0005$). Although this difference was significant, global wall motion was hypokinetic in all 18 patients. Regional dyskinesis was observed in three patients from group 2 and in no patients from group 1.

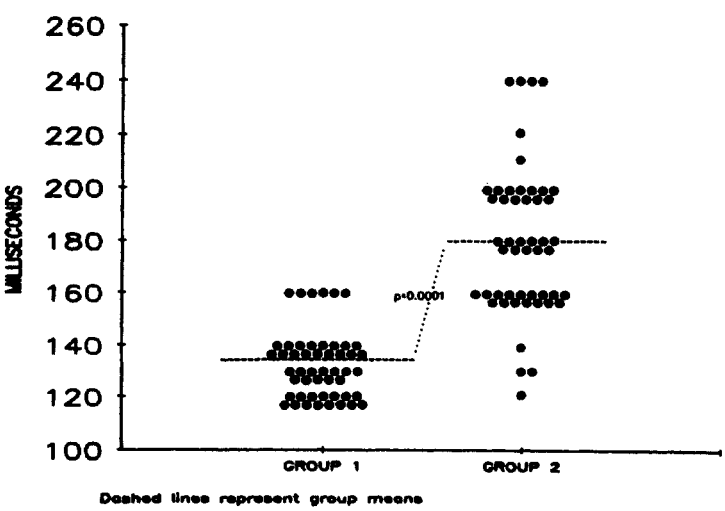

FIGURE 3. *Scatterplot showing group comparison of mean duration of PVC QRS complexes.*

TABLE 4. Angiographic and Radiologic Characteristics Associated With Types I and II Premature Ventricular Complex

Characteristic	Type I PVC	Type II PVC	*p*
Normal cardiac silhouette (%)	45 (90)	17 (34)	0.0003*
Cardiomegaly (%)	5 (10)	33 (66)	0.0003*
Normal left ventriculogram (%)	36 (73)	8 (16)	0.0007*
Global hypokinesis (%)	3 (6)	37 (76)	0.0001*
One or more hypokinetic segments (%)	10 (20)	10 (20)	NS
One or more akinetic segments (%)	1 (2)	18 (38)	0.0005*
EDVI (ml/m^2±SEM)	78±3	139±11	0.0000*
ESVI (ml/m^2±SEM)	34±2	98±10	0.0000*
Ejection fraction	0.59±0.02	0.34±0.03	0.0000*

PVC, premature ventricular complex; EDVI, end-diastolic volume index; ESVI, end-systolic volume index.
*Significant difference comparing group 1 vs. 2.

Ventricular volumes corrected to body surface area were significantly larger in patients with the type II PVC. EDVI was 78±5 ml/m^2 in group 1 as compared with 139±10 ml/m^2 in group 2 (p=0.0001), whereas ESVI was 34±2 ml/m^2 as compared with 98±10 ml/m^2 (p=0.0001) in groups 1 and 2, respectively.

EF also differed significantly with a mean value of 0.59±0.03 in group 1 as compared with 0.35±0.04 in group 2 (p=0.0001); 66% of patients in group 2 had an EF less than 0.40.

The relations of EDVI and ESVI to EF are illustrated in two scatterplots (Figure 4). The separate distributions of the two groups (open vs. closed circles) emphasizes the importance of both left ventricular size and function as discriminating variables.

Analysis of the coronary arteriograms revealed no significant group differences when considering the presence or absence of coronary artery disease (CAD). CAD was present in 24 of 42 patients (57%) from group 1 and in 24 of 36 patients (67%) from group 2 (p=0.6). As shown in Table 5, however, there seemed to be a trend for group 2 to contain a larger proportion of patients with more severe CAD and a smaller proportion of patients with less severe CAD as compared with group 1; three-vessel disease was found in 14 of 36 (39%) patients from group 2 as compared with six of 42 (14%) patients from group 1

TABLE 5. Coronary Artery Disease Prevalence in Types I and II Premature Ventricular Complex

Disease severity	Type I PVC	Type II PVC	*p*
n	42	36	
Normal coronaries (%)	18 (43)	12 (33)	0.57
Single-vessel disease (%)	11 (26)	4 (11)	0.57
Two-vessel disease (%)	7 (17)	6 (17)	NS
Three-vessel disease (%)	6 (14)	14 (39)	0.16
All coronary disease (%)	24 (57)	24 (66)	0.68

PVC, premature ventricular complex.

(p=0.54). Conversely, single-vessel disease affected only four of 36 (11%) patients from group 2 in contrast to 11 of 42 (26%) patients from group 1 (p=0.16). Importantly, normal coronaries were equally prevalent, that is, present in 18 of 42 (43%) patients from group 1 and in 12 of 36 (33%) patients from group 2 (p=0.57).

Sensitivity and Specificity

Examination of all variables showing significant differences between groups reveals that those variables representing indices of left ventricular size and global left ventricular function best distinguish between the two groups of patients. Accordingly, it was felt that the appropriate criteria defining these characteristics should include both EDVI (> or ≤90 ml/m^2) and EF (< or ≥0.5). Figure 5 shows that in patients with type I PVCs, 33 had both criteria within normal limits and only three had criteria that were both abnormal. In seven patients, discordant criteria were present, and in another seven patients, data was incomplete. In patients exhibiting a type II PVC, both criteria were abnormal in 34 patients, whereas both criteria were normal in eight patients. There were discordant criteria in four patients and incomplete data in another four patients. Thus, sensitivity was measured at 92%, and specificity was measured at 80%. Overall, accuracy was measured at 86%. When the criteria were redefined by substituting ESVI (> or ≤40 ml/m^2) for EDVI, sensitivity decreased to 87% but specificity increased to 82%. To ascertain the importance of PVC QRS duration, the patient database was sorted to control for this variable instead of morphology, and sensitivity decreased from 92% to 84%.

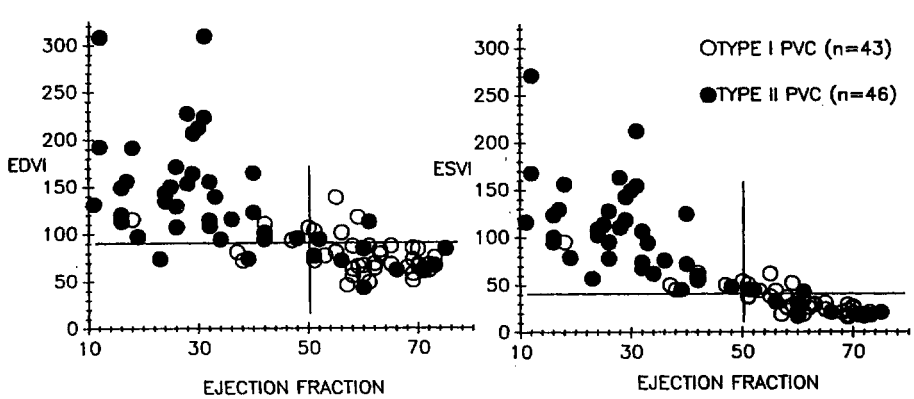

FIGURE 4. *Scatterplots of end-diastolic volume index (EDVI)* (left panel) *and end-systolic volume index (ESVI)* (right panel) *versus ejection fraction. Open circles represent patients from group 1 (type I PVC), whereas closed circles correspond to group 2 (type II PVC). Horizontal lines represent upper limit of normal for volume index. Vertical lines separate ejection fraction values above and below 0.50.*

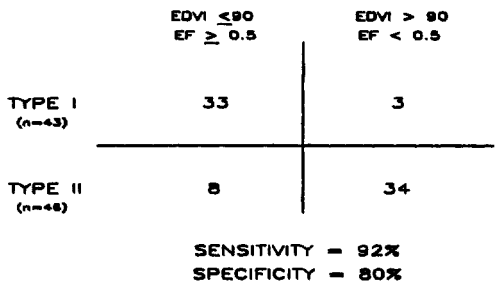

FIGURE 5. *Sensitivity and specificity of type I versus type II in PVC morphology as defined by end-diastolic volume index (EDVI) and ejection fraction (EF).*

Discussion

The clinical, noninvasive, and invasive data presented in this study verify an association between a dilated, globally hypokinetic ventricle and PVCs with morphology and duration differing from those occurring in normal-size hearts with good left ventricular function. Because all patients in both groups in our study had at least one form of heart disease, it is doubtful that the type II PVC (broad notches or shelves coupled with QRS duration ≥160 msec) is a useful discriminator between the presence or absence of heart disease, as previously suggested.[1-4] Rather, the type II PVC might serve as a reliable marker for a particular structural and functional state of a nonspecifically diseased myocardium: dilated and globally hypokinetic. On the other hand, the type I PVC (smooth contour or narrow notches as well as QRS duration <160 msec) is more likely to identify patients with normal-size hearts with normal or near-normal left ventricular function despite the presence of underlying cardiac disease. The findings in this study might also have an associated prognostic value because it is well known that left ventricular EF smaller than 0.40 and cardiac enlargement are among the strongest predictors of survival in patients with cardiac disease.

No one etiological classification of myocardial disease predominated in either group, including coronary artery disease. Further, neither the presence nor severity of coronary artery disease discriminated between groups. The severity of this disease's end-organ damage, however, did seem important as reflected by the significantly greater prevalence of ischemic cardiomyopathy and akinetic wall motion abnormalities in group 2.

Other than PVC morphology and duration, the only electrocardiographic feature that provided discriminating information was the duration of sinus-conducted QRS complexes. This might reflect the same disturbance in conduction responsible for the wide and notched PVC as is herein discussed.

Reference to electrocardiographic markers of cardiac enlargement with coexisting impaired systolic function is rare in the literature. In two reports, the suggested electrocardiographic signs either lacked sensitivity[5] or neither sensitivity nor specificity were determined.[6] Other work on the subject has suggested that there are no reliable electrocardiographic signs in congestive heart failure[7] or cardiomyopathy.[8,9] We found the simple attribute of PVC morphology to provide relatively high sensitivity and specificity. Linked to the morphological feature is the importance of duration of the PVC QRS complex: no patient in group 1 had a duration longer than 160 msec. In only four patients from group 2 was duration shorter than 160 msec; however, all had global hypokinesis, and two had an EDVI greater than 90 ml/m^2 with an EF less than 0.50. Although a lower sensitivity was found when the database was sorted to control for PVC QRS duration, it is possible that PVC QRS morphology and duration are pathogenetically similar. The data suggest that the inclusion of PVC QRS duration with the morphology criteria will help minimize the number of false positives. The 160-msec cutoff value for PVC duration used to distinguish the PVC types was chosen by determining the midpoint between the means for both groups because these data are normally distributed.

There are several limitations of this study. First, because all PVC were spontaneous and captured during the recording of a standard 12-lead electrocardiogram, there was no opportunity to examine PVC morphology in all 12 leads simultaneously. For example, had more leads been available for analysis of the three false-negative patients, the sensitivity of the test might have improved beyond 92%. The reliability of the type II morphology as a manifestation of an "abnormal" ectopic excitation process is reflected by the fact that approximately three of four leads displaying a PVC in group 2 readily met the type II criteria, which required that only one lead demonstrate wide notching. The finding that the type II morphology was not demonstrable in all leads containing a PVC is not surprising because the scalar representation of the excitation process depends, in part, on lead orientation. Thus, the provision of three simultaneously recorded leads in conventional ECGs minimizes the chance of a missed notch or shelf if there is only one lead available. Second, the occurrence of multiformity cannot be excluded with certainty because multiple PVCs on a given ECG were recorded in different leads. Another limitation results from the fact that all patients in the study were referred for evaluation of known or suspected heart disease. Accordingly, patients without heart disease but in whom PVC exist were not evaluated.

Anatomic and Electrophysiological Basis of the Ugly PVC

Wide and broadly notched PVCs as well as nonspecific sinus QRS widening can reflect a global form of impaired ventricular conduction in which ventricular mass, chamber size, and global function play important pathogenetic roles. Chronicity of the underlying disease state might also be a mitigating factor.

A direct causal role for severe degrees of regional myocardial scarring in the genesis of the type II PVC cannot be ascertained from the present data because it is unclear whether discrete scarring per se or its eventual impact on global structure and function is

most important. Several factors, however, suggest that scarring itself might be a sufficient but not necessary substrate, and they are as follows: 1) all of the patients in group 2 with one or more akinetic segments also had global hypokinesis, 2) 42% (21/49) of the patients in group 2 did not have discrete regional wall motion abnormalities, 3) the type II PVC was present in patients with normal coronaries only when the ventricle was dilated and globally hypokinetic, and 4) regional wall motion abnormalities were present in 22% (11/49) of the patients with the type I PVC (i.e., patients with scars but without notching).

Precise anatomic and electrophysiological explanations for an exceptionally wide and distorted PVC are not available; however, Ferrans et al[10] suggest that dilation of the T-system and the presence of fibrillar material within the lumen of the T-tubules might somehow interfere with the conduction of the impulse from the cell surface through the T-system into the depths of the cell. If sodium conductance is impaired because of the altered microanatomy, dV/dt_{max} and, consequently, global intraventricular conduction velocity might be altered. Whether these changes are the consequence of a long-term mechanical overload state or the direct result of an unknown initiating agent is unknown and invites further investigation. Another factor might relate to the effect of diffuse interstitial collagen accumulation on the integrity of cell-to-cell communication by desmosomes. The role of myofiber orientation is uncertain because this feature seems relatively undisturbed in patients with idiopathic dilated cardiomyopathy.[10] It is conceivable, however, that the conditions governing anisotropic conduction are altered in such a way that longitudinal conduction dominates, resulting in a more unfavorable safety factor for propagation throughout regions of the ventricles. The relatively slower transverse propagation might become exaggerated because of diffusely impaired continuity between adjacent myofibers or myofibrillar bundles. Finally, the longer path length traversed by the ectopic impulse in dilated hearts with increased mass would result in longer total ventricular activation time and wider PVC QRS duration.

The net effect of globally impaired ventricular conduction on the morphology of a PVC might be to permit the vectorial representations of different regions of the heart undergoing ectopic excitation to manifest with greater asynchrony than would have occurred if conduction were less impeded. Hence, there are the broad notches, analogous to the asynchronous activation characterizing the patterns in bundle branch block. The same process could explain the wider sinus-conducted QRS duration but, because of the role played by the His-Purkinje system in rapidly and uniformly distributing the impulse to both ventricles, total ventricular activation time is shorter and the result is smaller notches if the frequency response of the recorder is high enough. This is consistent with the observations of Flowers and Horan[11] who found that the greatest diagnostic accuracy of sinus QRS notching seen in high-frequency ECGs was in the detection of biventricular enlargement, a population in which it is unusual to find normal EFs. Given a frequency-response upper limit of 100 Hz in conventional electrocardiographs, the result is a more "smoothed" QRS of greater duration.

The clinical value of distinguishing between the type I and II PVC can be as an inexpensive and noninvasive means of risk stratifying patients early during the initial evaluation period when used in conjunction with the history and physical examination. It can also serve to prompt additional caution when contemplating the use of drugs that significantly impair ventricular function, including certain antiarrhythmic agents. In this regard, care must be used when interpreting PVC morphology in patients undergoing therapy with sodium channel blocking drugs, especially class IC agents. Finally, it is of interest whether selective high-resolution electrocardiographic recordings of the two types of premature ventricular complexes might disclose differential vulnerability to sustained ventricular arrhythmias or to proarrhythmic consequences when using sodium channel blocking agents.

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