Arrhythmias in Patients with Mitral Valve Prolapse

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SUMMARY

Resting ECGs, exercise treadmill tests and 24-hour ambulatory ECGs were recorded and analyzed in 24 unselected patients with mitral valve prolapse. Arrhythmias were frequent. There were three distinct groups of patients, defined on the basis of total number of premature ventricular contractions (PVCs) during the 24 hours: there were no PVCs in 25%, infrequent PVCs in 25%, and frequent PVCs in 50%. Complex ventricular arrhythmias, including ventricular tachycardia in five patients, were found almost exclusively in the group with frequent PVCs. Fifteen of the 24 patients demonstrated atrial premature contractions (APCs) during the 24 hours. Complex atrial arrhythmias were found among patients with infrequent, as well as those with frequent, APCs. Supraventricular tachycardia was detected in seven of these patients. The incidence of PVCs decreased during sleep in 58% of the patients, increased in 17%, and showed no change in 25%. The incidence of APCs decreased during sleep in 67% of the patients and showed no change during sleep in 33%. A poor correlation was found between symptoms recorded in patient diaries and changes noted on 24-hour ECG recordings. The peak PVCs/15 min and peak APCs/15 min during a 24-hour period of monitoring was found to be an excellent guide to the total number of PVCs and APCs occurring during that period. This permits an accurate prediction of the total number of PVCs in 24 hours after performing an exact PVC count on only 15 minutes of ECG data. Finally, the 24-hour ambulatory ECG was more sensitive than the treadmill test and both were superior to the 12-lead ECG for detecting arrhythmias in these patients.

Additional Indexing Words:
Ambulatory ECG recordings
Mid-systolic click, late-systolic murmur syndrome
Arrhythmia detection

Patients with mitral valve prolapse have a wide variety of auscultatory findings, symptoms and ECG abnormalities. The ECG abnormalities include Q waves, abnormal ST segments and T waves, prolonged QT intervals, prominent U waves, conduction abnormalities and various arrhythmias. One preliminary report suggests a high incidence of ventricular arrhythmias in ambulatory patients with mitral valve prolapse. However, the majority of patients in that report had arrhythmias as the presenting problem. Several studies have documented an increase in these arrhythmias during exercise treadmill testing. Sleep has been shown to decrease ventricular ectopic activity in a group of patients in whom coronary artery disease was the predominant underlying heart disease. One study in patients with the Wolff-Parkinson-White syndrome showed a poor correlation between cardiac symptoms and arrhythmias detected by ambulatory monitoring.

The purpose of the present study was to evaluate atrial and ventricular arrhythmias in a group of unselected patients with mitral valve prolapse, using the 24-hour ambulatory ECG. The incidence and types of arrhythmias were determined, as well as their relationship to sleep, symptoms, and heart rate. The validity of using the peak premature ventricular contractions per 15 minutes (peak PVCs/15') during a given period of monitoring as a guide to the total number of PVCs occurring during that period was demonstrated in patients with mitral valve prolapse. Finally, arrhythmia detection by 12-lead ECG and exercise treadmill test is compared to the 24-hour ambulatory ECG to determine the most sensitive method for arrhythmia detection in these patients.

Methods

Patient Population

Twenty-four patients, five men and 19 women, with mitral valve prolapse diagnosed by echocardiogram and/or left ventricular angiogram were studied after informed consent was obtained. The average age of the patients was 45.9 years (range 23–70). Twenty-three of the 24 had various clicks and murmurs documented by phonocardiography. The patients were unselected as to presence or absence of...
known arrhythmias. The most common causes for referral to the Cardiology Clinic were the presence of abnormal auscultatory findings or unexplained chest pain. Only five of the patients were evaluated primarily because of symptoms suggesting arrhythmias. However, when questioned in detail at the time of initial evaluation, almost all of the patients admitted to occasional palpitations. There was a high incidence of dyspnea and fatigue among these patients, and their symptoms were in every way similar to the findings of other investigations in patients with mitral valve prolapse. Clinically, none of these patients were classified as having coronary artery disease, and all five patients whose chest pain was severe enough to warrant coronary arteriography had normal coronary anatomy.

Data Collection

All patients had a 12-lead ECG, a maximal treadmill exercise test, and a 24-hour ambulatory ECG performed. The 12-lead ECG was performed in the supine position on a single-channel Hewlett-Packard #1511 ECG machine, and each contained approximately two minutes of ECG recording. The treadmill tests were performed using the Bruce protocol, monitoring leads II and V6 during exercise and ten minutes of recovery. In order to detect all arrhythmias, a continuous ECG recording was made during the treadmill testing at 50 mm/sec paper speed, with frequent sampling at 25 mm/sec paper speed. The 24-hour ECGs were recorded using a single-channel Avionic ECG tape recorder.

A modified V1 lead system was used. Patients followed their normal daily routine and recorded a diary of symptoms and activity levels, which included periods of sleep. The patients received no antiarrhythmic medications during the period of, and for at least 72 hours prior to, the data collection.

Data Analysis

The magnetic tapes containing the 24 hours of ambulatory ECG were processed at 60 times real time, using a digital computer system developed at Stanford Medical Center. This system provides plots of R-R interval, QRS duration, and mean QRS vector for each heart beat during a 24-hour period. These data are plotted simultaneously as a function of time, allowing visual detection of all arrhythmias. Verification of arrhythmias is made by sampling a digitized ECG signal from unlimited numbers of one-minute segments during the day. The one-minute segments to be sampled are selected after visual inspection of the plots of R-R interval, QRS duration, and mean QRS vector by a physician or trained technician. Frequent or continuous sampling is possible when there is a noisy signal or when there are complex arrhythmias present, and infrequent or random sampling when there are no arrhythmias present. The 24-hour period of monitoring is divided into 90 consecutive 15 minute periods. The total number of PVCs in each 15 minute period is determined from the computer-generated plots by visual inspection. The peak PVCs/15' during the 24-hour period represents the maximum number of PVCs in any of these 15 minute segments. Total PVCs in 24 hours was determined by summing the number of PVCs in each 15 minute period. From the patient diary, the total number of 15 minute periods spent awake and asleep was determined. The total number of PVCs while awake was computed by summing the PVCs for each of the 15 minute periods spent awake, and the mean number of PVCs/15' while awake was calculated by dividing the total number of PVCs while awake by the number of 15 minute periods spent awake. The peak PVCs/15' while awake was defined as the highest number occurring during any of the 15 minute periods while awake. In a similar manner, the total number of PVCs while asleep, mean PVCs/15' during sleep, and the peak PVCs/15' while asleep were also determined. Identical data were tabulated for all atrial premature contractions (APCs). All episodes of ventricular tachycardia and supraventricular tachycardia were analyzed. The beats occurring during these tachyarrhythmias were not included in the above tabulation of total, mean, and peak APCs and PVCs. Each tape was analyzed for the presence or absence of atrial or ventricular pairs or bigeminy. The number of ectopic ventricular forms was determined for each patient. From the R-R interval plots the maximum and minimum heart rate during 24 hours was determined, as well as the relationship of the arrhythmias to changes in heart rate. Symptoms recorded in the patient diaries were correlated with findings on the ECG recording.

To determine arrhythmia detecting sensitivity, all 12-lead ECGs, treadmill tests (exercise and recovery) and ambulatory ECGs were analyzed for the presence or absence of atrial premature contractions (APCs), premature ventricular contractions (PVCs), atrial and ventricular pairs and bigeminy and ventricular and supraventricular tachycardia. The total number of patients having each type of arrhythmia was taken as the number demonstrating that arrhythmia on any one of the three types of arrhythmia screening tests. The percent sensitivity for each test was defined as the number of patients with a given type of arrhythmia detected by that test, divided by the total number of patients with that arrhythmia detected on any of the three types of tests.

Mathematical and statistical analysis of the data was performed on a Hewlett-Packard Model 9100 A calculator, using standard programs. Comparison of mean APCs/15' and mean PVCs/15' in each patient while awake and asleep was made, using a standard two-tailed t-test. Comparison of peak and minimum heart rate while awake and asleep was made using the two-tailed t-test for matched pairs. Chi squared analysis with the Yates correction was used for evaluation of the presence or absence of complex arrhythmias in the high and low frequency PVC groups. The mathematical relationship between the peak PVCs/15' and total PVCs in 24 hours was derived by determining the linear regression equation for ln (total PVCs) as a function of ln (peak PVCs/15'). The other total and peak ectopic relationships were derived similarly.

Results

All 24 patients demonstrated cardiac arrhythmias which ranged in severity from three APCs in 24 hours to an episode of ventricular tachycardia which lasted one and a half minutes. Eighteen of the 24 patients had ventricular arrhythmias on the ambulatory ECG, 15 had atrial arrhythmias, and nine patients had both atrial and ventricular arrhythmias.

Ventricular Arrhythmias on Ambulatory ECG

The total number of PVCs in 24 hours in the 18 patients with ventricular arrhythmias ranged from three to 14,800. The PVCs were uniform in eight patients, of two forms in eight patients, and of three or more forms in two patients. There was an excellent
correlation between peak PVCs/15' during the 24-hour monitoring period and the total number of PVCs in 24 hours (fig. 1, table 1). These 18 patients were divided into two distinct groups based on the total number of PVCs in 24 hours. Six showed infrequent PVCs (<50 PVCs in 24 hours), and 12 showed frequent PVCs (>425 PVCs in 24 hours) (fig. 1). Further supporting the concept of two populations of patients with ventricular arrhythmias among patients with mitral valve prolapse was the fact that complex ventricular arrhythmias occurred almost exclusively in the group with frequent PVCs. All 12 patients in this group had pairs, and the pairs occurred frequently in six of the 12. Bigeminy occurred in eight of the 12 and was frequent in six of these eight. Ventricular tachycardia occurred in five of the 12 patients, ranging from one to more than 20 episodes per patient during 24 hours. These episodes ranged from three beats to 1½ minutes of sustained ventricular tachycardia at rates ranging from 140 to 180 beats/min. Among the six patients with infrequent PVCs there were no episodes of ventricular tachycardia, and no pairs. The only complex ventricular arrhythmias seen in this group of patients with infrequent PVCs were brief episodes of bigeminy in one patient. The difference in incidence of complex arrhythmias in these two groups was statistically significant (P < .01).

Effect of Sleep on Ventricular Arrhythmias

The effect of sleep on PVC frequency was evaluated for the 12 patients with frequent PVCs (fig. 2). Seven of the 12 (58%) showed a statistically significant reduction in the mean PVCs/15' with sleep. Two (17%) showed a significant increase with sleep, and three (25%) were unchanged. Only one of the episodes of ventricular tachycardia occurred during sleep. There was a correlation between peak PVCs/15' while awake and the total number of PVCs while awake (fig. 3, table 1), and between peak PVCs/15' during sleep and the total number of PVCs during sleep (fig. 4, table 1). These relationships existed despite the fact that patients spent slightly different fractions of their day asleep.

Atrial Arrhythmias on Ambulatory ECG

The total number of APCs in 24 hours in the 15 patients with atrial arrhythmias ranged from three to 9,190. There was good correlation between peak

![Figure 1](http://circ.ahajournals.org/)

A ln-In plot showing the relationship between the total number of PVCs in 24 hours and the peak PVCs/15' for the 18 patients with PVCs. This is seen to be a linear relationship, thus allowing an exponential equation to be written for the data (table 1, equation 1). Also note that the patients fall into two distinct groups, 12 with frequent PVCs and greater than ten peak PVCs/15' and those with infrequent PVCs and less than ten peak PVCs/15'. Complex ventricular arrhythmias were found almost exclusively in the group with frequent PVCs.

<table>
<thead>
<tr>
<th>Mathematical relationship</th>
<th>Standard error*</th>
<th>r</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Total PVCs/24 hr = 4.66 (peak PVCs/15')</td>
<td>±0.68</td>
<td>0.97</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>2. Total PVCs while awake = 3.90 (peak PVCs/15' awake)</td>
<td>±0.63</td>
<td>0.97</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>3. Total PVCs while asleep = 1.72 (peak PVCs/15' asleep)</td>
<td>±0.51</td>
<td>0.98</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>4. Total APCs/24 hr = 3.39 (peak APCs/15')</td>
<td>±0.51</td>
<td>0.98</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>5. Total APCs while awake = 2.65 (peak APCs/15' awake)</td>
<td>±0.64</td>
<td>0.97</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>6. Total APCs while asleep = 3.18 (peak APCs/15' asleep)</td>
<td>±0.64</td>
<td>0.97</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

*Given as the power of the constant e = 2.72.

r = correlation coefficient.

Table 1

Summary of the Mathematical Relationships between the Peak PVCs/15' and Peak APCs/15' during Various Periods of Monitoring and the Total Number of PVCs or APCs during That Period

Circulation, Volume 52, July 1975
Effect of sleep on ventricular arrhythmias for the 12 patients with frequent ventricular arrhythmias. The level of statistical significance of the change in mean PVCs/15' is given for each patient. Seven patients had a significant reduction in PVCs with sleep, three showed no change, and two patients had an increase in PVCs with sleep.

Effect of Sleep on Atrial Arrhythmias

The effect of sleep on the mean PVCs/15' was evaluated for the nine patients with more than 50 APCs in 24 hours. APCs/15' during the 24 hours and the total number of APCs in 24 hours (fig. 5, table 1). There was not a tendency to segregate into high and low frequency groups as with the PVCs. Complex atrial arrhythmias were distributed evenly among the 15 patients, with no apparent relationship to total number of APCs in 24 hours. Paroxysmal supraventricular tachycardia occurred in patients with as few as 25 and 35 APCs in 24 hours. The patient with the largest number of APCs (9,190 in 24 hours) had no complex atrial arrhythmias. A total of 19 episodes of paroxysmal supraventricular tachycardia was seen in seven patients. Two patients had six episodes each, two had two episodes, and the remaining three patients had one episode each. All episodes were brief and self-limited, averaging 5.6 ± 2.6 beats per episode (range 3–11). The average heart rate for all episodes of supraventricular tachycardia was 134 ± 25 (range 90–210). Atrial pairs occurred in five patients but were infrequent in four of the five (single pair in 24 hours in three). Atrial bigeminy was seen in only one patient.
ARRHYTHMIAS IN MITRAL VALVE PROLAPSE

A linear plot showing the relationship between total number of APCs during 24 hours and the peak APCs/15' during 24 hours. The linear nature of this relationship allows an exponential equation to be written for the data (table 1, equation 4). Note that the patients do not segregate into two distinct groups as they do for PVCs (fig. 1).

significant decrease in mean APCs/15' during sleep, and three (33%) were unchanged (fig. 6). Four of these nine patients also had frequent ventricular arrhythmias, thus making it possible to determine the effect of sleep on atrial and ventricular arrhythmias in the same patient. Three of the four showed a different effect of sleep on atrial arrhythmias, as compared to its effect on ventricular arrhythmias in the same patient (table 2).

Eight of the 19 (42%) episodes of supraventricular tachycardia occurred during sleep. The average rate of the eight episodes during sleep was 117.5 ± 19 beats/min, compared to 146 ± 29 beats/min for the 11 which occurred while the patient was awake (P < 0.05). There was no difference in the average number of beats per episode while awake and asleep.

Heart Rate During 24-hour Monitoring

The peak heart rate during the 24-hour period of monitoring occurred while awake and the minimum heart rate occurred while asleep in all patients. The peak heart rate while awake was 121 ± 14 beats/min (range 89–150) and the peak while asleep was 95 ± 12 (range 71–120). The minimum heart rate while awake was 67 ± 9 (range 50–80) and while asleep was 56 ± 8 (range 41–71). Both these differences were significant (P < 0.001).

In three patients there was a definite increase, and in another a probable increase in the number of ectopic beats with increased heart rate. One of these patients developed ventricular tachycardia each time the heart rate exceeded 125.

Correlation of Symptoms with Ambulatory ECG Findings

The relationship of symptoms recorded in the patient diary to ECG findings is summarized in Table 3. Symptoms were recorded during 13 of the 24 monitoring sessions. Nine patients recorded 12 episodes of chest pain. One of these episodes was associated with a brief run of bigeminy. The other 11 showed no significant change in heart rate or rhythm.

Table 2

The Effect of Sleep on Arrhythmias in the Four Patients with Both Frequent APCs and Frequent PVCs

<table>
<thead>
<tr>
<th>Patient</th>
<th>Effect of sleep on mean PVCs/15'</th>
<th>Effect of sleep on mean APCs/15'</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>No change</td>
<td>(P &lt; 0.06)</td>
</tr>
<tr>
<td>2</td>
<td>(P &lt; 0.005)</td>
<td>(P &lt; 0.001)</td>
</tr>
<tr>
<td>3</td>
<td>(P &lt; 0.001)</td>
<td>(P &lt; 0.05)</td>
</tr>
<tr>
<td>4</td>
<td>(P &lt; 0.001)</td>
<td>No change</td>
</tr>
</tbody>
</table>

Abbreviations: APCs/15' = atrial premature contractions per 15 minutes; PVCs/15' = ventricular premature contractions per 15 minutes.
Changes

shortness of breath and fatigue. Severe chest pain was infrequent. Bigeminy was noted in 12 of the 26 episodes of symptoms recorded, only seven (27%) were associated with ECG rate of rhythm changes, and three of these seven were sinus tachycardia. If the ten episodes of symptoms suggestive of arrhythmias are considered alone (palpitations and dizziness), significant arrhythmias were associated with only three. In six of the seven patients, the supraventricular tachycardia was asymptomatic, and the single patient recording symptoms of palpitation also had several asymptomatic episodes of supraventricular tachycardia. In only one patient were the episodes of ventricular tachycardia associated with symptoms in the diary.

Ventricular Arrhythmia Detection

The detection of ventricular arrhythmias by the 12-lead ECG, treadmill exercise test, and the 24-hour ambulatory ECG is summarized in table 4. Twenty patients had PVCs detected on at least one of the three types of tests. Only eight of the patients with PVCs were detected by resting ECG, and all of these eight had PVCs on both the treadmill test and the ambulatory ECG. Seventeen of the 20 patients with PVCs (85%) were detected by treadmill testing, and 18 of the 20 (90%) were detected by the 24-hour ambulatory ECG recording. There was a correlation between the number of PVCs seen on the exercise treadmill test and the total number of PVCs on the 24-hour ambulatory ECG tape (fig. 7). All of the 12 patients in the high frequency PVC group, as detected by ambulatory monitoring, had more than one PVC during the treadmill test, whereas 10 of the 12 patients with infrequent or no PVCs on the ambulatory ECG had zero or only one PVC on their exercise treadmill test ($p < 0.01$). All eight patients with PVCs on resting ECG were in the high frequency PVC group.

Thirteen patients had ventricular pairs on at least one of the three tests. Routine ECG detected two, the treadmill test five, and 24-hour ambulatory ECG 12 of the 13. Ambulatory ECG detected all nine patients with bigeminy. The treadmill detected five and the 12-lead ECG only three of the nine. Five of the six patients with ventricular tachycardia were detected by the ambulatory ECG, three by the treadmill test, and one by the 12-lead ECG.

Atrial Arrhythmia Detection

The detection of atrial arrhythmias is summarized in table 4. The 24-hour ambulatory ECG detected all patients with APCs and with each type of complex atrial arrhythmia. The exercise treadmill test detected only five of the 15 patients with APCs, and the 12-lead ECG detected only two of the 15 patients with APCs. Of the seven patients with brief episodes of supraventricular tachycardia, only the two with six episodes each during the 24-hour ambulatory ECG had supraventricular tachycardia recorded during exercise treadmill testing. No episodes of supraventricular

### Table 3

<table>
<thead>
<tr>
<th>Symptom</th>
<th>No. of episodes</th>
<th>ECG findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest pain</td>
<td>12</td>
<td>11 – no changes</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 – associated with bigeminy</td>
</tr>
<tr>
<td>Shortness of breath</td>
<td>2</td>
<td>No changes</td>
</tr>
<tr>
<td>Severe fatigue</td>
<td>2</td>
<td>No changes</td>
</tr>
<tr>
<td>Palpitations or increased heart rate</td>
<td>8</td>
<td>3 – no changes</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 – increased PVCs</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 – VT</td>
</tr>
<tr>
<td>Dizziness</td>
<td>2</td>
<td>1 – no change</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 – VT</td>
</tr>
</tbody>
</table>

Abbreviations: PVCs = premature ventricular contractions; SVT = supraventricular tachycardia; VT = ventricular tachycardia.

at the time of chest pain. There were two episodes each of shortness of breath and severe fatigue, none of which were associated with significant changes in the patient’s rhythm. Eight episodes of palpitations or increased heart rate were recorded. There were no changes in the patient’s ECG rhythm during three of these episodes, three showed sinus tachycardia (rate 100–122), one had an increase in the frequency of PVCs, and one had several brief episodes of supraventricular tachycardia. There were two episodes of dizziness recorded, one of which occurred with the 1½ minute episode of ventricular tachycardia, while the other showed no ECG changes. Thus, of the 26 episodes of symptoms recorded, only seven (27%) were associated with ECG rate of rhythm changes, and three of these seven were sinus tachycardia. If the ten episodes of symptoms suggestive of arrhythmias are considered alone (palpitations and dizziness), significant arrhythmias were associated with only three. In six of the seven patients, the supraventricular tachycardia was asymptomatic, and the single patient recording symptoms of palpitation also had several asymptomatic episodes of supraventricular tachycardia. In only one patient were the episodes of ventricular tachycardia associated with symptoms in the diary.

### Table 4

<table>
<thead>
<tr>
<th>Table 4</th>
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<tbody>
<tr>
<td>12-lead ECG</td>
</tr>
<tr>
<td>Arrhythmia</td>
</tr>
<tr>
<td>PVCs</td>
</tr>
<tr>
<td>VT</td>
</tr>
<tr>
<td>Pairs</td>
</tr>
<tr>
<td>Bigeminy</td>
</tr>
</tbody>
</table>

Abbreviations: PVCs = premature ventricular contractions; VT = ventricular tachycardia; APCs = atrial premature contractions; SVT = supraventricular tachycardia.

$^*$Figures in parentheses represent sensitivity of each test for each type of arrhythmia.

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The incidence of serious ventricular arrhythmias in our series is slightly less than that of Kreisman et al. However, their series may be biased by the fact that arrhythmias were the presenting manifestation in 65% of their patients. On the basis of 24 hours of ambulatory ECG recordings in our unselected series of patients, three groups of patients with mitral valve prolapse may be defined with regard to ventricular arrhythmias: 25% show no PVCs on 24-hour monitoring; 25% show infrequent PVCs and no significant complex ventricular arrhythmias; the remaining 50% demonstrate frequent PVCs and complex ventricular arrhythmias. This incidence of frequent PVCs in patients with mitral valve prolapse is similar to the 40% incidence found by DeMaria et al., the slightly higher figure in our study probably reflecting the longer period of monitoring (24 hr vs 10 hr). In that study, and in our experience, such frequent PVCs and complex ventricular arrhythmias are rare in normal patients. Several of the patients in our study have had multiple ambulatory ECG recordings performed, and although the total number of PVCs varies somewhat from recording to recording, patients with frequent PVCs consistently show frequent PVCs, and those with infrequent or no PVCs remain in those categories.

With regard to atrial arrhythmias, ambulatory monitoring appears to define two groups of patients: those with atrial arrhythmias (63%) and those without (37%). The occurrence of complex atrial arrhythmias was not related to the total number of APCs recorded during 24 hours. An interesting finding in this study was the large number of patients with brief episodes of asymptomatic paroxysmal supraventricular tachycardia. These episodes occurred only in patients with APCs recorded at other times during their tape, and all were initiated by a premature supraventricular beat. A wide range of rates and a decreased rate of supraventricular tachycardia during sleep were observed.

The effect of sleep on ventricular arrhythmias in patients with mitral valve prolapse in our study is similar to the findings in other types of patients with ventricular arrhythmias. The majority of patients in our study showed a decrease in PVCs with sleep, and only one episode of ventricular tachycardia occurred during sleep. However, there exists a small subset of patients who demonstrate a significant increase in PVC frequency during sleep. This study also demonstrates that sleep may affect the frequency of atrial arrhythmias, with most patients showing a decrease in APCs during sleep. The occurrence of supraventricular tachycardia did not appear to be suppressed by sleep. The clinical and therapeutic implications and the mechanism of sleep alteration in arrhythmia patterns are still largely unknown. The finding of dissimilar effects of sleep on atrial and ventricular arrhythmia patterns in the same patient suggests that the alteration of cardiac electrophysiology during sleep may be complex.

The discrepancy between symptoms suggesting arrhythmias and ECG evidence for arrhythmias in patients with mitral valve prolapse has implications for the clinician who must evaluate these patients. Complaints such as palpitations and dizziness recorded during history-taking in patients with mitral valve prolapse should not necessarily be interpreted as suggesting arrhythmia nor should they be attributed to known arrhythmia unless the symptoms and the arrhythmia can be repeatedly documented to occur simultaneously. We have observed syncopal episodes in two patients with mitral valve prolapse which occurred when the patient showed a normal blood
pressure and with only occasional PVCs recorded during the syncopal episode. Both of these patients were known to have ventricular arrhythmias (including prior episodes of ventricular tachycardia in one). This discrepancy between symptoms and objective findings is parallel to that of Gooch et al. who found a poor correlation between symptoms of dyspnea and fatigue and treadmill exercise tolerance in patients with mitral valve prolapse.7

The validity of the peak PVCs/15' during 24 hours as an index of the total number of PVCs in 24 hours was demonstrated for patients with mitral valve prolapse. We have found a similar relationship between peak PVCs/15' during a 24-hour period and total PVCs in 24 hours in patients with a variety of types of underlying heart disease. This study also demonstrates that peak APCs/15' was a good index for the total number of APCs during a 24-hour period. In addition, the peak PVCs/15' and peak APCs/15' while awake or asleep are reliable guides to the total number of PVCs and APCs while awake or asleep. Utilizing the equation derived in this study, it is possible to estimate the total number of PVCs in 24 hours by scanning a 24-hour tape for the period of most frequent PVCs and performing an exact count on only 15 minutes of ECG data. This number of peak PVCs/15' is inserted into equation 1, table 1, and the total number of PVCs in 24 hours is computed. This technique allows one to assess the effect of antiarrhythmic therapy on PVC frequency without having to perform a time-consuming count of all PVCs occurring in 24 hours.

This study indicates that a 24-hour ambulatory ECG is superior to an exercise treadmill test for arrhythmia detection in patients with mitral valve prolapse. Both the exercise treadmill test and the ambulatory ECG are superior to the 12-lead ECG. If the patients had undergone only a 24-hour ambulatory ECG, the only arrhythmias remaining undetected would have been single PVCs which occurred on treadmill testing in two patients, an episode of ventricular tachycardia in one patient, and a pair of PVCs on the treadmill in another. If only an exercise treadmill test had been performed, the majority of atrial arrhythmias would have remained undetected and the incidence of all ventricular arrhythmias except PVCs would have been markedly underestimated. However, the clinical usefulness of the resting ECG and exercise treadmill test should not be underestimated. From a practical standpoint, it would be useful to know whether or not a given patient falls in the group of patients with mitral valve prolapse who have frequent PVCs or in the group with infrequent or no PVCs. If there are PVCs on the resting ECG or more than one PVC on a treadmill test, it is probably valid to assume that the patient is in the high frequency PVC group, all of whom in our study had complex ventricular arrhythmias during 24-hour monitoring. If the patient has zero or only one PVC on a treadmill test, he is probably in the low PVC group having less than 50 PVCs in 24 hours and only rare complex ventricular arrhythmias. Using these guidelines, many of the patients who might benefit from antiarrhythmic therapy may be identified even if ambulatory ECG recordings are not available. If 24-hour ambulatory ECG recordings are available, then the exercise treadmill test probably does not provide enough additional information to justify its routine use for arrhythmia detection in patients with mitral valve prolapse. However, in an occasional patient it may provide significant additional information. The treadmill should also remain as a useful tool to objectively evaluate the exercise tolerance of patients with mitral valve prolapse.

With the development of the echocardiogram for noninvasive diagnosis of mitral valve prolapse, this condition is being recognized with increasing frequency.13 Although its exact incidence is unknown, it appears to be a relatively common form of cardiac abnormality and may represent the most common abnormality in young adults. Although the group of patients in this study may be weighted in favor of patients with more symptoms and more impressive auscultatory phenomena, they are probably representative of the spectrum of patients in whom the diagnosis of prolapse is made, since asymptomatic patients with minimal or no auscultatory phenomena probably are never detected. Little is known about the natural history of mitral valve prolapse or the prognostic significance of the arrhythmias seen in these patients. Sudden death has occurred on occasion in these patients, including at least one patient in whom no arrhythmias were detected on intensive monitoring.14 Whether or not the types of complex ventricular arrhythmias and frequent PVCs detected in 50% of the patients in this study have the same ominous prognosis as for patients with coronary artery disease15,16 will need to be determined by prospective long-term follow-up studies involving large numbers of patients.

References


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