The Effects of Right Atrial and Ventricular Pacing on the Auscultatory Findings in Patients with Mitral Valve Prolapse


SUMMARY
Fifteen patients with midsystolic clicks associated with mitral valve prolapse were studied in order to assess changes in auscultatory findings produced by pacing-induced variations in cardiac rate, rhythm, and conduction. As the heart rate was increased in stepwise intervals to the maximum possible extent by right atrial pacing (RAP) in 14 patients, the interval between the Q wave and the click (Q-C) decreased in all cases (21 ± 2 msec/10 beats/min; P < 0.001). In two patients, RAP at rates of and above 118 and 159 per minute, respectively, resulted in disappearance of the click.

During right ventricular pacing (RVP) without evidence of atioventricular (A-V) dissociation on the surface ECG in ten patients, the click was inaudible at all pacing rates in three instances. In all seven of the cases in which the click was audible at the lowest rate of RVP with VA conduction, Q-C was greater and C-S₂ less than that in sinus rhythm. As the rate of RVP was increased, Q-C was noted to decrease (26 ± 4 msec/10 beats/min) and C-S₂ to increase (19 ± 7 msec/10 beats/min) in all patients. In three patients in whom RVP induced atrioventricular dissociation, the click was seen only in beats closely preceded by a P wave.

In ten of 11 patients the click occurred earlier in systole with a postextrasystolic or post tachycardia beat as compared to its appearance after a sinus beat when a shorter preceding diastolic filling period was present (P < 0.001). In the eleventh patient a loud systolic murmur was present during postextrasystolic cycles.

It is concluded that pacing-induced rhythm disturbances can result in disappearance of a midsystolic click or can alter its timing and cause it to mimic sonic phenomena seen in other disease states. The possibility of similar changes taking place as a result of spontaneously occurring disturbances of rate, rhythm, and conduction should be recognized in order that the possible diagnosis of mitral valve prolapse not be overlooked.

Additional Indexing Words:
- Systolic murmurs
- Left ventricular volumes
- Systolic clicks
- Left ventricular contractility
- Mitral regurgitation

MIDSYSTOLIC CLICKS occurring alone or in conjunction with late systolic murmurs have been proven by left ventriculography to be caused by systolic prolapse of a portion or portions of the mitral valve into the left atrium.¹⁻³ Mild mitral regurgitation has been noted in most cases.

This combination of auscultatory and angiographic findings may occur in patients with ischemic heart disease, presumably on the basis of papillary muscle dysfunction.⁴ **Midsystolic clicks of mitral valve origin may also be seen with rheumatic mitral valve disease,⁵ after mitral valveplasty⁶ or heterograft replacement of the mitral valve,⁷ and in patients with connective tissue disorders such as Marfan's syndrome.⁸ In most cases, however, systolic prolapse of the mitral valve is seen in patients without any associated cardiac or systemic disease. Such patients are frequently asymptomatic, but some complain of atypical chest pain, palpitations, or dyspnea.⁹

In some of the latter group of patients with the "systolic click syndrome" we have observed periods when the click and murmur were absent despite all attempts to bring them out. We have also been impressed by the rather paroxysmal nature of symptoms in some of our patients with the syndrome. The present study was undertaken in an effort to assess the
effects of changes in cardiac rate, rhythm, and conduction on the auscultatory findings in this condition.

**Methods**

Fifteen patients with midystolic clicks with or without associated late systolic murmurs were studied. Their clinical findings are listed in table 1. Eleven patients had left and right heart catheterization which revealed normal hemodynamic findings at rest. In ten of these eleven patients, retrograde left ventriculography in the right anterior oblique or lateral projection demonstrated systolic prolapse of one or both mitral leaflets. In two patients who had atypical precordial pain, selective coronary arteriography was performed and revealed no abnormalities. Another patient with classic angina pectoris was found on coronary arteriography to have significant obstructive lesions of the right and anterior descending coronary arteries.

In each patient a bipolar pacing catheter was placed sequentially in the right atrium and ventricle. Standard lead II of the electrocardiogram was recorded along with a surface phonocardiogram from the apex. An intracardiac phonocardiogram (Statham SF-1 manometer tip catheter) from the left ventricle near the mitral valve was recorded in two patients. Central aortic pressure or an indirect carotid artery tracing was recorded simultaneously. Recordings were made with the patient in sinus rhythm and at various rates during atrial and ventricular pacing. The following time intervals were measured: Q-S$_2$ and Q-S$_3$, the interval from the onset of the QRS to the earliest high pitched vibration of the first and second heart sounds respectively; Q-C, the interval from the onset of the QRS to the major component of the systolic click; left ventricular ejection time (LVET), the interval from the onset of the rapid upstroke of the aortic or carotid pulse tracing to the trough of the incisura; C-S$_2$, the interval between the click and second heart sound, was derived by subtracting Q-C from C-S$_2$. The interval from onset of left ventricular ejection to the systolic click (E-C) was obtained by subtracting C-S$_2$ from LVET. All measurements were obtained from recordings made at 100 mm/sec paper speed on an Electronics for Medicine photographic recorder. Each interval was obtained by averaging the measurements of from five to ten cardiac cycles.

**Results**

**Right Atrial Pacing (RAP)**

In all 14 patients in whom the measurements could be obtained, stepwise increases (10 beats/min) in heart rate by RAP resulted in a progressive decrease in the Q-C interval by a mean of 21 ± 2 (standard error) msec (range 7.5 to 37.6) ($P < 0.001$) (table 2; figs. 1 and 2). In most patients the early rate of decline of Q-C was slower than that of Q-S$_2$, leading to an initial decrease in C-S$_2$. At faster rates of RAP, however, C-S$_2$ increased. Thus, in 12 of 13 patients, the over-all slope of Q-C decrease was steeper than that of Q-S$_2$, resulting in a mean increase (9 ± 2 msec/10 beats/min; (range −0.5 to +22) of C-S$_2$ as the heart rate was increased by RAP (fig. 1).

In the case of patient 2, in whom two midystolic clicks were present, both moved earlier in systole as the rate was increased by RAP. The earlier of the two clicks was used for measurements shown in table 2.

In some patients progressive increase in the rate of pacing resulted in a progressive decrease in intensity as well as earlier occurrence of the click.

In patient 4, a critical atrial pacing rate above which the click was not audible and below which it

**Table 1**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age</th>
<th>Cardiac symptoms</th>
<th>LV angio</th>
<th>Auscultatory findings</th>
<th>Other diagnosis</th>
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<td>1</td>
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<td>MSC</td>
<td>Hiatus hernia</td>
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<td>2</td>
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<td>MSC, LSM</td>
<td>None</td>
</tr>
<tr>
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<td>36</td>
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<td>Yes</td>
<td>MSC</td>
<td>None</td>
</tr>
<tr>
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<td>50</td>
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<td>MSC, LSM</td>
<td>None</td>
</tr>
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<td>M</td>
<td>47</td>
<td>Chest pain (atypical)</td>
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<td>MSC</td>
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</tr>
<tr>
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<td>F</td>
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<td>No</td>
<td>MSC</td>
<td>Essential hypertension; heroin addiction</td>
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<td>Syncope</td>
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<td>MSC, LSM</td>
<td>None</td>
</tr>
<tr>
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<td>F</td>
<td>23</td>
<td>Atypical chest pain</td>
<td>No</td>
<td>MSC</td>
<td>? Sarcoïdosis</td>
</tr>
<tr>
<td>15</td>
<td>M</td>
<td>58</td>
<td>Dyspnea, chest pain‡</td>
<td>Yes</td>
<td>MSC, LSM (intermittent)</td>
<td>Chronic lung disease</td>
</tr>
</tbody>
</table>

*Two sisters with midystolic click, late systolic murmur.
†Status post left pneumonectomy one year prior to admission.
§Normal selective coronary arteriograms.

Abbreviations: LV angio = retrograde selective left ventriculogram (this procedure verified mitral valve prolapse in all patients in whom it was performed); MSC = midystolic click; LSM = late systolic murmur.
was heard and recorded was demonstrated. In patient 10 the click was heard and recorded at a rate of 142 beats/min but was absent at a rate of 159 beats/min. In patient 12, the click was heard only in expiration at rates exceeding 138 beats/min. In all other patients stepwise increments in the rate of RAP resulted in failure of atrioventricular (A-V) conduction while the click was still audible.

In patient 3, whose click had been present intermittently but was absent at the outset of the study, rapid RAP resulted in the appearance of both a mid-systolic click and a prominent diastolic gallop.

In the seven patients who had measurement of Q-C at five or more rates of RAP, plotting of the Q-C interval against heart rate showed a wide variation in the rate of slope between individual patients (−1.45 to −3.62; \( N = 8 \); mean = −2.21 ± 0.67) but the correlation coefficients of all patients were quite strong (−0.857 to −0.995; mean = 0.959 ± 0.04).

In the case of patient 15 severely compromised pulmonary compliance causing extreme swings in pleural pressure may have been responsible for marked variation in click position noted clinically. At rest in the supine position, the click was audible only during inspiration. Atrial pacing at increasing rates resulted in a progressive increase in the percentage of cycles during which the click was recorded.

**Right Ventricular Pacing (RVP)**

The effects of RVP could be adequately assessed in 12 patients. During RVP A-V dissociation was present

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*Figure 1*

Effect of increasing heart rate during right atrial pacing on the Q-S2 (open circles) and Q-C (closed circles) intervals in six patients. C = click; S2 = second heart sound.

*Figure 2*

Phonocardiogram recorded at apex from single microphone (three different filter ranges) in sinus rhythm (SR) and with right atrial pacing (RAP) at rates from 80 to 136 beats/min (patient 12).
at all rates in two patients (patients 3 and 12) while in nine patients retrograde ventriculoatrial (VA) conduction was felt to be present at all rates based on the absence in the surface ECG of visible P waves, sinus capture beats, and fusion complexes. In one patient (patient 4) VA conduction was seen at higher pacing rates but A-V dissociation could be induced by pacing at rates only slightly above the resting sinus rate.

Of ten patients in whom VA conduction occurred at one or more rates of RVP, the click was neither heard nor recorded in three (patients 4, 10, 11). In the remaining seven patients (patients 1, 2, 5, 6, 7, 9, 13), the click occurred closer to the second sound and longer after the onset of the QRS than during sinus rhythm at the slowest rate of RVP carried out (table 3). In patient 13 the click was late systolic during ventricular pacing at rates below 100/min and absent at higher rates. In patient 14, in whom the click position was markedly affected by respiration, RVP at a rate just above sinus markedly reduced the number of cycles with clicks and further increases in the rate abolished the click.

As the rate of RVP with VA conduction was increased in six patients, (patients 2, 5, 6, 7, 9, 13), Q-C was noted to decrease (26 ± 4 msec/10 beats/min; range 6 to 61) and C-S₄ to increase (19 ± 7 msec/10 beats/min; range 4 to 48) in all cases (P < 0.001).

Concern that this late systolic sound might represent pulmonary closure with reverse splitting being secondary to premature activation of the right ventricle during RVP was allayed by the fact that the late systolic sound was always loudest at the apex. In addition, the click was noted to move earlier during inspiration.

During A-V dissociation in three patients, (patients 3, 4, 12), the click occurred only during those systoles in which the P wave closely preceded the QRS, regardless of whether there was normal QRS configuration (sinus captures) or not (fusion or ventricular paced). In general those cycles in which clicks were recorded during A-V dissociation were those with relatively louder first heart sounds (fig. 3). Occasionally the click would appear on a cycle not preceded by a P wave. A critical P-R interval above which the click was absent could not be exactly determined.

In patient 3 the click was seen to occur closer to the second heart sound with beats of right ventricular origin (mean C-S₄ = 27 msec) as compared to sinus beats (72 msec) during A-V dissociation (fig. 4). With fusion beats that position was intermediate (40 msec). Measurement of the time intervals of 25 consecutive cycles revealed only slight differences in the time interval from the onset of ejection to the click (E-C) between the beats of different origin. (sinus, 219 msec; RV paced, 228 msec). The left ventricular ejec-
Continuous recording of phonocardiogram recorded at apex in patient 10 during atrioventricular dissociation induced by right ventricular pacing at a rate slightly above the sinus rate. In the first three cycles of the top panel the P wave follows the QRS and a soft first heart sound (1) is recorded and no click is seen. As the P wave begins to precede the QRS (fourth cycle, top panel), the first sound becomes accentuated and the click appears. The click is then seen on the remainder of the cycles, all of which are preceded by P waves regardless of whether the beats are ventricular paced, fusion (second cycle, bottom panel) or sinus (last four cycles, bottom panel) in origin.

Continuous tracing of indirect carotid pulse (CAR), phonocardiogram recorded at apex (PCG) and electrocardiogram (EKG) from patient 3 during A-V dissociation induced by right ventricular pacing. A mid-systolic click (C) is easily seen on sinus beats but occurs very late in systole on ventricular paced beats with short P-R intervals (as evidenced by loud first sound). In the last two cycles of the bottom panel in which the P follows the R and is seen in the ST segment, the first sound is diminished in amplitude and the click is absent.

Post Premature Beat or Post Tachycardia Response

In 12 patients the effects of postextrasystolic or post rapid stimulation potentiation were studied. Systolic time intervals were measured in a cycle occurring immediately after the compensatory pause following a premature contraction or the pause following cessation of rapid pacing (table 4). In each instance the intervals of a sinus beat with a shorter or similar preceding R-R interval were used for comparison.
Table 4

Systolic Time Intervals of Sinus Beats Versus Post Premature or Post Rapid Stimulation Beats with Longer Preceding R-R Interval

<table>
<thead>
<tr>
<th>Patient</th>
<th>Cycle (S-X)</th>
<th>R-R</th>
<th>Q-S</th>
<th>LVET</th>
<th>PEP</th>
<th>Q-C</th>
<th>E-C</th>
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<tbody>
<tr>
<td>3</td>
<td>Sinus</td>
<td>1140</td>
<td>407</td>
<td>305</td>
<td>102</td>
<td>335</td>
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<tr>
<td></td>
<td>Potentiated</td>
<td>1325</td>
<td>365</td>
<td>280</td>
<td>85</td>
<td>280</td>
<td>195</td>
</tr>
<tr>
<td>4</td>
<td>Sinus</td>
<td>808</td>
<td>394</td>
<td>288</td>
<td>106</td>
<td>309</td>
<td>203</td>
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<td></td>
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<td>335</td>
<td>285</td>
<td>50</td>
<td>250</td>
<td>200</td>
</tr>
<tr>
<td>5</td>
<td>Sinus</td>
<td>818</td>
<td>421</td>
<td>355</td>
<td>66</td>
<td>325</td>
<td>259</td>
</tr>
<tr>
<td></td>
<td>Potentiated</td>
<td>1120</td>
<td>400</td>
<td>350</td>
<td>50</td>
<td>300</td>
<td>250</td>
</tr>
<tr>
<td>6</td>
<td>Sinus</td>
<td>1227</td>
<td>461</td>
<td>364</td>
<td>97</td>
<td>357</td>
<td>260</td>
</tr>
<tr>
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<td>Potentiated</td>
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<td>400</td>
<td>340</td>
<td>60</td>
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<td>265</td>
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<tr>
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<td>1126</td>
<td>417</td>
<td>—</td>
<td>—</td>
<td>327</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>Potentiated</td>
<td>1500</td>
<td>410</td>
<td>—</td>
<td>—</td>
<td>265</td>
<td>—</td>
</tr>
<tr>
<td>9</td>
<td>Sinus</td>
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<td>415</td>
<td>310</td>
<td>105</td>
<td>330</td>
<td>225</td>
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<td></td>
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<td>290</td>
<td>50</td>
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<tr>
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<tr>
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<td>335</td>
<td>—</td>
<td>—</td>
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<td>—</td>
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<tr>
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<td>317</td>
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<tr>
<td></td>
<td>Potentiated</td>
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<td>300</td>
<td>—</td>
<td>—</td>
<td>150</td>
<td>—</td>
</tr>
<tr>
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<td>Sinus</td>
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<td>280</td>
<td>128</td>
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<td>1166</td>
<td>337</td>
<td>265</td>
<td>72</td>
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<td>181</td>
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</table>

Abbreviations: LVET = left ventricular ejection time; PEP = pre-ejection period; E-C = ejection to click.

Thus, the left ventricle would be expected to have a similar or greater volume at the beginning of systole with the potentiated cycle as compared to the sinus beat. In one patient (patient 8), not included in table 4, the click disappeared on the postextrasystolic cycles and a holosystolic murmur not present previously was heard and recorded at the apex.

In the other ten patients, the click was seen to occur earlier in systole (shorter Q-C and S1-C) during the potentiated cycle (fig. 5). Of the six patients in whom the E-C could be determined, that interval was significantly shorter in the potentiated cycles in four. In two patients of the ten, a late systolic murmur not present during sinus rhythm was recorded with the potentiated cycle. In all of these patients the click’s intensity was increased with the potentiated cycle.

Figure 5

Recording of phonocardiogram from apex, first derivative of left ventricular pressure (1st deriv LVP), aortic (Ao), and left ventricular (LV) pressures. Following a long postextrasystolic pause, the fourth cycle shows an augmented rate of rise of left ventricular pressure. Despite the longer diastolic filling period preceding this cycle, the click occurs earlier in systole than during normal sinus rhythm (first and fifth cycles).
The effect of a long diastole was also apparent during RVP-induced A-V dissociation in patient 10. The click was only seen on cycles preceded by a P wave. An exception was noted when a paced beat was closely coupled to a preceding sinus beat. On these occasions the second ventricular paced beat, which was in effect postextrasystolic (long R-R following short R-R), had an easily recordable midsystolic click despite the absence of a preceding P wave (fig. 6).

Discussion

Mitral valve prolapse in its more severe forms (ruptured major chorda tendinea or papillary muscle) is characterized by a holosystolic murmur without a click. If the subvalvular apparatus maintains competence during the initial portion of the systole but gives way before ejection is completed, a late systolic murmur is produced as a portion of the valve prolapses into the left atrium. In many such individuals the murmur is initiated by a systolic click. In other patients only the click is audible. Criley et al. have shown by angiographic studies that this click corresponds in time to the point of maximal valve prolapse.11

Left ventricular volume has been presumed to be a major factor determining the timing in systole of the click and the length of the murmur.7 12

Thus, interventions which decrease left ventricular volume such as inhalation of amyl nitrite or standing move the click closer to the onset of ejection and lengthen the murmur, while maneuvers which increase left ventricular volume such as squatting delay the click and shorten the murmur.13

Moderate increases in heart rate produced by atrial pacing result in decreases in left ventricular end-diastolic volume, end-systolic volume, and stroke volume with little change in cardiac output and systemic arterial pressure.14-19 An increase in contractile element velocity is associated with elevations of heart rate by atrial pacing (Bowditch effect).19

Movement of the click closer to the onset of the QRS and the first heart sound as the heart rate is increased by right atrial pacing is consonant with the theory that reduction of left ventricular volume leads to earlier valve prolapse. However, increases in heart rate by atrial pacing also shorten the total electromechanical time of systole so that the C-S2 interval will be determined by relative changes in the Q-C and Q-S2. At lower atrial pacing rates the shortening of the Q-S2 may be proportionately greater leading to a reduction in the C-S2 interval in some patients, while at higher rates the C-S2 invariably lengths as the relationship is reversed.

An unexpected finding was the marked shortening of the C-S2 interval with right ventricular pacing as compared to sinus rhythm or right atrial pacing at similar or slower rates. This change may be partially, or in some cases entirely due to abbreviation of left

![Figure 6](http://circ.ahajournals.org/)

*Phonocardiogram (apex), respirogram (nasal thermistor), and right atrial electrogram from patient 10 recorded during atrioventricular dissociation induced by RVP. The beats preceded by P waves (first two cycles) have midsystolic clicks, while the last cycle, in which the P wave follows the QRS, has no click. The third cycle is a ventricular paced beat which is so closely coupled to the preceding beat that no heart sounds are produced. Thus, the fourth beat, which has a longer R-P than the fifth, yet has a click, is in effect a potentated (postextrasystolic: long following short R-R interval) cycle, as evidenced by the loud first sound.*

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ventricular ejection time rather than shortening of the S1-C or E-C interval. These observations would suggest that left bundle branch block can cause the click–second sound separation to be minimal. Two clinical examples of a very late systolic click in association with delayed left ventricular conduction were reported by Mercer et al. in 1970.29

In the case of patient 14, severely compromised pulmonary compliance associated with extreme swings in pleural pressure caused marked variation of click position. At rest in the supine position the click was audible only during inspiration. Atrial pacing at increasing rates resulted in a progressive increase in the percentage of cycles during which the click was audible, while ventricular pacing at a rate only slightly above sinus abolished the click. We would postulate that rapid atrial pacing by reducing the Q–click interval allowed mitral valve prolapse to occur during a greater number of cycles, while right ventricular pacing resulted in a reduction of LVET and possibly a slowing of ejection velocity and/or an increase in end-systolic volume, thus preventing mitral valve prolapse from occurring at any phase of the respiratory cycle.

An increase in contractility has been noted with cardiac cycles following a premature beat or cessation of rapid stimulation.30 Such beats usually occur after a prolonged diastole, and therefore, would be expected to have an end-diastolic volume similar to or larger than a normal sinus beat, as well as a reduced impedance to left ventricular ejection. A combination of unchanged or increased preload, decreased impedance to ejection, and increased contractility would be expected to increase the rate of left ventricular ejection. If the interval between the onset of left ventricular ejection and the click were dependent mainly upon left ventricular end-diastolic volume, as has been suggested by Barlow et al.7 and others,13 the click would be expected to occur at a similar or later time in post tachycardia or post premature beat cycles as compared to a sinus beat, with a shorter preceding diastolic filling period.29 To test this hypothesis, post premature or post tachycardia cycles were compared with beats occurring during normal sinus rhythm which had shorter diastolic filling periods and presumably similar or lesser end-diastolic left ventricular volumes. The finding that the click occurred earlier (shorter Q-C interval) in the potentiated cycles and/or produced a regurgitant apical systolic murmur in all 11 patients so studied indicates that left ventricular end-diastolic volume is not the sole factor governing position of the click and suggests that an increase in contractility, and/or a reduction in aortic impedance, may enhance mitral valve prolapse even without a reduction in end-diastolic volume.

We would suggest the following hypothesis as an explanation of our findings. If one assumes that in individuals with a systolic click of mitral valvular origin there exists a critical left ventricular volume at which prolapse of the mitral valve and thus a systolic click occur (critical prolapsing volume–CPV), then several factors might influence the point during systole at which that volume will be attained, or prevent that volume from being achieved during systole, thus abolishing the click.

Reduction of end-diastolic volume (LVEDV) by rapid atrial pacing would, assuming the same rate of left ventricular ejection, result in more rapid attainment of the critical volume for prolapse. Very rapid rates of RAP might place LVEDV near the CPV, thus causing fusion of the click with S1.

Inspiration by reducing LVEDV would cause earlier arrival at CPV or in cases where CPV is very close to end-systolic volume (during RVP), cause the click to appear only during the inspiratory portion of the respiratory cycle.

Occurrence of the click earlier in systole on potentiated cycles following a long diastole can be partially explained by a reduction in the length of the pre-ejection period due to abbreviation of isometric contraction by increased contractility and reduced aortic diastolic pressure. However, the reduction of the E-C interval which occurred in two of our patients may be a reflection of a more rapid ejection rate leading to attainment of the critical prolapsing volume at an earlier point during the ejection phase of systole, despite a probably unchanged or increased LVEDV.

Absence of the click can be explained by a critical prolapsing volume that is greater than left ventricular end-diastolic volume or less than left ventricular end-systolic volume. However, the rather marked changes in click intensity noted with various interventions, as well as the fading out in mid systole of the click as the atrial pacing rate was gradually increased in one of our patients, might suggest that a decrease in click intensity below the range of human audibility can cause the click to be absent even though CPV is attained during left ventricular systole.

Although we made no attempt to quantitate click intensity, certain changes were obvious. In all instances of potentiation of contractility, combined with reduced aortic impedance, click intensity was increased. In most instances of RVP with or without a fall in aortic pressure, click intensity diminished or the click disappeared. As the click moved close to the first sound with atrial pacing, click intensity often, but not always, diminished. These findings would cause us to suggest that contractility, rate of ejection at the time of prolapse, or other hemodynamic factors relate directly to click intensity and when sufficiently di-

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