Early Systolic Clicks due to Mitral Valve Prolapse

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SUMMARY

Four patients had evidence that mitral valve prolapse was the etiology of isolated early systolic clicks that were "ejection" in timing, that is, within 80 msec of the first heart sound (S1). Phonocardiography demonstrated movement of the click closer to S1 (three patients) with no change (three patients) or an increase in click amplitude (one patient) on inspiration. Earlier movement of systolic clicks on inspiration is a known feature of midsystolic clicks related to mitral valve prolapse but not of true aortic ejection sounds. In three patients the early systolic click showed a variable relation to the carotid upstroke, suggesting that the two events were not related. The click of the fourth patient occurred after the carotid upstroke. In one patient the click could be brought out by standing. Apical holosystolic murmurs could be elicited in all patients. Left- and right-heart catheterizations and left ventricular angiograms revealed no evidence of semilunar valve or great vessel root abnormality. Three patients had mitral valve prolapse and mitral regurgitation on left ventriculogram. The cineangiogram on one patient demonstrated maximal prolapse of the mitral valve just before opening of the aortic valve and coincident with the timing of the click. An isolated early systolic click may be due to mitral valve prolapse rather than to the usual ejection phenomena.

Additional Indexing Words:
Mitral regurgitation Ejection sounds First heart sound Heart murmurs

EARLY systolic clicks, occurring within 80 msec of the first heart sound, are generally considered to be related to ejection phenomena, that is, opening of the aortic or pulmonic valve or the ejection of blood into the aorta or pulmonary artery. On the other hand, systolic clicks related to mitral valve prolapse are classically described as occurring in middle or late systole.

Reported here are four patients with isolated, early systolic clicks in the absence of midsystolic clicks in whom phonocardiographic, cardiac catheterization, and angiographic data all indicate that these clicks were due to mitral valve prolapse rather than to ejection phenomena.

Description of Patients and Methods

The four patients were two men and two women aged 26 to 49 years. None had a history of rheumatic fever and three had had systolic murmurs since childhood. One patient experienced prior congestive heart failure and exhibited both ventricular and atrial gallop sounds. Her electrocardiogram showed normal sinus rhythm and left bundle-branch block. Chest X-ray revealed a normal-sized heart. Idiopathic cardiomyopathy was suspected clinically and cardiac catheterization revealed elevated end-diastolic
Table 1

Summary of Phonocardiographic and Angiographic Features of Patients

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<td>Mild prolapse of mural leaflet on cineangiogram occurring before opening of aortic valve and approximately coincident with C on phonocardiogram</td>
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Abbreviations: C = click; I = inspiration.

The relationships of the systolic clicks (C) to the first heart sound (S1) in each of the four patients are diagrammed in expiration and in inspiration. The timing in milliseconds is shown at the bottom of the figure. The height of the vertical bars depicts the relative intensity of the sounds. In the lower three patients, the click is seen to move closer to the first heart sound with inspiration. In patient M. H., the click also increased in intensity with inspiration. In patient J. O., the click remained in the same position.

Effects of Breathing on Early Systolic Clicks: Phonocardiography was performed in a soundproof room with a Sanborn 560 machine, utilizing Leatham suction microphones. Recordings were taken at the apex left lower border at frequencies ranging from 25 Hz to 400 Hz. Simultaneous electrocardiograms, respiration recordings, and indirect carotid pressure recordings were made. Right and retrograde left heart catheterizations were done in each case. All patients had left atrial flutter. The chest X-rays of all three patients showed left ventricular hypertrophy by silhouette and the third exhibited mild dilatation.

Pressures in both ventricles with elevated pulmonary artery pressures and normal pulmonary arterial resistance. Two patients complained of nervousness, palpitations, and mild dyspnea, and vague chest pains; one complained only of palpitations. The electrocardiograms of these three patients showed left ventricular hypertrophy by voltage, and the third exhibited incomplete right bundle-branch block. Two were in normal sinus rhythm and one in atrial fibrillation. The chest X-rays of all three were essentially unremarkable, except for the angiographic findings.
ventricular angiograms (two biplane at 6 frames per second; two single-plane cineangiograms at 60 frames per second).

**Results**

The results are summarized in table 1. Early systolic clicks were present in three patients in the supine position and in one patient in the standing position. In expiration, the click followed the first heart sound (S₁) by 70 msec in two patients, 50 msec in one patient, and 35 msec in the fourth patient (fig. 1). The click amplitude remained constant with inspiration in three patients and increased with inspiration in one. Inspiration also moved the clock closer to S₁ by 10 to 15 msec in three patients.

The relationship of the click timing to carotid upstroke is shown in figure 2. In three patients (H. B., W. G., and M. H.) the click occurred before the carotid upstroke and moved even earlier before the upstroke on inspiration. In M. H. the click also moved from before the carotid upstroke with the patient in the standing position to after it with the patient in the sitting position. The click occurred just after the carotid upstroke in both inspiration and expiration in the fourth patient (J. O.). Thus, the timing of the clicks showed little relationship to the carotid upstroke. Amyl nitrite inhalation and the Valsalva maneuver were done in patient H. B. (fig. 3). With both maneuvers the click moved close to S₁; with amyl nitrite it also became louder.

Diagrammatic representation of the murmurs recorded at the apex is shown in figure 4. Two patients had holosystolic murmurs at rest.

*Figure 2*

Timing of early systolic clicks (C) in relation of the carotid upstroke. In three patients the timing of the click can be seen to move earlier before the carotid upstroke with inspiration. M. H. also demonstrated a movement of the click from before the carotid upstroke while standing to after the carotid upstroke while sitting. In J. O. the click occurred after the carotid upstroke. S₁ is the first heart sound, S₂ the second heart sound.
in the supine position. In the other two patients the apical murmurs were midsystolic, crescendo-decrescendo with the patients in the supine position at rest. In one patient (M. H.) the murmur became holosystolic on standing, and was accompanied by the appearance of an early systolic click. In the fourth (H. B.) the murmur became holosystolic after amyl nitrite inhalation. Thus, in all four patients murmurs clinically suggestive of mitral regurgitation could be elicited.

No evidence of pulmonary valve, pulmonary artery root, aortic valve, or aortic root abnormality was found on catheterization or angiography. On the other hand, three of the patients had mitral regurgitation and mitral valve prolapse on left ventricular angiogram. These three had prolapse of the mural leaflet and one (fig. 5) also had prolapse of the anterior leaflet. The fourth patient had the characteristic phonocardiographic changes, but in only the single-plane angiocardiogram that was performed an adequate assessment of mitral valve movement was not possible (R. E. Dinsmore, A. M. Hutter, Jr., and C. A. Sanders: In preparation). In one patient the actual timing of the maximal prolapse could be ascertained by analysis of the cineangiogram (fig. 6). The mural leaflet was seen to begin prolapse when the aortic valve was still closed (fig. 6B). Maximum prolapse was achieved when the aortic valve was just beginning to open (fig. 6C). This occurred one frame, or 17 msec, before the aortic valve

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**Figure 3**

Effects of the Valsalva maneuver and amyl nitrite inhalation on the early systolic click in patient H. B. The upper series of tracings shows the effects of the Valsalva maneuver and the lower series of tracings, those of amyl nitrite inhalation. The interval between the first heart sound and the early click is shown in fraction of seconds with each sound recording. The early click moves 10 msec early with the Valsalva maneuver. After the release there is an overshoot of the click timing away from the first heart sound, finally returning to its control position. The same phenomenon is seen with amyl nitrite inhalation. The relative intensity of the click is increased with amyl nitrite (beat 59). HR = heart rate.
Diagrams of the systolic murmurs in the four patients with early systolic clicks (C) as taken from the phonocardiograms. In the supine position, M. H. and H. B. had midsystolic murmurs and W. G. and J. O. holosystolic murmurs. As shown on the right, however, holosystolic murmurs were produced by standing in M. H. and by amyl nitrite in H. B. $S_1$ is the first heart sound, $S_2$ the second heart sound.

opened fully and was approximately coincident with the timing of the early systolic click on the phonocardiogram.

Discussion

Clicks occurring in early systole, that is, within the first 80 msec after the first heart sound, are usually referred to as “ejection” clicks since they are generally considered to be due to either the opening of the semilunar valve or the ejection of blood into the pulmonary artery or aorta. The present report suggests that early systolic clicks can also originate from the mitral valve.

Clicks originating from the left side of the heart can be heard in aortic valvular stenosis or in aortic valvular abnormalities without apparent stenosis, such as a bicuspid aortic valve. Such a click connotes pliability of the valve and is probably due to sudden tensing of the valve leaflets as they open during systole.\textsuperscript{1} Clicks associated with aortic root dilatation or pure aortic regurgitation occur very slightly later and may arise in the aorta itself.\textsuperscript{1} Aortic ejection clicks coincide with the beginning of the upstroke of the carotid pulse.\textsuperscript{1, 2} Although we were not able to find documentation in the literature, it has been our experience, as determined in 10 patients, as well as that of others (J. Perloff: Personal communication) that aortic ejection clicks do

Figure 4

Biplane left ventricular angiogram of patient J. O. The anteroposterior view (left) shows prolapse of both the anterior leaflet (upper arrows) and the mural leaflet (lower arrows) of the mitral valve. The lateral view (right) shows mitral regurgitation and the marked degree of prolapse.
EARLY SYSTOLIC CLICKS

not vary in timing or in intensity with inspiration.

Ejection clicks may also arise from the right heart, especially the pulmonary valve or pulmonary artery. Since a pulmonic valvular click occurs at the end of the isovolumic contraction period of the right ventricle, its timing is of use in evaluating the severity of pulmonic valvular stenosis. The more severe the pulmonary stenosis, the closer the click will be to the first heart sound.8 The pulmonic valvular click diminishes and may even disappear on inspiration. This is thought to be the result of the increased venous return to the right heart moving the closed pulmonic valve upward toward a more open position, therefore reducing the excursion of the valve with systole and diminishing the intensity of the opening click. Inspiratory diminution of the sound is a very useful bedside clue to the detection of the pulmonic valvular click.4

In contrast, clicks related to pulmonary artery hypertension occur slightly later due to a longer isovolumic contraction period of the right ventricle working against a higher pressure. Furthermore, the click is usually not greatly diminished by inspiration.4 Occasionally, a pulmonic click can be related to ejection of blood into a pulmonary artery through a normal pulmonic valve under normal pressures, as in idiopathic dilatation of the pulmonary artery. This click occurs earlier in systole but is also unaffected by inspiration. The timing of pulmonic clicks originating from either the pulmonic valve or a dilated pulmonary artery is not usually affected significantly by respiration.

Clicks arising from the mitral valve have attracted a great deal of attention in the past decade with delineation of the so-called “midsystolic click–late systolic murmur” syndrome.5, 6 The clicks in this syndrome are felt to be due to the sudden tensing of elongated chordae tendineae or a prolapsed mitral
leaflet. The murmur is due to the resultant mitral regurgitation.

Several features of the clicks recorded in the four patients described here suggest that early systolic clicks may also be due to mitral valve prolapse. First, although the clicks occurred 40 to 70 msec after the first heart sound, they moved even closer to the first heart sound with maneuvers known to so affect clicks related to mitral valve prolapse: inspiration,\textsuperscript{5} Valsalva maneuver,\textsuperscript{5} or amyl nitrite.\textsuperscript{5, 6} Second, in one patient the click increased in amplitude on inspiration in contrast to pulmonic valvular clicks, and could be accentuated or brought out with standing as can mitral valve prolapse clicks.\textsuperscript{5-7} Third, the clicks were variable with respect to the beginning of the carotid upstroke, suggesting that the click is related to phenomena independent of aortic ejection. Fourth, no evidence for semilunar valve or great vessel root abnormality was found by catheterization or angiographic studies. Fifth, mitral valve prolapse with mitral regurgitation was documented in three cases. Finally and importantly, the timing of the mitral valve prolapse in one patient occurred just prior to the opening of the aortic valve on cineangiogram and approximately coincident with the timing of the click.

Thus, the differential diagnosis of an early systolic click must be broadened to include mitral valve prolapse as well as the various ejection phenomena. Furthermore, the detection of six additional patients within the past year with isolated early systolic clicks that behaved in the same way on the phonocardiogram suggests that mitral valve prolapse may even be a common cause of early systolic clicks. As in the middle or late systolic clicks originating from the mitral valve, the etiology of the early click is also presumably related to sudden tensing of the chordae tendineae or the abrupt completion of prolapse of the leaflet itself. The differential diagnosis of early systolic clicks is further broadened by the recent documentation of early systolic clicks due to systolic movement of the tricuspid valve in Ebstein’s anomaly,\textsuperscript{8, 9} and the occurrence of a click at the end of systolic movement of an aneurysm of the membranous portion of the ventricular septum associated with small ventricular septal defects.\textsuperscript{10}

References

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