The Effect of Congesting Cuffs on the Echo-phonocardiographic Findings in Mitral Valve Prolapse

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Changes in left ventricular volume appear to be important in changing the timing and duration of mitral valve prolapse. In this study, simultaneous phonocardiograms and echocardiograms were recorded in 16 patients with late systolic mitral valve prolapse before and after the inflation of congesting cuffs (tourniquets) on the upper and lower extremities. The tourniquet application, known to reduce the venous return, caused a statistically significant movement of the phonocardiographic recorded click toward the first heart sound, associated with reduction of the echocardiographic left and right ventricular and left atrial dimensions. These findings confirm the relationship between the mitral valve prolapse and left ventricular dimension and suggest that a decrease in left ventricular volume is a tenable explanation for the earlier onset of prolapse.

Mid-systolic clicks and late systolic murmurs are associated with systolic prolapse of the mitral valve leaflet (MVP) into the left atrium. Several articles noted the ability of postural changes and tilting and nitrite administration to move the clicks and murmurs earlier in systole. The explanation proposed for this movement is a decrease in left ventricular volume (LVV), which allows earlier prolapse of the mitral valve.

The application of constricting pneumatic cuffs (tourniquets) represents a simple, noninvasive procedure whereby venous return is diminished without any drug intervention, since the latter may have a direct effect on myocardial contractility. Review of the English literature failed to disclose data concerning the influence of tourniquets on LVV; however, reduction of venous return is expected to reduce LVV.

The present study evaluates the effect of tourniquets on the phonocardiographic findings and echocardiographic heart dimensions in patients with MVP.

Materials and Methods

Sixteen patients, 12 females and four males, were studied after informed consent had been obtained. All patients had a late systolic echocardiographic pattern of MVP. No patient had evidence of heart disease other than the mitral valve abnormality, and all were in sinus rhythm and receiving no medication. All patients had one or more systolic clicks, and 15 had also systolic murmurs. Simultaneous lead 2 ECGs, phonocardiograms, and echocardiograms were recorded with patients in the supine position and slight rotation to the left. The echocardiogram was performed using an Electronics for Medicine VR-12 ultrasonoscope and a 2.25 MHz, 13-mm diameter transducer with beam collimation to 5 cm in depth. The transducer was positioned in the third or fourth left intercostal space at the left sternal border, avoiding angulation when recording mitral valve echoes. The phonocardiogram was recorded at the apex using an Electronics for Medicine PSA-53 microphone, which was filtered to display frequencies of 120 to 500 Hz. The resting strip-chart recording was repeated ten minutes after inflation to 80 mm Hg of four congesting cuffs that were fitted around the upper and lower extremities close to the trunk.

From these recordings cardiac dimensions were measured. Left ventricular end-diastolic and end-systolic diameters were defined as the maximal and minimal separation of the left interventricular septal and left ventricular posterior endocardial echoes.

All measurements were averaged over five consecutive beats to minimize respiratory variation. The left ventricular end-diastolic and end-systolic volumes were determined by cubing these dimensions. Stroke volume was determined as the difference between both volumes, and ejection fraction was derived by dividing stroke volume by end-diastolic volume. Left atrial dimension was measured at the end of ventricular systole. Right ventricular dimension was measured at end-diastole.

On the phonocardiogram, \( S_1 \) was defined as the onset of the first high-frequency vibration of the first heart sound, and \( S_2 \) and \( C \) similarly for the second heart sound and the earliest recorded click. After measuring the intervals \( S_1-S_2 \) and \( C-S_2 \), the fraction of systole occurring after the click was determined by \( (C-S_2)/(S_1-S_2) \). The two-tailed \( t \) test for matched pairs was used for comparison of resting and tourniquets data, and linear regression and correlation coefficient for the relationship between volume and click position changes.

Results

The mean duration of the \( S_1-S_2 \) interval during...
Use of tourniquets also decreased stroke volume, but this average was only of borderline statistical significance, 78±19 ml to 71±15 ml (P < 0.08). The calculated ejection fraction remained unchanged. Tourniquets decreased the right ventricular dimension from 2.43±0.47 ml to 2.07±0.43 ml (P < 0.001) and left atrial dimension from 3.77±0.34 to 3.45±0.42 ml (P < 0.01). Eight of 15 patients showed a measurable decrease in end-diastolic LVV. All these patients showed an earlier onset of the click during use of the tourniquets. Regression analysis of the relation between changes in diastolic LVV and C-S2/S1-S2 revealed a statistically significant correlation (P < 0.001; Fig 3).

Two patients showed a minor earlier onset of the click, although no measurable decrease in end-
diastolic LVV was found. Three patients showed a minor increase in end-diastolic LVV with no variation in the click timing. In the remaining two patients tourniquet application did not lead to measurable changes in LVV and click position.

**Discussion**

Various authors have reported the migration of the click toward the first sound and prolongation and frequent intensification of the murmur following assumption of upright posture and inhalation of amyl nitrite. The most widely accepted hypothesis for these changes has been earlier prolapse of the mitral valve due to a smaller LVV or increased left ventricular contractility. The LVV was shown to be significantly reduced during passive tilting and amyl nitrite inhalation associated with an earlier onset and a greater degree of MVP.

The presumed mechanism for this phenomenon is that the chordae tendineae are of relatively fixed length, and the shortening of left ventricular axis allows the mitral valve leaflets to prolapse earlier in systole. Analysis of simultaneous echocardiograms and phonocardiograms in patients with MVP demonstrated that the click and onset of MVP were
temporarily related to a constant critical left ventricular dimension. The timing of the prolapse was found therefore to be dependent on the end-diastolic LVV and velocity of circumferential fiber shortening in the preprolapse period.10

Presumably, increase of LVV should produce the opposite effect, and indeed, prompt squatting routinely results in movement of the clicks back to a later systolic position.2 However, increasing LVV by propranolol11 and phenylephrine6 did not alter the timing of MVP and clicks. Sudden termination of rapid pacing, expected to increase LVV, resulted nevertheless in movement of the click earlier in systole.12 It is possible that simultaneous changes in contractility account for these findings.

No data are available to date on the quantification of the LVV changes during tourniquet application with which to compare our results. The application of the congesting cuffs at 80 mm Hg of pressure results in pooling of 500 to 1,000 ml of blood in the extremities of healthy people.13 With low-pressure congestion (20 to 40 mm Hg) the pooling is less effective and reaches a plateau after three minutes.14 At higher pressures further pooling occurs following capillary filtration and fluid diffusion into interstitial tissues.14 In this study tourniquet application caused a statistically significant decrease in left ventricular end-systolic and end-diastolic dimensions. Despite the relatively small reduction in LVV, it was associated with a statistically significant movement of the click toward the first heart sound (Fig 4).

In our experience the click was temporally related to the systolic posterior movement of the mitral valve echo, a finding similar to that reported by others.8 The eight patients in whom reduction in end-diastolic LVV occurred following tourniquet demonstrated the most marked changes in click position. The remaining seven patients did not show sufficient echocardiographic changes in LVV following application of the tourniquet. The fact that the regression line in Figure 3 does not cross the common zero point may be related to the relative insensitivity of the echocardiogram in detecting minor changes in LVV. However, effects other than volume changes consequent to tourniquet application cannot be ruled out.

The demonstration of a reduction in LVV together with earlier MVP does not necessarily mean that they are related as cause and effect. As cardiac agents may alter the after load, myocardial contractility, coronary perfusion and other parameters, singly or in combination, their effect on MVP cannot be attributed to alterations of LVV only. The possibility that the change in MVP occurring in the upright position may be related to a change in the position and axis of the heart must be considered as well.2 However, after excluding these factors by using the tourniquets technique in our study, the smaller volume-earlier prolapse concept is the most likely, although enhanced left ventricular contractility could not be excluded in our patients.

In conclusion, congesting cuffs caused a reduction in left atrial, left ventricular, and right ventricular size. Patients with demonstrable reduction in end-diastolic LVV revealed earlier onset of MVP. Our findings are in agreement with the opinion that LVV changes are major determinants of the onset and duration of MVP.

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