Mechanism of Production of Midsystolic Click in a Prolapsed Mitral Valve

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SUMMARY

In order to examine the production mechanism of the midsystolic click in cases of a midsystolic click and a late systolic murmur, the relationship between the click and the motion of the mitral apparatus was studied by means of ultrasono-cardiotomography and the simultaneous recording of phonocardiograms and ultrasono-cardiograms (UCGs).

It was found that the systolic click occurred in exact coincidence with the time at which the hump of the echo of the unusual early systolic anterior motion (early SAM) took its backmost position. This coincidence was found when the click was shifted either by postural changes or inhalation of amyl nitrite.

The present ultrasono-cardiotomographic study demonstrated that the echo source of the early SAM could be attributed to the protrusion into the left ventricular outflow tract of the slackened elongated chordae tendineae in systole. The anterior leaflet moved suddenly to the position of maximal prolapse when the slackened chordae tendineae were stretched taut in midsystole. In other words, the midsystolic click occurred when the anterior leaflet prolapsed and the tension exerted on the chordae tendineae was at its maximum.

Additional Indexing Words:
Prolapsed mitral valve Midsystolic click and late systolic murmur Elongated chordae tendineae Ultrasono-cardiogram (UCG: echocardiography) Ultrasono-cardiotomogram (UCT) Early SAM
THE click in cases having a midsystolic click and a late systolic murmur syndrome has been considered to be of an extracardiac origin.1)

Reid (1960) ascribed the origin of the systolic click to the fact that the slack chordae are snapped taut.2) Recent evidence obtained by autopsies,3)4) operative findings,5) left ventricular cineangiography,6)-8) a fundamental model experiment,9) and ultrasono-cardiographic studies10)-16) shows that the midsystolic click in cases of mitral insufficiency is associated with the prolapse of the mitral valve.

There are 2 different opinions concerning the mechanism of the occurrence of the systolic click.

Barlow (1968) supported the theory of chordal origin.3) Dock ascribed the midsystolic click to the sudden lateral stretch of the anterior cusp.9)

In any event, the mitral apparatus has been believed to be the source of the systolic click. However, the mechanism of the production of the click has not yet been fully understood.

The present paper is concerned with a case having a midsystolic click and a late systolic murmur. The shape and motion of the mitral leaflets and chordae tendineae were examined by ultrasono-cardiotomography and simultaneous recordings of the phonocardiogram and UCG.

The relationship between the recorded data and the click was examined, and the mechanism of the production of the systolic click was discussed.

**Material and Methods**

The patient was 35-year-old man with a typical midsystolic click and a late systolic murmur. He was found to have cardiac murmurs at the age of 20, but has lived a normal life without any further symptoms.

The ECG revealed only a slight left ventricular hypertrophy. The heart was normal in size on the chest X-ray film. The findings of left cine-ventriculography were interpreted as those of a prolapse of the posterior leaflet of the mitral valve.

Apparatus: An ultrasonic transmitter-receiver unit and a synchronizer for automatic control of the ultrasonic apparatus were the same as those used in our previous studies.17)-31)

A sensitivity time control circuit24) and a fast time constant circuit25) were built into the receiver unit in order to increase resolution of the ultrasono-cardiotomogram (UCT) and the UCG.

The frequency of the ultrasonic pulse was 2.25 MHz and the repetition rate 1,000/sec. A transducer was made of a concave disc of barium titanate. It has a diameter of 30 mm and a radius of curvature of 100 mm. This concave transducer was designed on the basis of preliminary investigation.31)

In transthoracic ultrasono-cardiotomography, a manual sector scan was made from the anterior chest wall. The transducer attached to this scanner could be affixed easily to the chest surface at any angle of incidence.
A thin vinyl bag was filled with warm degassed water and attached to the chest wall of the patient in a supine position. Olive oil was used as the acoustic couplant. The transducer was placed as close as possible to the chest surface (proximity immersed method). While the scanner made continuous sector swings without synchronization with the pulsation, the ultrasonic apparatus was operated in synchronization with the cardiac pulsation to record a stationary ultrasono-cardiotomogram (UCT).

The indicator (cathode ray tube) was repeatedly put into operation for about 20 msec at an arbitrary phase of the cardiac cycle selected by an ECG signal. The echo patterns obtained during 20 to 30 successive pulsations were assembled on a cathode ray screen and on film. Thus 10 to 20 UCTs could be obtained in one cardiac cycle. The UCGs were recorded after the echo sources were identified by ultrasono-cardiotomography.

**Results**

1. **Phonocardiogram**

The midsystolic click and a late systolic murmur were recorded clearly at the apex or in the left 4th intercostal space. Usually there was only one click but occasionally there were two clicks (Fig. 1). When there were two clicks, the second click was more intense than the first one.

![Fig. 1. Phonocardiogram. There is usually one click but occasionally there are two clicks. When there are two clicks, the intensity of the second click exceeds that of the first one.](image-url)
systolic murmur started with the first click and become more intense after the second click. In the present study, the second click, which was constantly audible, was the subject of investigation.

2. The relationship between the midsystolic click and the UCG

The UCG and the phonocardiogram were recorded simultaneously in order to find out from what part of the mitral apparatus the click was originated (Fig. 2).

The systolic click coincided with the time at which the hump of the characteristic echo of the chordae tendineae or anterior mitral leaflet in early systole (early systolic anterior motion, early SAM) came to its backmost position. Fig. 3 shows the change in timing of the appearance of the click by changing the posture of the patient.\(^3\) \(^2\) When sitting, the systolic click of the first cardiac cycle moved toward the second heart sound. In the second cardiac cycle, the click moved toward the first heart sound. However, the click always coincided with the time at which the early SAM took its backmost position in spite of the change of the posture of the patient.

Five sec after inhalation of amyl nitrite,\(^3\) \(^2\) the click moved toward the first heart sound and after 60 sec toward the second heart sound (Fig. 4). Sixty sec after inhalation, it was found that the early SAM persisted almost
throughout the systolic phase. Also the amplitude of the early SAM increased and the head of the early SAM hit against the ventricular septum. Even after such maneuvers, the click always coincided with the time at which the early SAM came to its backmost position.

3. The echo source of the early systolic anterior motion (early SAM)

The echo source of the early SAM which was observed to be coincident with the midsystolic click was confirmed by ultrasono-cardiotomogram (UCT) as shown in Fig. 5. That is to say, the early SAM results from the protrusion toward the left ventricular outflow tract and approaches toward the ventricular septum of the slackened elongated chordae tendineae in midsystole.
Fig. 5. The echo source of the early SAM. The UCT and UCG of the same magnification were compared in order to confirm the echo source of the early SAM on the UCG. As shown in UCG-2, the early SAM is related to the slackened elongated chordae tendineae. The echoes behind the early SAM emanate from the chordae tendineae and from the anterior mitral leaflet. The prolapse of the anterior mitral leaflet is shown as a midsystolic buckling on the UCG. The midsystolic buckling of the chordae tendineae of minor degree does not indicate a prolapse. †: prolapsing anterior mitral leaflet, ‡: elongated chordae tendineae.
4. The change in shape and movements of the chordae tendineae and the mitral valve

a) Chordae tendineae: The chordae tendineae protruded gradually during the course of systole toward the left ventricular outflow tract and moved toward the ventricular septum (Fig. 6). The prolapse of the anterior mitral leaflet had already begun at the time when the chordae tendineae were at their position of maximal protrusion (Fig. 6–4). Immediately there-
Fig. 7. The relationship between the chordae tendineae and the anterior mitral leaflet at the position of maximal prolapse. This figure shows the UCT's of a heart section different from the one in Fig. 6. The prolapse of the anterior leaflet is already appreciably detectable in the early systolic phase (2). At midsystole, the anterior leaflet comes abruptly to a maximal prolapse. At the same time the slackened chordae tendineae are stretched taut toward the left atrium by a pull from the prolapsing anterior mitral leaflet (3).

†: prolapsing anterior mitral leaflet.

after, the anterior mitral leaflet protruded to its position of maximal prolapse. Simultaneously the slackened elongated chordae tendineae were pulled by the prolapsing portion of the anterior mitral leaflet and were stretched taut (Fig. 7–3).

b) The anterior mitral leaflet: The prolapse of the anterior mitral leaflet toward the left atrium occurred early in systole (Fig. 7–2).

Then at midsystolic phase, the leaflet moved suddenly to its position of maximal prolapse (Fig. 7–3). In normal subjects, the mitral leaflet and the annulus move downward to the apex during systole. In this case, however, the anterior mitral leaflet moved upward because of prolapse.

On the other hand, the posterior leaflet moved downward the same as a normal posterior leaflet. Therefore, there was a lack of coaptation of both
DISCUSSION

There have been many studies of the midsystolic click and a late systolic murmur syndrome associated with the prolapse of the mitral leaflet and the pathological changes of the chordae tendineae.3),5),6),32),33)

In the present investigation, we have examined the shape and motion of the mitral leaflet and the chordae tendineae, and the relationship between the movement of the mitral apparatus and the cause of the click was studied. Also the mechanism responsible for the occurrence of the click was discussed.

The findings by operation or autopsy on patients with a midsystolic click and a late systolic murmur have demonstrated the presence of the elongated chordae tendineae in a majority of cases.3) In our study the presence of the elongated chordae tendineae was confirmed by UCT. In systole the elongated chordae tendineae moved anteriorly toward the ventricular septum. The time at which the early SAM came to its backmost position coincided exactly with the midsystolic click. The time relationship between the end of the early SAM and the click never changed even when there was a shift in the time of the appearance of the click by postural changes and inhalation of amyl nitrite. An amplitude of as much as 3 cm of the early SAM and a maximal downward velocity of 350 mm/sec of the chordae tendineae in this patient were almost the same as those of the anterior mitral leaflet in normal individuals.

Thus it was strongly suggested that the markedly exaggerated movement of the elongated chordae alone is sufficient to explain the occurrence of the click. Moreover, the time at which the anterior mitral leaflet moved to its position of maximal prolapse coincided with the time at which the slackened chordae moved to their position of maximal stretch. These findings indicate that the elongated chordae tendineae slackened and protruded into the left ventricular outflow tract during ejection phase.

On the other hand the anterior mitral leaflet began its prolapsing movement at the time when the chordae tendineae were at their position of maximal protrusion. In midsystole, the anterior mitral leaflet moved abruptly to a position of maximal prolapse. At the same time, the slackened chordae tendineae were stretched taut toward the left atrium by the prolapsing anterior leaflet. Consequently, the tension exerted on the anterior leaflet and the chordae tendineae became strongest at the same time, causing the click. Reid (1961)3) and Barlow (1963)3) stated that the occurrence of the click is related to the chordae tendineae, while Dock refuted the chordal snap theory.
of Reid, because it was inconceivable that the chordae tendineae became slackened in systole. In the present study, on the other hand, the presence of the slackened chordae during systole was demonstrated in vivo. Thus the present study is not in agreement with the objection to the theory of Reid presented by Dock. Furthermore we have found a prolapse of the anterior mitral leaflet (not one of the posterior leaflet) by UCT in another case of a midsystolic click and a late systolic murmur. Hence Dock’s theory which ascribes the occurrence of the click to a prolapse of the posterior leaflet seems to be invalid.

It was also observed that the first click was coincident with the end of the slight early SAM before the second click occurred as shown in Fig. 2.

Thus, it is possible that the first click comes from elongated chordae other than those related to the greatest early SAM. However, a coincidence in time between the end of the slight early SAM and the first click could not be detected after postural changes and inhalation of amyl nitrite, because the first click became invisible after such maneuvers. The results of cineangiocardiographic studies up to the present time in cases of the midsystolic click and a late systolic murmur syndrome have always suggested a prolapse of the posterior leaflet. On the other hand, our results indicated a prolapse of the anterior leaflet. Thus, the data obtained by ultrasono-cardiotomography were found to be quite opposite to those obtained by cineangiocardiography. However, the present case was not operated on, because surgery was not indicated. Therefore, there was no direct evidence of the presence of the slackened chordae tendineae. However, it is likely that the structure referred to the posterior leaflet in cineangiocardiography is the anterior leaflet.

A discussion on this point will be published elsewhere.

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