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Echophonocardiographic Study of the Initial Low Frequency Component of the First Heart Sound

YOSHIYUKI HADA, MD, KATSU TAKENAKA, MD, TOSHIYUKI ISHIMITSU, MD, TSUNEO YAMAGUCHI, MD, KEIKO AMANO, MD, HISAKO TAKAHASHI, MD, REIKO TAKIKAWA, MD, TSUGUYA SAKAMOTO, MD, FACC

Tokyo, Japan

To investigate the genesis of the initial low frequency component of the first heart sound that precedes the high frequency vibrations associated with closure of the atrioventricular valves, echophonocardiograms of 36 persons were recorded. These included 10 normal subjects and 26 patients with various types of heart disease including mitral valve replacement. Electrocardiograms demonstrated normal sinus rhythm in 23 subjects, atrial fibrillation in 9, complete atrioventricular block in 2 and atrial flutter in 2. In the phonocardiogram, the low frequency component of the first heart sound followed the onset of the QRS complex and preceded the first high frequency component of this sound. The low frequency component occurred simultaneously with the beginning of the final fast closing movement of the mitral valve on the echocardiogram and was found both in normal rhythm and in arrhythmias. However, in arrhythmias its intensity varied on a beat to beat basis, being loudest after a short RR interval or when atrial systole occurred very close to the expected time of ventricular systole. In patients in whom apexcardiograms were recorded, the low frequency component was coincident with or very close to the onset of ventricular systole.

It is concluded that the low frequency component of the first heart sound represents vibrations caused by contraction of the left ventricle and deceleration of antegrade blood flow across the mitral valve. Neither atrial contraction nor mitral valve tension is necessary for the production of this soft initial component.

The valvular origin of the high frequency vibrations that make up the first heart sound has been previously supported (1–5). These vibrations are preceded by an initial low frequency component, the source of which is unclear. This low frequency component has been attributed to residual atrial vibrations (6,7), tricuspid valve closure (8) and muscular factors (9,10). In 1972, Lakier et al. (11) observed that the low frequency component occurred at the point of crossover of left ventricular and left atrial pressures and postulated a mitral valve origin. Hence, the vibration was labeled an "M sound." Armstrong and Gotsman (12) subsequently recorded the initial low frequency component in patients with prosthetic mitral valve replacement or atrial fibrillation, and speculated that the low frequency component is produced by the resonance of myocardium, blood and valve ring at the onset of ventricular systole. Bonner and Tavel and co-workers (13,14) also supported the view that muscle and blood contributed to the production of the low frequency component.

All of these studies were based on mechanocardiograms (phonocardiograms and apexcardiograms) or on intracardiac pressure recordings. The recent development of echocardiography performed in conjunction with phonocardiography and pulse wave recordings provides an opportunity to observe the timing of the low frequency component and its relation to movements of atrioventricular (AV) valves. In 1978, Armstrong et al. (15), using this new method, concluded that the low frequency component could emanate from the ventricular wall as it becomes taut and decreases its compliance at the onset of systole. The purpose of the present study was to investigate the relation between this low frequency component and AV valve motion in patients with various types of arrhythmia in order to gain further understanding of the genesis of these soft apical vibrations.

From The Second Department of Internal Medicine, Faculty of Medicine, University of Tokyo, Japan. Manuscript received October 20, 1982; revised manuscript received April 5, 1983, accepted April 6, 1983.

Address for reprints: Yoshiyuki Hada, MD, The Second Department of Internal Medicine, Faculty of Medicine, University of Tokyo, Hongo 7-3-1, Bunkyo-ku, Tokyo 113, Japan.

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Table 1. Summary of Patient Data

Case	Age (yr) & Sex	Diagnosis	HR (beats/min)	P-R (ms)	Q-LFC (ms)	Q-ACG (ms)	IVs	Q-M (ms)	M-T (ms)
				Patients With Sinus Rhythm					
1	18M	Normal	86	150	25	25	–	55	25
2	20M	Normal	60	150	20	–	–	60	30
3	29F	Normal	71	140	30	–	–	50	20
4	44F	Normal	68	170	40	–	–	60	–
5	21M	Normal	54	180	20	–	–	60	40
6	24F	Normal	78	140	20	–	–	55	25
7	25M	Normal	60	170	30	–	–	60	30
8	35M	Normal	75	140	15	–	–	55	–
9	45F	Normal	67	160	30	–	+	65	–
10	23M	Normal	59	190	20	15	–	60	45
11	22M	Sinus bradycardia	43	150	15	–	–	55	25
12	15M	Systolic click	68	150	20	–	–	50	35
13	34M	Behçet's disease	57	190	45	–	+	80	10
14	31M	Hypertension	80	170	20	–	+	45	–
15	52M	Angina pectoris	77	160	10	–	–	40	–
16	25M	Myocarditis	80	160	25	25	+	75	25
17	43M	Nephrotic syndrome	91	200	20	–	+	65	0
18	23M	Systolic click	48	160	20	–	–	70	40
19	26F	Aortitis syndrome	61	180	30	–	–	65	–
20	28F	Pulmonary tuberculosis	69	170	25	–	–	50	–
21	35M	MR	57	170	30	–	+	65	–
22	42F	AR	59	130	30	30	–	65	–
23	23M	Systolic click	60	160	30	–	–	60	–
				Patients With Atrial Fibrillation					
24	70F	Lone AF	67	–	30	30	–	65	25
25	69F	Lone AF	72	–	30	–	–	70	30
26	60F	PSS	88	–	20	20	–	55	35
27	37F	MVR	75	–	15	15	–	55	30
28	38F	MVR,AVR	79	–	20	30	–	75	25
29	64F	MR,TR	44	–	30	20	–	60	35
30	46M	COCM	75	–	20	–	–	70	–
31	56F	Hypertension	79	–	30	–	–	60	–
32	32M	MR	80	–	30	–	–	70	30
				Patients With Complete Atrioventricular Block					
33	58M	Hypertension	27	–	0	–	+	65	–
34	29M	MVP,TVP	34	–	5	–	+	80	0
				Patients With Atrial Flutter					
35	41M	Lone AFl	78	–	40	30	–	75	35
36	71M	Lung cancer	133	–	35	–	–	80	55
				Total Group					
Mean	37.6		68.3	163.2	24.3	24.0		62.4	28.0
± SD	16.1		18.4	18.1	9.3	6.1		9.7	12.8

AF = atrial fibrillation; AFl = atrial flutter; AR = aortic regurgitation; AVR = aortic valve replacement; COCM = congestive cardiomyopathy; F = female; HR = heart rate; IVs = a fourth heart sound or an atrial sound; M = male; MR = mitral regurgitation; M-T = time interval from mitral to tricuspid valve closure; MVP = mitral valve prolapse; MVR = mitral valve replacement; P-R = PR interval of electrocardiogram; PSS = progressive systemic sclerosis; Q-ACG = interval from the Q wave of electrocardiogram to the onset of upstroke of the apexcardiogram; Q-LFC = interval from the Q wave to the onset of the low frequency component; Q-M = interval from the Q wave to mitral valve closure; SD = standard deviation; TR = tricuspid regurgitation; TVP = tricuspid valve prolapse.

Methods

Definition. The initial low frequency component is defined as apical low frequency vibrations that occur after the onset of the QRS complex of the electrocardiogram, but before the high frequency component of the first heart sound. The detection of low frequency sound usually depends on the characteristics of the filter settings used for sound recordings. Therefore, in this study we utilized a high pass filter with a cut off frequency of 100 Hz and a slope of 12 dB per octave to determine the presence and timing of the low frequency component.

Study patients. The low frequency component was recorded in 10 normal individuals and 26 patients with various types of disease (Table 1). The 36 subjects had an average age of 37.6 years; 22 were male and 14 were female. Their electrocardiograms demonstrated sinus rhythm in 23, atrial fibrillation in 9, complete AV block in 2 and atrial flutter in 2. PR intervals ranged from 130 to 200 ms during sinus rhythm. We excluded patients with mitral valve stenosis or bundle branch block, but included two patients with mitral valve replacement.

Phonocardiography and echocardiography. Each patient was studied by routine multichannel phonocardiography and two-dimensional echocardiography, followed by M-mode echocardiography using an IREX system II imager, equipped with a 2.25 MHz, 0.5 inch (1.27 cm) diameter and 7.5 cm focused transducer. The echocardiogram was recorded simultaneously with an apical phonocardiogram with two filter settings and, when possible, an apexcardiogram. All recordings were made on a strip chart recorder with the patients in the supine or left lateral position in held midexpiration. A paper speed of 100 mm/s was used for all measurements. We determined the timing of the onset of low frequency component, the upstroke of the apexcardiogram, AV valve closure and the beginning of the QRS complex of the electrocardiogram (Fig. 1). In tricuspid valve echograms, the point of sudden halting of the closing movement (point C) was taken as valve closure because of difficulties in visualization of two leaflets of the tricuspid valve (1–3,5). We also investigated the time relation among these measurements. When the apexcardiogram was recorded, the pickup device for sound recordings was moved slightly medial to the apex. The measurements were made to the nearest 5 ms. In atrial fibrillation, a cardiac cycle preceded by an RR interval of approximately 800 ms was selected for measurements.

Results (Table 1)

Timing of the low frequency component. The onset of the low frequency component occurred at an average of 24 ms after the Q wave of the electrocardiogram. This component appeared during the closing limb of the mitral valve echogram in all patients, whether they had sinus rhythm, atrial fibrillation, AV block or atrial flutter. In mitral valve echograms with a distinct point B on the descending or closing movement of the anterior mitral leaflet, the onset of the low frequency component was synchronous with this point (Fig. 1). Subsequent mitral leaflet coaptation (point C) coincided with the first high frequency component (mitral component) of the first heart sound (Fig. 1, left panel). In atrial fibrillation, the mitral valve showed an accelerated closing motion during the low frequency component (Fig. 2). The low frequency component was observed even in patients with mitral valve replacement and atrial fibrillation (Fig. 3). In complete AV block or atrial flutter, this component appeared during the descending limb of the closing mitral valve echogram in every beat, regardless of the timing of preceding P or F waves, respectively (Fig. 4 to 6).

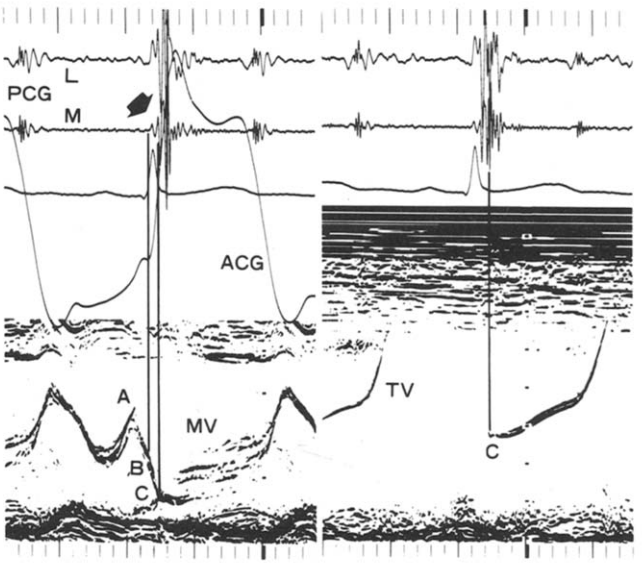

Figure 1. Case 1. Echophonocardiograms of a normal individual. The low frequency component in the phonocardiogram (PCG) **(arrow)** that appears just after the onset of the QRS complex is coincident with the onset of systolic rise of the apexcardiogram (ACG). The component is noted to occur during the rapid closing movement (BC slope) of the mitral valve (MV) echogram. This sound is followed by the initial high frequency component of the first heart sound, which corresponds to the coaptation of both mitral valve leaflets (point C). Note also that mitral valve closure precedes tricuspid valve (TV) closure (**right panel**). The lower medium frequency (M) phonocardiogram was recorded with a high pass filter with a frequency response of 100 Hz/12 dB used to determine the timing of the sounds in this study. The upper low frequency (L) phonocardiogram was recorded with a filter of 50 Hz/6 dB for reference. Dot intervals are 1 cm apart and time intervals are 40 ms apart.

Tricuspid valve closure was recorded in 23 patients and occurred at an average of 28 ms after the mitral valve closure (Fig. 1). In no patient did tricuspid valve closure precede that of the mitral valve.

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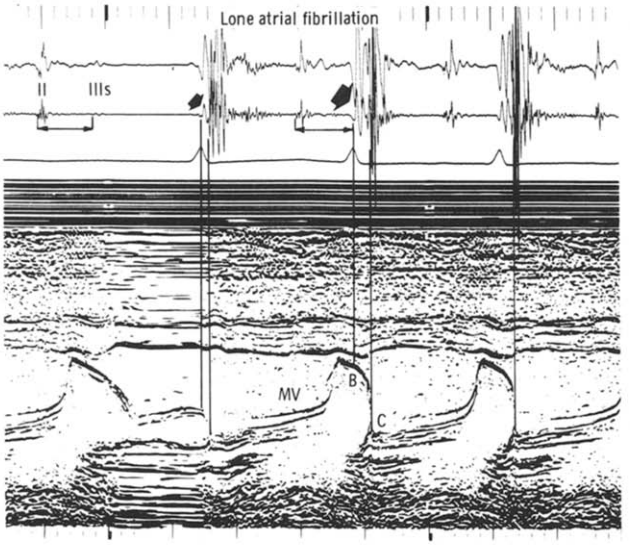


Figure 2. Case 24. The low frequency component in atrial fibrillation. The loudest low frequency component **(large arrow)** occurs when the cycle length is abbreviated such that the duration of diastole approximates the expected time interval from the second heart sound (II) to a third heart sound (III) as seen in the long diastole at the **left.** Note that there is a maximally opened position of the mitral valve (MV) and its subsequent delayed closure in that beat. The **first vertical line** shows that the mitral valve begins to close (point B) at the onset of the low frequency component, and the **second vertical line** relates mitral valve closure (point C) to the first high frequency component of the first heart sound.

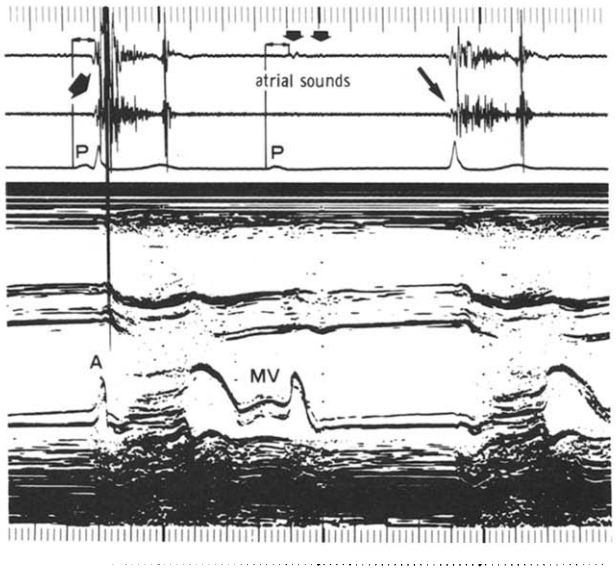
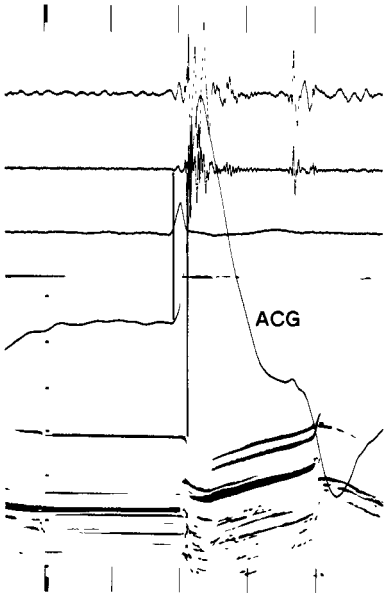


Figure 4. Case 33. Echophonocardiogram of a patient with complete atrioventricular block with sinus rhythm. The second P wave is followed by two atrial sounds **(small arrows).** An abbreviated PR interval (110 ms) is shown to induce a loud low frequency component **(left, large arrow)** during the rapid closing motion of the mitral valve (MV). This apical sound is of small amplitude when it is not preceded by a P wave **(right, long arrow),** but is augmented when an atrial sound occurs critically at the early systolic phase and becomes blended in with the low frequency component. This event occurs just before, but not after, mitral valve closure.

Figure 3. Case 27. The low frequency component of a patient with mitral valve replacement (Hall-Kaster prosthesis) and atrial fibrillation. The onset of this sound is synchronous with that of the apexcardiogram (ACG), and is followed by a loud first heart sound or the closure of the mitral valve prosthesis.



The apexcardiogram was recorded simultaneously with the mitral valve echogram and the low frequency component in 10 patients. The onset of the upstroke of the apexcardiogram was synchronous with the beginning of the low frequency component in six patients and within 10 ms in the other four.

Intensity of the low frequency component. The low frequency component was smaller in amplitude than the remainder of the first heart sound in all patients. It was inaudible in almost all patients and therefore could not be separated from the first heart sound by auscultation. The amplitude of its vibration varied from patient to patient. In three patients with atrial fibrillation and one with complete AV block, the intensity of the low frequency component varied greatly on a beat to beat basis.

In atrial fibrillation, this component was loudest in beats preceded by a short RR interval (Fig. 2). Maximal intensity of the sound was noted when it occurred at the expected time of a third heart sound after the preceding beat. This moment coincided on the echogram with a maximally opened position of the mitral valve leaflets. The intensity of the low frequency component was correlated with the interval of the Q wave to mitral valve closure. This interval was prolonged in beats of the loud component (Fig. 2).

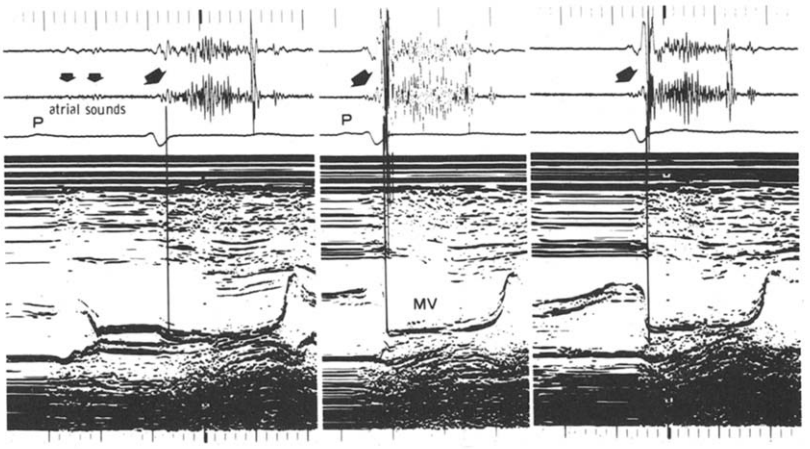


Figure 5. Case 34. Echophonocardiograms of another patient with complete atrioventricular block. In complete heart block when crucial PR intervals are not recorded, the apical low frequency component **(large arrow)** is constantly present without a change in loudness in every beat. In the **left panel,** the low frequency component occurs at the time of a nearly closed position of the mitral valve (MV) independent of atrial sounds **(small arrows).**

In complete AV block, the amplitude of the low frequency component was influenced by the duration of the PR interval (Fig. 4). With a very short PR interval, a loud first heart sound was noted and the low frequency component was similarly augmented in intensity. In one patient with complete AV block, but without critical short PR intervals, however, the component was constantly present without the change in its loudness (Fig. 5).

Low frequency component and the atrial heart sound. We were able to investigate the time relation among the low frequency component, atrial sound and the mitral valve echogram in eight patients. The peak of the atrial sound during sinus rhythm occurred at or just after that of the mitral valve A wave, but before the QRS complex (Fig. 7). In three patients with congestive heart failure (Cases 13, 16 and 17), the small low frequency component occurred at the timing of a plateau (B notch) of the closing mitral valve echogram. In one of these patients whose recording included a simultaneous apexcardiogram, the low frequency component started at the initial upstroke of the apical beat (Fig. 7).

Discussion

Origin of the low frequency component. The low frequency component was best recorded at or just medial to the apex, which is the best location for left-sided cardiac events. We found it difficult to recognize this sound at the left lower sternal-right ventricular area. This component began at the onset of the apexcardiogram, but before mitral valve closure. From the present echophonocardiographic study, it is evident that the low frequency component occurs in the absence of atrial contraction and even when the mitral valve has been replaced by a prosthesis. These observations are consistent with those of Counihan et al. (9) and Armstrong and Gotsman (12). After the onset of left ventricular systole, there is a crossover of left ventricular and atrial pressures, followed by mitral valve closure (16,17). It has

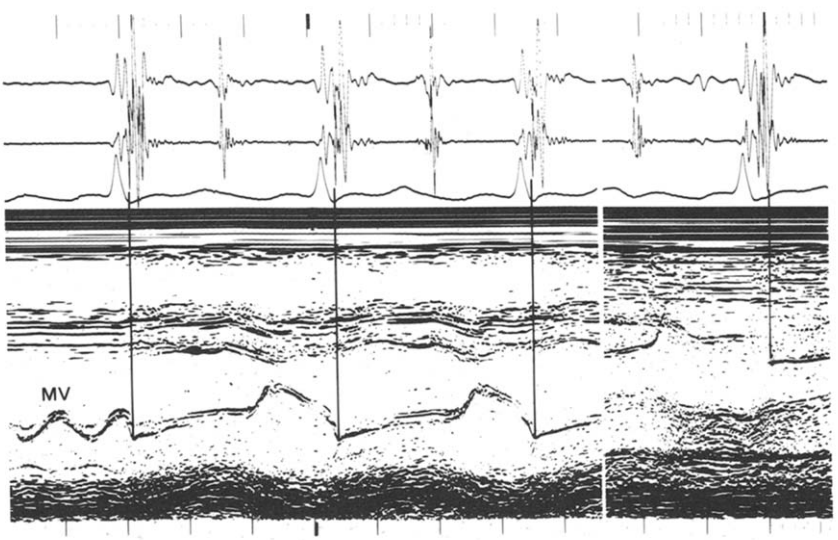


Figure 6. Case 35. The low frequency component in atrial flutter. The low frequency component is observed independent of the timing of the F wave of the electrocardiogram. The mitral valve (MV) begins to close after the onset of this soft sound. The **vertical line** relates the mitral valve closure to the first high frequency component of the first heart sound **(left panel).** The tricuspid valve closes later than the mitral valve **(right panel).**

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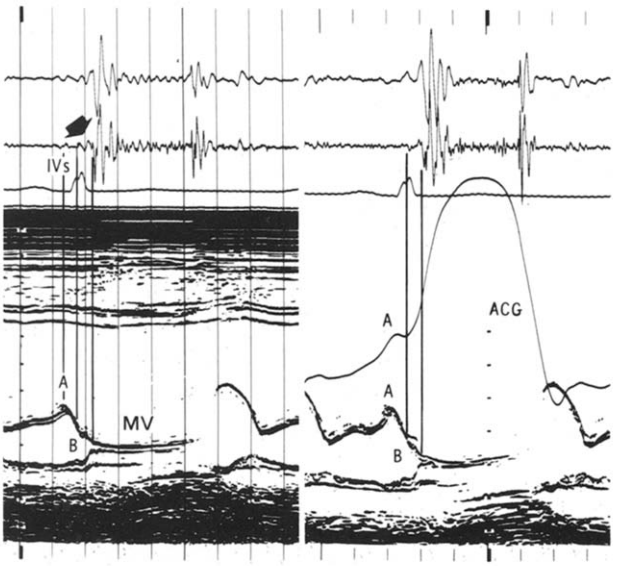

Figure 7. Case 16. The low frequency component and a fourth heart sound (IVs) of a patient with congestive heart failure. The low frequency component **(arrow)** is nearly synchronous with the B notch of the mitral valve (MV) echogram **(left)** and the beginning of the apexcardiogram (ACG) **(right).** A fourth heart sound appears at the timing of the mitral valve A wave in the echogram. A fourth heart sound in the **right panel** is obscured because a sound microphone is replaced by the transducer for recording the apex beat. Time intervals are 100 ms apart in this figure only.

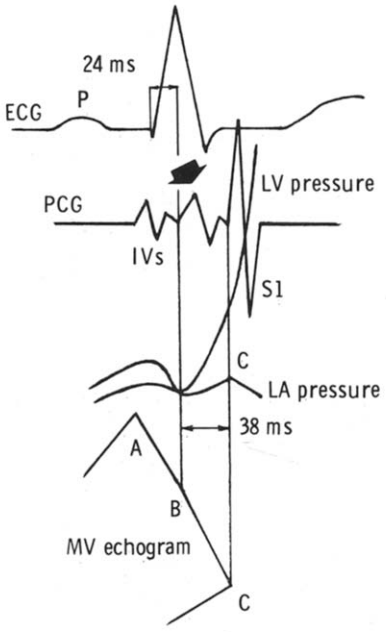

Figure 8. Schematic representation of the timing of the low frequency component of the phonocardiogram (PCG) in relation to a fourth heart sound (IVs), the first heart sound (S_1), mitral valve (MV) echogram and intracardiac pressures. A fourth heart sound occurs at the time of the A wave of the mitral valve echogram, but before the QRS complex of the electrocardiogram (ECG). The low frequency component **(arrow)** begins at the upstroke of left ventricular (LV) pressure and lasts until the initial high frequency component of the first heart sound. See text for details. LA = left atrial.

been described that mitral closure precedes tricuspid closure (1–5), and that both valve closures are synchronous with the C waves of corresponding atrial pressures (18). The low frequency component, which Lakier et al. (11), with the use of fluid-filled catheters found coincident with pressure crossover, must therefore be a cardiac event that precedes mitral valve closure. Right ventricular contraction or tricuspid valve closure can be excluded as a main contributor to the low frequency component, which we believe to be left-sided in origin.

In view of the reports (19,20) that the initial rise in the apexcardiogram is coincident with the onset of left ventricular pressure, we can illustrate our concept of the relation of the low frequency component to the rapid sequence of events in early systole by a diagram (Fig. 8). The sound occurs synchronously with the increase in left ventricular pressure at an average of 24 ms after the electrical systole, but 38 ms before mitral valve closure or point C of the left atrial pressure tracing. Therefore, these apical low aplitude deflections are an early systolic or pre-isovolumic sound (13, 14), and mitral valve tension does not contribute to the production of this sound as demonstrated by our patients with a mitral valve prosthesis.

Mechanisms of production. Tavel and Bonner recorded early systolic accentuation of the low frequency component, a third heart sound or diastolic rumble during the short cycles of atrial fibrillation on phonocardiograms (13), and proposed two mechanisms for its genesis (14): 1) active flow of blood into the ventricle, and 2) an abrupt change in compliance of the left ventricular wall in early systole acting to decelerate this blood. Thereafter, using echophonocardiography, Armstrong et al. (15) recorded the sound coincident with the upstroke of the apexcardiogram, but before the mitral valve closure. This observation is confirmed by our present study, and supports the concept that the left ventricular muscle plays a dominant role in the genesis of the low frequency component. However, it is evident from two examples from our study that the deceleration of anterograde flow across the mitral orifice also has a major role in its genesis. The first example of this phenomenon is the intensification of the low frequency component in atrial fibrillation when it appeared in early diastole, coincident with the expected time of a third heart sound of a preceding beat (Fig. 2). This fluctuation in intensity was apparent when beat to beat variations in RR intervals were noted in atrial fibrillation. Thus, when flow from atrium to ventricle in early diastole is decelerated by left ventricular contraction, the low frequency component will be magnified. The other example of the importance of AV flow is a fusion of the

low frequency component and atrial sound (Fig. 4). This sound became loudest when a critically timed atrial sound occurred at early systole. Both examples suggest that the low frequency component results from a summation of left ventricular mass tension (contractility) and the deceleration of AV flow (volume of flow and its deceleration).

It should also be noted that the weak beat after a short preceding diastole in atrial fibrillation had the strongest sound. This is contrary to the muscular theory of Armstrong et al. (15). Therefore, we believe that the arbitary muscle contraction as the origin of the "M sound," as we call it, is inadequate to explain its variability on a beat to beat basis in arrhythmias, and propose that two mechanisms—deceleration of anterograde flow and muscular contraction—are necessary to the genesis of the low frequency component.

Implications. From the limited number of patients in this study, we found it difficult to compare the low frequency component among the patients. We need to investigate many more patients to draw any conclusion about the clinical significance of the intensity of this small sound and the role of bundle branch block, chest wall vibrations, coronary artery disease, ischemia and left versus right ventricular events.

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References

1. Burggraf GW, Craige E. The first heart sound in complete heart block. Phono-echocardiographic correlations. Circulation 1974;50:17–24.
2. Leatham A, Leech G. Observations on relation between heart sounds and valve movements by simultaneous echo- and phonocardiography (abstr). Br Heart J 1974;37:557.
3. Mills PG, Chamusco RF, Moos S, Craige E. Echophonocardiographic studies of the contribution of the atrioventricular valves to the first heart sound. Circulation 1976;54:944–51.
4. Craige E On the genesis of heart sounds Contributions made by echocardiographic studies (editorial). Circulation 1976;53:207–9.
5. Brooks N, Leech G, Leatham A. Factors responsible for normal splitting of the first heart sound. High-speed echophonocardiographic study of valve movement Br Heart J 1979,42:695–702.
6. Kincaid-Smith P, Barlow J The atrial sound and the atrial component of the first heart sound. Br Heart J 1959;21:470–8.
7. Rappaport MB, Sprague HB. The graphic registration of the normal heart sounds. Am Heart J 1942;23.591–623.
8. Hultgren HN, Hubis H. A phonocardiographic study of patients with the Starr-Edwards mitral valve prosthesis. Am Heart J 1965;69:306–19
9. Counihan T, Messer AL, Rappaport MB, Sprague HB. The initial vibrations of the first heart sound. Circulation 1951;3.730–2.
10. Luisada AA, MacCanon DM, Coleman B. New studies on the first heart sound. Am J Cardiol 1971;28.140–9
11 Lakier JB, Fritz VU, Pocock WA, Barlow JB. Mitral components of the first heart sound. Br Heart J 1972;34:160–6.
12. Armstrong TG, Gotsman MS Initial low frequency vibrations of the first heart sound. Br Heart J 1973;35:691–6.
13. Bonner AJ, Stewart J, Tavel ME. "Presystolic" augmentation of diastolic heart sounds in atrial fibrillation. Am J Cardiol 1976;37:427–31.
14. Tavel ME, Bonner AJ Presystolic murmur in atrial fibrillation. Fact or fiction? (editorial) Circulation 1976,54:167–8.
15. Armstrong TG, Mitha AS, Matisonn RE, Weir EK, Chesler E. Noninvasive observations on initial low frequency vibrations of the first heart sound—correlation with the 'presystolic' murmur in mitral stenosis. Br Heart J 1978;40:750–7
16 Rubenstein JJ, Pohost GM, Dinsmore RE, Harthorne JW. The echocardiographic determination of mitral valve opening and closure Correlation with hemodynamic studies in man. Circulation 1975;51:98–103.
17. Shiina A, Matsumoto Y, Kawasaki K, et al. Analysis of opening and closing motion of the valve by simultaneous echocardiogram and pressure tracing across the valve J Cardiogr 1976;6:661–72.
18 O'Toole JD, Reddy SP, Curtiss EI, Griff FW, Shaver JA. The contribution of tricuspid valve closure to the first heart sound. An intracardiac micromanometer study. Circulation 1976,53:752–8.
19. Tavel ME, Campbell RW, Feigenbaum H, Steinmenz EF. The apex cardiogram and its relationship to haemodynamic events within the left heart. Br Heart J 1965,27:829–39
20 Willems JL, De Geest H, Kesteloot H On the value of apexcardiography for timing intracardiac events Am J Cardiol 1971;28.59–66