The Clinical Value of Frequency Analysis of the First Heart Sound in Myocardial Infarction

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SUMMARY

A sensitive technic for frequency analysis of the first heart sound (S_1) during isovolumic contraction time (ICT) was developed. Recorded heart sounds were filtered with a dynamic analyzer. Amplitudes of frequencies between 30 and 70 cps were plotted as a percentage of peak total energy of S_1 against frequency. A consistent and reproducible frequency "fingerprint" was obtained in 74 normal subjects. Frequencies of S_1 were shown to be directly proportional to ventricular elasticity (VE) and inversely proportional to combined ventricular mass (VM). VM is constant during ICT. Amplitude at 40 cps was less than at 30 cps because of reduced VE (myocardial infarction), increased VM (athletes), or combined reduction in VE and increased VM (myocardiopathy). Normal patterns were found in aortic insufficiency (increased VE and VM). Diagnostic patterns were found in 21 of 24 patients with acute myocardial infarction (MI) but were similar to patterns found in patients with healed infarcts and myocardiopathy and athletes. Acute pulmonary embolism could be differentiated from MI.

Additional Indexing Words:

Acute myocardial infarction	Aortic insufficiency	Athletes
Isovolumic contraction time	Mitral stenosis	Pulmonary embolism
Prosthetic mitral valves	Ventricular elasticity	Ventricular mass

USING a new technic for the frequency analysis of heart sounds, we have attempted to investigate the validity of Rushmer's[1] hypothesis of the origin of heart sounds. We have also evaluated the clinical usefulness of this technic in the diagnosis of myocardial infarction.

According to Rushmer's[1] hypothesis, analysis of the frequency content of the first heart sound (S_1) should give valuable information regarding the anatomy and physiology of the structures which generate that sound. The initial audible components of S_1 begin with abrupt tension of the closed mitral valve which decelerates the moving blood. These initial audible vibrations, which occur during the isovolumic contraction phase of the cardiac cycle, result from oscillatory overdistention of the valve and the myocardium to produce a "water-hammer" effect and not mitral valve closure per se.

The loudness of S_1 should depend on the rate of deceleration of blood at the onset of isovolumic contraction. The frequency content of the isovolumic contraction phase of S_1, however, should depend on the relative

contributions of mass and elasticity associated with the oscillating left ventricle. The term "mass" refers to the left ventricular muscle mass and the volume of blood in the left ventricle at end diastole. Mass remains constant during isovolumic contraction, the period of measurement. Left ventricular elasticity varies during isovolumic contraction, but gross decreases in the average value caused by a loss of contractile elastic muscle elements, as in myocardial infarction, should be detectable.

This hypothesis was tested by predicting how well-defined clinicopathologic disease entities should change the normal frequency pattern of S_1, and then by validating the prediction in patients with known heart disease. We reasoned that the frequency content of S_1 should be decreased by disease states which reduce ventricular elasticity (myocardial infarction), increase ventricular

mass (trained athletes), or both reduce ventricular elasticity and increase combined ventricular mass (myocardiopathy). Similarly the frequency content of S_1 should remain unchanged if elasticity and mass both increased proportionally (aortic insufficiency). The frequency versus amplitude patterns in all of these conditions were consistent with our predictions.

In addition, we found the frequency pattern of S_1 in myocardial infarction to be of diagnostic value, enabling the differentiation of myocardial infarction from acute pulmonary embolism.

Methods

A Model of the Elastic-Mass System

The term *ventricular elasticity*, $k(t)$, is used to represent a type of spring constant of the left ventricle which is changing in value with time during isovolumic contraction as the tension in

Figure 1

The natural frequencies of the first heart sound, $f_{vent.}$ (t), during the isovolumic contraction period are related to the ratio of ventricular elasticity, k (t), and combined ventricular mass, w/g, according to the equation shown in this diagram. Diagrammatic representations of the model for a normal ventricle are shown (a) and for an infarcted ventricle (b).
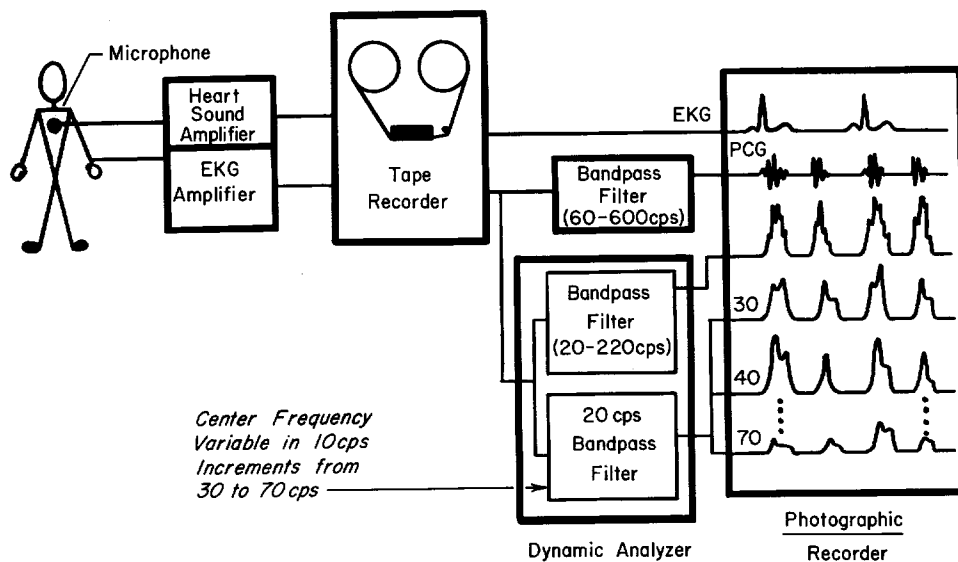
Figure 2

Data acquisition and analysis system. Heart sounds and the electrocardiogram were recorded on magnetic tape. Taped heart sounds were subsequently filtered in two ways. A standard bypass filter produced the standard phonocardiographic tracing (PCG). A dynamic analyzer was used to determine frequency content. One subsystem of this dynamic analyzer passed all heart sound frequencies between 20 and 220 cps. This output was considered "total heart sound voltage." The other analyzer subsystem was a 20-cps bandpass filter in which the center frequency setting could be varied in 10-cps increments from 30 to 70 cps. The electrocardiogram and the outputs of the bandpass filter and dynamic analyzer were graphically displayed. See text for details.

the ventricle increases. The term *combined ventricular mass,* w/g, represents the sum of the mass of the left ventricle and the blood it contains. This value should remain constant during isovolumic contraction; hence, it is not expressed as a function of time. The frequencies at which the ventricle will vibrate, $f_{vent.}$ (t), are assumed to be proportional to the ratio of ventricular elasticity and combined ventricular mass according to the equation:

$$f_{vent.}\ (t) \sim \frac{k(t)}{w/g}.$$

(isovol. cont.)

This model is illustrated diagrammatically in figure 1a. In myocardial infarction (fig. 1b), a reduction of myocardial contractile elastic units should result in a decrease in the frequency content of S_1.

The Recording System

Equipment used in data acquisition and analysis is shown in figure 2. All recordings were made with the subject in the supine position during normal continuous respiration. Heart sounds were recorded by a Hewlett-Packard/San-

born* contact microphone (model 350-1700-C10) placed at the cardiac apex and a Hewlett-Packard* heart sound preamplifier (model 350-1700B). The frequency response of the microphone and preamplifier combination was flat from 20 to 1000 cps (or Hz). An electrocardiogram was simultaneously recorded using a Hewlett-Packard* high gain preamplifier (model 350-2700C). Heart sounds and the electrocardiogram were recorded on a dual channel Crown† magnetic tape recorder (model SX-700) with the following specifications: frequency response flat from 20 to 25,000 cps at a tape speed of 7½ ips; signal to noise ratio, −55 db; and flutter and wow, 0.09%. The electrocardiogram was recorded on an AM tape channel converted to FM using an A.R. Vetter‡ FM recording adaptor (model 2). Frequency response of the FM conversion was 0 to 1000 cps.

The heart sounds on tape were subsequently filtered and displayed as described below and

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*Sanborn Division of Hewlett-Packard Corporation, Waltham, Massachusetts.

†Crown International, Inc., Elkhart, Indiana.

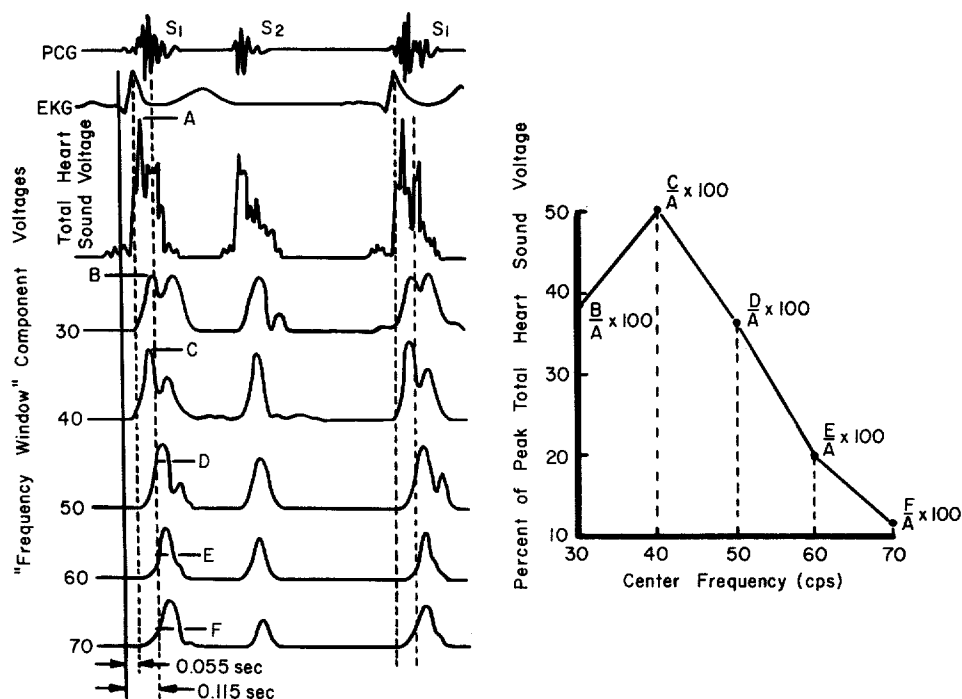‡A. R. Vetter Co., Rebersburg, Pennsylvania.

Figure 3

A normal frequency analysis is shown graphically in the left panel. The isovolumic contraction period was drawn on each first heart sound. The peak voltage output during this period was determined for the total heart sound and each of the component frequencies as shown. The ratio of the peak voltage output of each "frequency window" component to the peak total heart sound voltage permits a plot of the per cent of peak total heart sound voltage versus component center frequency as shown in the right panel. See text for details.

shown in figure 2. A Krohn-Hite* bandpass filter (model 310C, center frequencies set at 60 cps and 600 cps) filtered the heart sounds for display as the standard phonocardiographic tracing. A dynamic analyzer† (Spectral Dynamics analyzer, model SD-101A) containing two different bandwidth filters, one 200 cps wide, the other 20 cps wide, was also used to filter the heart sounds. The dynamic analyzer is a bandpass filter whose positive ordinate amplitude is proportional to the loudness of the frequency components occurring within the bandwidth. This analyzer heterodynes the input signal and then operates on a center frequency offset principle, so that the filter time constant does not vary with the center frequency setting. With the center frequency of the 200 cps bandwidth filter in the dynamic analyzer set at 120 cps, this filter passes all frequency components from 20 to 220 cps. This output was considered "total first heart sound voltage." Frequencies below 20 cps may be considered

precordial movements[2] rather than heart sounds since they are inaudible. The upper cutoff frequency of 220 cps was selected because we found little first heart sound energy above 200 cps or indeed above 100 cps. This finding agrees with that of Burton.[3] The center frequency of the 20 cps bandwidth component filter of the dynamic analyzer was set initially at 30 cps and then varied stepwise in 10 cps increments from 30 cps to 70 cps.

The final outputs for analysis were recorded with a multichannel Hewlett-Packard photographic recorder (series 560) at a paper speed of 100 mm/sec. The outputs consisted of an electrocardiogram, a standard phonocardiogram, "total first heart sound voltage," and 20 cps "frequency windows" with center frequencies of 30, 40, 50, 60, and 70 cps, respectively (figs. 2 and 3). The interval from Q to onset of S_1 was measured in all instances, using the electrocardiogram and standard phonocardiogram. Isovolumic contraction time (ICT) was assumed to be 0.06 sec.[4] Both intervals were drawn on each of the filtered heart sound read-outs (fig. 3). The

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*Krohn-Hite Corp., Cambridge, Massachusetts.

†Spectral Dynamics Corp., San Diego, California.

practical validity of these estimates of systolic interval was confirmed in six subjects according to the method of Weissler and co-workers.[5] The peak voltage output during the time of isovolumic contraction was determined for the total first heart sound voltage and each of the 20 cps bandwidth component frequencies. The same 10 consecutive beats were analyzed for each of the filtered outputs, and the results were averaged. The ratio of the average peak voltage output of each of the 20 cps bandwidth components to the peak total heart sound voltage versus component center frequency was plotted as shown in figure 3. The intensity (loudness) of S_1 may vary significantly from one patient to another. The plot of the ratio of the peak intensity of the frequency components to the peak total heart sound voltage versus frequency allows qualitative comparison of patients regardless of the intensity of their first heart sounds.

Patient Selection

We recorded heart sounds from 74 normal healthy men, ranging in age from 20 to 63 years. Frequency distribution patterns were analyzed by decades and for possible effects of height and weight. Repeatability studies were performed on 14 normal subjects after 2 days and in five of the same subjects after 1 year. Normality was established by history, physical examination, blood pressure, and an electrocardiogram.

Frequency distribution patterns were determined in 24 patients with acute myocardial infarctions confirmed by history, electrocardiograms, and myocardial enzyme elevations. We studied 16 patients with electrocardiographic criteria for healed myocardial infarction (diagnos-

tic Q waves); 14 college athletes, including 10 long-distance runners; 10 patients with chronic primary cardiomyopathy; five patients with severe aortic insufficiency confirmed by left heart catheterization and aortography; 11 patients with mitral stenosis confirmed by cardiac catheterization or surgery, or both; and four patients with mitral Starr-Edwards prosthetic valves.

Patients with myocardiopathy were selected from a larger group of patients screened and followed by Dr. Noble O. Fowler at the Cincinnati General Hospital. In the absence of angina or myocardial infarction, the following features suggested the diagnosis of cardiomyopathy rather than coronary disease: absence of diabetes mellitus, hyperlipemia, or hypercholesterolemia; lack of family history of coronary artery disease; pronounced and persistent ventricular and atrial gallop sounds; radiologic evidence of a striking decrease in heart size after treatment for heart failure; and prolonged survival after the onset of congestive heart failure. Because of the potential hazard to the patient and, in most instances, the lack of specific therapy, we have not employed coronary arteriography to differentiate coronary disease from myocardial disease in these patients.

Results

Normal Subjects (Fig. 4)

The normal frequency signature of S_1 derived from analyses in 74 normal adult men is shown in figure 4c. The average normal pattern was characterized by a greater contribution of energy at 40 cps than at 30 cps and progressively decreasing energy levels at
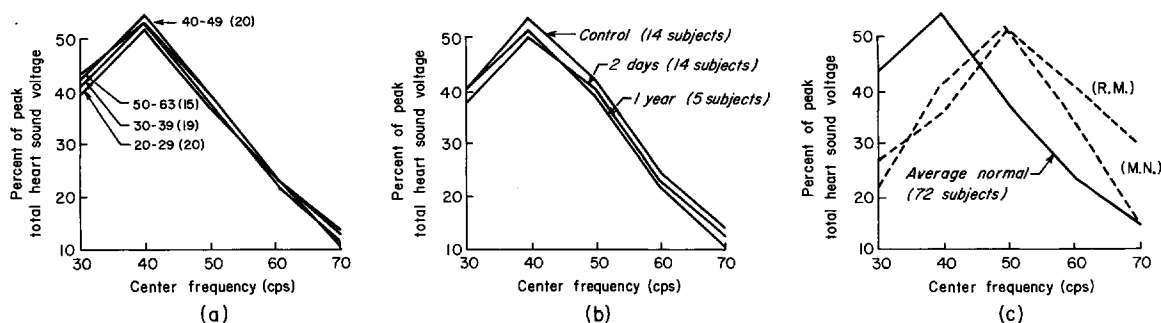

Figure 4

The normal frequency signature of the first heart sound (S_1) derived from 74 normal adult men. (a) Frequency analysis by decades. There was no significant effect of age on the shape of the curve. (b) The reproducibility of frequency analyses was determined 2 days apart in 14 subjects and 1 year apart in five of the 14 subjects. No significant change in the shape of the curve was found. (c) The averaged normal frequency signature is shown. The normal pattern is characterized by a greater percentage of energy at 40 cps than at 30 cps and progressively decreasing levels above 40 cps. Two variants are shown by dashed lines.

frequencies above 40 cps. In two normal subjects, the pattern differed somewhat (fig. 4c). Although the energy content at 40 cps exceeded that at 30 cps, the greatest energy level was found at 50 cps. At higher frequencies, the energy decreased progressively as in other normal subjects. In all subjects, the sum of the individual percentages of peak total heart sound voltage determined at center frequencies between 30 and 70 cps exceeded 100%. This was caused by the frequency overlapping of the 20 cps bandpass component filter which was stepped at 10 cps increments through the desired frequency range. Even so, it is apparent that normally

more than 85% of the energy content of the isovolumic phase of S_1 could be accounted for at frequencies below 80 cps. It was noted also that although the frequency analysis curves were dispersed along the ordinate, they retained the same general shape. The cause of this dispersion can be explained by the finding in many subjects of a rapid burst of heart sound voltage during the isovolumic contraction phase. In these instances, the 200 cps filter which recorded peak total heart sound energy was capable of responding fully to the transient voltages, while the 20 cps component filter could not. It follows then, that the ratio of component frequency voltage to total peak
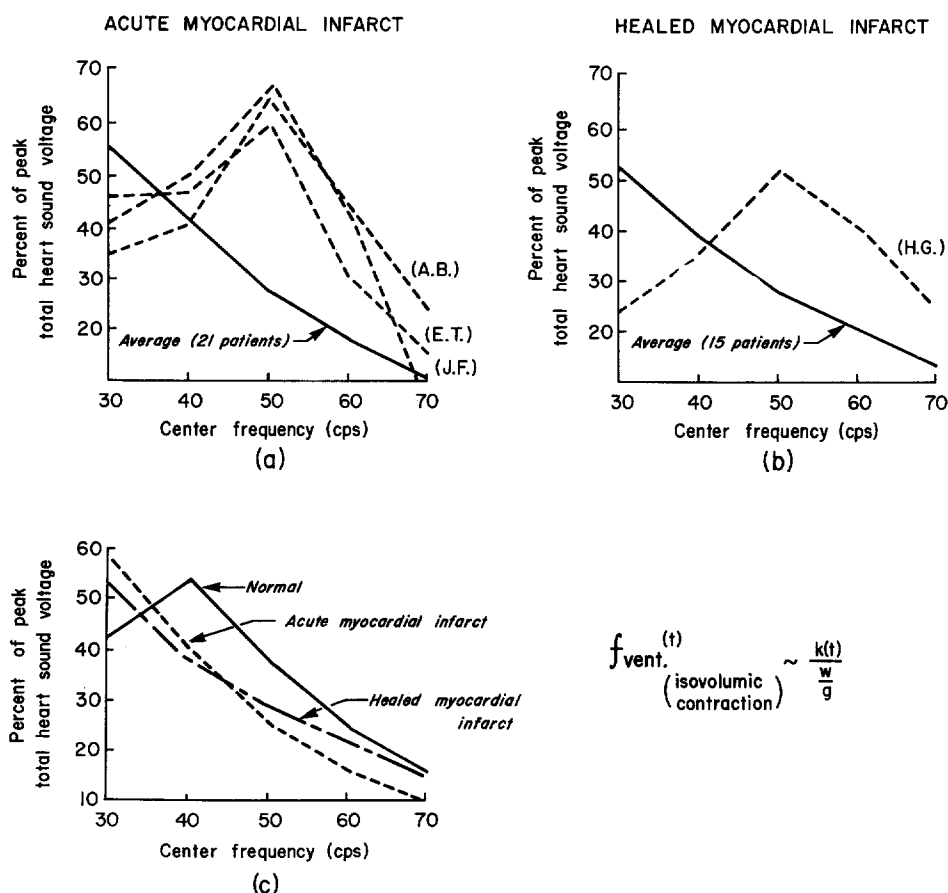

$$\int \text{vent.}^{(t)} \begin{pmatrix} \text{isovolumic} \\ \text{contraction} \end{pmatrix} \sim \frac{k(t)}{\frac{w}{g}}$$

Figure 5

Frequency analyses from patients having acute myocardial infarction (a) and healed myocardial infarction (b). Variants are shown by dashed lines. Unlike normal subjects, these two groups of patients exhibited increased percentages of low frequency components (c). These findings are consistent with reduced ventricular elasticity, k(t).

energy was reduced, resulting in a lower ordinate position of the final frequency analysis but no change in its shape.

The effect of age on the shape of the voltage-frequency curve was studied by decades in 74 normal men, 20 to 63 years of age. The age groups were as follows: 20 to 29 (20 men), 30 to 39 (19 men), 40 to 49 (20 men), and 50 to 63 (15 men). There was no significant effect of age on the shape of the curve (fig. 4a).

Ordinate values and shape of the frequency analysis curves showed little, if any, correla-

tion with body build (that is, mesomorph, ectomorph, or endomorph) or heart rate in the range of 60 to 110 beats/min.[6]

The reproducibility of the frequency analysis was determined. Frequency analyses were performed 2 days apart on 14 subjects, and 1 year apart on five of the 14 subjects. No significant change in the ordinate position or shape of the curve was found (fig. 4b).

Myocardial Infarction
(Decreased Elasticity, Normal Mass)

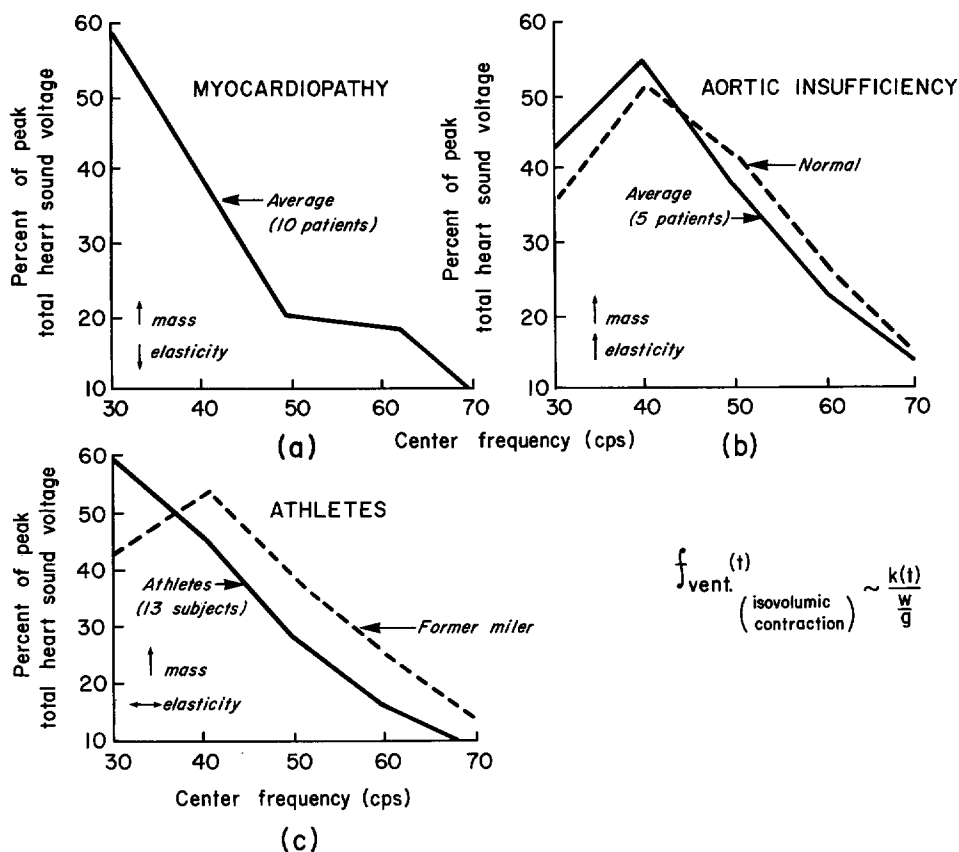Frequency analyses were obtained from 24 patients with evidence of acute myocardial

Figure 6

Frequency analyses from two groups of patients in whom both combined ventricular mass and ventricular elasticity have been varied by disease (a and b). The increase in mass and generalized reduced elasticity found in myocardiopathy (a) resulted in a greater percentage of low frequency components. In severe aortic insufficiency (b), the increase in ventricular wall tension was balanced by an increase in combined ventricular mass, producing a normal pattern. The increase in combined ventricular mass in college athletes (c) increased the low frequency components. The frequency pattern of a former miler was normal 2 years after cessation of training.

infarction (1 to 3 days previously). None of the patients were in clinical congestive heart failure. A similar pattern, that is, a greater percentage of low frequency components than that found in the normal signature, was found in 21 of the 24 patients. The greatest percentage of heart sound energy was found at 30 cps, and the percentage progressively decreased at higher frequencies (fig. 5a). The other three patients exhibited patterns similar to the two normal variants (fig. 5a).

Sixteen patients with electrocardiographic and historical evidence of myocardial infarction sometime in the past, that is, pathologic Q waves in the electrocardiogram, were studied. Heart size was normal according to chest roentgenograms. The average frequency analysis curve (fig. 5b) closely paralleled that found in patients with recent infarction. In one patient (H.G.), the percentage of total heart sound voltage was greater at 40 cps than at 30 cps with further peaking at 50 cps (fig. 5b). This variant pattern was also seen in two normal subjects and three patients with acute myocardial infarction. All of these patients have survived so that pathologic correlations were not performed.

Myocardiopathy
(Decreased Elasticity, Increased Mass)

Frequency analyses were obtained in 10 patients with chronic primary myocardiopathy (fig. 6a). All had enlarged hearts and persistent third and fourth sound gallops. The frequency pattern was similar to that found in myocardial infarction except that the ordinate values at each frequency were lower. This finding suggests greater percentage loss of elastic tissue and the additive factor of an increased combined ventricular mass. All patients showed this pattern.

Severe Aortic Insufficiency
(Increased Elasticity, Increased Mass)

Five patients had severe aortic insufficiency characterized by a wide pulse pressure, left ventricular enlargement, a third sound gallop, and an Austin-Flint murmur at the apex. All patients underwent diagnostic cardiac catheterization. Marked left ventricular dilation and elevation of the left ventricular end-diastolic

pressure were found, and other valve lesions were excluded. Normal frequency curves were found in all patients (fig. 6b). We have assumed that the increase in ventricular wall tension was balanced by an increase in combined ventricular mass.

Trained Athletes
(Constant Elasticity, Increased Mass)

Data were obtained from 14 college athletes. The average frequency pattern in 13 of the 14 athletes resembled that of myocardial infarction (fig. 6c). The increased frequency content at 30 cps presumably represents increased combined ventricular mass in these healthy young men. All of the 10 long distance runners had abnormal patterns as did three of four athletes trained for other sports. One football player had a normal pattern. Another subject, who had been a distance runner but had not run for 2 years, had a normal frequency curve. The average heart rate in the 10 distance runners was 54.4 beats/min (range, 38 to 60) and was 70.2 (range, 60 to 78) in four other athletes.

Mitral Stenosis and Mitral
Starr-Edwards Prosthetic Valves

A normal frequency signature was obtained in each of 11 patients with isolated mitral
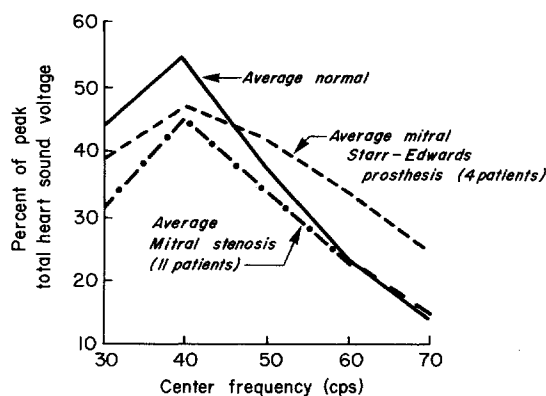

Figure 7

A normal frequency signature was obtained in all patients with isolated mitral stenosis or mitral Starr-Edwards prosthetic valves. It seems unlikely that the frequency content of the first heart sound is significantly altered by abnormal valve structure and function in the range of 20 to 80 cps.
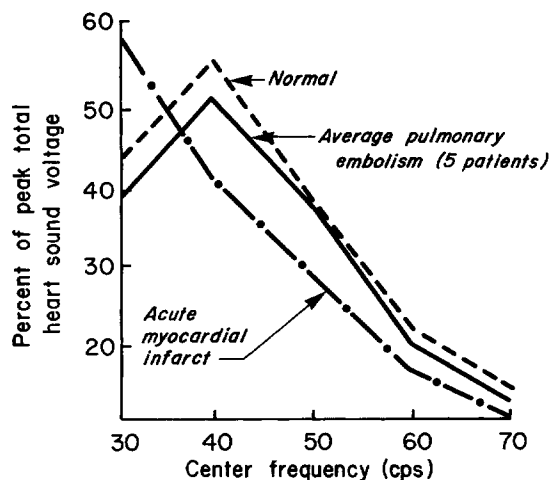
Figure 8

The frequency pattern was normal in all of five patients with proven massive pulmonary embolism. Frequency analysis was of value in differentiating this condition from acute myocardial infarction.

stenosis even though S_1 was loud and seemed "high in pitch" on auscultation (fig. 7).

Frequency analysis was performed on four patients with mitral Starr-Edwards prosthetic valves. The frequency curve was normal in all patients (fig. 7).

Since the frequency signature was normal in these two categories of patients, it seems unlikely that the frequency content of S_1 is significantly altered by abnormal valve structure and function in the range of 20 to 80 cps. The apparent loudness of S_1 in these patients attests to the sensitivity of the ear to low energy vibrations at higher frequency levels, that is, above 200 cps.

Acute Pulmonary Embolism

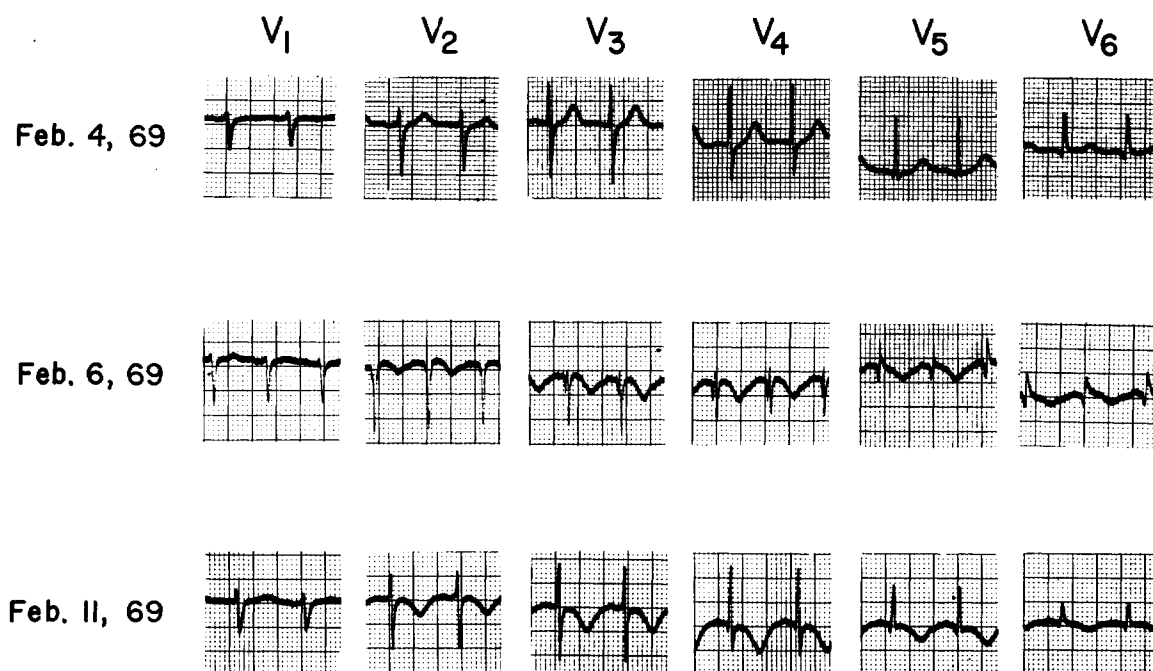A frequency analysis was applied to five patients with proven large pulmonary emboli

Figure 9

A routine electrocardiogram taken on patient F.R. was normal on February 4, 1969. On February 6, 1969, he complained of severe chest pain. This was associated with a fall in blood pressure and the appearance of abnormal Q waves in the electrocardiogram in leads V_2 to V_6. The frequency analysis was normal at the same time. The abnormal Q waves disappeared after 5 days. Massive pulmonary embolism and no myocardial infarction were found at postmortem examination.

to test the efficacy of the technic in the differential diagnosis of myocardial infarction versus pulmonary embolism. Massive pulmonary embolism was confirmed by pulmonary angiography. The frequency patterns in all patients with pulmonary embolism were normal (fig. 8). Of interest, one patient (F.R.), who had a normal electrocardiogram on February 4, 1969, developed electrocardiographic changes consistent with acute myocardial infarction (Q waves in leads V_2 to V_6) during an episode of chest pain and hypotension on February 6, 1969 (fig. 9). The frequency analysis was normal at the same time. The abnormal Q waves disappeared after 5 days. Massive pulmonary embolism and no myocardial infarction were found at postmortem examination despite multiple microscopic sections.

Discussion

A reduction in the intensity of the first heart sound after myocardial infarction has been suspected clinically for many years. Recently, Price and Brown[7] confirmed this clinical impression by a systematic study.

Another clinical impression, and one which prompted this study, was the finding of a muffled first heart sound after myocardial infarction. We suspected that S_1 was lower than normal in pitch as well as loudness. It is difficult sometimes for the human ear to distinguish pitch from loudness. For example, the loud S_1 of mitral stenosis seems of higher than normal pitch, but frequency patterns obtained from patients in this study seem to deny this impression. It is possible, however, that the small percentage of total first heart sound energy remaining above 100 cps (that is, 3%) which is easily sensed by the ear, gives the illusion of a high-pitched sound.

We are aware of only one other application of filtering to the study of heart sounds during myocardial infarction. Agress and Fields[8] used a bandpass filter with a bandwidth of 5 cps to filter heart sounds of dogs before and after experimental myocardial infarction. They reported a reduction in intensity of frequency components between 3 and 50 cps after

infarction. The intensities of the frequency components of the entire first heart sound were compared. One must view with skepticism, however, the use of a bandpass filter having a bandwidth of 5 cps and a time constant of about 0.2 sec in the analysis of a heart sound of short duration.

Few investigators have studied the frequency content of heart sounds.[8-10] The Kay analyzer was used by McKusick[9] to give a three-dimensional display of frequency, amplitude, and time. In practice, frequency range, recording time, and resolution can be varied, but each variable is dependent on the others. Gross frequency pattern changes can be detected, but detailed frequency component analysis is difficult. These displays have been of little clinical value. The Spectral Dynamics analyzer used in this study is a more accurate filter having a higher dynamic range than the Kay analyzer in addition to having sharper filter "skirts." Use of this dynamic analyzer coupled with the magnetic tape recorder (used to record heart sounds) has allowed us to manipulate the frequency range, recording time, and frequency resolution as independent variables.

The normal adult frequency signature of S_1 during isovolumic contraction has been shown to be reproducible and independent of heart rate, body build, and age. Occasional variant patterns were found, but in all instances the percentage of peak total heart sound energy at 40 cps was greater than at 30 cps.

The consistent pattern found in each of 74 normal subjects has allowed us to investigate Rushmer's[1] hypothesis regarding the origin of the first heart sound and to examine the applicability of frequency analysis as a diagnostic test for myocardial infarction.

It has been stated[1] that there are more than 40 theories regarding the origin of the first heart sound. These various theories may be grouped into two basic concepts. Most investigators have attributed S_1 to the vibration of one or more substructures of the heart. According to the substructure concept S_1 may be caused by sudden tensing of the mitral valve curtain,[9] collision of the mitral valve

cusps during closure,[11, 12] or sudden tensing of the chordae tendineae,[13] to name a few of the substructures which have been incriminated. Rushmer has championed the concept that S_1 results from the vibration of the entire blood-filled heart as a dynamically coupled system. Basic to this "cardiohemic" concept is the assumption that acceleration or deceleration of blood initiates vibrations which result in heart sounds. Proponents have claimed to have demonstrated four components to S_1. The initial low-amplitude, low-frequency, inaudible vibrations occur at the onset of left ventricular contraction when blood is moved toward the atrium, thus closing the mitral valve. The second and audible component begins with the abrupt tension of the closed mitral valve which decelerates the moving blood. This second component may represent the oscillation of blood that results from overdistention of both the valve and the myocardium to produce a water-hammer effect. This sound corresponds with the isovolumic contraction phase of the cardiac cycle. The third component is often audible and may represent oscillations of the root of the aorta and myocardium during early ventricular ejection. The fourth component probably represents vibrations caused by turbulence of blood flowing through the aorta.

It is difficult to conceive of valve cusps or chordae tendineae producing the quantity of energy found at the chest wall during the time of the first heart sound. We have, therefore, examined the "cardiohemic" hypothesis by subjecting it to engineering vibration analysis, in which the left ventricle was represented by a simplified physical model. The model was further simplified by limiting the analysis to the isovolumic contraction period. Combined ventricular mass was, therefore, constant, and only ventricular elasticity varied with time. This model was tested by predicting how a well-defined clinicopathologic disease entity should change the normal frequency pattern of S_1, and then by validating this prediction in patients with known heart disease. That these predictions were generally confirmed in these disease states tends to add credence to the "cardiohemic" hypothesis although one cannot necessarily conclude that our initial assumptions were entirely valid.

The finding of normal frequency plots in mitral stenosis and in patients with mitral Starr-Edwards prosthetic valves is strong evidence against the substructure concept which assumes that S_1 is caused primarily by valve closure and that the myocardium acts as a transmitter of sound.

It may be questioned whether the frequency analysis pattern would be changed if either the onset or duration of ICT were prolonged by disease. Since the interval from Q to the onset of S_1 was always measured, the onset of ICT was known. Although the duration of ICT was not routinely measured, it is unlikely that prolongation of ICT would significantly change the frequency plot. The duration of ICT was assumed to be 0.06 sec, a value based on results obtained at cardiac catheterization of normal subjects.[4] The average duration of ICT derived from external measurements of systolic intervals in patients with heart failure is about 0.06 sec.[5] A single voltage peak was always seen in both the 30 cps and 40 cps frequency windows as shown in figure 3. A second peak was always found following ICT. This second peak probably represents an early systolic ejection sound. Energy levels at frequencies between 50 and 70 cps assumed either of two distinct patterns. In 53% of individuals, two peaks were found; one occurred during ICT and a second peak followed the assumed isovolumic interval. In 47% of individuals, energy levels increased during ICT, but the single peak occurred shortly after the assumed interval (fig. 3). If ICT were prolonged by disease, a frequency analysis curve could show a somewhat greater percentage of total heart sound energy above 40 cps in the latter group, but not in the former group. The overall pattern would be relatively unchanged. The diagnostic value of frequency analysis of S_1 resides in the peak energy level at 30 cps compared to that at 40 cps. These values would be unaffected by prolongation of ICT.

The sensitivity and specificity of frequency analyses in the diagnosis of acute myocardial infarction deserve comment. The pattern was diagnostic in 21 of 24 patients selected because of historical, electrocardiographic, and enzyme evidence of acute infarction. Evaluation of the sensitivity of frequency analysis in a larger group of unselected individuals must await pathologic correlations. Frequency analysis may improve our accuracy in the recognition of myocardial infarction. Following acute myocardial infarction, electrocardiographic changes are often nondiagnostic, perhaps in as many as 40 to 50% of patients, although Zinn and Cosby[14] found diagnostic changes in 80% of patients with acute infarction. An accuracy rate of only 54% has been reported in patients with healed infarction proven at autopsy.[15] The specificity of frequency analysis in acute infarction was also examined. A pattern indistinguishable from acute infarction was found in healed myocardial infarction, myocardiopathy, and conditioned athletes. Frequency analysis was of value in differentiating acute pulmonary embolism from acute myocardial infarction.

Frequency analysis is a noncannulating technic which can be automated and simplified for more general clinical application. Its ultimate usefulness can be evaluated only after more extensive clinical trial.

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