

[54] METHOD AND APPARATUS FOR DIAGNOSING MYOCARDIAL INFARCTION IN HUMAN HEART

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[73] Assignee: **Board of Directors of the University of Cincinnati**, Cincinnati, Ohio

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[63] Continuation of Ser. No. 44,281, June 8, 1970, abandoned.

[52] U.S. Cl. **128/2.05 S, 128/2.06 R**

[51] Int. Cl. **A61b 5/02**

[58] Field of Search..... **128/2.05 Q, 2.05 R, 2.05 S, 128/2.06 R, 2.1 R, 2 K; 179/1 ST; 181/24**

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Primary Examiner—William E. Kamm

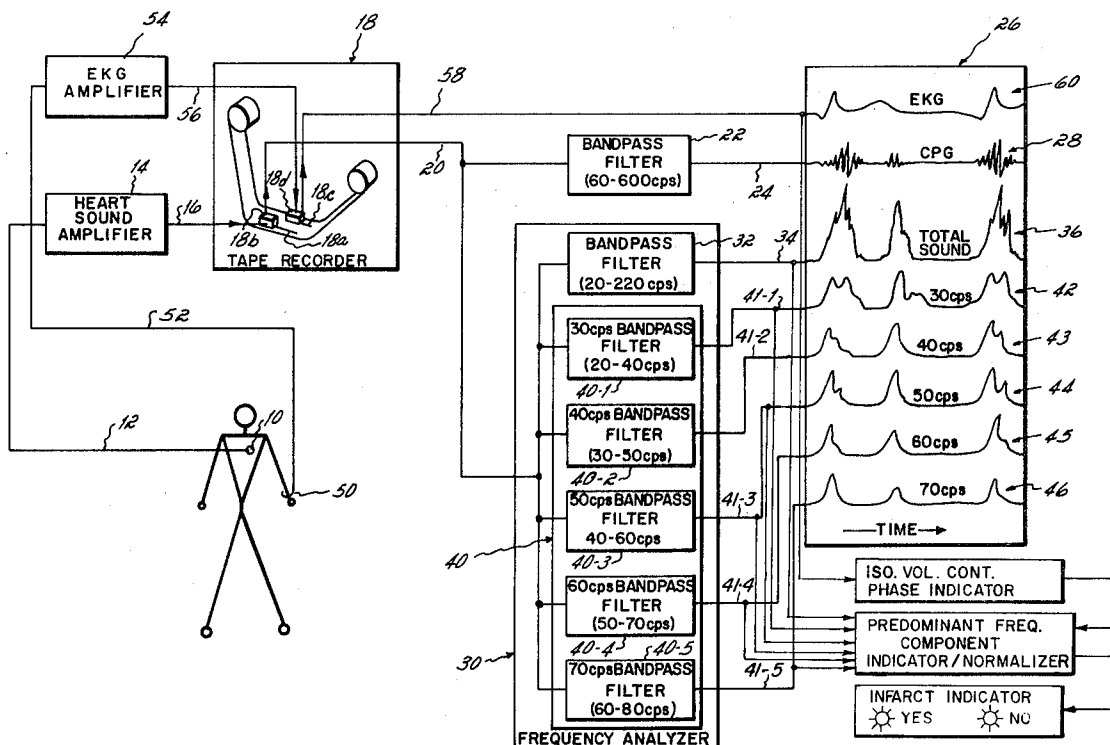
Attorney, Agent, or Firm—Wood, Herron & Evans

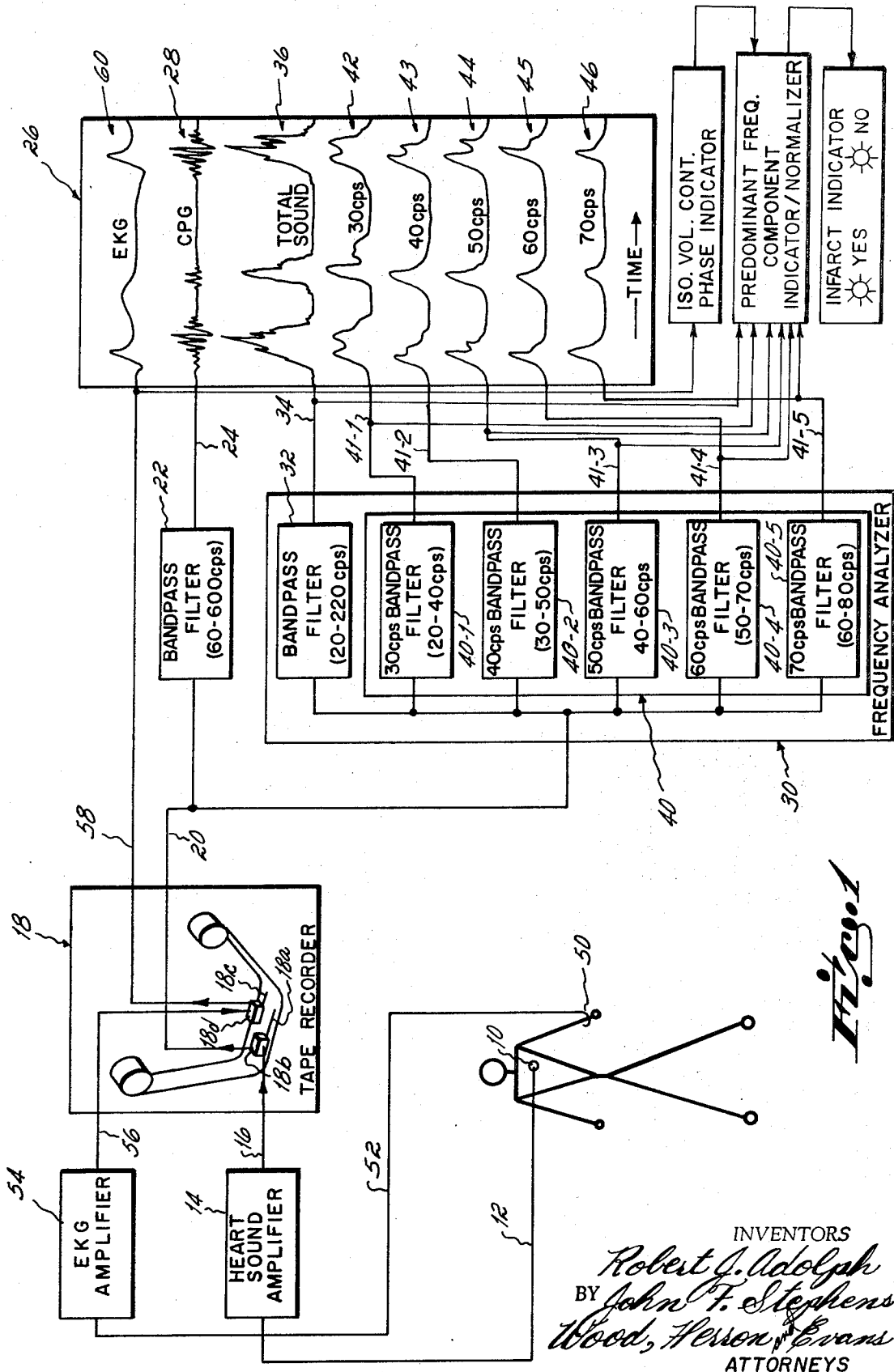
[57]

ABSTRACT

Apparatus and method for diagnosing myocardial infarction predicated on analysis of the acoustical frequency spectrum of the human heart during the isovolumic contraction phase of the cardiac cycle for the purpose of detecting a shift in the predominant frequency component of the spectrum from a 30-50 cps frequency band, typical of a noninfarcted heart, to a 20-40 cps frequency band, indicative of an infarcted heart.

10 Claims, 11 Drawing Figures





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Fig. 1

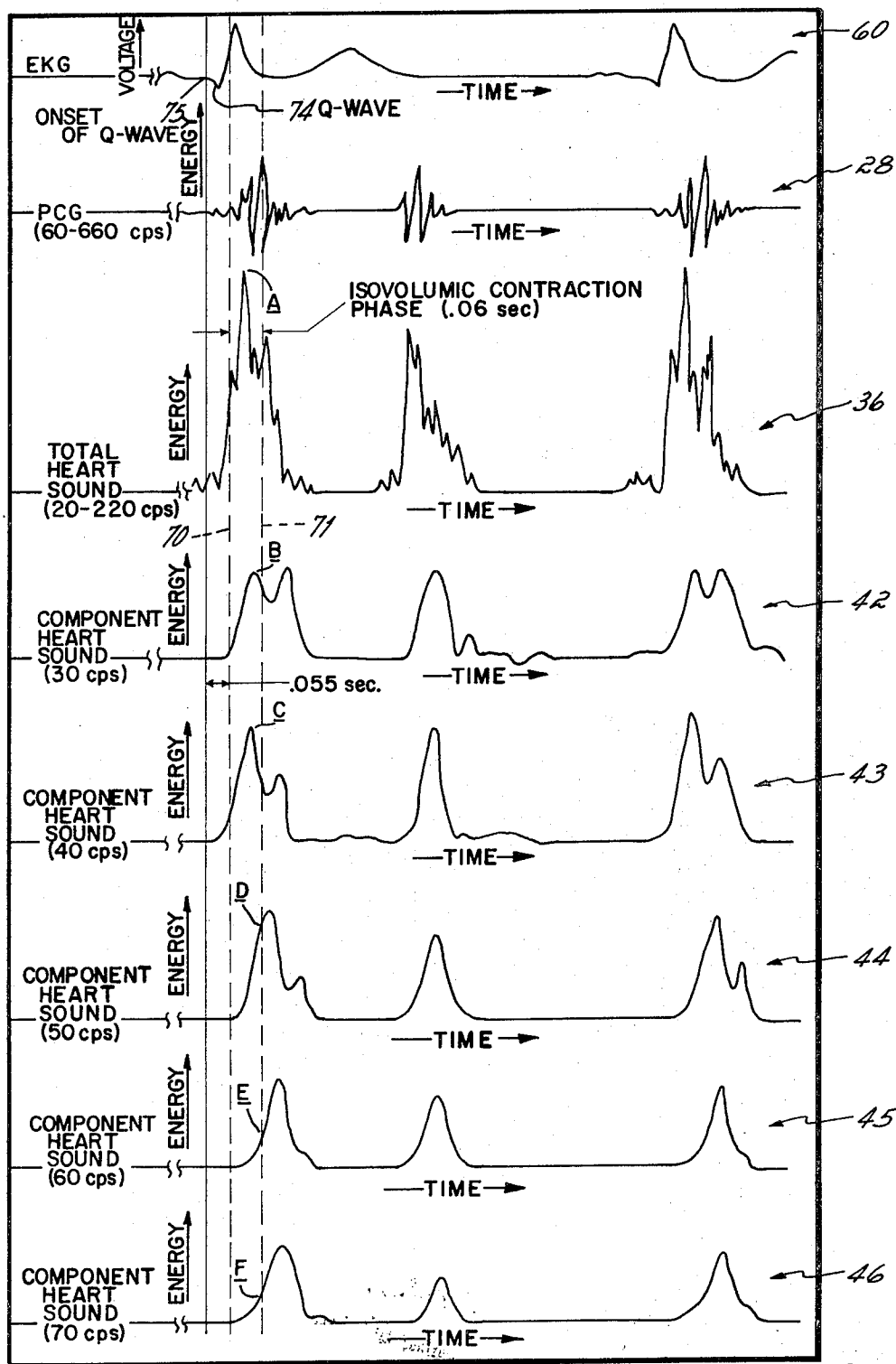


Fig. 2

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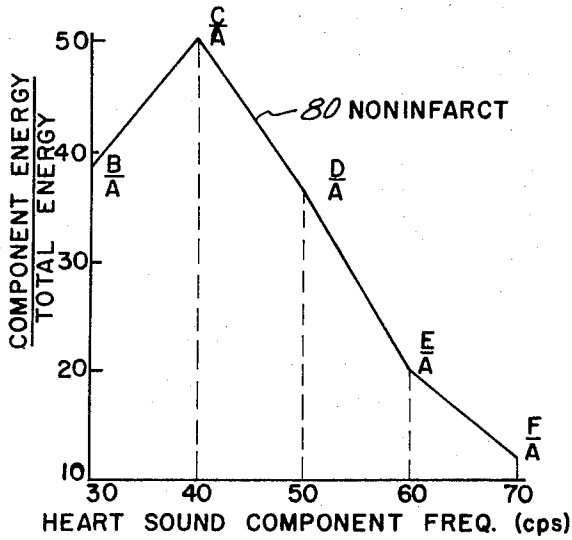


Fig. 3

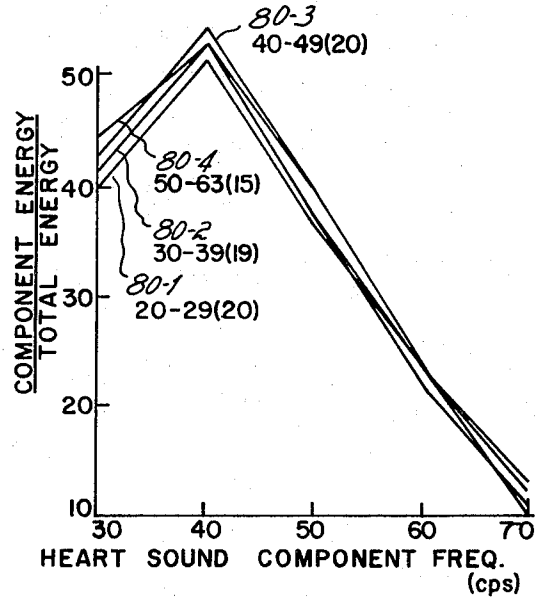


Fig. 4

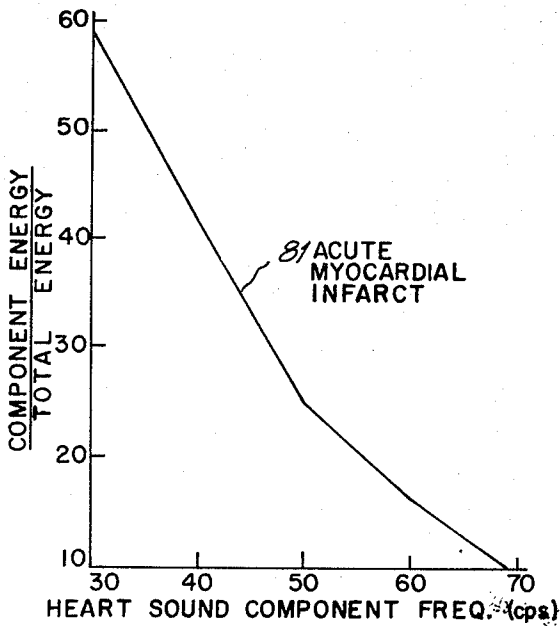


Fig. 5

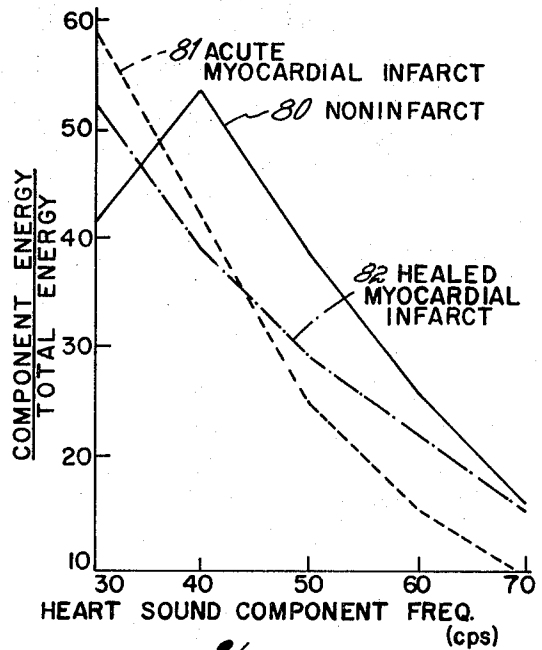


Fig. 6

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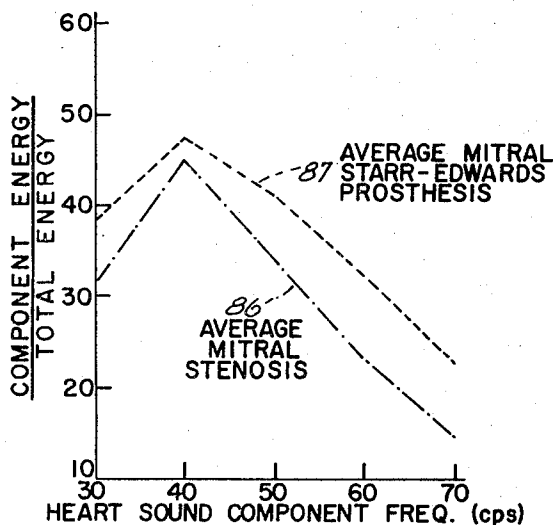


Fig. 1A

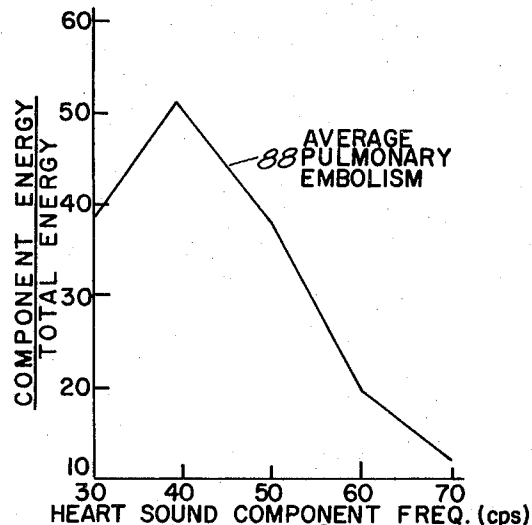


Fig. 1B

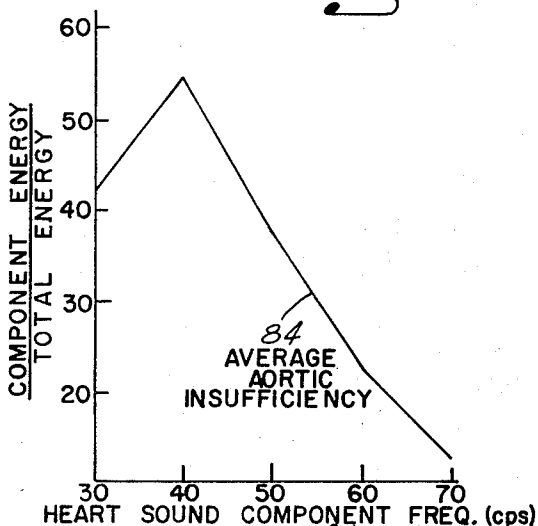


Fig. 1C

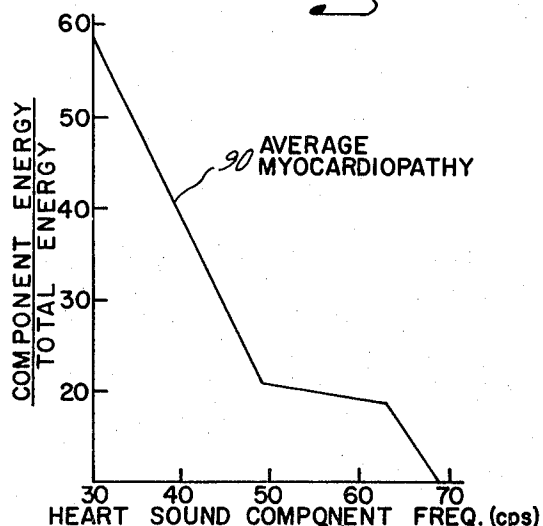


Fig. 1D

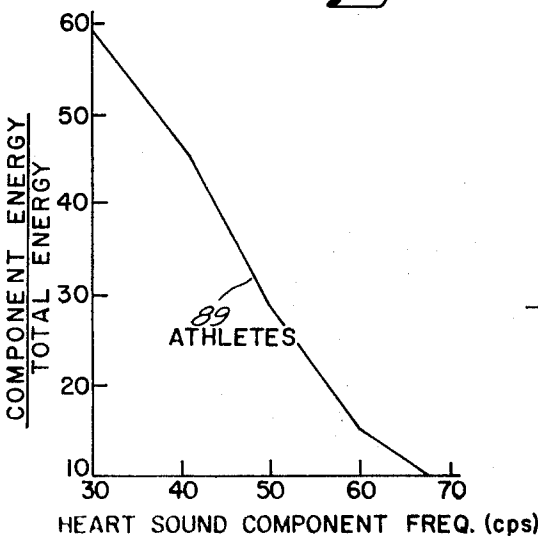


Fig. 1E

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METHOD AND APPARATUS FOR DIAGNOSING MYOCARDIAL INFARCTION IN HUMAN HEART

This is a continuation of application Ser. No. 44,281, filed June 8, 1970, now abandoned in the names of Robert J. Adolph et al.

The invention described herein was made in the course of work under a grant or award from the Department of Health, Education, and Welfare.

This invention relates to a method and apparatus for diagnosing disorders of the human heart, and more particularly to a method and apparatus for diagnosing myocardial infarction in the human heart.

To facilitate a more complete understanding of this invention, particularly the problem solved and the preferred method and apparatus of this invention used to solve it, knowledge of the anatomy and dynamics of the human cardiovascular system is useful. The principal component of this system is the heart which is a thin-walled, multi-chambered conical organ located slightly to the left of the center of the chest, with its apex directed downwardly and to the left. The heart wall consists of an inner layer known as the endocardium; an intermediate layer constituting the heart muscle, or myocardium; and an outer layer termed the epicardium or visceral pericardium. Surrounding the heart is the pericardium. The pericardium is divided into two parts or sub-layers, namely, an inner lining, or visceral pericardium, and an outer loose sac in which the heart is suspended, termed the parietal pericardium. Between the sublayers of the outer layer, or pericardium, exists a small amount of lubricating fluid.

The interior of the heart is divided into four chambers, namely, the right atrium, right ventricle, left ventricle, and left atrium. The right and left atria serve as receptacles for blood returning to the heart. Specifically, the right atrium serves as a receptacle for unoxygenated, or venous, blood returning to the heart from the body, while the left atrium serves as a receptacle for oxygenated, or arterial, blood returning to the heart from the lungs via the pulmonary veins. The right and left ventricles, which consist of several layers of interlacing muscle or myocardium, serve as the primary pumps of the heart. More particularly, the right ventricle pumps unoxygenated blood, which is returned from the body and temporarily stored in the right atrium, to the lungs via the pulmonary arteries whereat the blood is oxygenated. Interconnecting the right atrium and right ventricle is the tricuspid valve. The left ventricle pumps oxygenated, or arterial, blood, which temporarily stored in the left atrium following oxygenation in the lungs, to the body via the aorta. The mitral valve interconnects the left atrium and left ventricle, while the aortic valve interconnects the aorta and the left ventricle. A portion of the oxygenated, or arterial, blood pumped by the left ventricle to the body via the aorta branches off to the coronary arteries, supplying oxygenated blood to the myocardium, that is, to the major portion of the heart constituting the heart muscle.

From the standpoint of flow, unoxygenated blood enters the right atrium from the body. This unoxygenated blood then flows into the right ventricle through the tricuspid valve. From the right ventricle, the blood, which is still unoxygenated, is pumped to the lungs via the pulmonary artery. In the lungs the blood is oxygenated, following which it is returned via the pulmonary veins to the left atrium. The oxygenated blood stored in the left atrium flows into the left ventricle through the mi-

tral valve. It is then pumped by the left ventricle through the aortic valve to the aorta and thence to the body. Part of the blood in the aorta branches off to the coronary arteries which in turn supply the heart muscle, or myocardium, with oxygenated blood.

An occlusion, or blockage, of the coronary arteries may cause a myocardial infarction, commonly known as a coronary heart attack. Such an infarction will result if the blockage produces sufficient hypoxia, that is, lack of oxygen, of the myocardium to cause necrosis, or death, of myocardium cells.

The sequence of heart activity characterizing the pumping of blood by the heart is known as the cardiac cycle, and classically is divided into four phases. These phases are the filling, isovolumic contraction, ejection, and relaxation. Both the left and right sides of the heart function with this four-phase sequence, although not synchronously. A myocardial infarction is by-and-large a left side malady. Hence, the left side of the heart is of principal interest to an understanding of this invention, and accordingly the four phases of the cardiac cycle are considered only with reference to the left side. However, it will be understood that the right side operates similarly.

Left Ventricle Filling Phase (mitral valve open; aortic valve closed) — After relaxation of the left ventricle, the pressure of the oxygenated blood in the left atrium exceeds that in the left ventricle, permitting the mitral valve interconnecting them to open. This initiates rapid filling of the left ventricle with oxygenated blood. The filling becomes less rapid as left ventricular pressure rises due to increased elasticity of the left ventricular myocardium. Near the end of this period of slow filling, the left atrium contracts, "topping-off" the filling of the left ventricle with oxygenated blood. This filling phase ends abruptly upon initial contraction of the left ventricle, which causes the pressure in the left ventricle to exceed the pressure in the left atrium. When this occurs the mitral valve between the left atrium and left ventricle closes.

Left Ventricular Isovolumic Contraction Phase (mitral and aortic valves closed) — At the beginning of this phase the left ventricle is a closed chamber since both the mitral and aortic valves are closed. Throughout this phase the myocardium is contracting, increasing ventricular wall tension, and in turn increasing intraventricular pressure until it slightly exceeds the pressure in the aorta.

Left Ventricular Ejection Phase (mitral valve closed; aortic valve open) — The excess of pressure in the left ventricle relative to the aorta, caused by the continued contraction of the left ventricle, is effective to open the aortic valve. When this occurs, blood in the left ventricle is ejected rapidly into the aorta for distribution to the body. Midway through the ejection phase, contraction of the left ventricle ceases. Fluid inertia continues the ejection from the left ventricle for a portion of this ejection period. The intraventricular pressure eventually drops far enough below the pressure in the aorta to permit the aortic valve to close.

Relaxation Phase (aortic valve closed; mitral valve open) — As the myocardium relaxes, intraventricular pressure continues to drop until it falls below the pressure of blood in the left atrium at which time the mitral valve opens and the cardiac cycle is repeated.

As noted earlier, blood which has been oxygenated in the lungs, temporarily stored in the left atrium, and

pumped by the left ventricle, is transmitted to the body via the aorta. Part of the flow through the aorta branches off to the myocardium via the coronary arteries. If an occlusion or blockage of the coronary arteries or their branches occurs, and the occlusion is sufficient in duration and extent, the cells of the myocardium, or heart muscles, surrounding the left ventricle are deprived of the necessary oxygen, and die. Death of myocardium cells due to oxygen starvation occasioned by coronary artery blockage is a myocardial infarction, commonly termed a coronary heart attack.

Diagnosis and treatment of a myocardial infarction is compounded by two problems. First, many of the symptoms of a myocardial infarction, such as crushing chest pain, shortness of breath and will, cold skin, sweaty pallor, rapid but weak pulse, abnormally low blood pressure, abnormal cardiac sounds, etc., are also common to other ailments. For example, many of the foregoing symptoms of myocardial infarction are also present in patients having acute pericarditis (inflammation of membrane surrounding the heart), pulmonary embolism (lung clot), pneumothorax (air imprisoned in pleura around lungs), acute pancreatitis (inflammation of pancreas), dissecting aortic aneurysm (escape of blood into aorta wall), etc. This similarity of myocardial infarction symptoms to those of a number of other ailments tends to obscure the real cause, and obviously is a source of difficulty to the diagnostician. A further problem inheres in the fact that approximately 50 percent of the patients who succumb from a myocardial infarction do so within the first 45-60 minutes after the onset of symptoms. Thus, if treatment of a myocardial infarction is to be initiated in time to afford the patient a chance for survival, the diagnosis, often elusive for the reasons noted above, must be made with little or no delay after the onset of symptoms.

To facilitate making a correct diagnosis of myocardial infarction and to do so without unnecessary delay, a number of tests and techniques have been proposed as a supplement to the often nonconclusive evaluation of the patient's symptoms. Principal among these is subjecting the patient to an electrocardiogram (EKG). The EKG provides information regarding the electrical activity of the heart which, when interpreted by a skilled cardiologist, often provides the necessary additional information to enable the diagnostician to distinguish a myocardial infarction from other ailments having similar symptoms. Use of the EKG as a major diagnostic tool, however, has one principal disadvantage. In a significant percentage of patients, an EKG taken within 4-8 hours after the onset of symptoms is nonspecific, that is, does not provide the diagnostician with conclusive evidence of a myocardial infarction. The other diagnostic tools which have been proposed for facilitating diagnosis of myocardial infarctions are frequently unsatisfactory. For example, some provide indications which, while abnormal, are nondiagnostic to myocardial infarctions as is the case with conventional phonocardiograms, X-rays, laboratory studies, and the like.

Accordingly, it has been an objective of this invention to provide a method and apparatus for distinguishing a myocardial infarction from other ailments having similar symptoms, and for doing so immediately after the onset of symptoms, and yet without unnecessarily discomforting or jeopardizing the patient's health by blood vessel instrumentation or the like. This objective

has been accomplished in accordance with certain of the principles of this invention by utilization of a fundamentally different approach to the diagnosis of myocardial infarctions which is predicated on the discovery that the frequency spectrum of sounds emanating from the heart undergoes a characteristic and immediate change when a person suffers an acute myocardial infarction. More specifically, this invention is predicated on the discovery that following an acute myocardial infarction there is an immediate downward frequency shift in the dominant component of the heart sound frequency spectrum, believed due to the decreased elasticity of the left ventricular myocardium occasioned by the infarction. By examining the heart sound frequency spectrum for such downward shift in dominant frequency component, it is possible to diagnose myocardial infarctions. Since the frequency shift occurs substantially concurrently with the infarction, a diagnosis can be made immediately after the infarction when it is still early enough for treatment. Additionally, since the diagnosis involves analysis of sounds, a microphone pick-up secured to the chest can be used, obviating the need for discomforting and hazardous instrumentation of blood vessels and the like.

In a preferred form of the invention, analysis of the heart sound frequency spectrum for the downward shift in frequency of the predominant frequency component is made only during the isovolumic phase of the cardiac cycle, greatly simplifying the analysis. During this phase of the cardiac cycle the mass of the left ventricular complex is substantially constant due to the fact that both the mitral and aortic valves are closed. Since frequency and mass are related by the equation $f \propto e/m$ where f is the natural frequencies of the left ventricle, m is combined ventricular mass (left ventricular muscle plus contained blood), and e is myocardial elasticity, by analyzing the heart sound frequency spectrum during the constant mass, isovolumic contraction phase of the cardiac cycle, the effect on frequency of changing mass is eliminated and mass compensation therefor unnecessary. Observed shifts in dominant frequency are thus attributable solely to a change in the elasticity of the myocardium, and, hence, properly considered indicative of a myocardial infarction.

These and other advantages of this invention will become more readily apparent from a detailed description of the invention taken in conjunction with the drawings in which:

FIG. 1 is a schematic circuit diagram of a preferred apparatus embodiment constructed in accordance with the principles of this invention.

FIG. 2 is an enlarged view of the photographic record depicted in FIG. 1 and produced by the photographic recorder.

FIG. 3 is a plot, versus time, of the ratio of frequency component energy to total heart sound energy, for a patient having a noninfarcted heart.

FIG. 4 are plots, versus time, of the ratio of frequency component energy to total heart sound energy, for patients in different age groups having noninfarcted hearts.

FIG. 5 is a plot, versus time, of the ratio of frequency component energy to total heart sound energy, for patients having acute myocardial infarctions.

FIG. 6 are plots, versus time, of the ratio of frequency component energy to total heart sound energy, for

noninfarctions, acute infarctions, and healed infarctions.

FIG. 7A are plots, versus time, of the ratio of frequency component energy to total heart sound energy, for patients having isolated mitral stenosis and mitral Starr-Edwards prosthesis.

FIG. 7B is a plot, versus time, of the ratio of frequency component energy to total heart sound energy, for patients having pulmonary embolism.

FIG. 7C is a plot, versus time, of the ratio of frequency component energy to total heart sound energy, for patients having aortic insufficiency.

FIG. 7D is a plot, versus time, of the ratio of frequency component energy to total heart sound energy, for patients having myocardiopathies.

FIG. 7E is a plot, versus time, of the ratio of frequency component energy to total heart sound energy, for well-conditioned athletes.

Referring to FIG. 1, a preferred embodiment of the apparatus of this invention is seen to include an acoustical transducer, or microphone, 10 which in use is secured to the chest of the patient to facilitate monitoring of the sounds emanating from the heart. As previously noted, myocardial infarction is almost exclusively an ailment of the left ventricle. As such, heart sounds emanating from the left ventricle are of principal interest, and accordingly the microphone 10 is preferably placed relative to the patient's chest such as to pick up heart sounds emanating from the left ventricle. It has been found that proper location of the transducer for pick up of left ventricular sounds, known as the point of maximal impulse on the chest, is above the apex and antero-septal wall of the left ventricle.

It is generally known that sounds emanating from the heart can be divided into two categories. In the first category are sounds in the range of 0.1-20 cycles per second (cps). Such sounds are attributable to precordial movements, that is, gross movements of the chest wall. In the second category are sounds in the range of 20-1,000 cps. These sounds are audible, and generally associated with cardiac auscultation. In accordance with the principles of this invention, the heart sounds which are of interest to this invention are those in the second group, i.e., the group covering the frequency range of 20-1,000 cps, and herein are termed "heart sounds."

It has also been found that 95 percent of heart sound energy is below 100 cps. Accordingly, the microphone 10 preferably has a frequency response which is flat over at least the range of 20 cps — 100 cps. However, for reasons which will become apparent hereafter, the transducer 10 preferably has a flat response over a greater range at least including 20-600 cps. A transducer 10 found to be satisfactory in operation is one marketed by the Sanborn Division of Hewlett-Packard Corp., Waltham, Mass., designated Model 350-1700-C10, having a flat frequency response over the range of 20 cps — 1,000 cps.

To facilitate securing the transducer 10 to the patient's chest, an elastic belt or strap (not shown) to which the transducer is secured can be used. The strap is positioned to encircle the patient's body in the vicinity of the heart with the transducer 10 between the strap and patient's chest. The strap tension is then adjusted to insure good sound transmission between the patient's chest, and hence, heart and the transducer.

Strap tension should not be too large, otherwise low frequency sounds will be undesirably damped.

The output of the microphone 10 is connected via line 12 to the input of an audio amplifier 14. The amplifier 14 amplifies the transduced heart sounds on line 12 to a useful level, providing an amplified audio electrical output signal on line 16. The amplifier 14 preferably has a frequency response which is flat at least over the range of 20-600 cps. An amplifier, Model 350-1700-B, marketed by the Sanborn Division of Hewlett-Packard Corp., was found to operate satisfactorily.

A magnetic tape recorder 18 is connected to the output line 16 of the amplifier 14, and functions to produce a permanent record of the amplified microphone output signals. A recorder, Model SX-700, marketed by Crown International, Inc. of Elkhart, Indiana, has been found to operate satisfactorily. Such a tape recorder has a frequency response of 20-25,000 cps at 7-1/2 inches per second tape recording speed, a signal/noise ratio of -55db, and flutter and wow specifications of 0.09 percent. The tape recorder 18 preferably is a two-track recorder and on one tape track 18a stores in amplitude modulated form the amplified output signal of the microphone 10 which is present on line 16. The tape recorder transducer 18b associated with the magnetic tape track 18a on which the amplified heart sounds are recorded is connected to an output line 20. Use of the tape recorder 18 permits a patient's heart sounds to be permanently recorded for subsequent analysis at the convenience of the diagnostician. It also eliminates the need for transporting to the patient the electronic equipment, to be described later, which facilitates analysis of the heart sounds as is necessary for diagnosis.

Output line 20 of the recorder 18 is input to a frequency spectrum analyzer 30. The analyzer 30 includes a bandpass filter 32 having a pass band of 20-220 cps. The output of bandpass filter 32 present on line 34 is input to a multi-channel photographic recorder 26 which functions to provide, in response to the input on line 34, a permanent visual representation or trace 36, referenced to time, of the intensity of the heart sound energy present in the range of 20-220 cps. Since 95 percent of heart sound energy has been found to lie below 100 cps, the upper limit of the pass band of the filter 32 may be reduced 100 cps if desired. A photographic recorder satisfactory for use is the Oscillographic Recording System Console, Series 560, marketed by the Sanborn Division of Hewlett-Packard Corp., preferably operated at a paper speed of 100 mm per second.

The recorded output of the microphone 10, present on output line 20 of tape recorder 18 and which is input to the dynamic analyzer 30, is also passed through a bandpass filter 40. Filter 40 has a pass band 20 cps wide which can be selectively centered at 30 cps, 40 cps, 50 cps, 60 cps, and 70 cps to produce electrical outputs correlated to the sonic energy of the heart sounds in the bands of 20-40 cps, 30-50 cps, 40-60 cps, 50-70 cps, and 60-80 cps, respectively. For the purpose of clarity, the single, selectively variable center frequency, 20 cps wide bandpass filter 40 is depicted as comprising five separate 20 cps wide bandpass filters 40-1, 40-2, 40-3, 40-4 and 40-5 centered at 30, 40, 50, 60 and 70 cps, respectively, and having output lines 41-1, 41-2, 41-3, 41-4, and 41-5, respectively. Output lines 41-1, 41-2, 41-3, 41-4, and 41-5 are input to the photographic re-

corder 26, providing visual representations or traces 42, 43, 44, 45, and 46 of the intensity versus time of heart sound energy in the ranges of 20-40 cps, 30-50 cps, 40-60 cps, 50-70 cps, and 60-80 cps, respectively. Analyzer 30 is preferably of the type marketed by Spectral Dynamics Corp., San Diego, Calif., designated Model SD-101A. Analyzers of this type heterodyne the input signal and then operate on a center frequency offset principle to render the filter time constant independent of the center frequency of the bandpass filter 40.

For the purpose of time referencing the visual traces 36 and 42-46 to the various phases of the cardiac cycle, particularly the isovolumic contraction phase, an electrocardiogram (EKG) is made. The apparatus for making the electrocardiogram is conventional, and includes suitable electrodes indicated schematically by reference numeral 50 which are attached to the patient in a manner well known to those skilled in the field of cardiology. The EKG output of the electrodes 50 is connected via a line 52 to the input of a high-gain amplifier 54 which amplifies the EKG electrode signals present on line 52 to a suitable level, providing amplified EKG electrode signals on line 56. The amplifier 54 is preferably of the type marketed by the Sanborn Division of the Hewlett-Packard Corp., designated Model 350-2700-C. The output of the amplifier 54 on line 56 is input to the tape recorder 18 where it is recorded in frequency modulated format on a second tape track 18C. To accomplish conversion of the amplified EKG electrode signal on line 50 to frequency modulated form a conventional FM recording adapter is utilized. Such adapters are commercially available from A. R. Vetter Company, Rebersburg, Pennsylvania, designated Model 2, having a frequency response of 0-1,000 cps. The output of the tape recorder transducer 18d associated with the magnetic tape track 18C on which the EKG signal is recorded is connected to a tape recorder output line 58. Output line 58 in turn is input to the phonographic recorder 26 to produce a conventional visual EKG trace 60.

It has been discovered that when a patient has a myocardial infarction the predominant frequency component of the heart sound frequency spectrum shifts from a 30-50 cps band to a 20-40 cps band. This downshift in frequency of the predominant heart sound components is believed caused by the decreased elasticity of the left ventricle occasioned by infarction of the left ventricular myocardium. As earlier set forth, the heart sound frequency is believed related to myocardium elasticity by the equation

$$fae/m$$

where f is left ventricular natural frequencies, e is elasticity of the left ventricular myocardium, and m is the combined mass of the left ventricular complex and the blood contained therein. In a myocardial infarction the elasticity of the infarcted myocardium is reduced due to necrosis of the myocardium cells. Such reduction in elasticity reduces the frequency of the heart sounds emanating from the left ventricular complex, as can be noted by reference to the above equation. Specifically, it has been discovered that the predominant frequency component shifts from a 30-50 cps band to a 20-40 cps band.

In one preferred form of this invention, the frequency spectrum of the heart sounds of a patient is analyzed to

determine the frequency of the predominant component by measuring, at predetermined frequency intervals of the spectrum, the ratio of (a) the acoustical energy at said predetermined frequencies to (b) the total acoustical energy of the heart sound spectrum. This technique of using the ratio of (a) the energy at a given frequency to (b) the total energy has the advantage of normalizing the frequency measurements. By comparing the ratios to each other, it is then possible to conveniently determine the predominant frequency component of the heart sound frequency spectrum. If the predominant frequency component of the heart sound spectrum is in the neighborhood of the 30-50 cps band, the patient is deemed not to have had a myocardial infarction, i.e., the myocardium elasticity is normal due to the absence of infarcts. If the predominant frequency component of the heart sound spectrum is in the neighborhood of the 20-40 cps band, the patient is determined to have had a myocardial infarction, i.e., myocardium elasticity is abnormally low due to the presence of infarcts, unless the existence of the 20-40 cps band predominant frequency component can be justified on grounds other than the existence of a myocardial infarction. Illustrative of instances where the predominant frequency component of the heart sound spectrum is found to be in the 20-40 cps band, and yet the individual has not had a myocardial infarction, are (a) cases where the mass of the left ventricular complex is increased with no increase in elasticity which often occurs with very well-conditioned athletes, and (b) cases where the elasticity and mass of the left ventricular myocardium are reduced and increased, respectively, as is prevalent with cardiomyopathy.

As noted earlier, in a myocardial infarction cells in the left ventricular myocardium die. The deadened regions of the myocardium are termed "infarcted areas." Due to the presence of infarcted areas in the myocardium, the elasticity of the myocardium is reduced. The natural frequencies of the left ventricle, f , are related to the combined mass, m , of the left ventricular myocardium and blood contained therein and the elasticity of the left ventricular myocardium, e , by the equation fae/m . Since the mass of blood contained in the left ventricle varies during the cardiac cycle, it is desirable to analyze the frequency spectrum of the heart sounds at a point in the cardiac cycle when the combined mass of the left ventricular complex and contained blood is constant, namely, during the isovolumic contraction phase. In this way, observed changes in frequency of the predominant component of the heart sound spectrum due to a myocardial infarction are dependent solely upon the changes in the elasticity of the left ventricular myocardium occasioned by the infarcted areas thereof, the interdependence of frequency and mass having been eliminated by virtue of analysis during the isovolumic contraction phase of the cardiac cycle when the mass is constant.

With reference to FIG. 2, which is an enlargement of the photographic recorder 26 of FIG. 1, the isovolumic contraction phase of the cardiac cycle relative to the frequency component traces 42-46, the total heart sound trace 36, and the EKG trace 60 is depicted. Specifically, dotted vertical lines 70 and 71 represent the beginning and end, respectively, of the isovolumic contraction phase of the cardiac cycle. Line 70 representing the beginning of the isovolumic contraction phase is located by first locating the onset 75 of the Q-wave

74 in the EKG trace 60. From well known correlations of EKG traces to the cardiac cycle, the beginning 70 of the isovolumic contraction phase is known to occur approximately 0.055 seconds after the onset 75 of the EKG Q-wave 74. Also known is that the isovolumic contraction phase has a duration of approximately 0.06 seconds. Thus, the end 71 of the isovolumic contraction phase is easily located by measuring 0.06 seconds on the time scale from line 70 representing the beginning of the isovolumic contraction phase, locating the end 71 of the phase at a point 0.115 seconds after the onset 75 of the EKG Q-wave 74. Location of the isovolumic contraction phase on an EKG trace is a routine task for a skilled cardiologist, and further discussion herein of the technique is unnecessary. It is noted at this point only that the various traces of FIG. 2 are synchronized with respect to the time axis because the electrical signals from which the traces are derived were obtained from the patient concurrently and stored on tracks of the same magnetic tape to retain their synchronism.

A convenient index of the total sound energy present during the isovolumic contraction phase is the peak value of the total heart sound trace 36 occurring during the isovolumic contraction phase and which is indicated as point A on FIG. 2. An index of the sound energy in the 20 cps wide band centered at 30 cps during the isovolumic contraction phase, referred to as the 30 cps frequency component, is conveniently taken as the peak value of the 30 cps frequency component trace 42 occurring during the isovolumic contraction interval and which is indicated as point B. In a similar fashion convenient indices of the sound energy in the 20 cps wide frequency bands centered at 40 cps, 50 cps, 60 cps and 70 cps, herein referred to as the 40 cps, 50 cps, 60 cps and 70 cps frequency components, are the peak values of the 40 cps, 50 cps, 60 cps, and 70 cps traces 43, 44, 45 and 46 occurring during the isovolumic contraction phase, which peaks are designated on FIG. 2 as points C, D, E and F.

To facilitate convenient visual analysis of the frequency spectrum of the heart sound, and particularly of the predominant frequency component, a plot is made of the ratios of (a) the 30 cps, 40 cps, 50 cps, 60 cps and 70 cps frequency component peak values, designated points B, C, D, E and F, occurring during the isovolumic contraction phase to (b) the peak value of the total heart sound, designated point A, occurring during the same period. With reference to FIG. 3, a plot 80 is shown of the ratio of (a) the peak amplitude of the 30 cps, 40 cps, 50 cps, 60 cps and 70 cps frequency components to (b) the peak amplitude of the total heart sound energy, the ratio being derived from the total sound and frequency component traces 42-46 of FIG. 2 during the isovolumic contraction phase of the cardiac cycle. Specifically, the ratio of energy at 30 cps to total sound energy, B/A, is approximately 0.38; the ratio of energy at 40 cps to total sound energy, C/A, is approximately 0.5; the ratio of energy at 50 cps to total sound energy, D/A, is 0.46; the ratio of energy at 60 cps to total energy, E/A, is 0.20; and the ratio of energy at 70 cps to total energy, F/A, is 0.03. The plot shape represented by the foregoing ratios is termed herein the "heart sound signature" of the patient in question.

The total sound trace 36 and frequency component traces 42-46 of FIG. 2 represent actual traces for a pa-

tient not having a myocardial infarction. Accordingly, the heart sound signature 80 of FIG. 3 derived from the traces 42-46 of FIG. 2, corresponds to a heart which does not have a myocardial infarction. It is significant that in such a heart the predominant frequency component of the heart sound is the 30-50 cps band.

When traces (not shown) of the general type referenced as traces 42-46 in FIG. 2 are generated for a person having an acute myocardial infarction, and a heart sound signature 81 as shown in FIG. 5 is made for the acutely infarcted heart, the predominant frequency component of the acutely infarcted heart signature 81 is observed to have shifted downwardly from the 30-50 cps band to the 20-40 cps band. Thus, it is apparent that the predominant heart sound frequency component for a noninfarcted heart is the 30-50 cps band, whereas the predominant frequency for an acutely infarcted heart is the 20-40 cps band. Accordingly, by determining the predominant frequency component of the heart sound spectrum, it is possible to ascertain whether a person has an acute myocardial infarction.

It has been found that the heart sound signature for patients with noninfarcted hearts is substantially independent of patients' age. FIG. 4 depicts average heart sound signatures 80-1, 80-2, 80-3 and 80-4 for 20, 19, 20 and 15 patients, respectively, in age groups 20-29 years, 30-39 years, 40-49 years, and 50-63 years, respectively. By reference to FIG. 4 it is noted that signatures 80-1, 80-2, 80-3 and 80-4 are substantially identical in shape with a predominant frequency component at the 30-50 cps band. It has also been found that the heart sound signature for patients with non-infarcted hearts is independent of the patients' body build (mesomorph, ectomorph, or endomorph); and independent of the patients' heart rate (60-110 cycles/minute).

With reference to FIG. 6 which is a plot of heart sound signatures similar to FIG. 5, it is noted that the average heart sound signature 81 for acute myocardial infarcts is very similar to the average heart sound signature 82 for healed myocardial infarcts, both having a predominant frequency at the 20-40 cps band. Whereas, the noninfarcted heart has an average heart sound signature 80 with a predominant frequency at 30-50 cps band. Thus, myocardial infarctions, whether healed or acute, are distinguishable from noninfarcted hearts using the method and apparatus of this invention.

With reference to FIG. 7A, it is noted that patients with isolated mitral stenosis or mitral Starr-Edwards prosthetic valves have average heart sound signatures 86 and 87, respectively, with predominant frequency components at 30-50 cps band, which conform closely to the noninfarcted heart sound signature 80 (FIG. 3). This is to be expected because both of these maladies involve heart valve structures per se, and not heart mass/elasticity changes. The normality of the heart sound signature for mitral stenosis and Starr-Edwards valves confirms the theoretical basis of the invention herein, namely, that the nature of the heart sound signature is predicated on a mass/elasticity phenomenon and not on other sources of vibrations such as vibrating heart valve elements. Were the basis of the heart sound signature to be found in valve vibration, the heart sound signature for patients with mitral stenosis and Starr-Edwards valves, both heart valve abnormalities, would not have been normal. The normal nature of the

heart sound signature in these two types of patients also demonstrates that the method and apparatus of this invention is capable of distinguishing myocardial infarction from valve abnormalities not affecting heart mass/elasticity. In practice, however, such can be done by a skilled clinician without resort to heart sound signature analysis.

With reference to FIG. 7B, showing the average heart sound signature 88 of persons having massive pulmonary embolisms, it is noted that the heart sound signature 88, having a predominant frequency component at 30–50 cps band, conforms to the signature 80 (FIG. 3) of the noninfarcted heart and, hence, the method and apparatus of this invention is capable of distinguishing between patients with myocardial infarction and those with pulmonary embolism.

With reference to FIG. 7C, which shows an average heart sound signature 84 for patients having aortic insufficiency, it is noted that the predominant heart sound frequency component is the 30–50 cps band. Such is to be expected since in aortic insufficiency both heart mass and heart elasticity increase, tending to cause no change in heart sound frequency which is governed by the relation $f \propto \sqrt{m}$. The normality of the heart sound signature for aortic insufficiency also establishes that such can be distinguished from a myocardial infarction using the method and apparatus of this invention, although resort to the techniques of this invention in such cases is ordinarily unnecessary for a skilled cardiologist.

Two types of false positive situations have been found when the method and apparatus of this invention is used. Specifically, with reference to FIG. 7E showing the average heart sound signature 89 of very well-conditioned athletes, it has been observed that the heart sound signature 89 does not correspond to the signature 80 of the noninfarcted heart (FIG. 6) having a predominant frequency of 30–50 cps band, but rather more closely resembles the signatures 81 and 82 of the acutely and healed infarcted heart (FIG. 6) with a predominant frequency of 20–40 cps band. The 20–40 cps band predominant frequency component of the signature 89 of very well-conditioned athletes presumably represents the athletes' (a) abnormally large combined mass of the left ventricular complex and contained blood, coupled with (b) normal myocardial elasticity.

The second source of false positives has been found in patients with myocardial pathology. With reference to FIG. 7D showing the average heart sound signature 90 of the myocardial pathic patient, it is noted that the signature 90 resembles the signature 81 (FIG. 5) of the infarcted patient with a predominant frequency of 20–40 cps band. The myocardial pathic heart sound signature 90 presumably is due to the combination of (a) a loss of myocardial elasticity, coupled with (b) an increased combined left ventricular mass and contained blood due to an enlarged heart.

False positive diagnoses obtained using the method and apparatus of this invention in cases of myocardial pathies and well-conditioned athletes are discoverable using known diagnostic techniques such as the presence of persistent third and fourth sound gallops in the case of myocardial pathies, etc. Hence, such false positives do not significantly impair the utility of this invention.

If desired as a further means of checking for cardiac abnormalities, the diagnostician using the method and apparatus of this invention may produce a phonocardiogram (PCG) simultaneously with making the EKG trace 60 and total sound and frequency component traces 36 and 42–46. The PCG can be obtained by connecting the tape recorder output line 20 to the input of a bandpass filter 22 having a pass band of 60–600 cps. The output of the bandpass filter 22, which is present on line 24, is in turn input to the photographic recorder 26, producing a conventional PCG trace 28. The PCG trace 28 constitutes a plot of the intensity versus time of heart sounds in the range of 60–600 cps, and as indicated can be provided for the purpose of checking for abnormalities and extra sounds suggesting cardiovascular disease. The PCG, however, forms no part of this invention, and can be eliminated if desired.

If desired, the heart sound frequency spectrum can be analyzed with respect only to the intensity of heart sounds of the 20–40 cps band frequency component and the 30–50 cps band frequency component inasmuch as it is these two frequency components which change in a characteristic manner as a consequence of a myocardial infarction. The analysis of the heart sound frequency spectrum, if desired, can be automated by providing appropriate electrical circuitry designed in accordance with well-known techniques for automatically monitoring the 30–50 cps band frequency component and the 20–40 cps band frequency component and providing an output indicative of the frequency band of the predominant component. If the predominant frequency band component is the 30–50 cps band, the patient is deemed not to have had a myocardial infarction, whereas if it is 20–40 cps band, the patient is deemed to have had a myocardial infarction. Responsive to such output and associated with such circuitry would preferably be a suitable visual and/or audible indicating means for providing a visible and/or audible indication of the results of the predominant frequency component analysis. For example, a red lamp could be utilized to indicate a 20–40 cps band predominant frequency component typical of a myocardial infarction, while a green lamp could be used to indicate a 30–50 cps band predominant frequency component typical of a noninfarcted heart.

It is also possible to modify the preferred embodiment to utilize other approaches for determining the magnitude of acoustical energy of the various frequency components. For example, instead of utilizing the peak value of acoustic energy in a band as an indication of the magnitude of energy in the band, the integral of acoustic energy in the band could be utilized as an index of band energy. Also, in the preferred embodiment, the magnitudes of the heart sound frequency components are normalized by dividing each heart sound frequency component by the total heart sound energy. However, such normalization is not necessary in order to practice the method and apparatus of this invention. Finally, if desired, the analysis of the heart sound frequency components as an index of myocardial elasticity can be made during phases of the cardiac cycle other than the isovolumic contraction phase. If elasticity analysis is made during a different phase, suitable means should preferably be provided to determine the mass of the left ventricular complex and contained blood during the phase in question, thereby enabling

the elasticity analysis to be compensated for varying mass.

Having described the invention, we claim:

1. A method of diagnosing the condition of a living human heart to determine the probable existence of a myocardial infarction comprising the steps of:
 - monitoring the multiple frequency component sound emanating from the living heart in components centered approximately at 30 cps and 40 cps,
 - detecting the frequency component of said monitored components which predominates, and
 - indicating the probable existence of a myocardial infarction when the frequency of said predominant component is centered at approximately 30 cps.
2. A method of diagnosing the condition of a living human heart to determine the probable existence of a myocardial infarction comprising the steps of:
 - monitoring the multiple frequency sound emanating from the living heart in at least the range of frequencies lying below approximately 50 cps,
 - determining the occurrence of the isovolumic contraction phase of the cardiac cycle,
 - detecting the frequency component of said monitored sounds which predominates, in the range of frequencies below approximately 50 cps, during said isovolumic contraction phase of the cardiac cycle, and
 - indicating the probable existence of a myocardial infarction based on the frequency of said predominant component.
3. The method of claim 1 further including the step of determining the occurrence of the isovolumic contraction phase of the cardiac cycle, and wherein said detecting step includes detecting which of said components occurring during the isovolumic contraction phase of the cardiac cycle is predominant.
4. A method of diagnosing the condition of a living human heart to determine the probable existence of a myocardial infarction comprising the steps of:
 - monitoring the sounds emanating from the living heart,
 - measuring (a) the magnitude of said sound energy over a first specified frequency range including substantially all frequencies between approximately 20 cps and 220 cps, (b) the magnitude of sound energy at a first frequency band within said range and associated with an infarcted heart, said first frequency band including the frequency of approximately 30 cps, and (c) the magnitude of sound energy at a second frequency band within said range and associated with a noninfarcted heart, said second frequency band including the frequency of approximately 40 cps,
 - adjusting said second third mentioned magnitudes with said first mentioned magnitude to normalize said second and third mentioned magnitudes,
 - comparing said normalized magnitudes to determine which is predominant, and
 - indicating the probable existence of a myocardial infarction when said normalized second magnitude exceeds said normalized third magnitude.
5. The method of claim 4 wherein said measuring step includes measuring the magnitude of said sound in the range of at least 20 cps — 50 cps, the magnitude of said sound in a band centered at approximately 30 cps, and the magnitude of said sound in a band centered at approximately 40 cps, and wherein said indicating step

includes indicating the probable existence of a myocardial infarction when the normalized magnitude associated with said 30 cps band exceeds said normalized magnitude associated with said 40 cps band.

6. A method of diagnosing the condition of a living human heart to determine the probable existence of a myocardial infarction comprising the steps of:

transducing sounds emanating from the living heart,

concurrently recording on the same record member (a) transduced sounds emanating from the heart in at least the range of frequencies lying below approximately 50 cps and (b) electrocardiographic signals,

concurrently reproducing the recorded sound signals and electrocardiographic signals,

determining reproduced sound signals associated with the isovolumic contraction phase of the cardiac cycle using the reproduced electrocardiographic signals as a cardiac cycle phase reference,

measuring the predominant frequency component of the reproduced sound signals associated with the isovolumic contraction phase, and

indicating the probable existence of a myocardial infarction based on the frequency of said predominant component.

7. Apparatus for diagnosing the condition of a living human heart to determine the probable existence of a myocardial infarction comprising:

a transducer positionable in use in sound receiving relation to a living human heart for producing signals correlated to sounds emanating from the heart,

electrocardiographic means for producing an electrocardiographic signal, and

means responsive to said heart sound signals for measuring the relative magnitudes of said sound signals in frequency bands centered at approximately 30 cps and 40 cps and responsive to said electrocardiographic means for measuring the relative magnitudes of said 30 cps and 40 cps centered sound signal bands during the isovolumic contraction phase of the cardiac cycle for indicating the existence of a myocardial infarction based on the frequency of the predominant component band occurring during the isovolumic contraction phase of the cardiac cycle.

8. The apparatus of claim 7 wherein said measuring means includes recording means for producing a visually perceptible indication of the magnitude of said 30 cps and 40 cps centered sound signal bands during said isovolumic contraction phase.

9. Apparatus for diagnosing the condition of a living human heart to determine the probable existence of a myocardial infarction comprising:

a transducer positionable in use in sound receiving relation to the living human heart for producing signals correlated to sounds emanating from the heart,

electrocardiographic electrodes positionable in use to produce electrical signals correlated to electrical activity of the heart,

recording means for concurrently recording said sound and electrocardiographic signals,

reproducing means associated with said recording said sound and electrocardiographic signals,

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reproducing means associated with said recording means for reproducing said recorded sound and electrocardiographic signals,
indicating means responsive to said reproduced electrocardiographic signal to provide an indication of the isovolumic contraction phase of the cardiac cycle, and
means responsive to said indicating means and to said reproduced sound signals for detecting the predominant frequency component below approximately 50 cps of said sound signals occurring during the isovolumic contraction phase of the cardiac cycle.

10. Apparatus for diagnosing the condition of a living human heart to determine the probable existence of a myocardial infarction comprising:

a transducer positionable in use in sound receiving relation to the living human heart for producing signals correlated to multicomponent sounds emanating from the heart in a range at least including sounds below approximately 50 cps,

means for determining the isovolumic contraction phase of the cardiac cycle,
frequency selective means jointly responsive to said heart sound signals and to said isovolumic contraction phase determining means for measuring the relative magnitudes of said sound signals occurring during the isovolumic contraction phase of the cardiac cycle in a first frequency band which includes a first frequency below approximately 50 cps and in a second frequency band which includes a second frequency below said first frequency, and
indicating means for indicating the probable existence of a myocardial infarction when the signal magnitude associated with said second band exceeds the signal magnitude associated with said first band.

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