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ABSTRACT

A Study of Pulsation of Chest Wall Using Microwave Interferometer

by

Paul Zeno

The microwave interferometer is utilized to pick up vibrations of the anterior chest wall produced by the cardiac activity. The objective of this project is to find the causes of different peaks in the output of the microwave interferometer. The output of the microwave interferometer is recorded simultaneously with EKG and jugular pressure pulses. Eighteen subjects had their mechanical vibrations of the chest wall, EKG and jugular pressure pulses recorded. The processed interferometer data, when compared with EKG and jugular pressure pulses appear to have a close relation to the mechanical events in the cardiac cycle. Four peaks in the recorded output of the interferometer are identified by this comparison. Out of 18 subjects 15 confirmed this findings. All recordings are made from healthy male subjects, at normal heart rates. Further clinical studies are recommended for confirming these findings.

A STUDY OF
PULSATION OF CHEST WALL
USING MICROWAVE INTERFEROMETER

by
Paul Zeno

A Thesis
Submitted to the Faculty of
New Jersey Institute of Technology
in Partial Fulfillment of the Requirements for the Degree of
Master of Science

Biomedical Engineering

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A Study of

Pulsation of Chest Wall

Using Microwave Interferometer

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This thesis is dedicated to
Mary George

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CHAPTER 1
INTRODUCTION

1.1 Motivation

Movements of the heart and the great arteries are transmitted to the surrounding structures. When the pulsating organ is close enough to the chest wall it produce pulsations of the intercostal tissues as well as the bony structures. Low frequency movements of the thoracic wall can be investigated by recordings of a microwave interferometer. These records present a faithful picture of the movements of the chest wall. The recordings are somewhat complex, offering a multitude of waves. The recordings when correlated with EKG and jugular pressure pulses are of great interest.

1.2 Objective

The objective of this research is to find the causes of different peaks in the output of the interferometer, when used to record the pulsation of chest wall. The output of the interferometer are correlated with EKG and jugular pressure pulses to identify these peaks.

1.3 Mechanism of Pulsation of Chest Wall

Movements of the heart and the great arteries are transmitted to the surrounding structures. When the pulsating organ is close enough to the chest wall it may produce pulsations of the intercostal tissues as well as the bony structures. Changes in consistency of the myocardium, changes in shape and volume of the ventricles, and fall of intrathoracic pressure

associated with outflow of blood from the thorax seems to be involved in this pulsation.

Stiffening of the myocardium facilitates transmission of cardiac movements to the chest wall. When the blood is moved at the beginning of systole toward the atrioventricular valves, and later, during the ejection period into the great arteries, recoil of the heart takes place. This causes a movement of the ventricular cone directed forward and footward. This movement is enhanced by systolic stretching of the great arteries which allows the ventricular cone to move in a direction opposite to that of the blood stream in the great arteries.

Since the aorta is covered by a thick layer of lung, only large pulses are able to transmit their pulsations to the anterior chest wall. Systolic decrease in ventricular volume exerts an aspiratory effect upon the chest wall, due to drop of intrathoracic pressure which results from rapid outflow of blood from the chest.

CHAPTER 2
SYSTEM DESCRIPTION

2.1 Interferometer

The microwave interferometer was designed and constructed by Cornell Aeronautical Laboratories in 1967. This instrument operates on the principle of a phase-locked, coherent, lowpower radar. The phase-lock circuit uses a feedback gain of approximately 120,000. This instrument directly compares the phase of the signal reflected from the target with the phase of the signal illuminating the target. The phase comparison is done at an intermediate frequency, so as to reduce losses in sensitivity due to noise.

The microwave horn antenna of interferometer transmits a microwave of frequency 9.3 GHz, corresponding to a free-space wave length of 3.1 cm. This wave can pass through normal clothes and dry blankets and can reflect from the chest wall. The interferometer is theoretically capable of detecting .01 of a degree of carrier phase shift which corresponds to a target displacement of 0.08 microns.

The signal source is a reflex-klystron oscillator operating at 9.3 GHz. Most of the energy of this wave is fed through the main arm of a directional coupler to a 60 MHz. balanced modulator. The 60 MHz. for the balanced modulator is produced by a local oscillator. The output of the balanced modulator is passed through a single sideband filter to a microwave mixer-receiver. In the mixer it is introduced as a local oscillator input of frequency 9.3 GHz. + 60 MHz. A minor fraction of power from the side arm of the directional coupler is amplified in a travelling wave tube (TWT) amplifier and transmitted out through the transmitting horn antenna.

The transmitter horn antenna illuminates the chest wall of the subject with the 9.3 GHz wave and from the chest wall the wave is reflected back and is collected by receiving horn antenna. Any movement of the chest wall relative to the transmitter antenna produces a phase shift in the wave front reflected by the chest. Thus the reflected wave front has a phase shift directly proportional to the chest movement.

The receiver signal having the phase variation is mixed with the local oscillator signal of 9.3 GHz. + 60 MHz. frequency. The output of this mixer is a 60 MHz. intermediate frequency signal containing the phase information. This signal containing the phase variation is applied to a F-M receiver together with a 49.3 MHz. wave generated by a local oscillator. The output of the F-M receiver is a 10.7 MHz. signal with the phase information. This signal is amplified and is fed to a phase demodulator. Waves from 60 MHz. and 49.3 MHz. local oscillators are fed to a F-M receiver and the output 10.7 MHz. is amplified and is used as a reference wave in the phase demodulator. The output of this phase demodulator is a voltage whose amplitude is linearly proportional to the amount of phase information contained in the signal derived from the microwave receiver. This output voltage is then amplified and applied to the helix of the TWT phase shifter. TWT phase shifter instantly corrects the cause of phase shift by electrically "moving" the entire radar. This feed-back voltage to the helix of TWT is used to record the movement of chest wall. Therefore, the output of the microwave interferometer corresponds to the rate of change of position of the chest wall relative to the transmitting horn antenna.

2.2 DASH-16

DASH-16 is a high speed analog/digital I/O expansion board for the IBM personal computer. It turns the computer into a high speed data acquisition and signal analysis instrument. DASH-16 uses 12 bit successive approximation converter with a 25 microsecond conversion time. It provides a choice between 16 single ended channels or 8 differential channels. When operating in the 16 channel single-ended configuration the inputs are only suitable for "floating" sources. A floating signal source does not have any connection to ground at the signal source. If the signal source has one side connected to a local ground, the 8 channel differential configuration should be used. If there is a combination of floating and ground referred signal sources, the 8 channel differential configuration should be used and the floating signal should be connected with a jumper between LO and L.L. GND. The gain of the board can be selected by a DIP switch. DASH-16 has direct memory access capabilities to the IBM P.C. DMA is a method for moving data from the data acquisition system directly into memory using hardware capabilities of PC. This process allows acquisition to take place independent of the computer's processor. The board can be set for a unipolar or bipolar input using a slide switch.

2.3 ASYST

ASYST is a scientific system software from Macmillan Software Company. The ASYST system is divided into several different modules; System, Graphics, Statistics, Analysis, and Data Acquisition.

ASYST has an extensive set of graphics capabilities. Graphs can be customized by modifying the default parameters and options in the graphics configuration menu. Graphics formatting can be completely

controlled inside user programs. ASYST can permanently store numerical data in the form of a data file. In order to store numerical data one has to create a file; once the file is created that file may be accessed to read or write data .

Direct memory access (DMA) is a process which allows acquisition to take place independent of the computer's processor. Data can be transferred with extreme efficiency and increased acquisition rates using DMA. DASH-16 board supports direct memory access operation. An IBM PC, XT has four DMA channels. DMA channels 0 and 2 are used for memory refresh and floppy disk support. DMA channel 3 is used to support the hard disk. So only channel 1 is left for DMA acquisition. When the acquisition has been completed, the data is ready to be used. But the data is written to the disk in the same format as it is received from the data acquisition board. While storing data, the most 4 significant bits are used for the address of the memory location. So these 4 bits should be removed before processing data. Only integer type of data can be transferred during DMA operation.

The analysis package provides extensive manipulation and reduction capabilities for data obtained through the ASYST acquisition system, including differentiation, integration, smoothing, frequency analysis, and curve fitting. It also contains a number of analytic routines that will allow the resolution of many difficult mathematical problems.

2.4 Procedure

The purpose of this project is to correlate the output of the interferometer with ECG and jugular pressure pulses. For this the interferometer output, ECG and jugular pressure pulses from a subject are to be collected.

Therefor, an interferometer, ECG machine and a microphone (for jugular pulse) are used for this project. Since three different datas are to be collected at the same time, a three channels of data acquisition is needed for this project.

Power up the Microwave Interferometer after following the steps indicated in the setup chapter. The subject is provided with the "consent form" and requested to read its contents. A copy of the consent form is provided in the Appendix D. The subject, after reading and understanding the contents, has to sign the form.

The subject is made to lie on a table in a supine position under the microwave interferometer. Electrodes from the ECG machine are strapped on to the subject's left leg, right forearm and left forearm. A crystal microphone is strapped on to the right side of the neck, above the jugular vein, of the subject.

Start ASYST and run the data acquisition program "DISKACQ1", see Appendix B. This program would collect three channels of data using DMA, and store this data on hard disk. This program would ask for the number of buffers needed for collecting data, the sampling rate in kilo hertz and the file name for storing data. For all the 18 subjects whose data are recorded, two buffers and sampling frequency of 0.9 kilo hertz is used.

The three channels of data are stored in the data file as three columns of integer numbers. Run the program "RESHAPE", see appendix C. This program would separate three columns of data into three different arrays of data, each corresponding to the output of interferometer, EKG and jugular pulse, respectively.

Each data point is a 16 bit binary number. The most significant four bits of this 16 bits are address and the remaining 12 bits are the actual

sample value. The program "RESHAPE" (appendix C) would mask the 4 most significant digits.. The output of the interferometer is an amplified chest motion due to the subject's heart beat. The output filter of the interferometer has a bandwidth of 30 Hz., with the center frequency at 15 Hz. Therefore, the heighest frequency component in the interferometer output is 30 Hz. During data acquisition 60 Hz. noise are added into the signals, due to the surrounding 60 Hz. power supply. To remove this 60 Hz. noise a lowpass Blackman's filter with cutoff frequency 54 Hz. is used. This filter will eliminate all frequencies above 54 Hz. This program would also find the frequency spectrum of interferometer output and store it in the array named "INTERF".

2.5 Hardware Adjustments

On the Dash-16 board set the slide switch marked "A/D" to "BIP" position. This provides an input scaling from a negative to a positive full scale. Set the 6 slider switch marked "GAIN" with the switch marked 0.5 to "ON" and all other five switches "OFF". These settings will provide an input scaling of +/-10V. The I/O address is set by the Base Address DIP switch for 300 hex (768 decimal). The DMA mode is set to 1.

Install DASH-16 board inside the computer. Connect the ribbon connector of the interfacing board to the DASH-16 board. The ECG output of the interferometer is connected to the HI and LO of channel 1. The output from the ECG machine is connected to the HI and LO of channel 2. The output of the microphone is connected to the HI and LO of channel 3, with a jumper between LO and GND. Refer the operation manual of interferometer for the operating instructions of the interferometer. Adjust the sensitivity of the interferometer according to the manual.

CHAPTER 3
PHYSIOLOGY

3.1 Physiology of Heart

The heart is a four chambered, hollow, muscular organ lying between the lungs in the middle mediastinum. The primary function of the heart is to serve as a muscular pump propelling blood into and through vessels to and from all parts of the body. The heart is shaped like an inverted cone, with its apex pointed downward.

The heart is enclosed inside a double layered sac, the pericardium. The wall of the heart consists of three distinct layers - the epicardium (external layer), the myocardium (middle layer), and the endocardium (inner layer). The myocardium is responsible for the ability of the heart to contract. The thickness of the myocardium varies according to pressure generated to move blood to its destination. The myocardium of the left ventricle is therefore thickest, the myocardium of the right ventricle is moderately thickened, while the atrial walls are relatively thin. The heart is divided into right and left halves, with each half subdivided into two chambers. The upper chambers, the atria, are separated by the interatrial septum; the lower chambers, the ventricles, are separated by the interventricular septum.

The atria serve as receiving chambers for blood from the various parts of the body and pump blood into the ventricles. The ventricles in turn pump blood into the lungs and remainder of the body. The right atrium constitutes the right superior portion of the heart. It is a thin walled chamber receiving blood from all tissues except the lungs. The right ventricle constitutes the right inferior portion of the heart's apex. The

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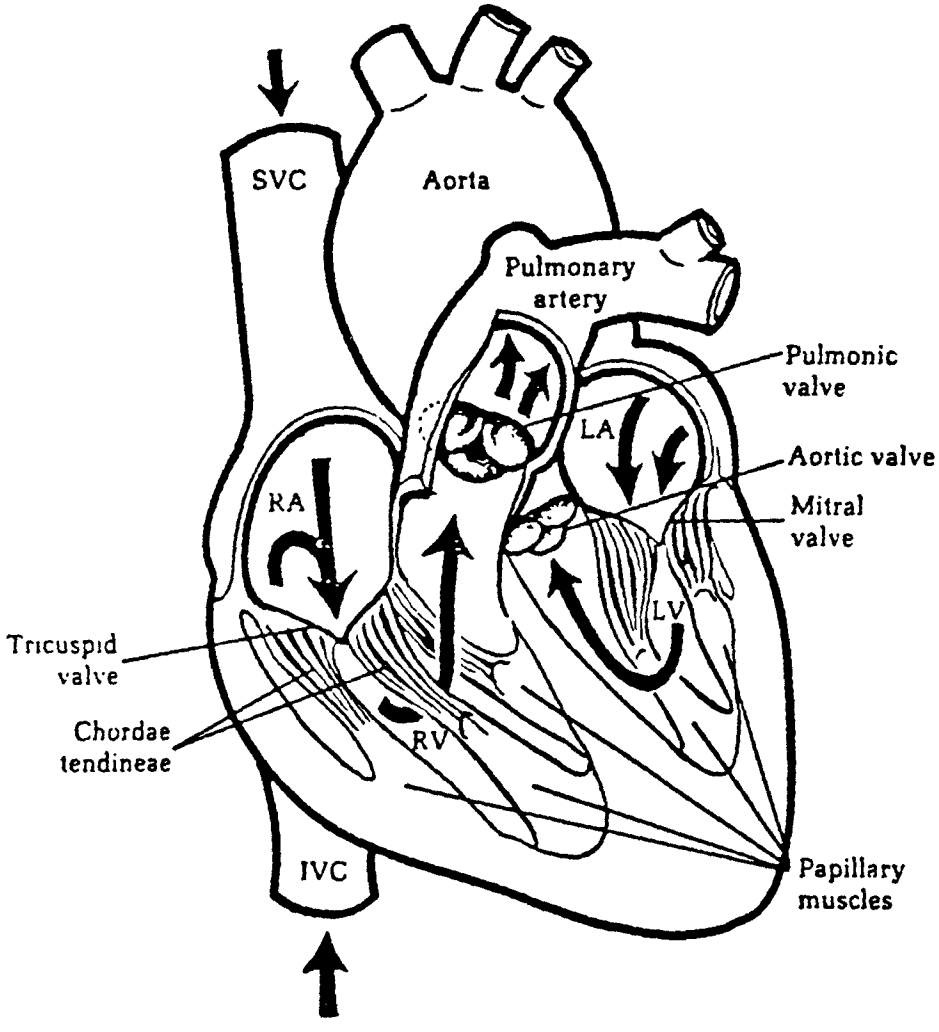

Figure 3.1 Heart.

pulmonary artery carrying blood to the lungs leaves from the superior surface of the right ventricle.

The left atrium constitutes the left superior portion of the heart. The left atrium receives the oxygenated blood from the lungs. Blood flows from the left atrium to the left ventricle. The left ventricle constitutes the left inferior portion of the apex of the heart. Blood from the ventricle is forced through the aorta to all parts of body.

There are two types of valves located in the heart: the atrioventricular valves (tricuspid and mitral) and the semilunar valves (pulmonary and aortic). The atrioventricular valves are located between the atria and ventricles. The right atrioventricular opening is guarded by the tricuspid valve. The left atrioventricular opening is guarded by the mitral or bicuspid valve.

Blood is propelled through the tricuspid and mitral valves as the atria contract. When the ventricle contracts, blood is forced backward to close the valves and form a complete partition between atria and ventricles. The pulmonary valve guards the orifice between the right ventricle and the pulmonary artery. The aortic valve guards the left ventricle and aorta.

3.2 Conduction System

The sino-atrial (S-A) node is a small mass of specialized myocardial tissues embedded in the atrial wall near the entrance of the superior vena cava (3). The S-A node is the normal pacemaker, spontaneously originating the spreading waves of excitation at a more rapid rate than any other part of the heart. A large number of fibers from parasympathetic and sympathetic nervous systems terminate in the vicinity of the S-A node. Discharge of

facing 11

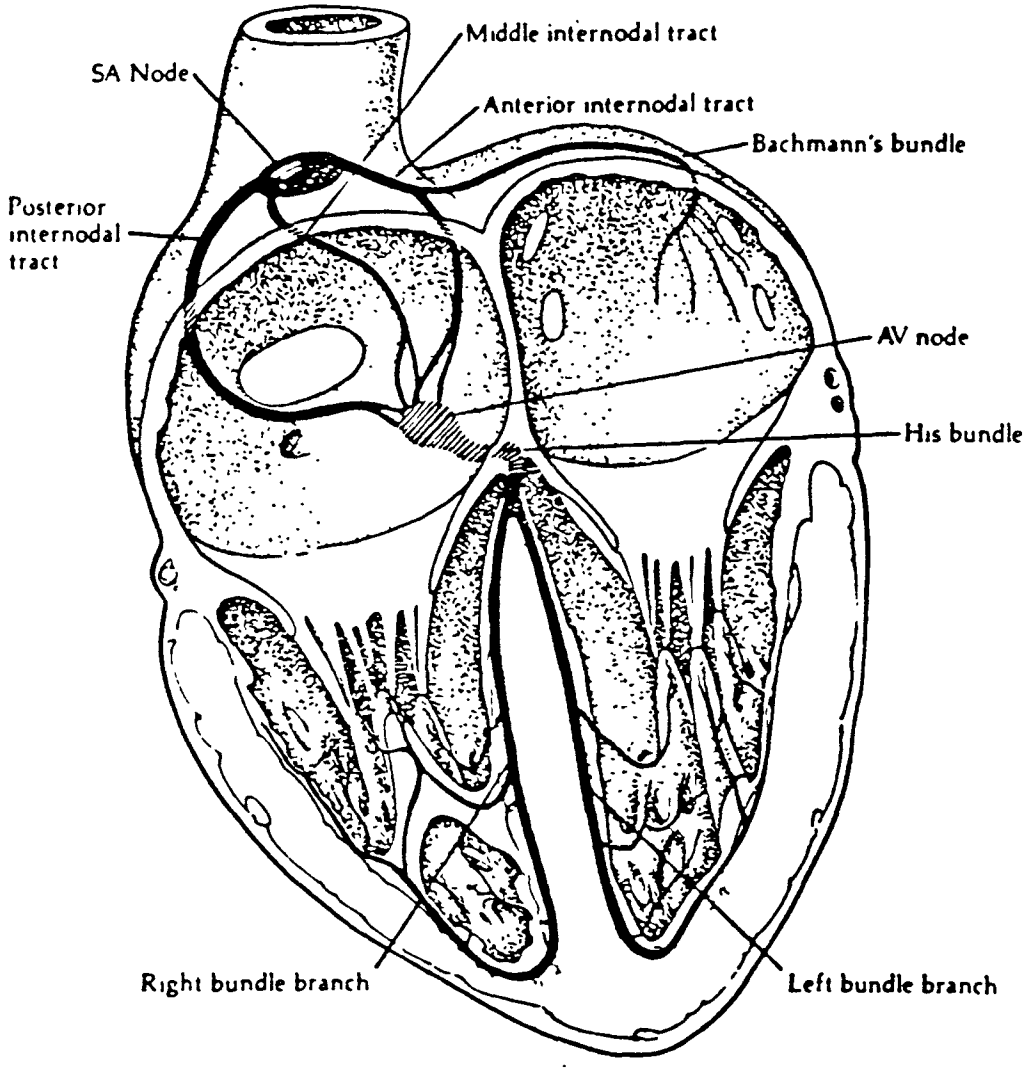

Figure 3.2 Conductive system of heart.

vagal parasympathetic fibers slow the rate of impulse formation and discharge of sympathetic fibers accelerate the impulse formation (3).

A wave of excitation originating in the S-A node spreads in all directions. As it approaches the interatrial septum, the wave of excitation reaches another mass of specialized conducting tissue, the atrioventricular (A-V) node. When the wave reaches the A-V node, it does not proceed directly to the ventricles but is delayed there. During the A-V nodal delay atrial contraction is largely completed. A bundle of Purkinje fibers - the bundle of His - starts from the A-V node. This bundle divides into two bundles, the left and right. The branches ramify into a network of Purkinje fibers which are distributed over the inner surface of the ventricular chambers.

After leaving the A-V node, the wave of excitation passes along the Purkinje fibers enters the endocardial surface of the ventricles. The wave of excitation then penetrates the ventricular walls from the endocardial to the epicardial surface.

3.3 Electrophysiology

The physiological events that precede the mechanical acts of contraction and relaxation are electrochemical in nature. The fluids both inside and outside the cell membranes of the body are electrolyte solutions made up of negative and positive ions.

When the muscle cells of the heart are at rest, the intracellular potential is negative with respect to the cell exterior. When a muscle cell of the heart is stimulated, there is change in polarity across the membrane of the cell. Its interior becomes positively charged due to the entry of positively charged sodium ions. A difference then exists between this cell

and its neighbor, and a current discharges, or flows, between the two until all cells in the same muscle mass have been stimulated to change their polarity.

In between electrical impulses the cell is said to be at rest. The electrical potential existing across the cell membrane during this time is called the resting membrane potential, which consists of an accumulation of negative ions along the inner surface of the cell membrane and an equal accumulation of positive ions on the outer surface of the membrane. This is the polarized state of the cell. An electrode inserted across the membrane into the inside of the cell records a difference in potential between the inside and the outside of the cell of roughly -90 millivolts. This electrical potential exists across the membrane of healthy cells in the atria and ventricles.

Depolarization is the process by which the inside of the cell becomes less negative. A cell depolarizes by the mechanism of a rapid influx of Na+ into the cell, in atrial and ventricular muscles.

The threshold potential is a level of membrane potential to which cells must depolarize before they can generate a propagating action potential. When this value of negativity is reached, there will be a rapid depolarization until the cell is at +30 mV on the inside with respect to the outside. Such a rapid depolarization is then propagated to neighboring cells, causing a current to flow.

Repolarization is that process by which a cell returns to its resting level of negativity. Depolarization in cardiac cells is initially rapid and brief, followed by a plateau and again a rapid but longer repolarization to bring the cell to its resting level.

The resting membrane potential depends on maintenance of a gradient of Na+ to K+ ions across the cell membrane. A mechanism actively pumps Na+ to the outside of the cell and K+ to the inside. These pumps function in an electrogenic fashion; that is, more Na+ is pumped out than K+ is pumped in. The result is a disparity of charge across the membrane.

Pacemaker cells are capable of self-initiated depolarization because of their unstable resting membrane potential. During diastole the pacemaker cell becomes less negative until a threshold potential of approximately -60 mV is reached and rapid depolarization ensues.

The action potential consists of depolarization and repolarization. The upstroke of the action potential is known as phase 0 and represents the rapid depolarization of the cell. When a threshold potential is reached due to an applied stimulus or to stimulus received from a neighboring cell, there is sudden change in cellular membrane permeability. Na+ then rushes into the cell, causing a reversal of potential, and produces the upstroke of the action potential.

Phase 1 is the initial stage of repolarization. This brief, rapid initiation of the repolarization process is believed to be due to an influx of chloride, a negative ion, as well as to an inactivation of the inward Na+ current.

During phase 2, approximately the next 10 msec the repolarization process slow down, causing a plateau in the action potential. This plateau allows the cardiac muscle a more sustained contraction and is thought to be the result of a complex interaction of a slow inward current (predominantly Ca++) with a slow outward K+ current. During phase 3 there is a sudden

acceleration in the rate of repolarization as the outward K+ current increases and the slow Ca++ current is inactivated.

Phase 4 diastolic depolarization distinguishes pacemaker cells from nonpacemaker cells. The pacemaker cell has the property of self-excitation. The nonpacemaker cell does not. Phase 4 in the pacemaker cell slopes up to a less negative potential until a threshold is reached at approximately -60 mV. The slow diastolic depolarization is caused by a time dependent fall in outward K+ current. This, combined with an increase in Na+ influx, causes a threshold to be reached. Depolarization is thus self-initiated in the pacemaker cells. This process is usually more rapid in the cells of the sinus node, which depolarize first and discharge the lower pacemaker cells before they have a chance to reach threshold on their own. The atrial and ventricular cells reach threshold potential more abruptly, since they are dependent on the pacemaker cells for their depolarization.

Not all regions of the heart are undergoing depolarization or repolarization at the same instant. Both processes spread from one heart region to another during a single cardiac cycle. The sum of all instant to instant vectors is referred to as the mean vector. The direction that the mean vector takes is the electrical axis of the heart.

The bipolar leads compare the electrical potentials between the two electrode (- and +) terminals. The axis of the lead is determined by the two electrodes. The ECG from a bipolar lead is a reflection of the orientation of the cardiac vectors to this axis.

The arms and legs are linear extensions of the electrical field surrounding the heart. Therefore an electrode placed on the right arm senses the same electrical potentials that would be sensed at the right

shoulder. The same relationship applies for electrodes on the other extremities.

The axis of lead 1 extends from shoulder to shoulder. The negative electrode is on the right arm, and the positive electrode on the left arm. The axis of lead 2 extends from the right shoulder to the left leg. The negative electrode is on the right arm and the positive electrode is on the left leg.

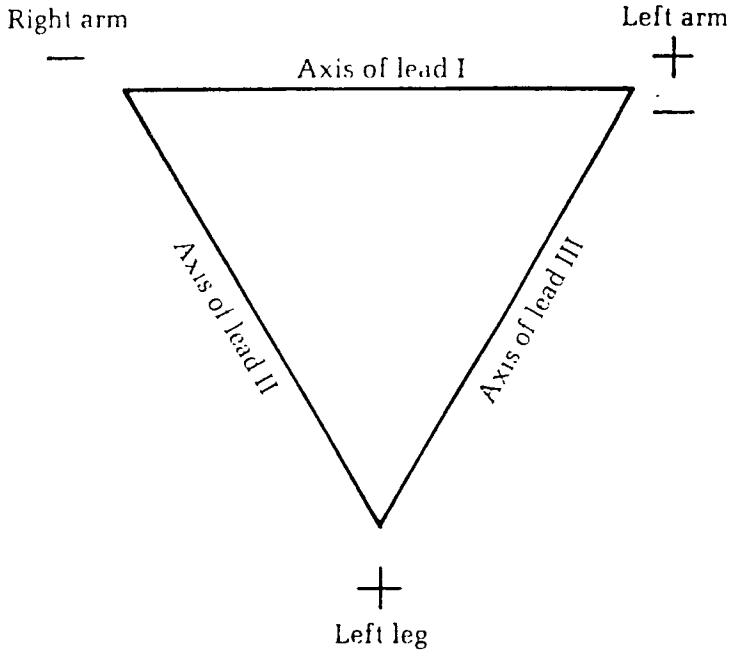

Figure 3.3 Axis of the 3 standard ECG leads.

The axis of the left lead extends from the left shoulder to the left leg. The negative electrode is on the left arm and the positive electrode is on the left leg. When a current flows towards a positive electrode, a positive polarity is developed in this electrode; when a current is oriented towards a negative electrode, a negative polarity is developed in this electrode.

The normal EKG is composed of a P wave, a QRS complex, and a T wave. The P wave represents atrial depolarization and the QRS complex, ventricular depolarization. The T wave reflects the phase of rapid repolarization of the ventricles.

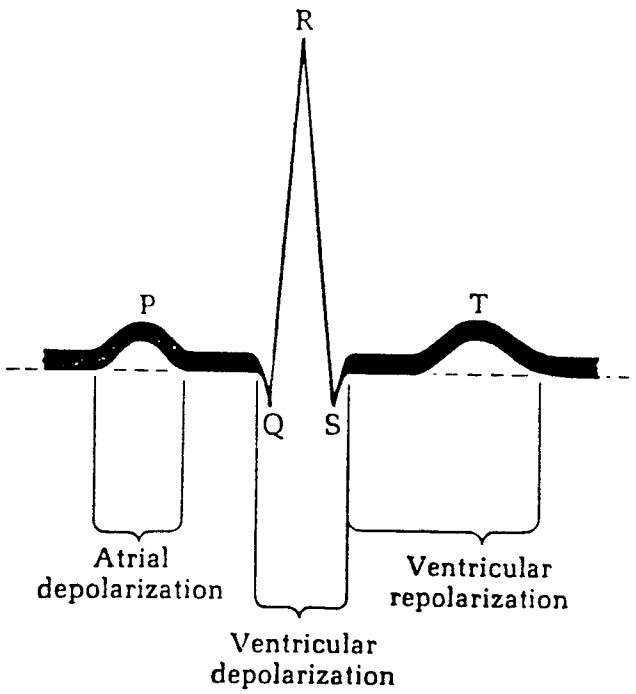

Figure 3.4 Normal ECG deflections.

The duration of the P wave indicates the time it takes for the depolarization current to pass through the atrial musculature. Because the atria are thin walled structures, the P wave is a small deflection not normally more than 3 mm in height. Since the P wave is travelling in a leftward and inferior direction, the current flows towards the positive terminals of the leads 1 and 2, therefore, the P wave is a positive deflection in these leads. The ventricular complex (QRS) indicates the depolarization of the ventricle.

3.4 Cardiac Cycle

The cardiac cycle is divided into four phases, according to what is going on in the ventricles. During isovolumic contraction, ventricular pressure rises above atrial pressure but does not yet reach arterial pressure (pulmonary or aortic). The atrioventricular (AV) valves (mitral and tricuspid) are closed. When aortic pressure is exceeded, the ejection phase begins; the AV valves remain closed, but the semilunar valves open. When ejection ceases, the aortic and pulmonary valves close, and ventricular pressure falls toward atrial pressure. During this phase of isovolumic relaxation all valves are again closed. When the ventricular pressures drop below atrial pressures, the AV valves open, and ventricular filling begins; aortic and pulmonary valves remain closed.

Electrical systole begins with the first component of the ventricular complex (QRS). Mechanical systole follows electrical systole. The first rise in ventricular pressure tenses the AV valves, which, at normal heart rates, are almost closed at the end of diastole. The AV valves are held in place by the chordae tendineae, but the leaflets bulge into the atria. This bulge produces a small rise in atrial pressure. Sudden tensing of the AV valves produces the main components of the first heart sound.

Shortly after the ventricle is fully depolarized (ventricular complex of ECG is completed), ventricular pressure exceeds aortic pressure, and ejection begins. The principal decrease in ventricular volume and peak rate of flow in the aorta occur early in systole. Outflow from the atria is prevented by high intraventricular pressure that holds the AV valves shut. As ventricular fibers shorten, they pull the base of the heart toward the apex. This stretches the atria and increases their capacity. The resulting fall

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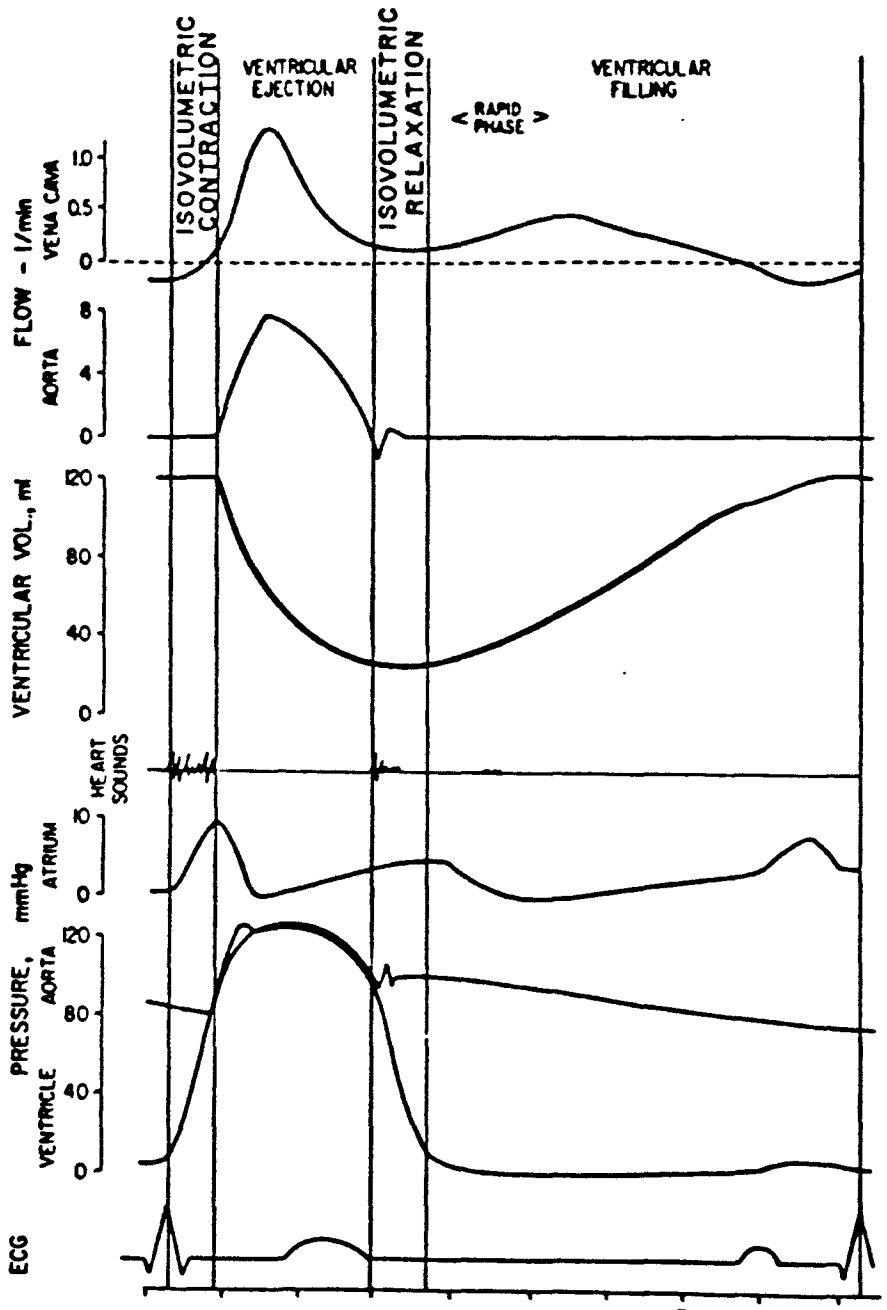

Figure 3.5 Events in cardiac cycle

in atrial pressure produces a brisk increase in vena caval flow. Atrial filling is responsible for the slow rise in atrial pressure and decrease flow rate in venae cavae during the later part of systole.

Ventricular pressure falls rapidly as active tension decays. Release of potential energy stored in elastic fibers during systole helps to expand the ventricles and accelerates the pressure drop. The large pressure gradient that develops between aorta and ventricle drives a small back-flow that slams the aortic valve shut. The abrupt tensing of the semilunar valve leaflets gives rise to the second heart sound and to a sharp oscillation in aortic pressure called the dicrotic notch. Meanwhile, venous return continues to expand the atria, so atrial pressure increases further.

After ventricular pressure drops below atrial pressure the AV valves open. Blood dammed up in the atria during systole rushes into the ventricles. Ventricular wall tension continues to fall during this time. Consequently, ventricular pressure remains slightly below atrial pressure. The rate of filling slows as atrial and ventricular pressure approach equilibrium. Flows in the venae cavae and pulmonary veins decrease in late diastole as rising pressure dissipates the gradient for venous return. At slow heart rates, filling practically ceases before atrial contraction begins.

Electrical activation of the atria (P wave of ECG) is followed by a rise in atrial and ventricular pressures. At slow heart rates this adds little additional volume to the ventricles, because the resistance to retrograde flow back into the great veins is less than resistance to forward flow if the ventricles are nearly full. At rapid heart rates, however, passive filling is incomplete, and atrial contraction becomes essential.

When the atrial fibers relax, atrial pressure falls, and the leaflets of the AV valves float into close proximity. The time for this prepositioning is

provided by the AV nodal delay. With the valves almost closed, the rise in ventricular pressure in early systole causes virtually no reflux of blood into the atria.

3.5 Heart Sound

Closure of the heart valves is associated with an audible sound. The first sound occurs when the mitral and tricuspid valves close, the second with the closing of the pulmonic and aortic valves. These sounds are caused by the vibration of the walls of the heart and major vessels around the heart.

The first heart sound is heard when the ventricles contract, causing a sudden back flow of blood, closing the valves and causing them to bulge back. The elasticity of the valves then causes the back-surging blood to bounce backward into each respective ventricle. This effect sets the walls of the ventricle into vibration, and the vibrations travel away from the valves. When the vibrations reach the chest wall where it is in contact with the heart, sound waves are created.

The second heart sound results from vibrations set up in the walls of the pulmonary artery, the aorta and the ventricles as the blood reverberates back and forth between the walls of the arteries and the valves after the pulmonic and aortic semilunar valves suddenly close. These vibrations are then heard as the chest wall transforms the vibrations into sound waves.

3.6 Jugular Pulse

When the subject is supine, pressure pulsations can be detected in the jugular vein in the middle of the neck. In normal subjects this consists of three waves in each cycle, a, c and v. The "a" wave is due to atrial

facing 20

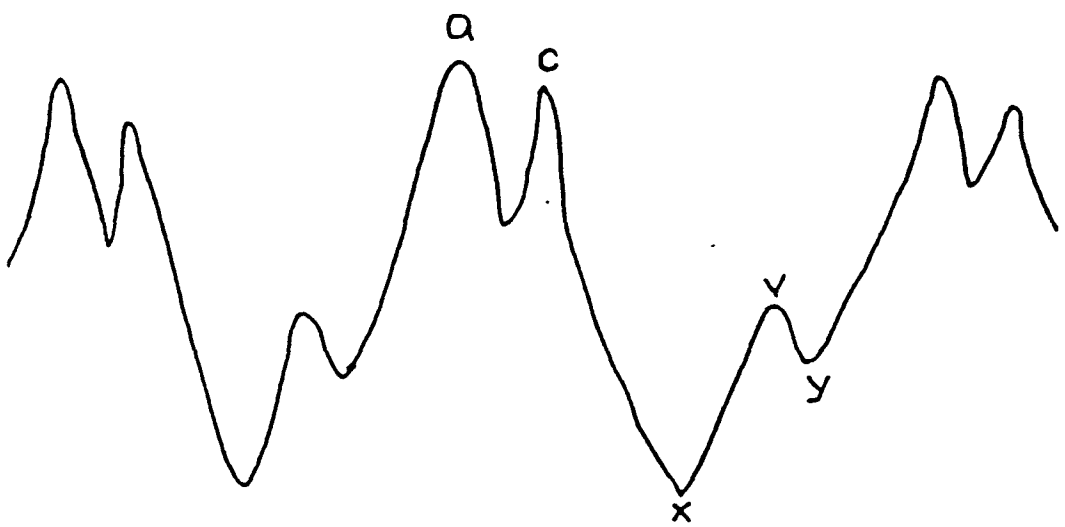

Figure 3.6 Jugular pulse.

contraction and is caused by the arrest of venous inflow to the heart by constriction of the mouth of the vein. This expansion of the vein which is observed in the presystolic phase is due to interference with entry of blood from the veins into the contracted atrium. This atrial contraction wave, "a", is seen just prior to the first heart sound. The summit of the wave marks the end of the mechanical systole of the atrium.

The "c" wave rises at the moment of isovolumic contraction of the heart. Therefore, the "c" wave indicates ventricular systole. The relaxation of the atrium subsequent to its contraction opens the way to venous flow into the heart, which results in collapse of the vein during ventricular systole. This effect is greatly enhanced by the aspiratory action of ventricular systole which contributes to the collapse of the vein in two ways. First, contraction of the right ventricle, by pulling the atrioventricular boarder toward the apex, exerts an effect similar to that of drawing back the piston of a syringe. Second, drop in intrathoracic pressure, which results from outflow of blood from the thoracic cavity. The descent of the systolic collapse is interrupted by the "c" peak. It is synchronous with the rise of the carotid pulse. The "c" wave is caused by impact of the pulsating aorta upon the right atrium.

At the end of ventricular systole the atrium is filled to capacity and the venous stream stagnates, distending the wall of the vein. Enlargement of the vein is enhanced at the beginning of diastole when the atrioventricular septum moves upward reducing the capacity of the atrium. Hence during the phase of isometric relaxation, the curve of the venous pulse ascends, forming a summit "v", which is lower than the "a" wave.

During the phase of isometric relaxation, the pressure in the ventricle falls. As soon as the atrial pressure becomes higher than the ventricular

pressure, under normal conditions, the tricuspid valve opens and rapid influx of blood from the atrium causes emptying of the vein. The "v" wave descends during the phase of rapid ventricular filling, forming the second trough "y" of the venous pulse.

3.7 Relation of ECG to Mechanical Events

Electrical changes in heart muscle precede their contraction by a short interval (fig.3.7). The P-wave begins slightly before the right atrial a-wave rises. The QRS begins shortly before the two ventricular pressures rise. The end of the T-wave occurs at about the end of ventricular ejection.

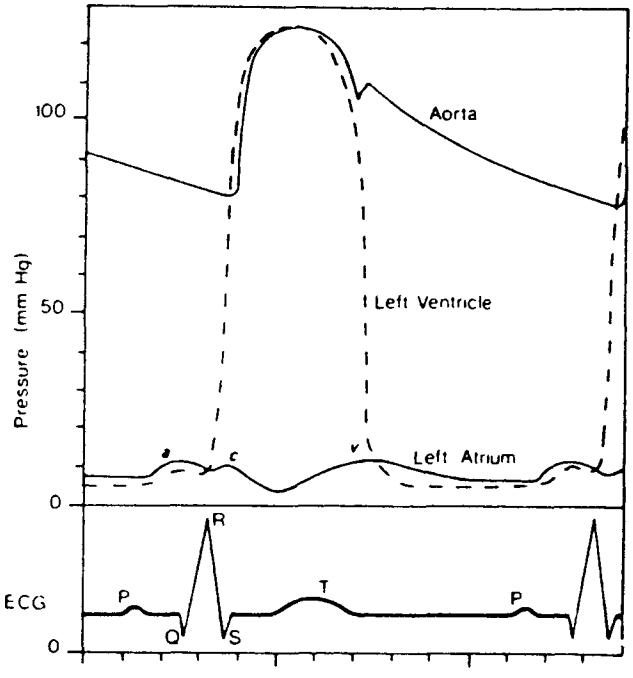

Figure 3.7 Relation of ECG to mechanical events.

CHAPTER 4
ANALYSIS

4.1 Correlation with EKG

Signals were recorded, using the interferometer, from the anterior chest wall of subjects lying in supine position. The interferometer deflections are a measure of the velocity of the anterior chest wall. The recordings of the fifteen subjects done with the microwave interferometer reveal distinct shape and same number of peaks refer the interferometer recordings of 14 subjects in appendix A. To identify the cause of these peaks, data from fifteen subjects were compared with EKG and jugular venous pulse. These two comparisons reveal the identity of some of the peaks of the interferometer output. These peaks are named K, U, Z and H for further references, see fig.4.2. Analysis done on one of 15 subject is explained in the following sections. All other recordings are given in appendix A.

Fig.4.1 is the sequence of events in the cardiac cycle. The lower three curves, representing the jugular pulse, the electrocardiogram, and the heart sounds, have been reconstructed from data obtained on human subjects. The vertical lines represent the following events: 3 = atrio-ventricular valves close, 4 = aortic valve opens, 5 = aortic valve closes. Vertical line 3 coincides with the peak of R wave, 4 coincides with the end of QRS complex, 5 coincides with the end of T wave. These vertical lines can be clearly identified on all electrocardiograms. So these three points 3, 4 and 5 are used throughout this analysis, to identify peaks in vibrational pattern of the chest wall.

The output of the interferometer is filtered through a lowpass Blackman's filter. The cutoff frequency used in this filter is 54 Hz.

facing 23

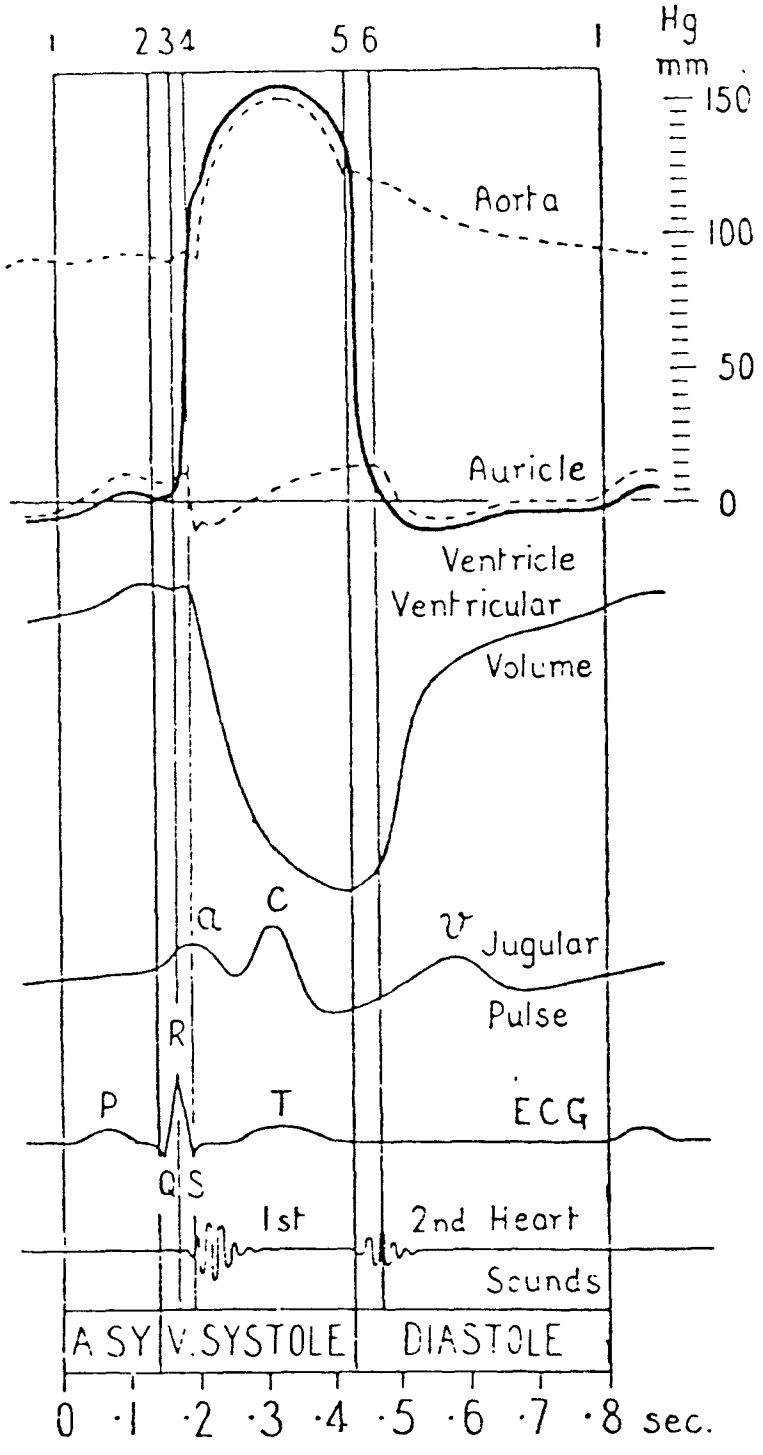

Figure 4.1 The sequence of cardiac cycle.

Therefor, the heighest frequency component in the interferometer output is 54 Hz. For sampling this data without the problem of aliasing a sampling rate, atleast twice the heighest frequency component in the signal must be used (sampling theorem). Therefor, the least allowed sampling rate without aliasing in this case is 108 Hz. In this project a sampling rate of 300 is used for each channel. Since three channels of signals are sampled at 300 Hz. each, a total sampling rate of 900 Hz. is used. When sampling, the first data point is collected from the first channel second from the second channel and the third from the third channel. Therefore, the time difference between two channels of data is .0011 seconds (1 divided by 900). This time lag is neglected for the comparison of the two traces, thereby enabling a common base for comparison.

The following analysis is true for all 15 subjects given in appendix A. The explanations following is for a single subject from the 15 subjects in appendix A. The wave K of interferometer output and the peak R of electrocardiogram coincide in fig.4.2. The peak R represents the closing of atrioventricular valves, that is the end of ventricular filling. So the wave K represents the ventricular filling.

The peak U of the interferometer output and the end of QRS complex wave in electrocardiogram occurs at the same time in fig.4.2. The end of QRS wave of the electrocardiogram corresponds to the opening of the aortic valve in fig.4.1. Therefore, the peak U represents the opening of aortic valve.

The peak H of interferometer output and the end of T wave of electrocardiogram occurs at the same time in fig.4.2. The end of T wave of electrocardiogram corresponds to the closing of the aortic valve in fig.4.1.

facing 24

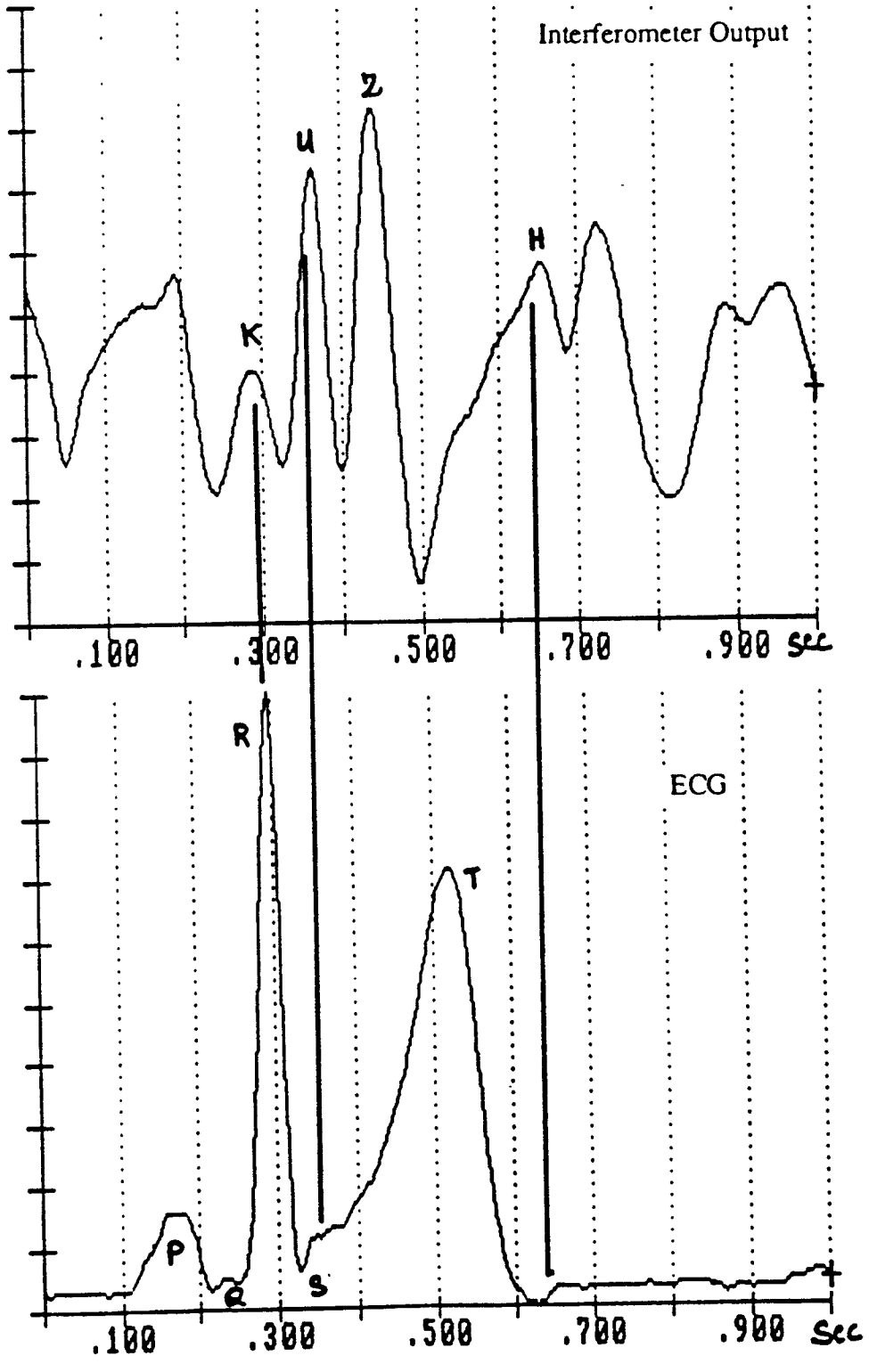

Figure 4.2 Interferometer output and ECG.

So the peak H represents the closing of aortic valve. Out of 18 recordings 15 recordings confirm these findings.

4.2 Correlation with Jugular Pulse

Fig 4.3 are the interferometer and jugular pressure readings from the same subject represented in Fig 4.2. The summit of the "a" wave of jugular pulse marks the end of the mechanical systole of the atrium . So the summit of the "a" wave marks the ending of ventricular filling. In fig.4.3 the K wave of interferometer and the summit of the "a" wave of the jugular pulse occur at the same time. This implies that the K wave of the interferometer represents the ventricular filling.

The "c" wave of jugular pulse is caused by the impact of pulsating aorta upon the right atrium . The pulsation of the aorta is caused by the opening of the aortic valve and the ejection of blood into aorta. So the beginning of "c" wave and opening of aortic valve occur at the same time. In fig. 4.3 beginning of "c" wave and U wave of interferometer output occur at same time. So the U wave of interferometer output represents the opening of aortic valve.

The opening of the aortic valve causes ejection of blood into the aorta and recoil of the heart takes place. This recoil produce a strong vibration on the chest wall. This vibration occurs after the aortic valve opens and before the summit of the "c" wave. In fig. 4.3 the Z wave of the interferometer output occurs between the opening of the aortic valve and summit of the "c" wave. Therefore, the Z wave of interferometer output is hypothesized to be caused by the recoil of the heart following the opening of the aortic valve.

facing 25

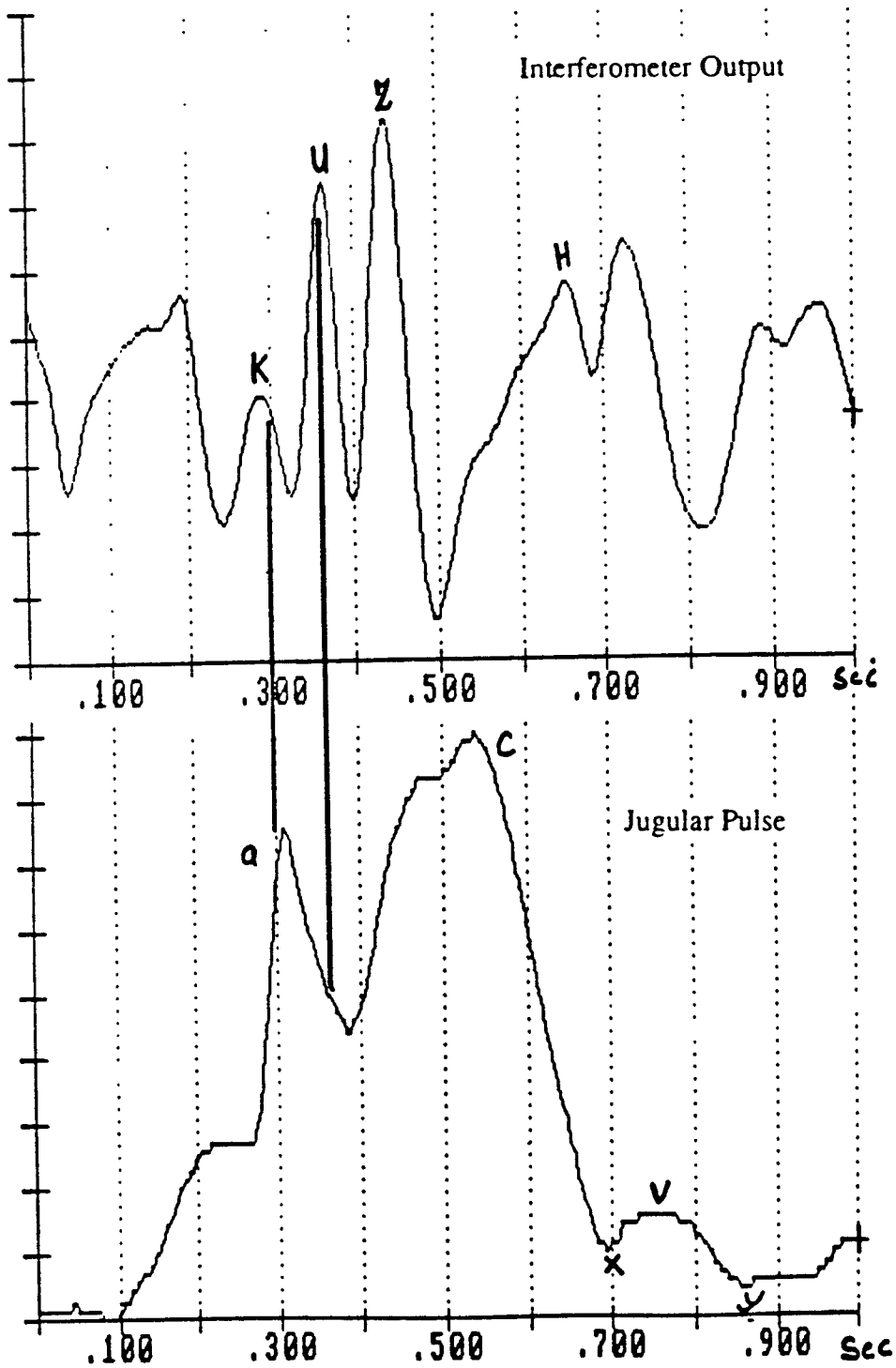

Figure 4.3 Interferometer output and jugular pulse.

In fig. 4.2the Z wave occurs in the beginning of ventricular systole. This also shows that Z wave represents recoil of heart. Out of 18 recordings 15 recordings confirm these findings.

4.3 Conclusions

The objective of the analysis is to identify the cause of different peaks in the output of the interferometer signal. Signals from 18 subjects are recorded, fifteen records have same number of peaks with a small shift in time (appendix A). These fifteen signals are compared with electrocardiogram and jugular pulses to identify causes of different peaks in it. Three recordings out of 18 recordings had a complex wave pattern, these three recordings are not used in this project.

The results of these studies clearly identifies causes of four peaks in interferometer output: peak K is caused by the ventricular filling, peak U is caused by the opening of the aortic valve, peak Z is caused by the recoil of heart in the beginning of ventricular systole, and peak H is caused by the closing of aortic valve.

There are small shift in time in the occurance of peaks in the interferometer outputs of diffferent subjects (appendix A). There are different physiological factors that effect the transmission of movements of the heart to the chest wall. Thickness of myocardium and orientation of the heart in the body are different for different subjects (1). This causes a difference in the time for transmitting movements of the heart to the chest wall. This difference appears as a time lag in the interferometer output. The different mechanical events that happens with the cardiac cycle are complex. So further clinical studies are needed for confirming these findings.

APPENDIX A

RECORDINGS OF ACQUIRED SIGNALS

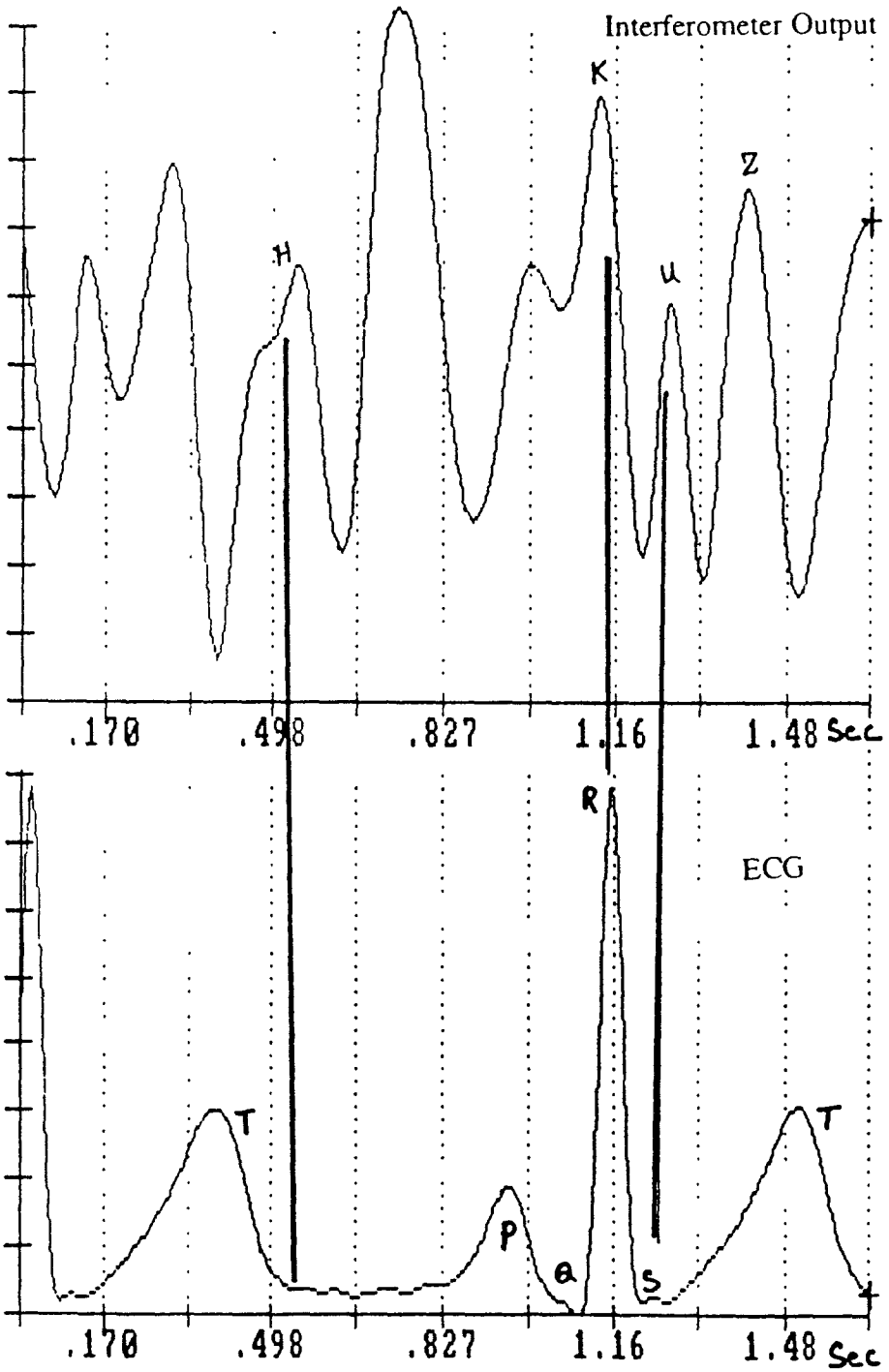

Figure A1.a Pa

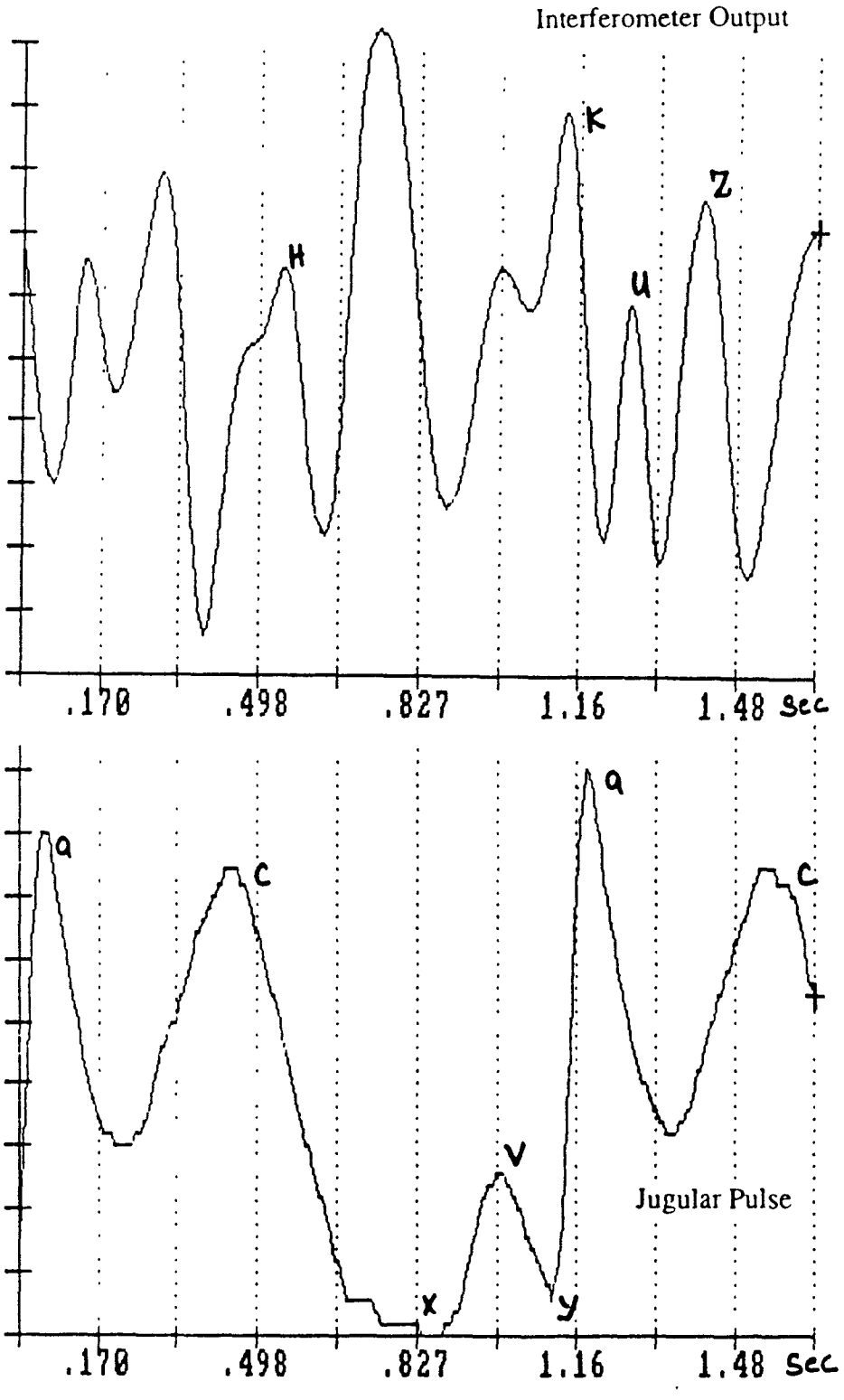

Figure A1.b Pa

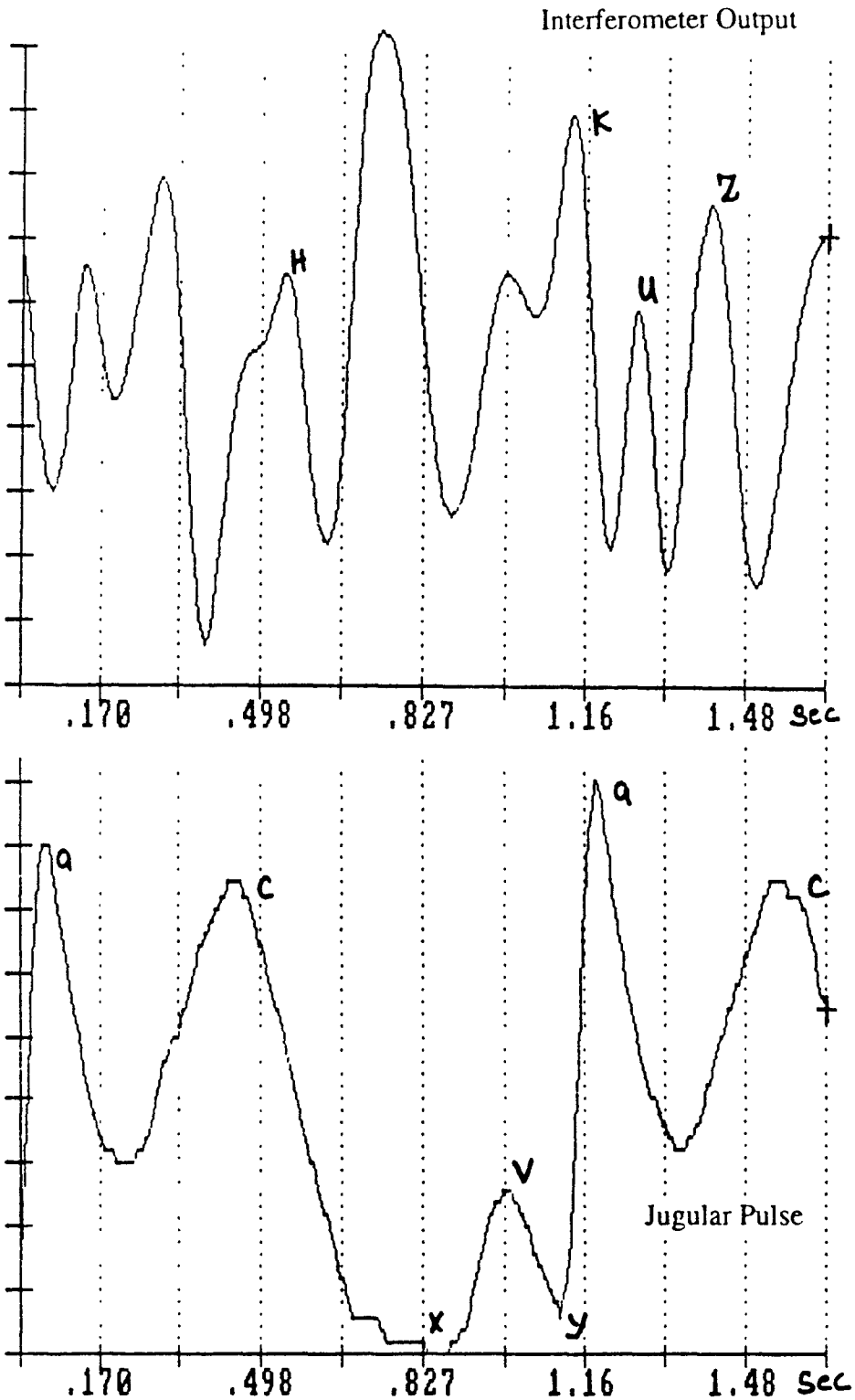

Figure A1.b Pa

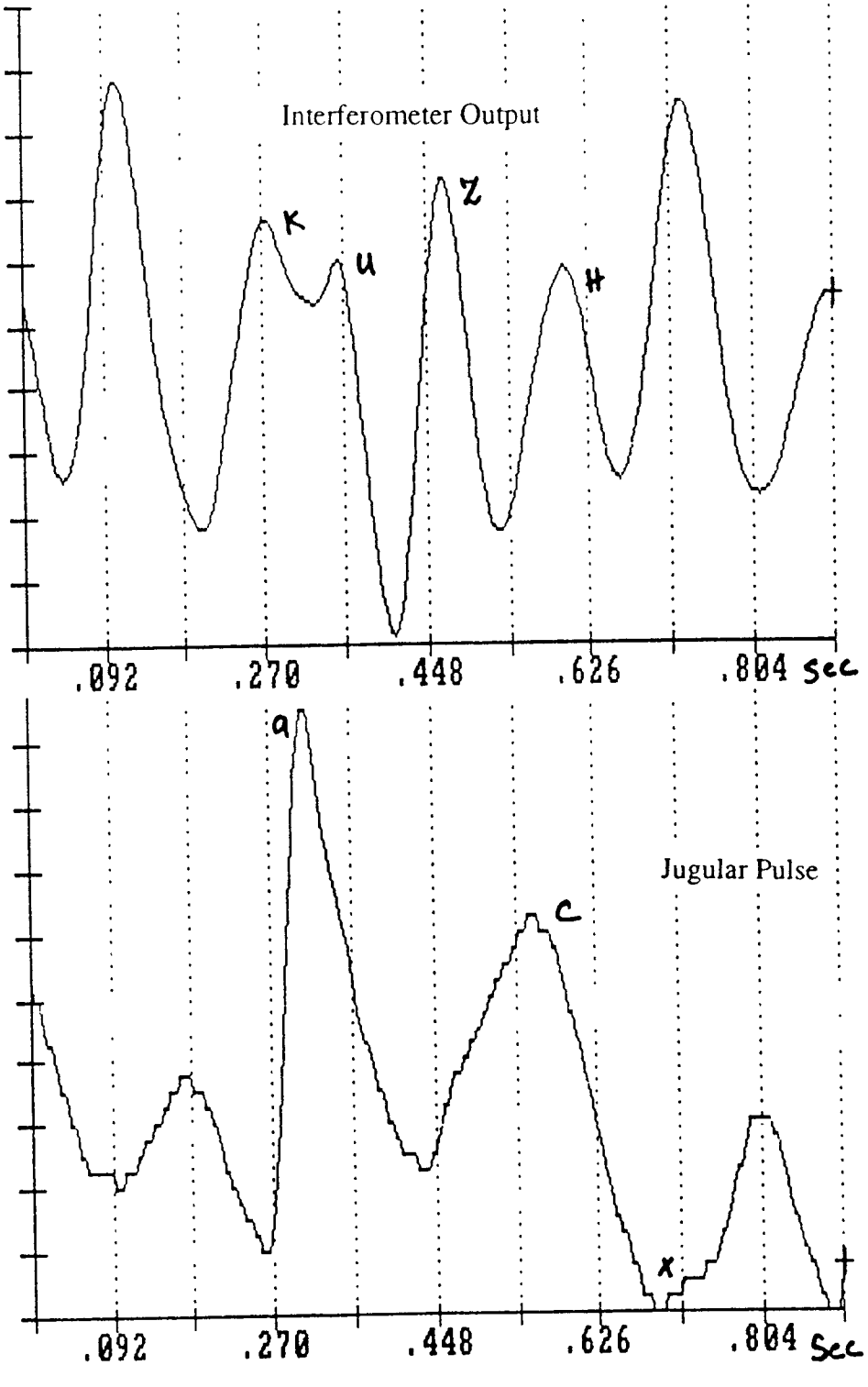

Figure A2.a Th

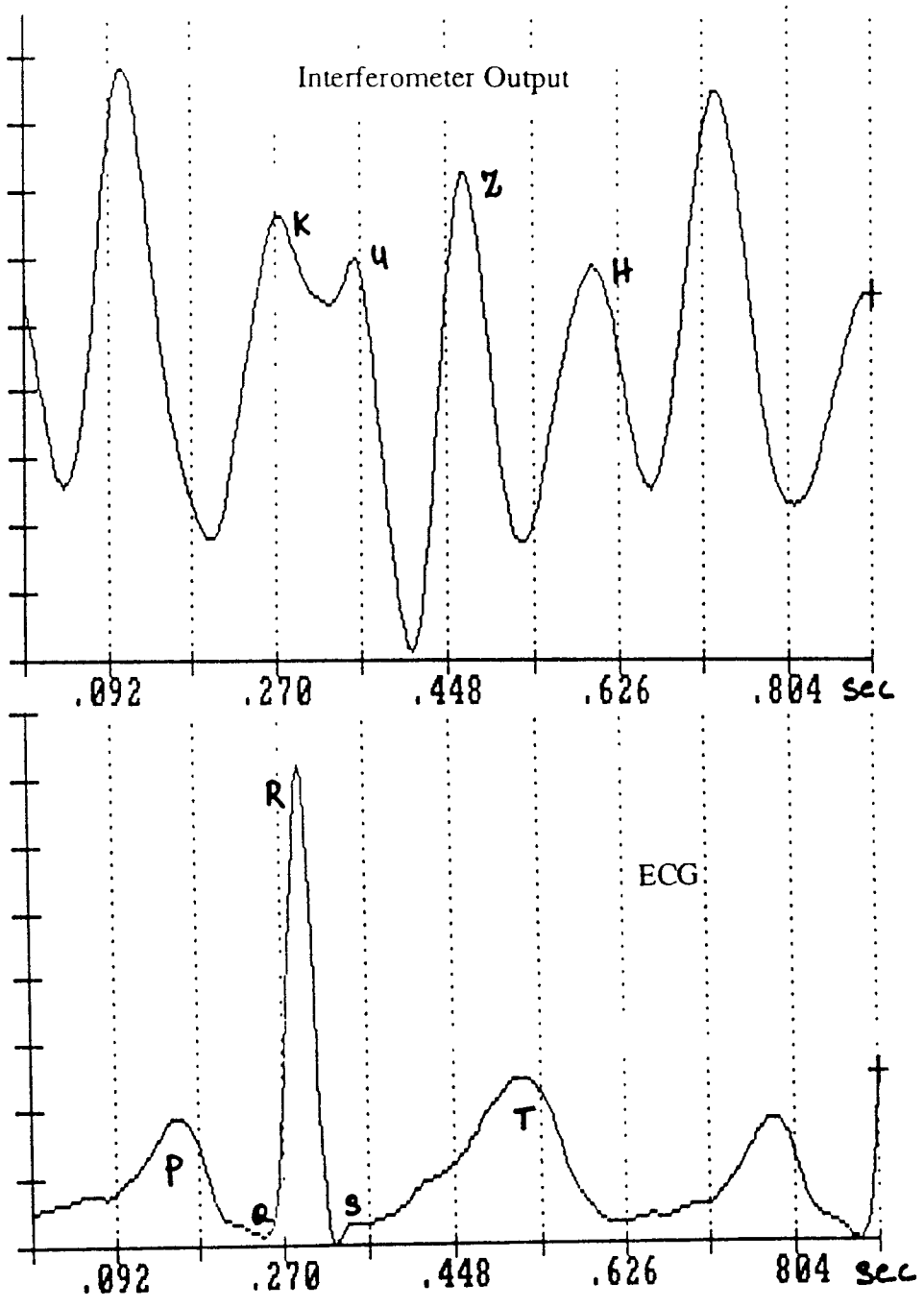

Figure A2.b Th

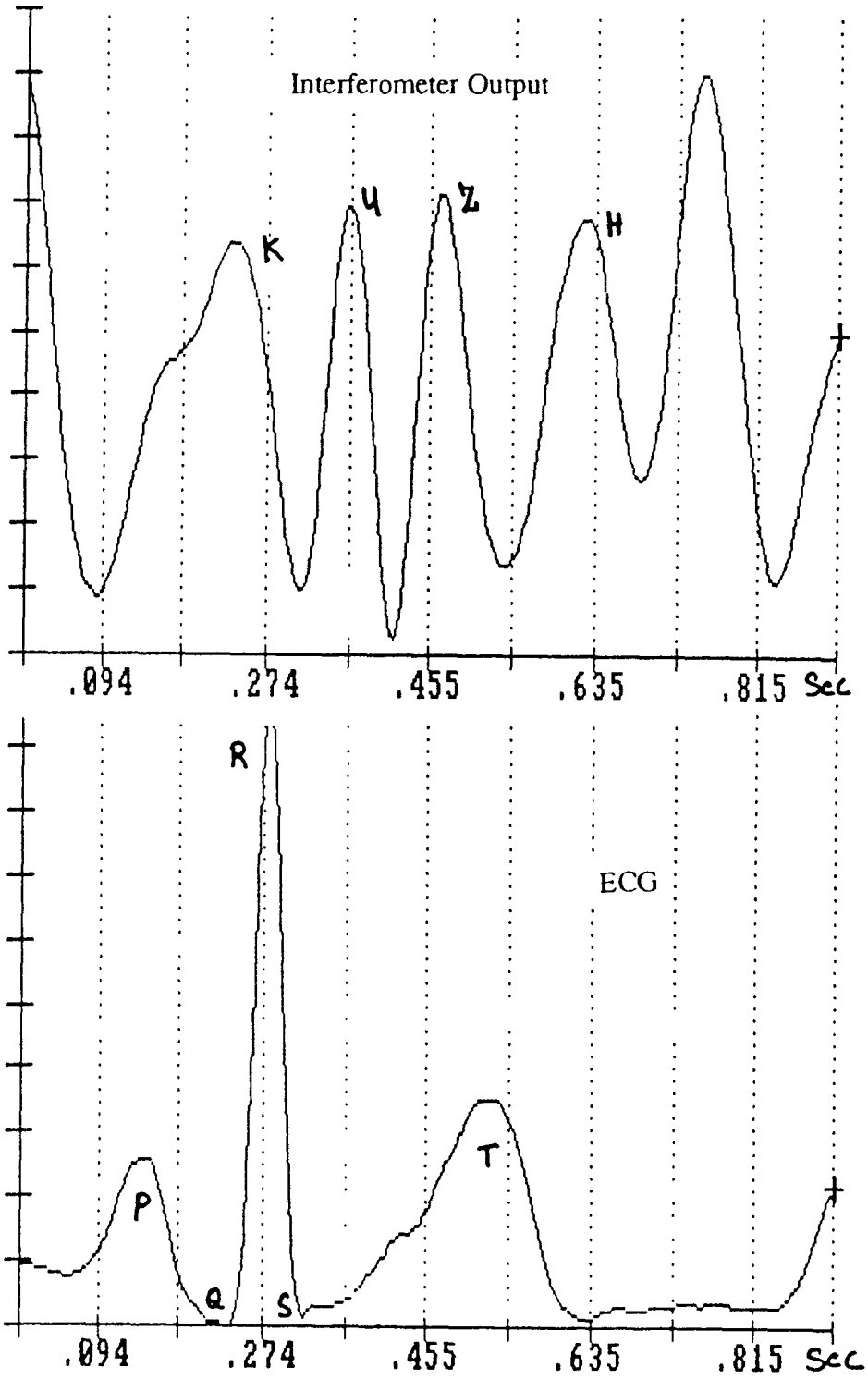

Figure A3.a To

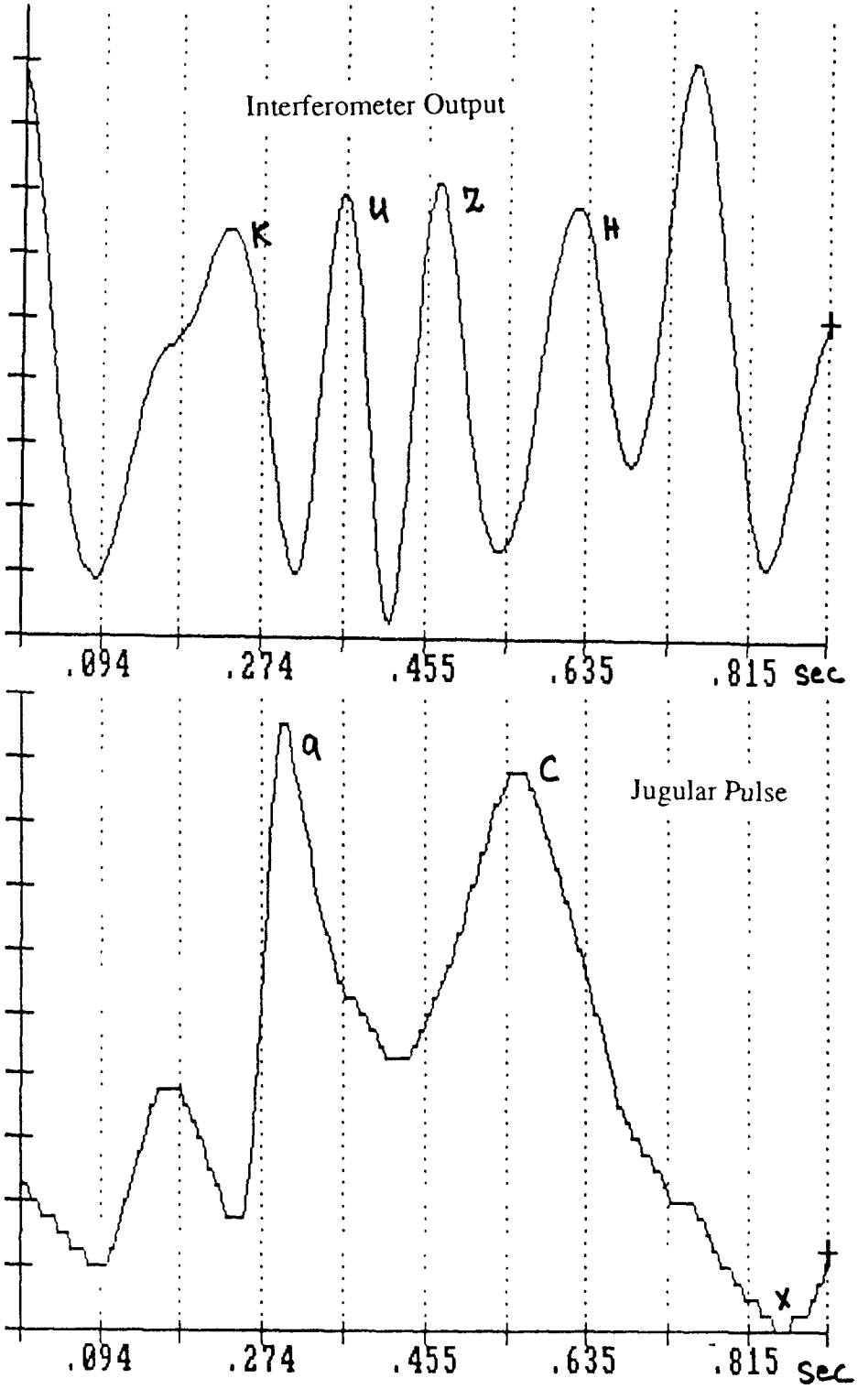

Figure A3.b To

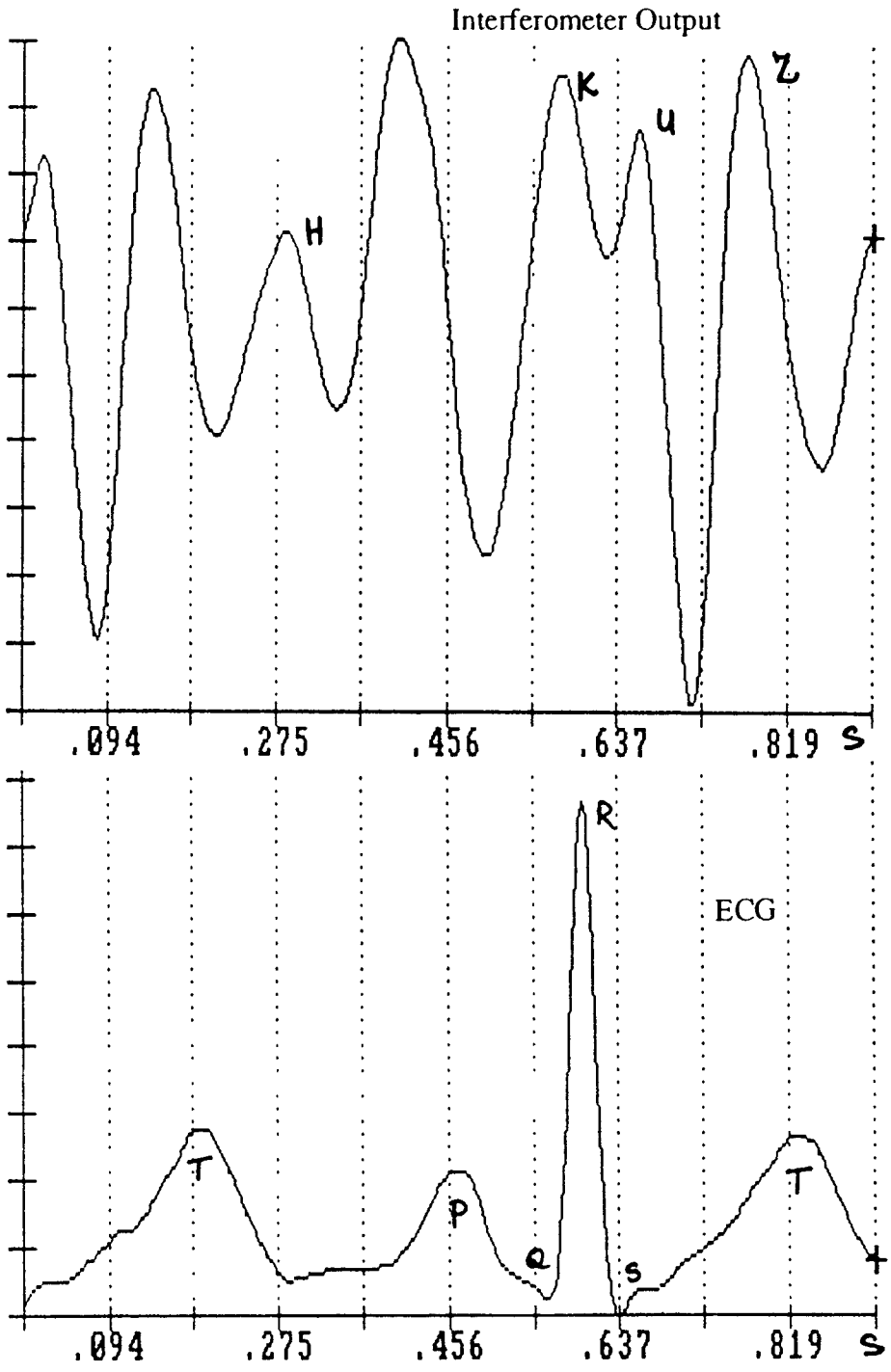

Figure A4.a Su

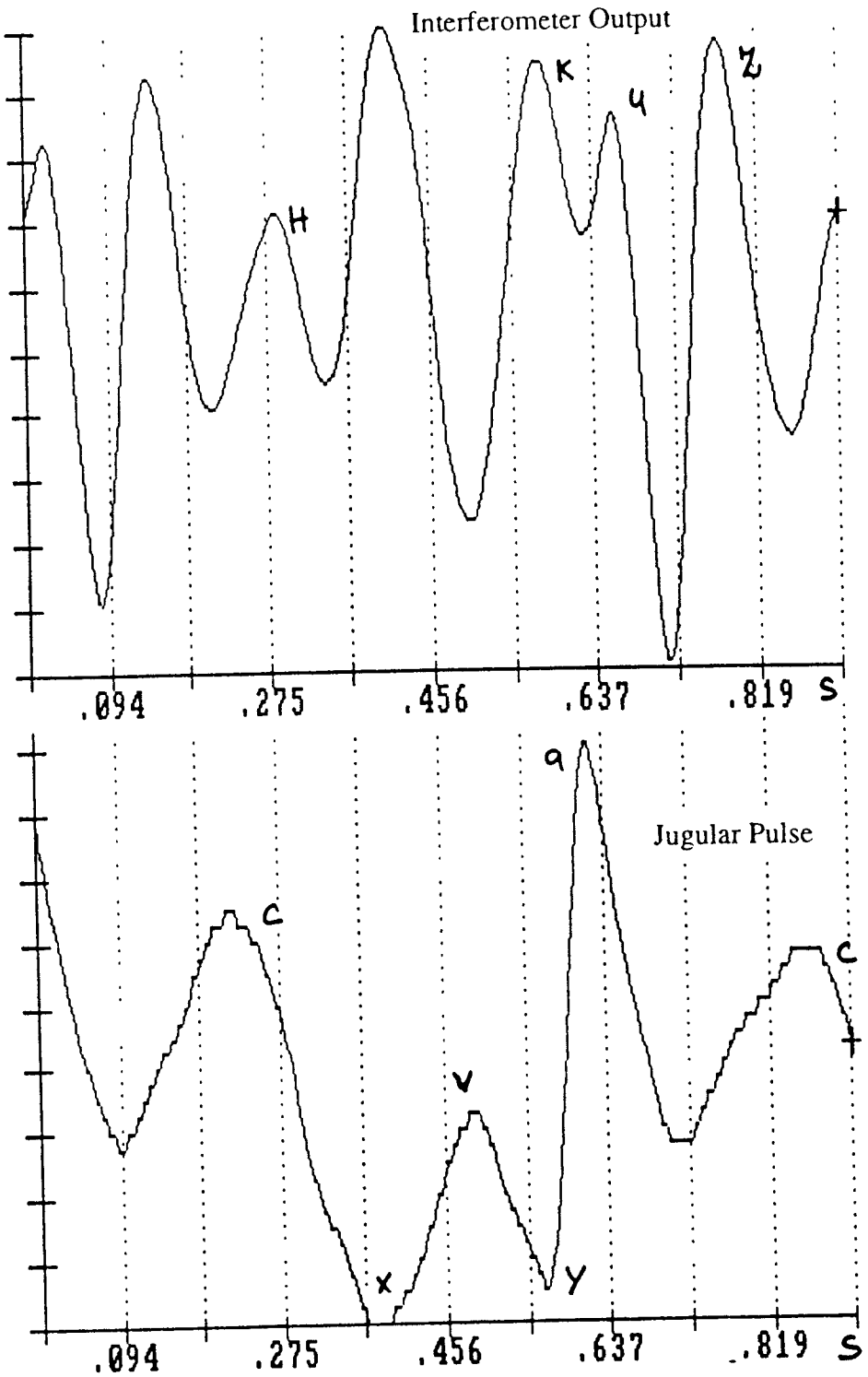

Figure A4.b Su

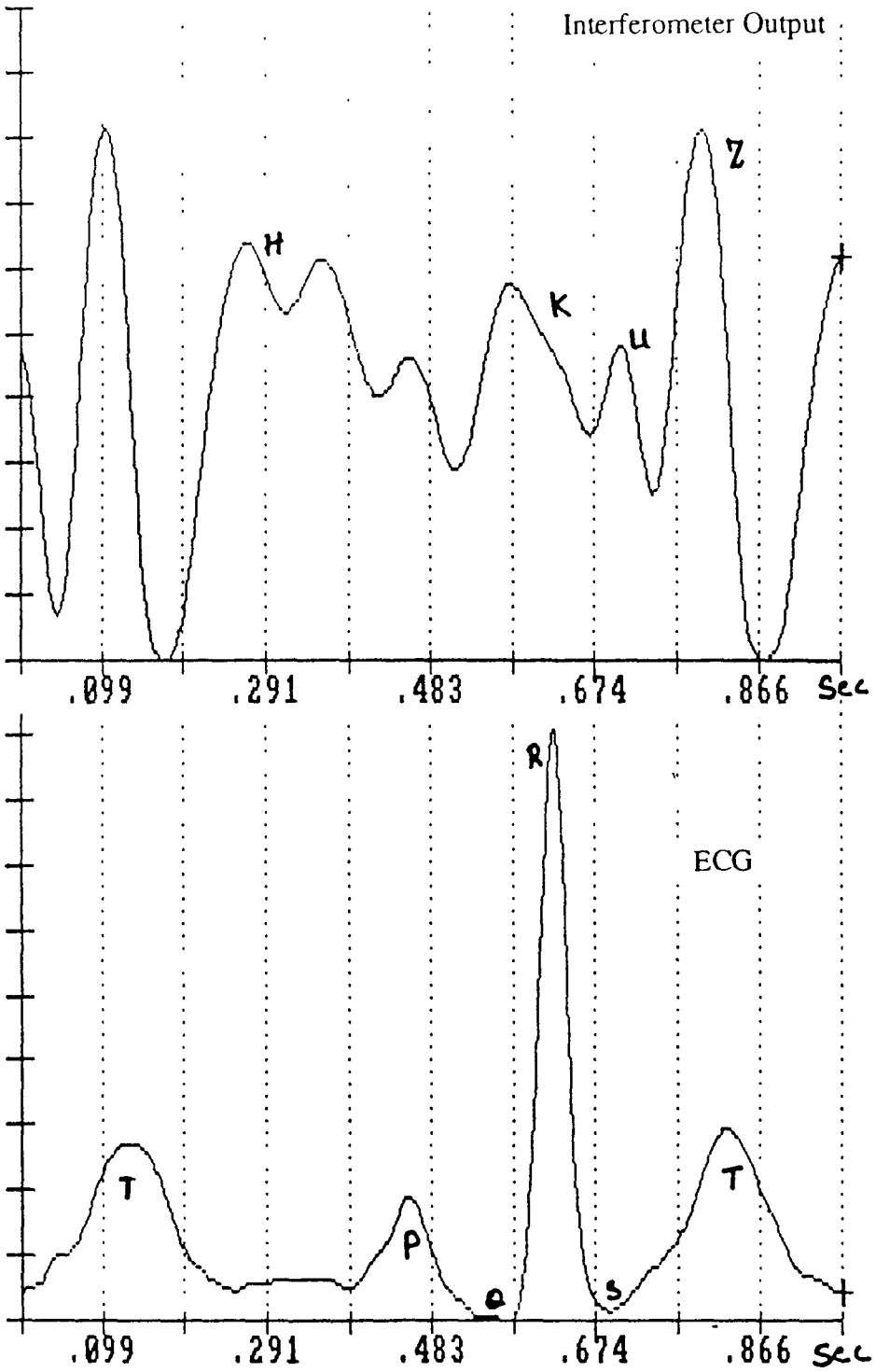

Figure A5.a Jo

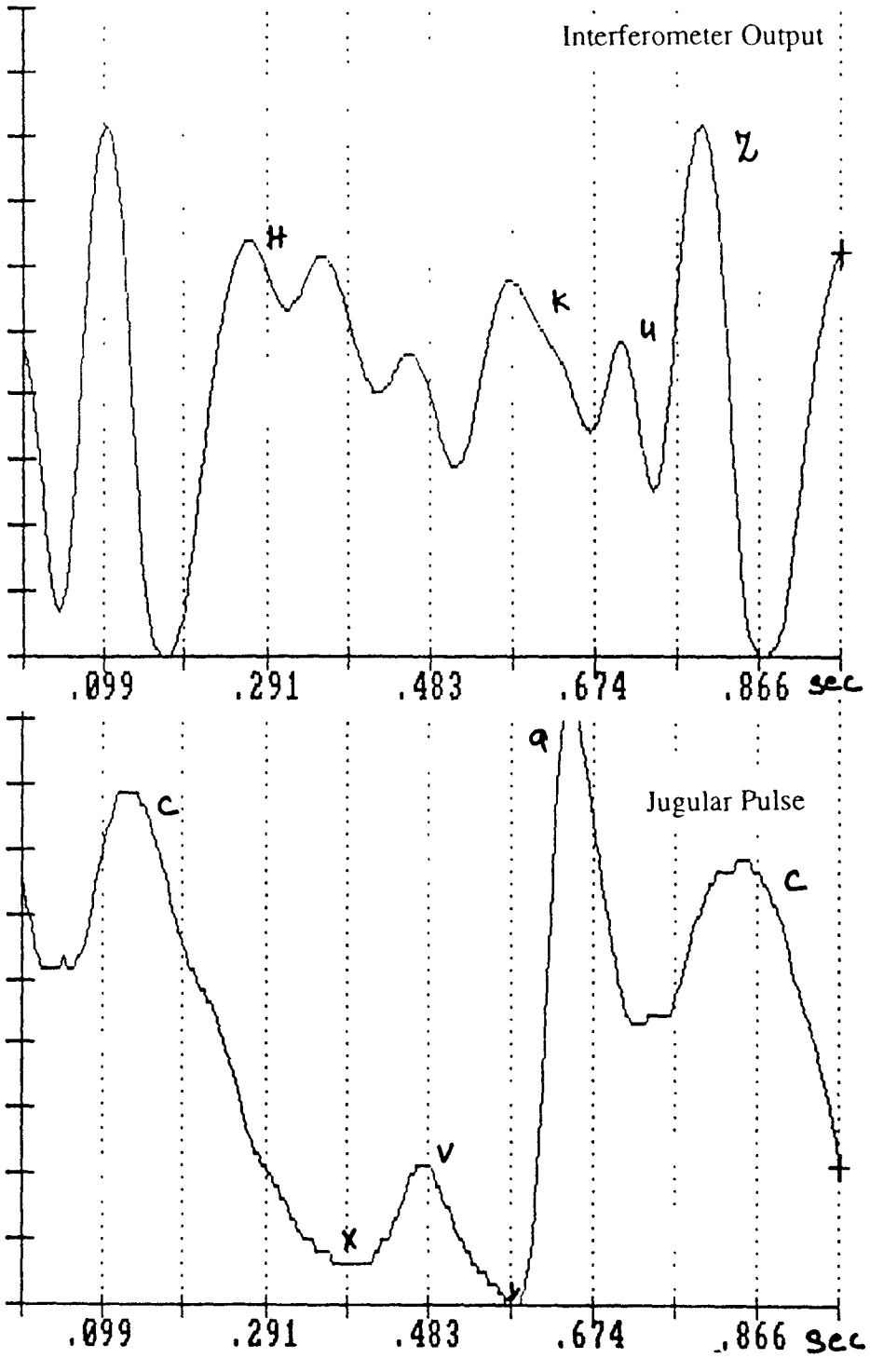

Figure A5.b Jo

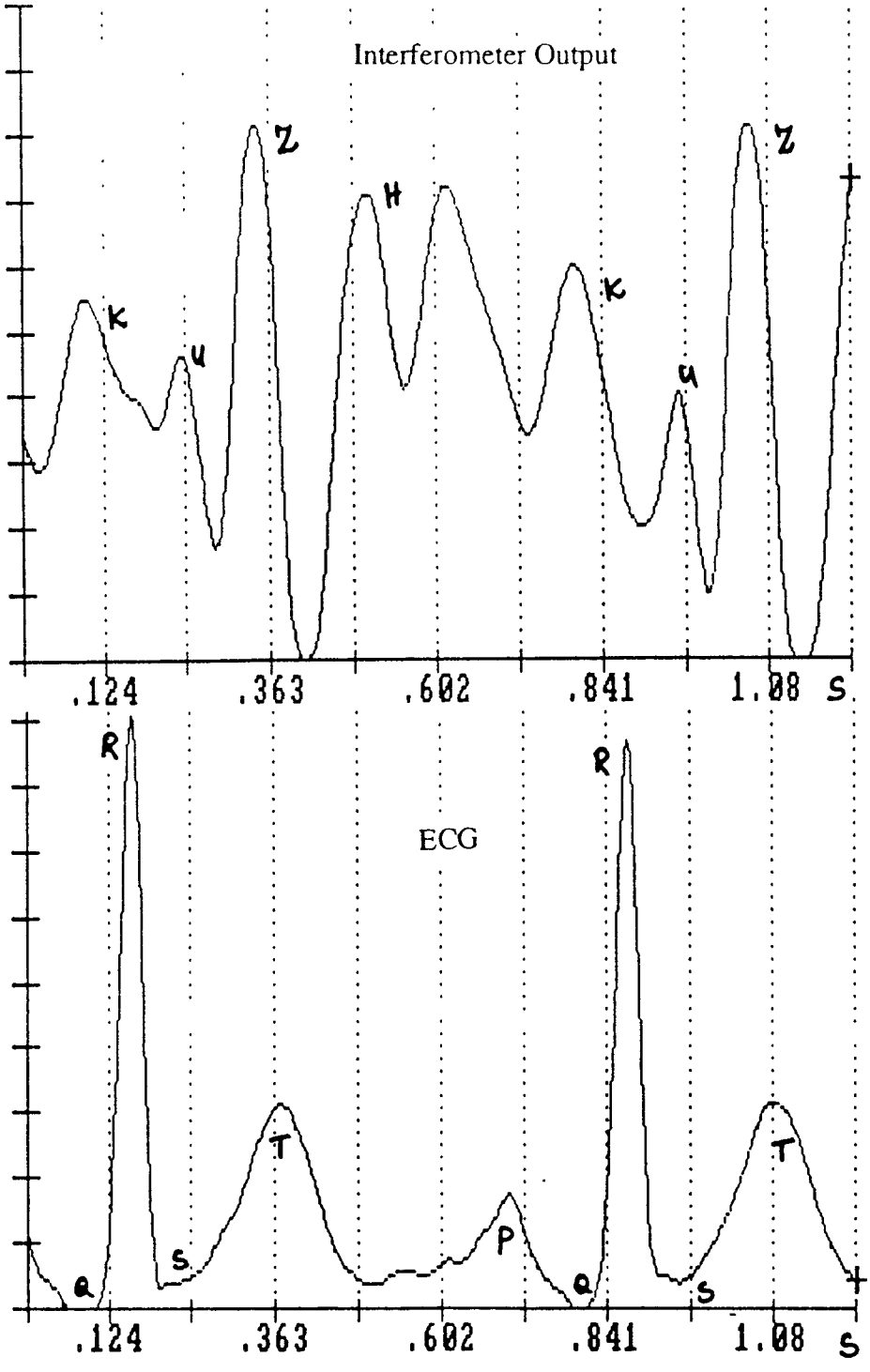

Figure A6.a To

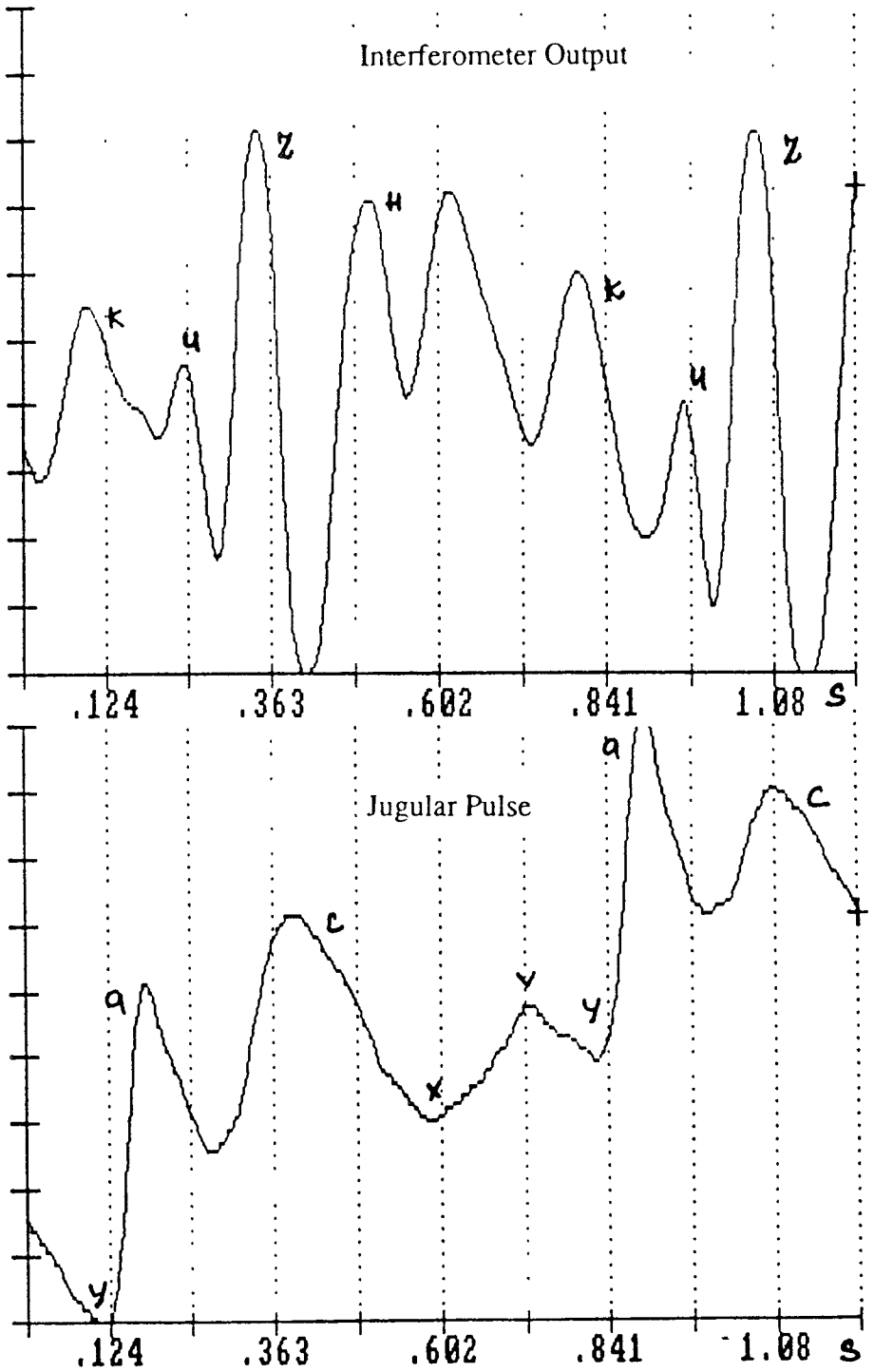

Figure A6.b To

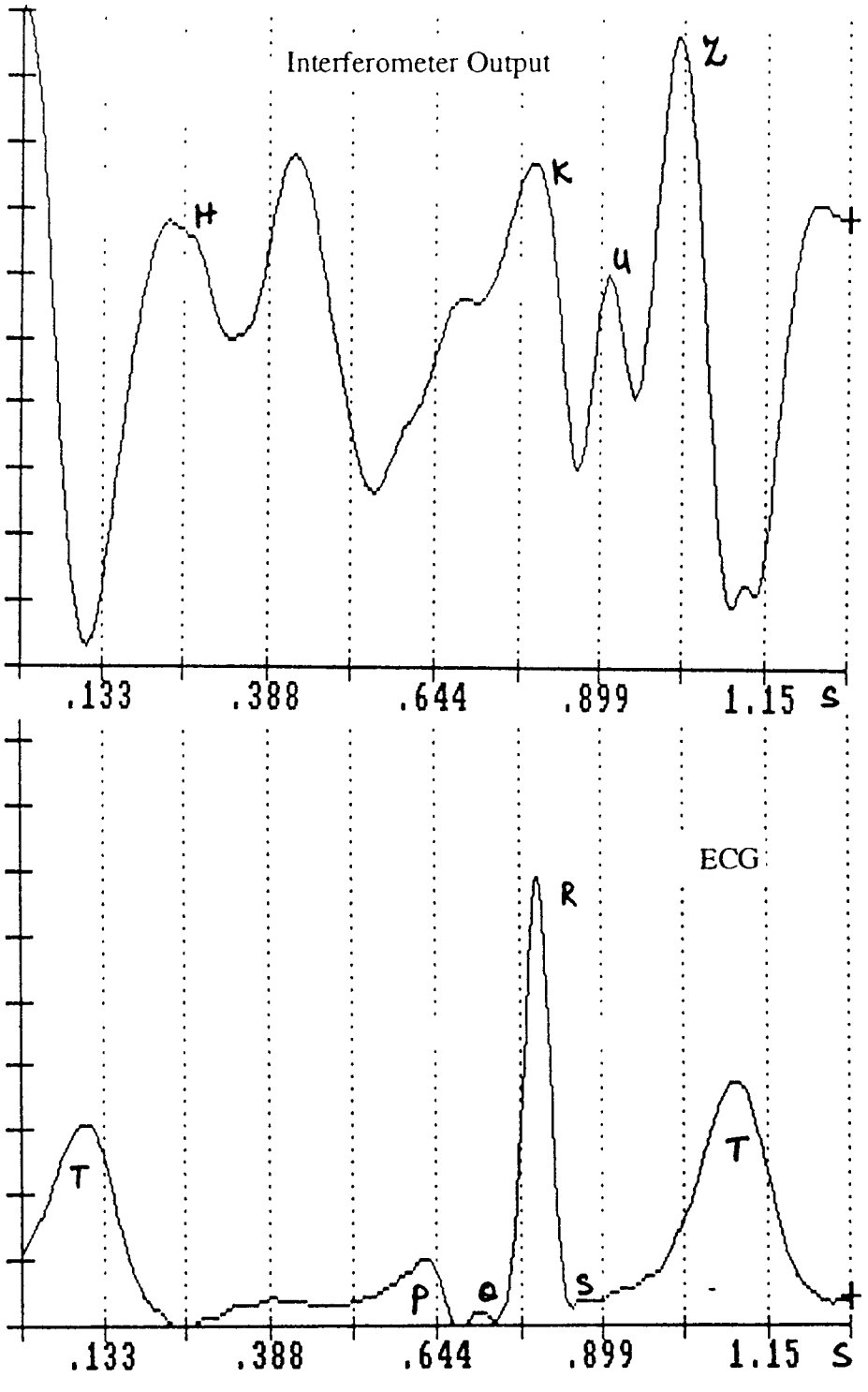

Figure A7.a Ko

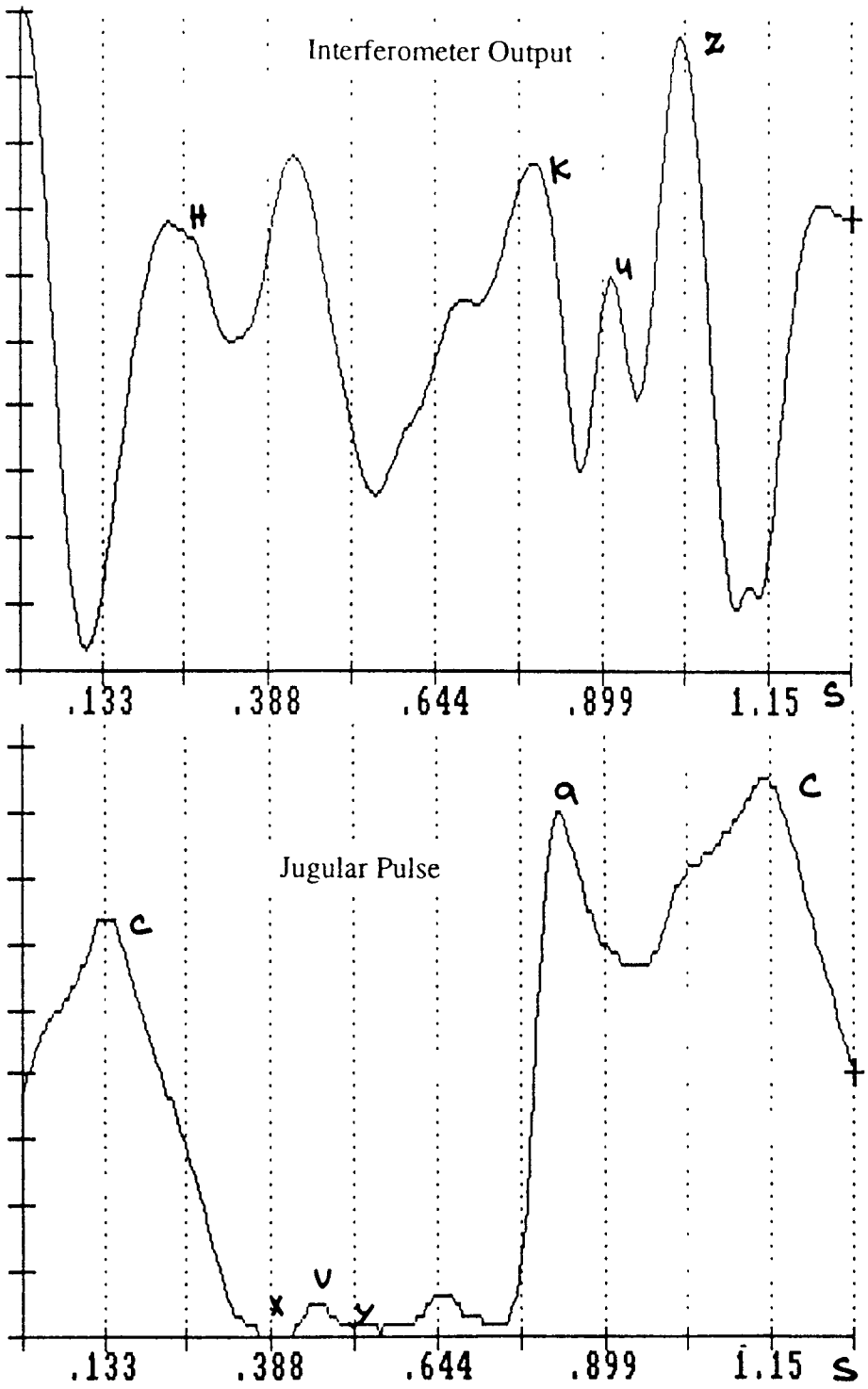

Figure A7.b Ko

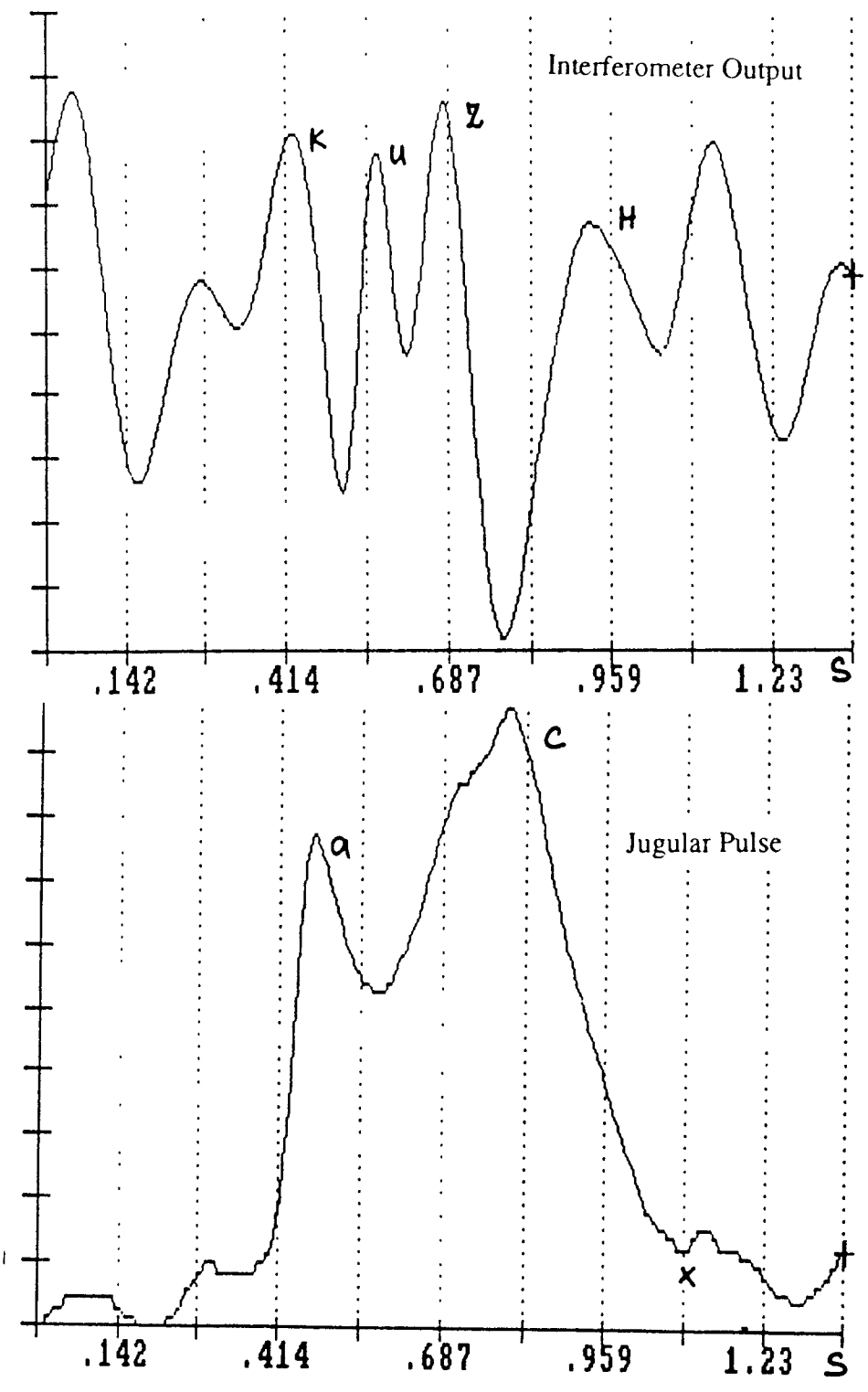

Figure A8.a Gi

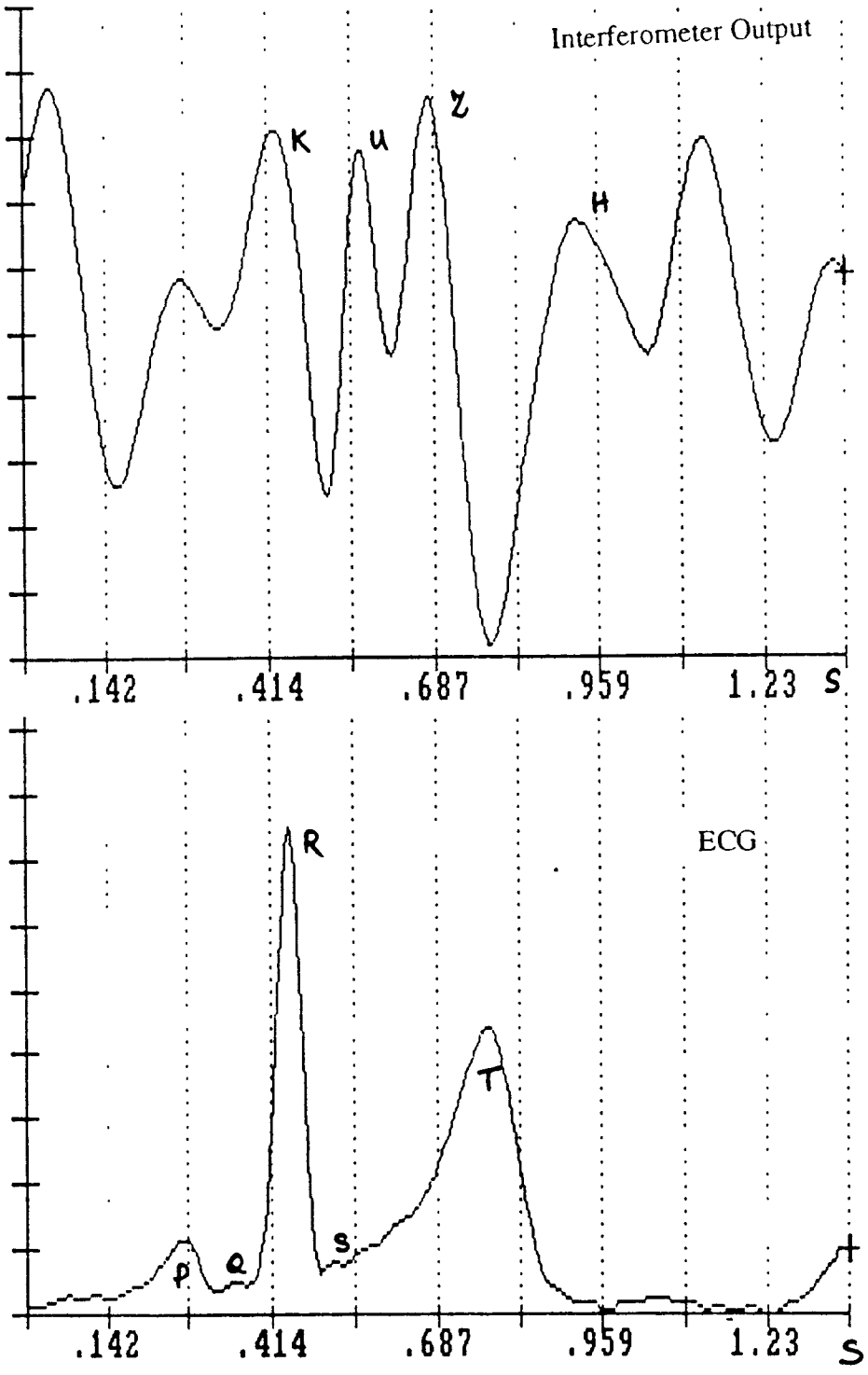

Figure A8.b Gi

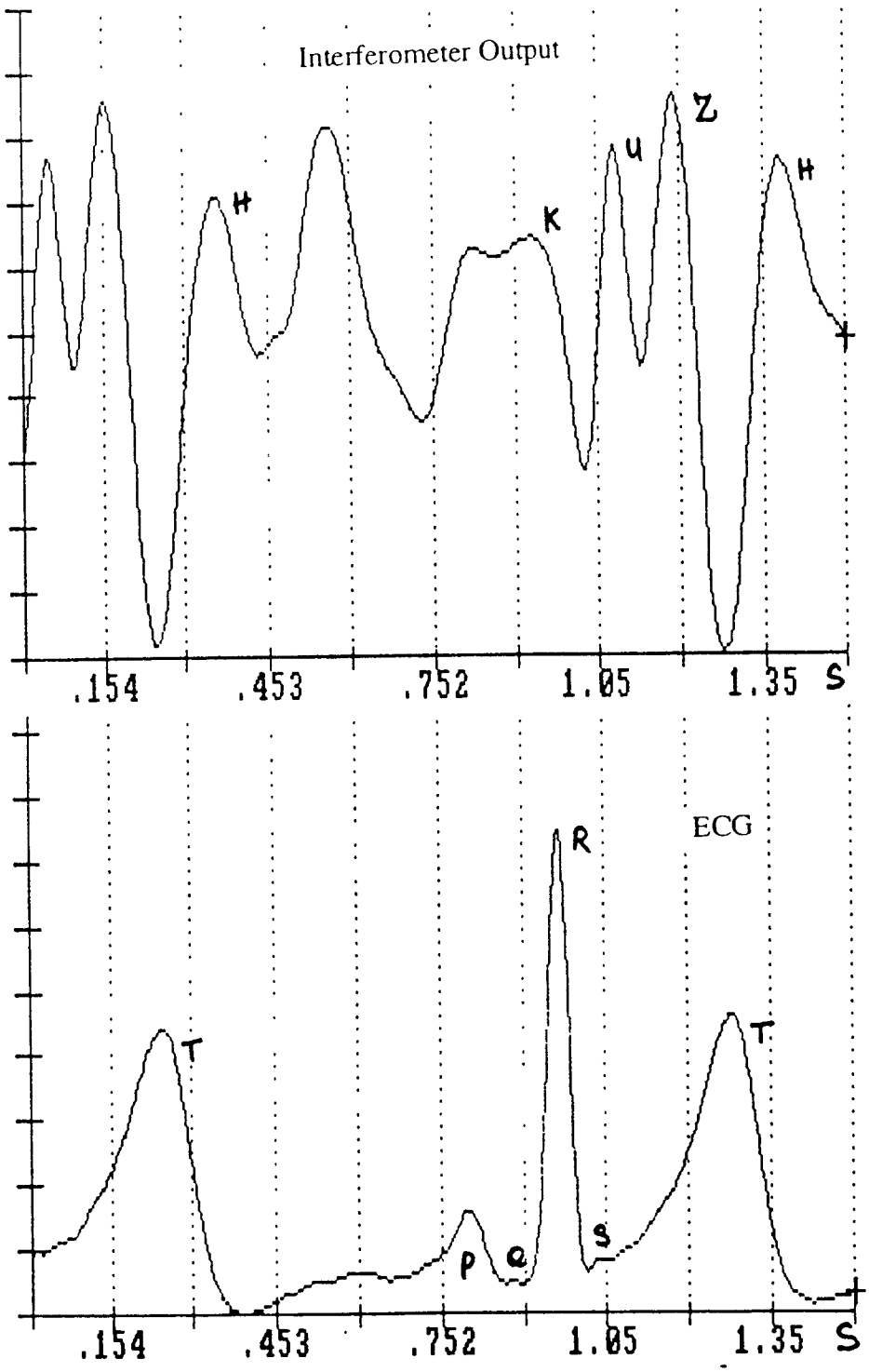

Figure A9.a Ju

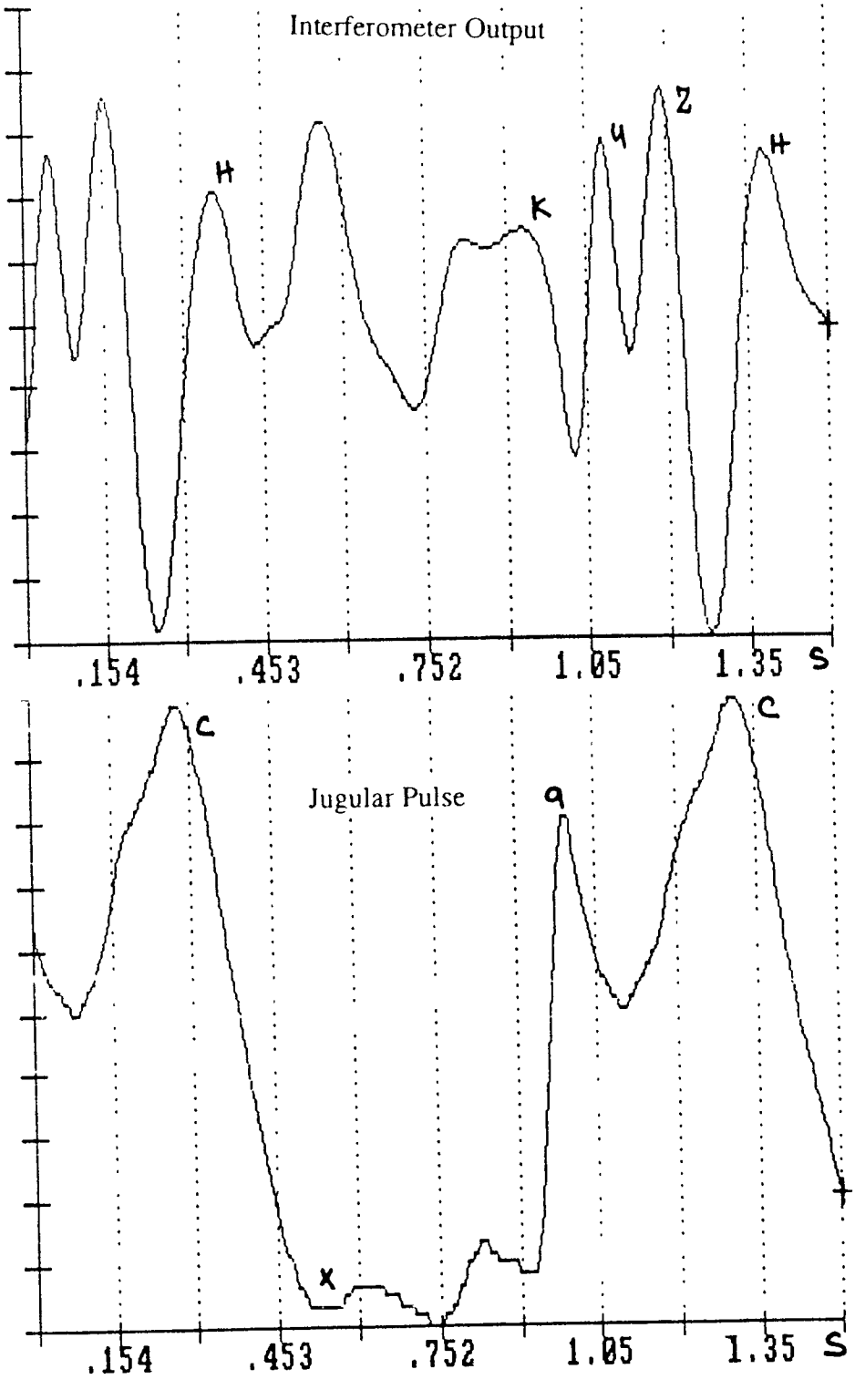

Figure A9.b Ju

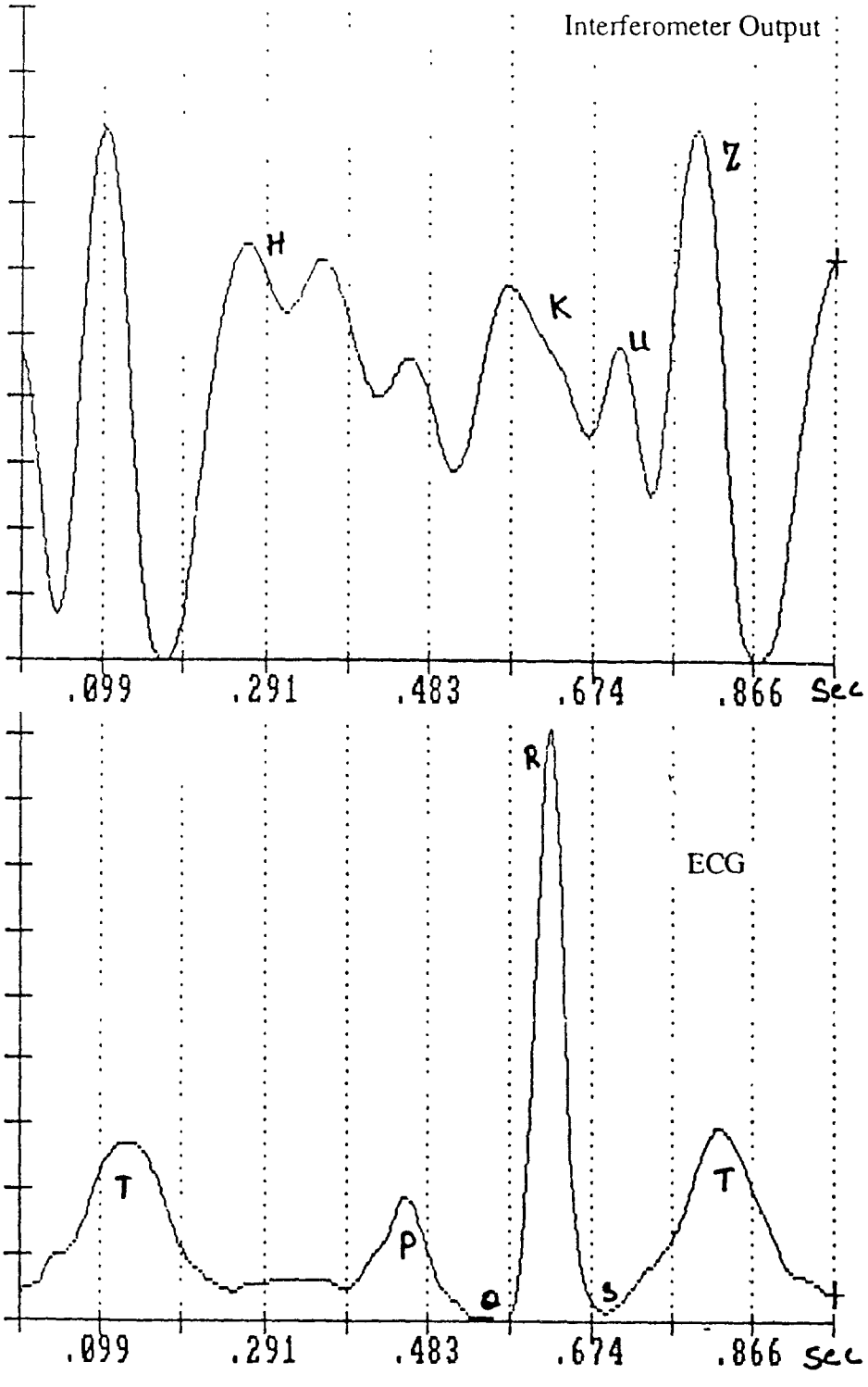

Figure A10.a Ma

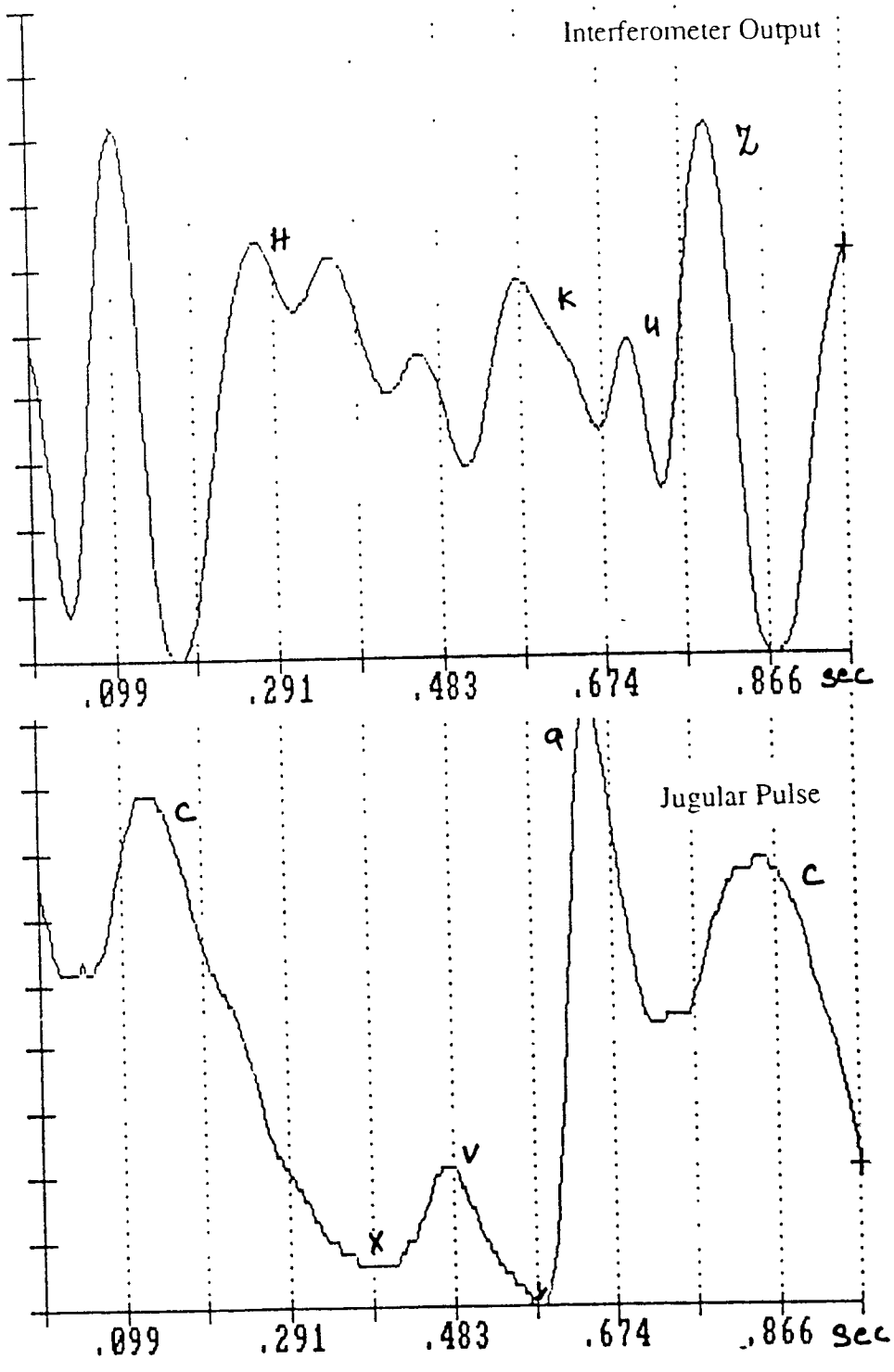

Figure A10.b Ma

APPENDIX B

DISKACQ1

PROGRAM FOR ACQUIRING 3 CHANNELS OF DATA

```
**********************************************************************
\ * Disk Acquisition and A/D.IN > FILE
\ *
**********************************************************************

15. STRING  FNAME                    \ for our filename...

" C:" FNAME  ": =                    \ set it up for

INTEGER SCALAR BUF.LENGTH
INTEGER SCALAR #BUFFERS

FNAME DEFER > ?CLUSTER.SIZE          \ note: ?CLUSTER.SIZE
needs to
4 * BUF.LENGTH : =                   \     see a drive spec and
                                     \     ignores the rest...
                                     \ let the buffers be four times
                                     \ the cluster size
                                     \ NOTE: this can be modified

DIM[ BUF.LENGTH ] DMA.ARRAY  DATA1
DIM[ BUF.LENGTH ] DMA.ARRAY  DATA2
```

```
DASH16
1 3 A/D.TEMPLATE AD0


: GO

  DATA1  DATA2  DMA.DOUBLE.TEMPLATE.BUFFERS    \
A/D.IN> FILE needs two !!

  BEGIN
    CR ." Enter the acquisition rate in Khz :  "
    #INPUT
  UNTIL


  1000. * INV DUP 1000 * CONVERSION.DELAY       \ kHz -> Msec

  A/D.INIT

  BEGIN
    CR ." How many buffers do you wish to collect:  "
    #INPUT
  UNTIL

  DUP  #BUFFERS : =
```

```
BUF.LENGTH * DUP ROT *          \ figure some statistics...

CR CR ." This acquisition will last " . ." seconds"
  CR ." and require " 512. / . ." kilobytes of disk space"
  CR
FILE.TEMPLATE
  AD0 FORM.DAS.SUBFILE              \ create a file template
  #BUFFERS TIMES                    \ #buffers from the stack...
END

CR ." Enter the file name (MUST OF FORM C:ppppppppp.xxx) : "
"INPUT FNAME ":=

CR CR ." Allocating " INVERSE.ON ." " FNAME "TYPE ." " INVERSE.OFF

FNAME DEFER> FILE.CREATE            \ pre-allocate the file

CR CR ." Collecting data .... "

100 SYSTEM.BUFFER.SIZE

FNAME DEFER> A/D.IN>FILE            \ do the

collection 32000 SYSTEM.BUFFER.SIZE

CR CR ." Acquisition complete. "
```

APPENDIX C

RESHAPE

PROGRAM TO SEPARATE 3 CHANNELS OF DATA

```
INTEGER DIM[ 2730 , 3 ] ARRAY PAUL.DAT
FILE.OPEN C:th4.dat
1 SUBFILE PAUL.DAT FILE> ARRAY
FILE.CLOSE

PAUL.DAT
SUB[ 1 , 2730 , 3 ]
DIM[ 2730 ] RESHAPE
INTEGER DIM[ 2730 ] ARRAY INTER
INTER :=
PAUL.DAT
SUB[ 2 , 2730 , 3 ]
DIM[ 2730 ] RESHAPE
INTEGER DIM[ 2730 ] ARRAY ECG
ECG :=
PAUL.DAT
SUB[ 3 , 2730 , 3 ]
DIM[ 2730 ] RESHAPE
INTEGER DIM[ 2730 ] ARRAY JUGU
JUGU :=
LOAD.OVERLAY WAVEOPS.SOV
INTER
```

```
DASH16.DMA.ALIGN
.18 SET.CUTOFF.FREQ
INTER SMOOTH
INTER :=
INTER FFT
COMPLEX DIM[ 2048 ] ARRAY INTER1
INTER1 :=
INTER1
ABS
INTER1 :=
INTER1 DUP *
REAL DIM[ 2048 ] ARRAY INTERF
INTERF :=

PAUL.DAT
SUB[ 2 , 2730 , 3 ]
DIM[ 2730 ] RESHAPE
INTEGER DIM[ 2730 ] ARRAY ECG
ECG :=
LOAD.OVERLAY WAVEOPS.SOV
ECG
DASH16.DMA.ALIGN
.18 SET.CUTOFF.FREQ
ECG SMOOTH
ECG :=
```

LOAD.OVERLAY WAVEOPS.SOV

JUGU

DASH16.DMA.ALIGN

.18 SET.CUTOFF.FREQ

JUGU SMOOTH

JUGU :=

APPENDIX D

SUBJECTS CONSENT FORM

Name of Project or Principal Investigator:

Dr. Peter E. Engler

Title of Project:

A Study of Pulsation of Chest Wall Using Microwave Interferometer

I acknowledge that on _____, I was informed by Dr. Engler of the New Jersey Institute of Technology of a project concerning or having to do with the following: USING A MICROWAVE INTERFEROMETER TO RECORD THE VIBRATION OF MY CHEST WALL.

I was told with respect to my participation in said project that:

(1) The following possible risk are involved: ILLUMINATION OF MY CHEST WALL WITH ELECTROMAGNETIC ENERGY AT A FREQUENCY OF 10 GHz, AND A POWER DENSITY THAT NOT EXCEED 0.01 mW/cm^2, PRECAUTION OF THE EYES WILL BE TAKEN.

(2) The following procedures are involved :

LYING QUIETLY ON A LAB BENCH, AND I MAY BE ASKED TO HOLD MY BREATH FOR A FEW SECOND, AND I MAY BE ASKED TO DO SOME MILD CALISTHENICS TO ELEVATE HEART RATE.

(3) The following benefits are expected by my participation:

DEVELOPMENT OF A UNIQUE CLINICAL INSTRUMENT AND DIAGNOSIS OF CARDIOVASCULAR PROBLEMS/ABNORMALITIES.

I am fully aware of the nature and extent of my participation in said project and possible risk involved or arising therefrom. I hereby agree, with full knowledge and awareness of all of the foregoing, to participate in said project. I further acknowledge that I have received a complete copy of this consent statement.

I also understand that I may withdraw my participation in said project at any time.

Date:_____ _____

Signature of Subject or

Responsible Agent

Place:_____

REFERENCES

Benchimol, A., and E. Diamond. 1963. "The Normal and Abnormal Apex Cardiogram." *Amer. J. Cardiol.* 12: 368.

Bigger, J., and B. Goldreyer. 1970. "The Mechanism of Paroxysmal Supraventricular Tachycardia." *Circ.* 42: 673.

Conover, M. 1980. "Understanding Electrocardiography." *The C.V. Moshby Company, St. Louis.* 4: 35-61.

Cranefield, P. 1977. "Action Potentials, After Potentials and Arrhythmias." *Circ. Res.* 41: 415.

Dressler, W. 1950. "Cardiac Diagnosis Without Laboratory Aid: Pulsation and Percussion Signs." *Med. Clin. N. Amer.* 34: 721.

Dressler, W., and E. Diamond. 1970. "Clinical Aids in Cardiac Diagnosis." *Grune and Stratton, New York.* 5: 50-101.

Gauer, O. 1970. "The Regulation of Extracellular Fluid Volume." *Ann. Rev. Physiol.* 32: 547.

Guyton, A. 1963. "A Concept of Negative Intestitial Pressure Based on Pressures in Implanted Perforated Capsules." *Circulation Res.* 12: 399-414.

Han, J. 1969. "Mechanisms of Ventricular Arrhythmias Associated With Myocardial Infarction." *Amer. J. Cardiol.* 24: 800.

Henry, J. 1971. "The Circulation." *Year book medical publishers, U.S.A.* 4: 65-78.

Mackenzie, J. 1902. "The Study of the Pulse, Arterial, Venous and Hepatic and of the Movements of the Heart." *The Macmillan Co., New York.* 6: 67-78.

McCally, M. 1968. "Hypodynamics and Hypogravics." *New York Academic Press, Inc.* 3: 12-45.

Narula, O. 1974. "Sinus Node Reentry." *Circ.* 50: 1114.

Overly, H., K. Vogel, and S. Blount. 1967. "Adaption Of the Right Ventricle to Increased Pressure Loads." *Cardiol. Digest.* 45: 29.

Rosen, K. 1973. "Junctional Tachycardia: Mechanisms, Diagnosis, Differential Diagnosis and Management." *Circ.* 47: 654.

Rushmer, R. 1970. "Cardiovascular Dynamics." *W. Saunders Company, Philadelphia.* 4: 67-90.

Selkurt, E. 1963. "Physiology." *Little, Brown and Company, Boston.* 13: 278-281.