5-31-1993

Comparison of heart rate with chest wall vibrations

Kofi M. Arbuah
New Jersey Institute of Technology

Follow this and additional works at: https://digitalcommons.njit.edu/theses

Part of the Biomedical Engineering and Bioengineering Commons

Recommended Citation
https://digitalcommons.njit.edu/theses/1719

This Thesis is brought to you for free and open access by the Electronic Theses and Dissertations at Digital Commons @ NJIT. It has been accepted for inclusion in Theses by an authorized administrator of Digital Commons @ NJIT. For more information, please contact digitalcommons@njit.edu.
Copyright Warning & Restrictions

The copyright law of the United States (Title 17, United States Code) governs the making of photocopies or other reproductions of copyrighted material.

Under certain conditions specified in the law, libraries and archives are authorized to furnish a photocopy or other reproduction. One of these specified conditions is that the photocopy or reproduction is not to be “used for any purpose other than private study, scholarship, or research.” If a user makes a request for, or later uses, a photocopy or reproduction for purposes in excess of “fair use” that user may be liable for copyright infringement.

This institution reserves the right to refuse to accept a copying order if, in its judgment, fulfillment of the order would involve violation of copyright law.

Please Note: The author retains the copyright while the New Jersey Institute of Technology reserves the right to distribute this thesis or dissertation.

Printing note: If you do not wish to print this page, then select “Pages from: first page # to: last page #” on the print dialog screen.
The Van Houten library has removed some of the personal information and all signatures from the approval page and biographical sketches of theses and dissertations in order to protect the identity of NJIT graduates and faculty.
ABSTRACT

Comparison of Heart Rate With Chest Wall Vibrations

by
Kofi M. Arbuah

A study is done using a Microwave Interferometer as a computer-assisted non-invasive cardiopulmonary monitor. The Microwave Interferometer is used to record the mechanical vibration of the anterior chest wall by recording the velocity of the displacement of the chest wall. The objective is to first, record mechanical vibrations of the anterior chest wall at different heart rates, and second, attempt to identify the relation between heart rate and chest wall vibration.

The system uses two channels, the first signal produced by the Microwave Interferometer is a record of the velocity of the displacement of the chest wall caused by the heart. The second signal is produced by the Electrocardiogram (ECG), serving as a timing reference signal.

The Interferometer and the ECG output signals are separately and simultaneously sent to an IBM-AT via an attached DASH-16 A/D board. The ASYST (A Scientific System) software package, is used to process and convert digitized data and then produce a graphic output of the results.

Results that have been obtained from testing the system have been encouraging. Repeated signals from the Interferometer are observed as the heart rate changes. The
amplitude of these signals vary with the heart rate. At this
time, it is observed that the relationship between heart
rate and the spectral distribution of chest wall vibration
cannot be clearly defined. However, it is believed that the
information gathered could be utilized in future research
involving the mechanical dynamics within the cardiac cycle
and chest wall vibrations.
COMPARISON OF HEART RATE WITH CHEST WALL VIBRATIONS

by
Kofi M. Arbuah

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 in Biomedical Engineering

Biomedical Engineering Committee

May 1993
Comparison of Heart Rate with Chest Wall Vibrations

by

Kofi M. Arbuah

Dr. Peter Engler, Thesis Adviser (date)
Associate Professor of Electrical Engineering, NJIT

Dr. Stanley S. Reisman, Committee Member (date)
Associate Chairperson and Professor of Electrical Engineering, NJIT

Dr. David S. Kristol, Committee Member (date)
Professor of Chemistry and Director of the Center for Biomedical Engineering, NJIT
BIographiesK Sketch

Author: Kofi M. Arbuah

Degree: Master of Science in Biomedical Engineering

Date: May 1993

Undergraduate and Graduate Education:

• Master of Science in Biomedical Engineering, New Jersey Institute of Technology, Newark, NJ, 1993

• American Society of Clinical Pathologists Certification-Medical Technology Category, University of Medicine and Dentistry of New Jersey, Newark, NJ, 1984

• Bachelor of Arts in Biology, Mansfield University, Mansfield, PA, 1982

Major: Biomedical Engineering
This thesis is dedicated to the memory of the late Mr. Joseph Edwin Arbuah.
I would like to thank Dr. Peter Engler for giving me the opportunity to take on this interesting research project. His guidance and moral support is greatly appreciated.

Special thanks goes to Dr. David Kristol for encouraging, and also, helping me to find the subjects for this project.

The help and suggestions on spectral analysis and statistical operations from Dr. Stanley Reisman is greatly appreciated.

I am very grateful to Mr. Jackie Bush from the University of Medicine and Dentistry of New Jersey library for his patience and support in researching this thesis project.

Special thanks goes to Mr. John Andrews from the Biomedical Engineering Laboratory for the HPPS software.

Finally, I would like to thank all of the subjects that took the time to volunteer for this project.
# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Chapter</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 CARDIOVASCULAR PHYSIOLOGY</td>
<td>1</td>
</tr>
<tr>
<td>1.1 Introduction</td>
<td>1</td>
</tr>
<tr>
<td>1.2 Cardiac Cycle</td>
<td>2</td>
</tr>
<tr>
<td>1.2.1 Atrial Contraction</td>
<td>2</td>
</tr>
<tr>
<td>1.2.2 Ventricular Isovolumic Contraction</td>
<td>4</td>
</tr>
<tr>
<td>1.2.3 Rapid Ventricular Ejection</td>
<td>5</td>
</tr>
<tr>
<td>1.2.4 Decreased Ventricular Ejection</td>
<td>7</td>
</tr>
<tr>
<td>1.2.5 Isovolumic Relaxation</td>
<td>8</td>
</tr>
<tr>
<td>1.2.6 Rapid Ventricular Filling</td>
<td>8</td>
</tr>
<tr>
<td>1.2.7 Diastasis</td>
<td>9</td>
</tr>
<tr>
<td>1.3 Systolic Time Intervals (Clinical Significance)</td>
<td>10</td>
</tr>
<tr>
<td>1.3.1 Preload</td>
<td>10</td>
</tr>
<tr>
<td>1.3.2 Afterload</td>
<td>11</td>
</tr>
<tr>
<td>1.3.3 Contractile State of the Myocardium</td>
<td>12</td>
</tr>
<tr>
<td>1.3.4 Myocardial Disease</td>
<td>13</td>
</tr>
<tr>
<td>1.3.5 Left-Ventricular Ejection Time (LVET)</td>
<td>14</td>
</tr>
<tr>
<td>1.4 Heart Valve Motion and Heart Sounds</td>
<td>15</td>
</tr>
<tr>
<td>1.5 Research Objective</td>
<td>16</td>
</tr>
<tr>
<td>2 SYSTEM CONFIGURATION</td>
<td>18</td>
</tr>
<tr>
<td>2.1 System Hardware Construction</td>
<td>18</td>
</tr>
<tr>
<td>2.2 Microwave Interferometer</td>
<td>19</td>
</tr>
<tr>
<td>2.3 DASH-16 Acquisition and Control Board</td>
<td>21</td>
</tr>
<tr>
<td>2.4 System Software-Introduction To Asyst</td>
<td>22</td>
</tr>
</tbody>
</table>
Chapter 3 SYSTEM SOFTWARE DESCRIPTION..............................................25
  3.1 Signal Acquisition.................................................................25
  3.2 Signal Processing Executed By Asyst.................................26
    3.2.1 Interferometer Output Signal of Chest Wall Vibrations........................................26
    3.2.2 Interferometer Signal Frequency Spectrum..............................26
4 EXPERIMENTAL PROCEDURE, DATA ANALYSIS AND DISCUSSION....28
  4.1 Measurement Parameters......................................................28
  4.2 Subject Description............................................................28
  4.3 Changes in Heart Rate and Chest Wall Vibration.........................29
  4.4 Interferometer Signal Frequency Distribution Analysis..................30
  4.5 Conclusion..............................................................................32
APPENDIX A Data Acquisition System Configuration.........................34
APPENDIX B Subject's Consent Form...............................................36
APPENDIX C Data Collection Procedure..........................................37
APPENDIX D Interferometer Power-Up Procedure...............................39
APPENDIX E Time Series Analysis (Power Spectrum Routine)..............40
APPENDIX F Glossary of Abbreviations..........................................43
APPENDIX G Disk Acquisition and A/D.IN>FILE Routine......................44
APPENDIX H Illustration of Other Frequency Spectra.........................46
APPENDIX I Illustration of the Effect of Zero Padding on Frequency Spectrum Analysis..........................51
BIBLIOGRAPHY................................................................................52
<table>
<thead>
<tr>
<th>Table</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Table of Moments from Frequency Spectrum of an Interferometer Signal Equivalent to One Cardiac Cycle Length</td>
<td>facing 31</td>
</tr>
</tbody>
</table>
LIST OF FIGURES

<table>
<thead>
<tr>
<th>Figure</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.1</td>
<td>Events of the Cardiac Cycle</td>
<td>facing2</td>
</tr>
<tr>
<td>1.2</td>
<td>Valve Movements</td>
<td>facing15</td>
</tr>
<tr>
<td>1.3</td>
<td>System Block Diagram</td>
<td>facing17</td>
</tr>
<tr>
<td>2.1</td>
<td>System Hardware Block Diagram for Microwave Interferometer Based Cardiopulmonary Monitoring</td>
<td>facing18</td>
</tr>
<tr>
<td>2.2</td>
<td>Basic Microwave Interferometer System Block Diagram</td>
<td>facing20</td>
</tr>
<tr>
<td>3.1</td>
<td>Example of Power Spectrum Analysis Using Two Separate Signals at Frequencies of 5 Hz and 18 Hz</td>
<td>facing27</td>
</tr>
<tr>
<td>4.1</td>
<td>Interferometer and ECG Signals from Subject(KMA). Heart Rate = 110 Beats Per Minute</td>
<td>facing29</td>
</tr>
<tr>
<td>4.2</td>
<td>Interferometer and ECG Signals from Subject(KK). Heart Rate = 135 Beats Per Minute</td>
<td>facing30</td>
</tr>
<tr>
<td>4.3</td>
<td>Frequency Spectrum of Interferometer Signal, with Zero Padding, from Subject(JM). Heart Rate = 86 Beats Per Minute</td>
<td>facing30</td>
</tr>
<tr>
<td>H1</td>
<td>Frequency Spectrum of Interferometer Signal, with Zero Padding, from Subject(JM). Heart Rate = 94 Beats Per Minute</td>
<td>46</td>
</tr>
<tr>
<td>H2</td>
<td>Frequency Spectrum of Interferometer Signal, with Zero Padding, from Subject(JM). Heart Rate = 115 Beats Per Minute</td>
<td>47</td>
</tr>
<tr>
<td>H3</td>
<td>Frequency Spectrum of Interferometer Signal, with Zero Padding, from Subject(KUEN). Heart Rate = 94 Beats Per Minute</td>
<td>48</td>
</tr>
<tr>
<td>H4</td>
<td>Frequency Spectrum of Interferometer Signal, with Zero Padding, from Subject(KMA). Heart Rate = 110 Beats Per Minute</td>
<td>49</td>
</tr>
<tr>
<td>H5</td>
<td>Frequency Spectrum of Interferometer Signal, with Zero Padding, from Subject(KK). Heart Rate = 135 Beats Per Minute</td>
<td>50</td>
</tr>
<tr>
<td>Ia</td>
<td>Frequency Spectrum of Interferometer Signal, without Zero Padding, from Subject(JM). Heart Rate = 86 Beats Per Minute</td>
<td>51</td>
</tr>
</tbody>
</table>
Figure

lb  Frequency Spectrum of Interferometer Signal, with Zero Padding, from Subject (JM). Heart Rate = 86 Beats Per Minute. ........................................ 51
CHAPTER 1

CARDIOVASCULAR PHYSIOLOGY

1.1 Introduction

The heart is formed from cardiac muscle. Its function is to receive blood at low pressure from the venous system and to deliver the blood to the arterial system at pressures adequate enough to maintain the circulatory needs of the body. It is divided into four chambers, two atria and two ventricles.

The heart operates as two pumps joined together in series. The pump seated to the right and front in the chest, is the right ventricle. It pushes blood via the pulmonary circulation and the left atrium into the left ventricle. The pump emplaced to the left and back in the chest, is the left ventricle. It pumps blood via the systemic circulation into the right atrium. Other little but prominent forces that bring about movement of blood include the skeletal muscle pump and the respiratory pump. In any case, the major cause of blood movement originates from the heart.

Both ventricles operate as periodic pumps that cease pumping to fill, and cease filling to pump. The way that the heart is excited and effectuated to contract is planned in such a manner that both pumps fill and drain simultaneously. Diastole period is the interval of time it takes for the ventricles to fill. The interval of time it takes for the ventricles to drain or pump blood is known as the systolic
Some of the most important events of the cardiac cycle. Phases: \( A \) = atrial contraction; \( B \) = ventricular isovolumic contraction; \( C \) = rapid ventricular ejection; \( D \) = decreased ventricular ejection; \( E \) = isovolumic relaxation; \( F \) = rapid ventricular filling; \( G \) = diastasis. Other designations: \( AP \) = aortic pressure; \( LV P \) = left ventricular pressure; \( L A P \) = left atrial pressure; \( LV \, Vol \) = left ventricular volume; \( AF \) = aortic flow; \( AV \) = aortic valve; \( MV \) = mitral valve; \( P \) = pulmonary valve; \( TV \) = tricuspid valve (a solid bar above the designation means that the valve is closed); \( E C G \) = electrocardiogram; \( P, Q, R, S \) = component waves of the ECG; \( 1, 2, 3 \) = heart sounds; \( RV P \) = right ventricular pressure; \( PA P \) = pulmonary arterial pressure; \( RA P \) = right atrial pressure.

**Figure 1.1** Events of the Cardiac Cycle (Reproduced from Vick, Contemporary Medical Physiology).
period. Diastole and systole together make up the cardiac cycle, which is the recurring entity of cardiac action. Diastole and systole are broken down into stages to enable us to examine all the significant events in the cardiac cycle.

1.2 Cardiac Cycle
1.2.1 Atrial Contraction (Phase A, Fig. 1.1)
The set of heart cells that begin the cardiac cycle, the cardiac pacemaker, is situated where the right atrium and the superior vena cave connect. This is known as the sinistral (SA) node. The SA node electrically excites the right atrium first; this electrical stimulation is distributed over both atria, which creates the P wave of the electrocardiogram (ECG). Electrical stimulation results in mechanical atrial compression, which creates both the right and left atrial pressure profiles, see figure 1.1 phase A.

Since the atrioventricular valves (AV-valves), which separate the passageway between the atria and the ventricles, are open during diastole and no valves are located between the atria and the venous system, diastolic pressure waves are dispatched onward into each ventricle and backward into the veins. The pressure waves created in the venous system by right atrial contraction is recorded as the a-wave of the jugular venous pulse.
During atrial contraction, blood is calmly moving from the large veins into the atria, and the momentum maintains the movement regardless of the counteracting atrial pressure wave. The atrial pressure wave facilitates blood flow from the atria into the ventricles. However, its effect in the gradually beating heart of a normal resting individual is negligible. During this period, the portion of time for diastole is comparatively long, and the ventricles fill up completely with blood from the large veins via a flaccid atria, which are simply operating as channels between the venous system and the ventricles. So in the normal resting heart, the ventricles are virtually filled at the time that the atria contract, and the effect of atrial pumping action is negligible.

On the other hand, whenever the cardiac output is increased from exercise for example, the heart rate is increased, decreasing the cardiac cycle period, consequently reducing the diastolic interval. Since there is less time for the heart to fill by venous inflow alone, atrial contraction becomes an important participant in ventricular filling. A significant effect of atrial contraction, regardless of an increase or a decrease in heart rate, is that it facilitates the suspension of the AV-valves into the correct position to be closed during ventricular contraction. Generally, the pressure waves resulting from atrial contraction are not sensed, but they can be recorded.
The fourth heart sound is associated with these rumbles, when they are heard.

1.2.2 Ventricular Isovolumic Contraction (Phase B, Fig. 1.1)
Ventricular systole is initiated by ventricular isovolumic contraction, see figure 1.1 phase B. Ventricular systole goes on for about 15 milliseconds in the right ventricle and around 40 milliseconds in the left ventricle. Isovolumic contraction starts close to the peak of the R-wave of the ECG, and increases swiftly. In each ventricle, blood is pushed backward upon the atrium to close the AV-valve. The pressure wave resulting from this activity and the impact of the ventricular blood mass upon the rigid tricuspid and mitral valves, is detected as the beginning constituents of the first heart sound. Supporting fiber strands, known as the chordae tendineae, join the borders of the valve leaflets to muscular pillars that protrude from the walls of the ventricles. These strands prevent the valves from backing up into the atria. The papillary muscles contract immediately following the start of ventricular excitation, and they provide supplementary support to the chordae tendineae. The AV-valves bulge into the atria, and the atrial pressures increase. This increase in pressure creates a venous pressure wave which is recorded in the jugular venous pulse as the c-wave.

Since the pulmonic valve and the aortic valve are closed, ventricular blood volumes remain unchanged, hence
the term "isovolumetric". At the same time, the pressure in each ventricle increases rapidly. The blood in the ventricles build up potential energy as a result, and higher pressures are recorded during extended periods of isovolumetric contraction. Isovolumic contraction is complete when pulmonary artery pressure and aortic artery pressure reach their low minimum, that is, diastolic pressure.

1.2.3 Rapid Ventricular Ejection (Phase C, Fig.1.1)
Opening of the aortic valve and the pulmonic valve mark the beginning of ejection. At this time, the pressure in the ventricle is greater than that in the artery, see figure 1.1 phase C. The valves open suddenly as the ventricular pressure builds up very quickly, thus ejecting blood out of the heart in a trajectory manner. Left ventricular ejection, also known as aortic flow, goes on for 50 to 100 milliseconds, and then diminishes. Most of the blood delivered from the ventricle to the aorta occurs during the rapid ejection period. The blood that is delivered when the pulmonic valve and the aortic valve open collides with a static mass of blood in each of the great arteries- this is consistent with prevention of arterial collapse subsequent to the previous closing of the valves.

Some of the blood flowing into the arteries is stopped at the point of collision with the static mass of blood, and its kinetic energy is converted into potential energy. Arterial volume and pressure increases, and movement of
blood is accelerated. The pulmonary artery and the aorta quickly dilate, causing a vibratory effect, which is detected as the extension of the first heart sound. As the blood flow in the great arteries increases, it becomes turbulent. This is recorded as the last part of the first heart sound.

Since the tricuspid valve and the mitral valve are closed during this phase, the atria act as the storage for venous inflow. Despite the increase in atrial volume, left atrial pressures are shown to decrease during phase C in Fig.1.1. The decrease is the result of thoracic blood volume depression caused by a decrease in left ventricular volume. Thus, the blood outflow from the thorax via the arterial system expedites blood inflow into the thorax via the venous system.

Ventricular pressure surpasses arterial pressure during the rapid ventricular ejection phase, thus blood flow in the pulmonary artery and the aorta is increased. The region of laminar flow near the walls of these large vessels is small with respect to their cross-sectional area. Hence, impedance to blood flow is very small and higher flow speed is reached by mainly overcoming the inertia of the blood mass. Consequently, as blood is set in motion, a tendency for continuous flow due to their propulsion is observed.

As energy is invested in the blood to bring about flow, energy is extracted from the blood to effect cessation of flow. The declining ratio of aortic volume with respect to
time of aortic flow subsequent to reaching maximum flow rates, demonstrates a loss of kinetic energy, and the increase in pressure in the pulmonary artery and the aorta illustrates the energy conversion from kinetic energy to potential energy. Hence, energy transferred to the blood, originally by ventricular contraction, is collected in the elastic walls of the large arteries in early systole and utilized later to maintain flow during diastole. The rapid ventricular ejection phase is completed when the left ventricular pressure is equal to the aortic pressure.

1.2.4 Decreased Ventricular Ejection (Phase D, Fig.1.1)

The start of decreased ejection phase is characterized by a decline in left ventricular pressure below that of aortic pressure. Blood is calmly flowing into the aorta driven by the force of the previous ventricular contraction. The aortic pressure decrease is slow, because the aorta's elastic walls are using some of the energy that is needed to complete the decrease in ventricular outflow and initiate the closure of the aortic valve. The pressure gradient between the aorta and the ventricles, at the end of ejection, causes blood to flow back toward the ventricles. The result of this backflow is the closure of the aortic valve. The same sequence of events occurs in the pulmonary artery, right ventricle, and the pulmonic valve. The vibration produced from the cessation of backflow in the
aorta and in the pulmonary artery by the valves is perceived as the second heart sound.

Since the amount of outward blood flow from the thorax via the aorta decreases and the atria are being filled with blood streaming in from the systemic and pulmonary veins, atrial pressures build up throughout the course of decreased ventricular ejection phase. Electrical repolarization of the ventricles is represented by the T-wave of the ECG. Closure of the aortic and the pulmonic valves mark the end of the decreased ventricular ejection phase.

1.2.5 Isovolumic Relaxation (Phase E, Fig.1.1)
Subsequent to the closure of the aortic and the pulmonic valves, ventricular pressures quickly dip as the myocardium loosen. There is no volume change in the ventricles, and the aortic pressure bounces back after the fall caused by aortic valve closure. The pressures in the great arteries decrease uniformly as they are separated from additional intake. Isovolumic relaxation ends when ventricular pressure falls to less than that of the corresponding atrial pressure, and also, when the AV-valves open.

1.2.6 Rapid Ventricular Filling (Phase F, Fig.1.1)
In the course of ventricular contraction, coupled with shortening of the muscle fibers and reduction of ventricular size, some stress is created in the connective tissue that ties the muscle layers together. Letting go of this stress, during isovolumic relaxation, helps to increase ventricular
volumes. This stress is greater in the thick-walled left ventricle, where the pressure can fall below that of the atmospheric pressure.

So, in the beginning of diastole, there is an increased pressure gradient between the aorta and the ventricles, hence the ventricles fill rapidly. During the rapid ventricular filling phase, the AV-valves' opening are wider, the atria and the great veins are filled more efficiently, and the ventricles are more flexible.

Consequently, a small pressure gradient is enough to speed up blood flow into the ventricles. Ventricular flexibility decreases as the ventricles bulge up. This stops blood inflow and the resulting vibration produced is sensed by auscultation as the third heart sound. Generally, ventricular filling occurs in early diastole in the normal heart at rest. This is a significant adaptation, since a physiological increase in cardiac output increases the heart rate, which results in the abbreviation of the diastole period.

1.2.7 Diastasis (Phase G, Fig. 1.1)
This phase is marked by relatively little activity. Blood flows under the driving force of the pressures in the elastic arteries, and the ventricles fill up slowly. The atria and great veins discharge the blood accumulated during systole and then, load up again with blood flowing in from the systemic circulation. No electrical activity is taking
place and no sounds are perceived. The duration of diastasis depends on the heart rate. Diastasis ends at the P-wave of the ECG, where another cardiac cycle begins (1).

1.3 Systolic Time Intervals (Clinical Significance)

Systolic time intervals (STI) apply to the different phases of systole, whose periods are identified by the hemodynamic and mechanical modulations occurring in that time period. There are four vital elements that influence these hemodynamic and mechanical modulations:

1. **Preload** (Diastolic fiber length, Diastolic volume or Left-ventricular end-diastolic pressure)

2. **Afterload** (Aortic diastolic pressure or wall tension after the aortic valve opens)

3. **Contractile state of the myocardium**

4. **Abnormal wall contraction due to disease**

1.3.1 Preload

The term preload applies to the extent of diastolic strain on the ventricle just before systole. The first report on factors governing preload and the effect of a deviation in this factor was reported by Otto Frank in 1895, and afterwards, in the studies done by Wiggers and Starling. Frank's studies on frog hearts revealed that an increase in diastolic volume resulted in the elevation of cardiac output. Wiggers resolved, in 1914, that the rate of pressure rise during Isovolumic contraction and the maximum systolic pressure, were influenced by alterations in initial
diastolic strain, provided the intrinsic contractility was unchanged.

These studies, however, did not explain why cardiac output was essentially associated with initial diastolic tension, the left ventricular end-diastolic pressure or with the diastolic volume. Using a heart-lung preparation, Starling et al. came to a determination that the mechanical energy produced was influenced by the diastolic fiber length. This is known as the Frank-Starling mechanism.

1.3.2 Afterload
This is the pressure in major vessels when the pulmonary and aortic valves open. A higher diastolic pressure results in a longer interval between the start of diastolic pressure rise and the opening of the pulmonary valve. On the other hand, a lower diastolic pressure results in a shorter interval between the start of diastolic pressure rise and opening of the pulmonary valve, hence, shortening the Isovolumic contraction (IVC) time. Isovolumic contraction time is a measure of the ventricular contraction rate. Protraction of diastole during atrial fibrillation, for example, results in a larger outflow from the peripheral vessels, hence decreasing the diastolic pressure or afterload. Abbreviating isovolumic contraction time, in these instances, does not necessarily increase the inherent contraction velocity.
Clarification of this problem was made in studies leaving heart rates and peripheral resistance constant. It was determined that at constant heart rate and peripheral resistance, a faster diastolic filling rate resulted in a decreased Isovolumic contraction time, and a slow filling rate protracted the contraction time. Contraction velocity was influenced by the inherent velocity of each myocardial fiber, except if there was a deviation in contractility.

1.3.3 Contractile State of the Myocardium

Studies done by Wiggers found that epinephrine reduced IVC time. It was also discovered that comparable results were obtained with sympathetic stimulation and with digitalis (inotropic agent). Contrary to the measurable changes in myocardial operation, like IVC time, caused by changes in Preload and Afterload, an improvement in myocardial contractility results in changes in the nature of contraction of the myocardial fibers. There is an increase in the force and velocity of myocardial contraction. In other words, increased contractility facilitates the Force-Velocity relationship and alters the maximal velocity of contraction ($V_{\text{max}}$). Myocardial contractility is determined at cardiac catheterization. The rate of left-ventricular pressure rise ($\text{dp/dt}$), time to maximal left-ventricular pressure (maximum $\text{dp/dt}$), and maximum $\text{dp/dt}$ ratio, which is maximum $\text{dp/dt}$ divided by the instantaneous left-ventricular pressure, are measured.
1.3.4 Myocardial Disease
The final element determining the duration of STI's is disease type and severity. In the case of Ischemic heart disease, hypertrophy, cardiomyopathy, and myocardial infarction, it has been determined that the pressure elevation rate (ventricular dp/dt) is reduced, and the duration of Isovolumic contraction is extended.

Abnormal wall motion (Akinesis, Dyskinesia, or Asynchrony) causes myocardial asynergy, which results in impaired contractility and increased wall tensions. The overall effect is extension of Isovolumic contraction time during ventricular systole. Thus, a change of rate of pressure rise (dp/dt) results in the protraction of Isovolumic contraction time in ventricular systole, due to the effect of myocardial disease.

Protraction of IVC time is mostly indicative of inadequate rate of ventricular pressure rise, due to myocardial disease. On the other hand, a decrease in IVC time is generally caused by increased velocity of myocardial contraction. Mitral and aortic valvular diseases are exceptions to the findings above, when reduced resistance to ejection and reduced diastolic pressure, or both, are presented. Where we have indications of both valvular lesions and myocardial disease, Isovolumic contraction period is influenced by the prevalent lesion.
1.3.5 Left-Ventricular Ejection Time (LVET)

Contrary to Isovolumic contraction period, Left-ventricular ejection time does not always precisely define the functional aspects of heart operation, as does IVC time. LVET is however influenced by the heart rate. From previous studies, LVET decreases as heart rate increases and forms a direct relationship with Stroke volume. The four elements that influence Isovolumic contraction time also control LVET. So in the absence of any deviation in myocardial contraction, LVET is protracted when Preload period is increased, when Afterload period is decreased, and when the Stroke volume increases.

LVET is also affected by myocardial disease. A healthy ventricle unloads two-thirds of its End-diastolic volume per beat to produce a Stroke volume of about fifty milliliters. A twice enlarged ventricle, due to disease, would only unload one-third of its End-diastolic volume to produce the same Stroke volume. So in this case, the extent of fiber contraction is reduced, which eventually decreases LVET. Aortic and mitral valvular diseases have complementary effects on LVET. Aortic valve disease protracts LVET, and mitral valve disease decreases LVET. The extent of LVET prolongation is the determinant for aortic stenosis and insufficiency, lacking the presence of a pre-existing heart failure. The extent of LVET reduction is the determinant for mitral stenosis or regurgitation.
 Movements of the valves. A shaded box indicates that the valve is closed; an open space indicates that the valve is open. The electrocardiogram is shown to permit reference to the cardiac cycle. The heart sounds (1, 2, 3) are shown to permit relation to the motions of the valves. The overlapping of the boxes (above sounds 1 and 2) indicates the periods of isovolumic contraction and isovolumic relaxation of each ventricle. Note that the period of right ventricular ejection exceeds the period of left ventricular ejection. The difference shown here probably is minimal. Increase of the right ventricular stroke volume would move the closure of the pulmonic valve to the right and cause splitting of the second heart sound.

Figure 1.2 Valve Movements (Reproduced from Vick, Contemporary Medical Physiology).
Providing that Preload and Afterload are normal, and there is no pre-existing myocardial and valvular disease, a direct relationship is seen between IVC time, LVET, and myocardial function. A reduction of the IVC time, prolongs LVET, which means better contractility and increased cardiac output.

Because sudden changes in Preload, Afterload, and certain inotropic conditions, like shock, hypotension, trauma etc., occur in the clinical setting, it is evident that deviations in IVC time, and LVET or both, are indicative of Left-ventricular disease (2).

1.4 Heart Valve Motion and Heart Sounds (Figure 1.2)
The first heart sound is complicated and rather prolonged. It is produced when the mitral and the tricuspid valves close, the aortic and the pulmonic valves open, and blood initially gushes into the aorta and the pulmonary artery. Valve movements are not synchronous: The mitral valve closes first, followed by the closing of the tricuspid valve, then the opening of the pulmonic valve, and then the opening of the aortic valve, shown in figure 1.2. So the pattern here is that, right ventricular events occur amid left ventricular events.

The second heart sound is produced when the aortic and the pulmonic valves close. The aortic valve closes first, however the gap between that event and closing of the
pulmonic valve depend on the respiratory cycle. At the end of quiet expiration, the events are only a few milliseconds apart. During inspiration, the second heart sound consist of two separate elements, referred to as "split". The second heart sound "splits" in conditions that prolong LVET and slow the closing of the pulmonic valve. During inspiration, "splitting" occurs when there is an increase in right ventricular Stroke volume (1).

1.5 Research Objective

The thesis objective is to record the velocity of the displacement of the anterior chest wall at different heart rates, and then try to find out if there is a relationship between the mechanical dynamics within the cardiac cycle and chest wall vibration. Analysis of the relationship between heart rate and chest wall vibration is done by examining the frequency spectrum of the Interferometer signal at different heart rates. It is hypothesized that at various heart rates, the associated anterior chest wall vibration should be related to the frequency characteristics of heart motion. ECG and Microwave Interferometer outputs are acquired simultaneously by the DASH-16 A/D board.

With the help of the ASYST software system, data is acquired at different heart rates from consenting human subjects lying in the supine position. Data for the different heart rates are collected after having the
Figure 1.3 System Block Diagram.
subjects walk briskly on a treadmill to elevate their heart rates. Data are also acquired intermittently as the heart rate goes down to their respective normal rates.

The Asyst system software configuration for data acquisition and processing, and hardware configuration are described, in detail, in the following chapters. The system block diagram is illustrated in figure 1.3.
Figure 2.1 System Hardware Block Diagram For Microwave Interferometer Based Cardiopulmonary Monitoring.
2.1 System Hardware Construction

The system hardware block diagram for monitoring chest wall vibration due to cardiac activity is shown in figure 2.1. The system is built around the Microwave Interferometer and an IBM-AT computer. A DASH-16 data acquisition and control board attached to the IBM-AT computer is a 12 bit A/D converter, which is capable of processing 8 different analog I/O channels for data acquisition. The computer executes the task of data acquisition and signal processing with the help of the ASYST software package by Macmillan Software Company.

The analog signals from the Microwave Interferometer and the ECG machine are acquired and digitized by the DASH-16 A/D converter. A 12 bit binary output from the A/D converter is stored as integers in decimal ASCII code in a file, which is used for processing by the ASYST system. The algorithms used by ASYST for signal acquisition, filtering and processing are described in the next chapter. From figure 2.1, analog data from the two channels are acquired with the help of the DASH-16 converter. One channel is the chest wall signal from the Microwave Interferometer, and the other is the ECG signal. The ECG signal serves as the timing reference signal; one time-unit being an R-R interval from the ECG signal.
The interferometer analog voltage output, is presented to the DASH-16 converter for digitizing. The ECG output is filtered with a 0.5 Hz high-pass analog filter to prevent signal baseline drift.

2.2 Microwave Interferometer

The Calspan Corporation of Buffalo, New York, now Cornell Aeronautical Laboratories, designed and built the Microwave Interferometer in 1966. The basic operating principle of the instrument is illumination of the chest wall with a low intensity, highly coherent microwave beam, and then comparing the phase of the incident energy with the phase of the reflected wave, which fluctuates as the chest wall moves in response to cardiac activity. Phase comparison is made at an intermediate frequency to prevent changes in sensitivity due to noise.

The Microwave Interferometer was designed to monitor heart and respiratory functions of a resting individual without placing electrodes or leads on the person's skin. Also, it was not essential that the individual disrobe, since microwave energy easily traverses normal clothing.

Vibration of the chest wall causes a phase difference between the microwave energy incident upon, and the energy reflected from, the moving chest wall. This phase difference is displayed on an oscilloscope and then recorded for computer analysis.
Figure 2.2 Basic Microwave Interferometer System Block Diagram.
The Interferometer is characterized as a phase-locked, coherent, low power radar. It operates at 9.3 GHz, which is equivalent to a free-space wavelength of 3.2 cm. The system has the ability to resolve, theoretically, 1/100 of a degree of carrier phase shift, which is equal to a target displacement of 0.84 microns.

A basic Interferometer system block diagram is shown in figure 2.2. The signal source is provided by a reflex Klystron oscillator operating at 9.3 GHz. This oscillator output is fed through a directional coupler, through a 60 MHz balanced modulator, and through a single sideband filter to a microwave mixer receiver. At this point, it becomes a local oscillator input operating at a frequency of 9.3 GHz + 60 MHz. A small fraction of the power from the directional coupler is amplified in a Travelling Wave Tube (TWT) phase shift amplifier. This power is then transmitted out via the Transmitting Horn Antenna.

The power from the transmitter illuminates the subject's chest wall, where it is reflected as the chest moves in response to cardiac activity. As the chest wall moves with respect to the transmitting horn, the amount of phase shift of the reflected wave front is proportional to the change in distance between the horn aperture and the surface of the chest wall. This time varying phase shift is superimposed upon the coherent 9.3 GHz carrier signal.

The reference 9.3 GHz carrier signal is offset by a 60 MHz Intermediate frequency (IF) signal to produce a 9.3 GHz +
60 MHz reference signal which is mixed with the phase varying carrier signal from the receiving antenna in a mixer/receiver, whose output is then the 60 MHz IF signal with the superimposed time-varying phase shift. The reference IF signal and the phase, containing IF signal, are further reduced to 10.7 MHz. Phase shift information is detected from these two signals to create a voltage which is proportional to the amount of phase shift produced by the moving target (chest wall). This voltage is amplified and fed back to the TWT phase shifter in such a way as to preserve a specified phase shift at the microwave receiver output.

This feed-back voltage is recorded as the time varying phase shift produced as the anterior chest wall moves. To exclude the large and slow phase shifts created by respiration, a high pass filter with a cut off frequency of 0.5 Hz is used. This filters out breathing and differentiates the displacement signal to produce the velocity of chest wall motion (3).

2.3 Dash-16 Data Acquisition and Control Board

Supplied by the Metrabyte Corporation, the DASH-16 A/D converter board is a IBM PC compatible multifunction high speed analog to digital I/O expansion board. Its multilayered construction with integral ground plane reduces noise and crosstalk at high frequencies. DASH-16 uses an industry standard 12 bit successive approximation converter
with a 25 microsecond conversion time. Channel input configuration is switch-selectable on the board, and a choice between 16 single-ended channels and 8 differential channels is provided. The board also provides 90 db common mode rejection and ±10 volt common mode range.

The DASH-16 is configured as a 8 channel, ±2.5 volt bipolar mode with GAIN equal to 2 and internal clocking. Direct memory access operation is enabled by setting the DMA switch to level 3 (4).

2.4 System Software- Introduction to Asyst

Asyst provides a broad range of capabilities and only a few applications are able to utilize all of them at the same time. Asyst version 2.0's overlay structure permits loading into memory only those capabilities that are required for a special application, and unloading them at the time that they are no longer wanted. An overlay is a precompiled program that can be interchanged in and out of memory. It is one of the most practical ways of fitting a large program into a limited memory space.

Asyst supports three types of overlays: System overlays, Application overlays, and Configuration overlays. System overlays are files comprising functionally related Asyst commands. Application overlays are user-created overlays, and they can only be loaded transiently into memory. Configuration overlays are used alongside a
configuration menu to build a particular system. Configuration overlays are internal to Asyst, so they cannot be changed by the user. The system software structure customized for this research makes use of the system overlays and the configuration overlays.

There are different configuration choices available to the Asyst user. The following include the configuration choices utilized in this research for: signal acquisition, signal processing, and graphic output. This selects, among other things, a Data file overlay, Editor overlay, and Analysis overlay, which also contains the waveform operation system overlay.

**MEMORY CONFIGURATION**: This selects the size of different memory areas like the user dictionary, and the data acquisition system (DAS) buffer segment. It should be noted that these selections are effective only if they are saved in a save version of Asyst.

**DAS CONFIGURATION**: This selects the specific configuration of data acquisition hardware to be used. For this research, it is the Metrabyte DASH-16 A/D - D/A converter board.

**HARDWARE CONFIGURATION**: This makes Asyst aware of the type of microprocessor, the system clock speed, the display adapter, and the printer type that will
be utilized in the system. This is discussed in the Computer hardware construction section.

GRAPHIC CONFIGURATION: This is used to set or change the default graphics display style according to user requirement.

SAVE/EXIT: This makes provisions for saving the configurations chosen by the user in a customized save version of Asyst(5).
3.1 Signal Acquisition

Configuring the Asyst system for signal acquisition involves a three step process. In the first step, the data acquisition overlays are selected and loaded into memory. The second step involves setting the data acquisition system (DAS) buffer size. In the third step, the DAS configuration program is executed to acquaint Asyst with the DASH-16 A/D - D/A board's hardware specifications.

DAS OVERLAY SELECTION:
For system data acquisition, two DAS overlays are loaded into memory. These overlays are the DAS Master overlay and the DAS Device Driver overlay menu. Overlay configuration in the configuration menu is started by pressing the < F2 > key.

DAS BUFFER SIZE CONFIGURATION:
DAS buffer is that memory area used for intermediate storage of data by all Asyst data acquisition I/O functions, with the exception of direct memory access (DMA) functions. Since this system uses only DMA data acquisition, the DAS buffer is set at the default value of zero bytes. Memory configuration option found in the Asyst configuration menu is used to set DAS buffer size.
DAS BOARD CONFIGURATION:
This configuration process is used to tell Asyst about DASH-16 board's specific characteristics, of which its I/O address is the most important. The DASH-16 board is configured for I/O address 300 HEX which is 768 DECIMAL.
DAS configuration procedure is found in appendix G (6).

3.2 Signal Processing Executed by Asyst
3.2.1 Interferometer Output Signal of Chest Wall Vibrations
A low pass filtering operation using the Asyst software basically eliminates higher frequency oscillations including 60 Hz noise.

The low pass filtering process is a smoothing operation done by convolution of the Interferometer signal with the inverse Fourier transform of the Blackman's window function(6).

3.2.2 Interferometer Signal Frequency Spectrum
The interferometer signal frequency distribution of the chest wall vibrating at various heart rates is analyzed by obtaining the frequency content of the signal.

Firstly, data points representing one cardiac cycle are extracted from the parent Interferometer signal. Secondly, to improve the frequency resolution of the
Figure 3.1 Example of Power Spectrum Analysis Using Two Separate Signals at Frequencies of 5 Hz and 18 Hz.
extracted signal, zero padding is used. The extracted signal is zero padded to 2048 sample points. By so doing, the frequency resolution of the signal is increased. Appendix I shows the effect of zero padding on the frequency resolution of a spectrum. Thirdly, the fast fourier transform of the signal with zero padding is obtained. The absolute value of the transformed signal data is taken and then, the square of the absolute value is plotted(7).

To find out if the asyst software correctly performed the mathematics involved with spectral analysis, two known frequency signals, provided by a signal generator, are analyzed. Two separate signals at 5 Hz and 18 Hz are acquired simultaneously using asyst acquisition software. Spectral analysis is performed using the asyst program DT3PLOT.DMO, found in appendix E. Figure 3.1 illustrates graphically, the outcome of the analysis. The fundamental frequencies of the two signals are clearly shown at 5 Hz and 18 Hz.

The first, second, and third moments of the frequency distribution are utilized to evaluate or establish a relation between chest wall motion (of one cardiac cycle length) and changes in heart rate(8).
CHAPTER 4
EXPERIMENTAL PROCEDURE, DATA ANALYSIS AND DISCUSSION

4.1 Measurement Parameters
The Interferometer and the ECG records were obtained simultaneously. Other data recorded included subject's age, sex and chest circumference. Signal data were recorded before and after the subject had walked on a treadmill set at 2 miles per hour, for approximately 5 minutes. Total signal data acquisition time for each subject was approximately 3 minutes.

4.2 Subject Description
Ten healthy males between the ages of 17 and 35 volunteered for this research study. Their chest circumference ranged between 31 and 40 inches, and none of the subjects had any known preexisting cardiopulmonary conditions. Each subject read and signed a consent form before taking part in the experiment. A consent form is found in appendix B. The subjects lay in the supine position throughout the entire signal acquisition period. This aided in the evaluation of anterior chest wall movement due to cardiac activity. Data collection procedure is found in appendix C.
Figure 4.1a and b Interferometer and ECG Signals from Subject(KMA). Heart rate = 110 beats per minute.
4.3 Changes in Heart Rate and Chest Wall Vibration

Figure 4.1a graphically demonstrates the output signal from the Interferometer, which represent chest wall motion. Three cardiac cycles are shown.

In order to describe this signal, it should be noted that outward movements of the atria and the ventricles toward the anterior chest wall are registered graphically as upward deflections, and movement away from the chest wall are recorded as downward deflections. The time between consecutive samples is 0.001 seconds, since a signal acquisition rate of 900 Hz is used.

The cardiac cycle starts at the p-wave of the ECG, when the atria are electrically stimulated. Atrial contraction is the mechanical response to the stimulation. The a-wave in figure 4.1a is believed to represent atrial contraction since it is directed downward.

Immediately following atrial contraction is ventricular loading, where the pressure and fluid volume in the ventricles increase. This is observed as the upward moving wave (Vf) in figure 4.1a.

Ventricular ejection starts at the R-wave of the ECG signal. This period is marked by ejection of blood into the aorta and the pulmonary system. The mechanical response to this event is compression of the ventricles, in other words, the ventricles move away from the anterior chest wall. The
Figure 4.2a and b Interferometer and ECG Signals from Subject (KK). Heart rate = 135 beats per minute.

Figure 4.3 Frequency Spectrum of Interferometer Signal, with Zero Padding, from Subject (JM). Heart rate = 86 beats per minute. n = 2048 points. Frequency Resolution = 0.15 Hz.
downward moving (Ve) wave in figure 4.1a represents this event.

The period between ventricular ejection (Ve) and the next atrial contraction (a-wave), is marked by
1. Increase and decrease in aortic blood flow.
2. Increase and decrease in aortic pressure.
3. Mitral and tricuspid valve closure.
4. Opening of aortic and pulmonic valves.
All of these events result in specific patterns of signal wave deflections which are represented in the Interferometer signal.

In figure 4.2a, where the heart rate is 135 beats per minute, the change in signal pattern is observed in the amplitudes of the signals. For instance, it is observed that the amplitude of (Vf) decreases as the heart rate increases. This is indicative of the system's increased circulatory requirements, that is, increased cardiac output and decreased diastolic period.

4.4 Interferometer Signal Frequency Distribution Analysis
The frequency spectrum of chest wall vibration is illustrated in figure 4.3. It consist of the frequencies represented in one cardiac cycle.

Within one cardiac cycle, the spectrum is distributed in a range of up to 8 Hz. The distribution of frequencies in the spectrum does not follow any specific pattern, as the heart rate changes. However, the first peak in the spectrum
# Table 1: Table of Moments from Frequency Spectrum of an Interferometer Signal Equivalent to One Cardiac Cycle Length

<table>
<thead>
<tr>
<th>SUBJECT'S INITIAL/AGE</th>
<th>HEART RATE BEATS/MIN</th>
<th>FIRST MOMENT (MEAN)</th>
<th>SECOND MOMENT (VARIANCE)</th>
<th>THIRD MOMENT (SKEWNESS)</th>
</tr>
</thead>
<tbody>
<tr>
<td>JM/28</td>
<td>86</td>
<td>4.4</td>
<td>9.6</td>
<td>-0.40</td>
</tr>
<tr>
<td>JM/28</td>
<td>94</td>
<td>4.0</td>
<td>10.6</td>
<td>+0.13</td>
</tr>
<tr>
<td>JM/28</td>
<td>115</td>
<td>3.1</td>
<td>11.6</td>
<td>+0.10</td>
</tr>
<tr>
<td>KMA/32</td>
<td>110</td>
<td>2.5</td>
<td>8.7</td>
<td>+0.45</td>
</tr>
<tr>
<td>KMA/32</td>
<td>115</td>
<td>2.4</td>
<td>17.7</td>
<td>-0.10</td>
</tr>
<tr>
<td>KMA/32</td>
<td>125</td>
<td>2.1</td>
<td>20.7</td>
<td>+0.30</td>
</tr>
<tr>
<td>KUEN/24</td>
<td>94</td>
<td>1.4</td>
<td>1.2</td>
<td>+0.76</td>
</tr>
<tr>
<td>KUEN/24</td>
<td>140</td>
<td>3.7</td>
<td>9.2</td>
<td>-0.20</td>
</tr>
<tr>
<td>PZ/24</td>
<td>86</td>
<td>3.0</td>
<td>8.2</td>
<td>-0.99</td>
</tr>
<tr>
<td>PZ/24</td>
<td>140</td>
<td>6.1</td>
<td>13.2</td>
<td>-0.57</td>
</tr>
<tr>
<td>PZ/24</td>
<td>150</td>
<td>6.1</td>
<td>8.5</td>
<td>-0.64</td>
</tr>
<tr>
<td>KK/35</td>
<td>100</td>
<td>2.3</td>
<td>7.6</td>
<td>+0.60</td>
</tr>
<tr>
<td>KK/35</td>
<td>135</td>
<td>3.8</td>
<td>10.9</td>
<td>+0.35</td>
</tr>
<tr>
<td>KK/35</td>
<td>150</td>
<td>4.4</td>
<td>9.6</td>
<td>-0.14</td>
</tr>
</tbody>
</table>
contributes to the frequency of the heart rate at that instant. In addition to this finding, peaks other than the heart rate frequency peak are observed in the spectrum. These peaks may be the result of changes in vascular motion within each cardiac cycle. Appendix H shows the other frequency spectra obtained at various heart rates.

Table #1 consists of the first, second, and third moments of the frequency spectrum in an Interferometer signal equivalent to one cardiac cycle. Five of the subject's chest wall signal frequency spectra are evaluated.

It should be noted that, the results were not obtained from controlled experimentation, since this thesis was only a preliminary study. For instance, the heart rate range was different in all of the subjects.

The first moment (mean) does not stay the same as the heart rate changes. In three out of the five subjects, the mean frequency within the length of one cardiac cycle increases as the heart rate increases. This is observed at heart rates greater than 130 beats per minute. Data from two subjects whose heart rates were below 130 beats per minute showed the mean frequency decreasing, as the heart rate increased.

The second moment, which is the variance, is not constant throughout changes in the heart rate. In four out of the five cases, the variance increases as the heart rate increases. This is observed in data with heart rates smaller than 130 beats per minute and heart rates greater than 130
beats per minute. A possible explanation for this finding could be, the dispersion is greater at higher heart rates. In other words, the response to higher heart rates is increased mechanical motion.

The third moment (skewness), which is a measure of the symmetry about the mean frequency, does not show any specific pattern as the heart rate changes. However, it is observed that there is a tendency to a change from either positive skewness to negative skewness and vice versa. This is observed in four out of five cases.

4.5 Conclusion

Even though this thesis was a preliminary investigation of the relationship between the mechanical dynamics within a cardiac cycle (translated on to the anterior chest wall) and changes in heart rate, it also evaluated the reliability of the microwave Interferometer as a non-invasive cardiopulmonary monitoring system.

Despite limited sample size, using central moments to analyze the frequency distribution of the chest wall signals brought out very useful information. The average frequency of the mechanical vibrations within a cardiac cycle did not remain constant as the heart rate changed. Also, the frequencies were not confined about the mean frequency or symmetrically distributed as the heart rate changed.
It should be noted that significant conclusions cannot be drawn from this thesis project due to the following reasons:

1. Controlled experimentation, where data are obtained at the same heart rate in all of the subjects, is not considered.

2. Limitations in gender diversity: All the subjects were male volunteers.

3. The effect of age on changes in heart rate and chest wall motion is not considered.

4. The statistical significance of each data is not taken into consideration.

Perhaps, future frequency distribution analysis of chest wall vibrations using the Interferometer and significant measuring parameters, could provide a much more better understanding of the mechanical dynamics within the cardiac cycle.
APPENDIX A

DATA ACQUISITION SYSTEM CONFIGURATION

I/O ADDRESSING OPTIONS:

SET SWITCHES:

| BASE ADDR-9   | TO | OFF |
| BASE ADDR-8   | TO | OFF |
| BASE ADDR-7   | TO | ON  |
| BASE ADDR-6   | TO | ON  |
| BASE ADDR-5   | TO | ON  |
| BASE ADDR-4   | TO | ON  |

Configuration is done by selecting DAS configuration in the Asyst configuration menu.

The first three prompts shown by the DAS configuration program include the following:

1. **Clear all devices from table ? ( Y/N )**
   
   A yes clears the table of all devices without removing from memory the driver overlay that was loaded earlier.

2. **Specify table entry to use:**

   < 1 > Table entry is chosen.

3. **Specify device to use:**
At this prompt, a list of all the DAS boards that Asyst supports is shown on screen. The highlighted entry should correspond to the driver overlay that was loaded from the overlay configuration menu. The board is configured to match the chosen driver overlay and the number for the DAS device type is entered. The word <DASH-16> starts configuration of the DASH-16 board. Metrabyte DASH-16 board is described in chapter II.
APPENDIX B

SUBJECT'S CONSENT FORM

Name of Project or Principal Investigator: Dr. Peter E. Engler

Title of Project: COMPARISON OF HEART RATE WITH CHEST WALL VIBRATION

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/cm². 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:__________________________
APPENDIX C

DATA COLLECTION PROCEDURE

The following list is adhered to in the collection procedure:

1. All the cable connections are checked.
2. Turn on the Interferometer. See appendix D for the description of the power-up procedure.
3. Oscilloscope, ECG machine, and computer are turned on.
4. ECG electrodes are attached to subject's left arm, right arm, and right leg. This provides the configuration for a LEAD I ECG signal.
5. Subject lies supine on bench, and the Interferometer is pointed on the subject's chest region.
6. Interferometer and LEAD I ECG signals (waveforms) are examined on the oscilloscope for a regular continuous rhythm.
7. Load signal acquisition software program into computer by typing \texttt{load diskacql.dmo} at the OK command prompt. The source program for signal acquisition is found in appendix G. Sampling rate is set at 0.9 Kilohertz, 2 buffers are used, and the signal data are stored in the C: directory under the .dat extension.
8. Wait for the OK prompt after acquisition is completed.
9. Put the Interferometer in the standby mode.
10. Detach connections to electrodes on the subject and direct him onto the treadmill.
11. Turn on the treadmill and allow subject to walk on it for approximately 5 minutes.
12. Turn off treadmill and direct subject back onto the recording bench.
13. Switch the Interferometer back on to operational mode.
14. Attach connections to electrodes on the subject and repeat steps #7 and #8 at least two more times. Allow approximately 1 minute before doing step #7 each time.

15. Copy all data files on to floppy disks for permanent storage.
APPENDIX D

INTERFEROMETER POWER-UP PROCEDURE

1. Make sure the klystron modulation power switch is off.
2. Turn on the main switch.
3. Turn on the power switch for the fluke power.
4. Turn on the power supply to the klystron.
5. Wait for approximately three minutes.
6. Turn the high voltage switch on fluke to the right.
7. Turn the klystron modulation to CW.
8. A microwave receiver crystal current should register.
9. After usage, power-down the Interferometer by turning off the switches in the following order: 7 then 6 then 4 then 3 then 2 then 1.
APPENDIX E

TIME SERIES ANALYSIS
(POWER SPECTRUM ROUTINE)

INTEGER DIM[2730, 3] ARRAY YDATA
REAL DIM[2048] ARRAY TIME
INTEGER DIM[1792] ARRAY CADATA
INTEGER DIM[2048] ARRAY FODATA
INTEGER DIM[256] ARRAY Y3DATA

DP.COMPLEX DIM[2048] ARRAY YCDATA
DP. REAL DIM[2048] ARRAY YADATA
REAL SCALAR OFFSET
REAL SCALAR CONSTANT

FILE.OPEN A:KK20.DAT
1 SUBFILE YDATA FILE>ARRAY
FILE.CLOSE
YDATA

21 0 24 79 WINDOW {BOTTOM}
\ VERTICAL 0 9000000000000. WORLD.SET
\ HORIZONTAL 0. 20. AXIS.FIT.OFF WORLD.SET

TIME [ ]RAMP
0.1464844 CONSTANT :=
CONSTANT TIME *
CONSTANT -
TIME :=

: INSTRUCTIONS
CR ." <F1> PLOT CHNL#1 DATA  <F2> PLOT CHNL#2 DATA "
CR ." <F3> PLOT CHNL#3 DATA "
;

: CHNL#1.DATA \ INTERFEROMETER SPEC.
YDATA SUB[890, 256; 1, 1] \ ANALYSIS

DIM[256] RESHAPE \ DATA EQUIVALENT TO ONE CARDIAC
\ CYCLE
Y3DATA :=
.07 SET.CUTOFF.FREQ
Y3DATA SMOOTH
Y3DATA :=
0
CADATA :=
Y3DATA CADATA CATENATE \ ZERO PADS DATA TO 2048 SAMPLE
\ POINTS
FODATA :=
FODATA FFT \ FAST FOURIER TRANSFORMS
\ SIGNAL WITH ZERO PADDING
YCDATA :=
YCDATA ABS \ ABSOLUTE VALUE OF TRANSFORM
YADATA :=
YADATA DUP \ TAKES THE SQUARE OF THE
* \ ABSOLUTE VALUE OF TRANSFORM
YADATA :=
\ .4 SET.CUTOFF.FREQ
\ YADATA SMOOTH
\ YADATA :=
\ YADATA
\ 5 *
\ YADATA :=

0 .20 VUPORT.ORIG
1 .65 VUPORT.SIZE
WORLD.COORDS

XY.AXIS.PLOT
TIME SUB[ 1 , 120 , 1 ]
YADATA SUB[ 1 , 120 , 1 ] XY.AUTO.PLOT
0 0 POSITION
;

: CHNL#2.DATA
\ YDATA SUB[ 1 , 2730 ; 2 , 1 ]
\ OFFSET +
\ DIM[ 2730 ] RESHAPE
\ Y2DATA :=

0 .20 VUPORT.ORIG
1 .32 VUPORT.SIZE
XY.AXIS.PLOT
\ TIME SUB[1, 555, 4] \\ Y2DATA SUB[1, 555, 4] XY.AUTO.PLOT \\
\ NORMAL.COORDS
.82 .95 POSITION "TIME (sec)" LABEL 0 0 POSITION

: CHNL#3.DATA
 YDATA SUB[1, 2730; 3, 1]
 OFFSET +
 DIM[2730] RESHAPE
 Y3DATA :=
 0 .20 VUPORT.ORIG
 1 .32 VUPORT.SIZE
 XY.AXIS.PLOT
 TIME SUB[1, 250, 4]
 Y3DATA SUB[1, 250, 4] XY.AUTO.PLOT

: GO GRAPHICS.DISPLAY
 (BOTTOM)
 VUPORT.CLEAR
 INSTRUCTIONS
 F1 FUNCTION.KEY.DOES CHNL#1.DATA
 F2 FUNCTION.KEY.DOES CHNL#2.DATA
 F3 FUNCTION.KEY.DOES CHNL#3.DATA
 INTERPRET.KEYS
 ONERR:
 SCREEN.CLEAR BELL
 CR."UNKNOWN ERROR: TYPE CONTROL-BREAK TO RESTART"
 CR."OR ANOTHER KEY TO EXIT"
 KEY BYE


### GLOSSARY OF ABBREVIATIONS

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Full Form</th>
</tr>
</thead>
<tbody>
<tr>
<td>AV VALVE</td>
<td>Atrioventricular valve</td>
</tr>
<tr>
<td>SA NODE</td>
<td>Sinoatrial node</td>
</tr>
<tr>
<td>ECG</td>
<td>Electrocardiogram</td>
</tr>
<tr>
<td>IVC</td>
<td>Isovolumic contraction</td>
</tr>
<tr>
<td>LVET</td>
<td>Left ventricular ejection time</td>
</tr>
<tr>
<td>STI</td>
<td>Systolic time interval</td>
</tr>
<tr>
<td>DAS</td>
<td>Data acquisition system</td>
</tr>
<tr>
<td>DMA</td>
<td>Direct memory access</td>
</tr>
</tbody>
</table>
15. STRING FNAME  \\
    " C:" FNAME " :=  \\
    INTEGER SCALAR BUF.LENGTH  \\
    INTEGER SCALAR #BUFFERS  \\
    FNAME DEFER> ?CLUSTER.SIZE  \\
    ?CLUSTER.SIZE needs to  \\
    4 * BUF.LENGTH :=  \\
    drive spec and  \\
    the rest...  \\
    buffers be four times  \\
    size  \\
    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: "  

44
#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. "

;
Figure H1 Frequency Spectrum of Interferometer Signal, with Zero Padding, from Subject (JM). Heart rate = 94 beats per minute. n = 2048 points. Frequency Resolution = 0.15 Hz.
Figure H2 Frequency Spectrum of Interferometer Signal, with Zero Pading, from Subject(JM). Heart rate = 115 beats per minute. n = 2048 points. Frequency Resolution = 0.15 Hz.
Figure H3 Frequency Spectrum of Interferometer Signal, with Zero Padding, from Subject (KUEN). Heart rate = 94 beats per minute. $n = 2048$ points. Frequency Resolution = 0.15 Hz.
Figure H4 Frequency Spectrum of Interferometer Signal, with Zero Padding, from Subject (KMA). Heart rate = 110 beats per minute. $n = 2048$ points. Frequency Resolution = 0.15 Hz.
Figure H5 Frequency Spectrum of Interferometer Signal, with Zero Padding, from Subject (KK). Heart rate = 135 beats per minute. n = 2048 points. Frequency Resolution = 0.15 Hz.
APPENDIX I

ILLUSTRATION OF THE EFFECT OF ZERO PADDING ON FREQUENCY SPECTRUM ANALYSIS

Figure 1a Frequency Spectrum of Interferometer Signal, without Zero Padding, from Subject (JM). Heart rate = 86 beats per minute. n = 350 points. Frequency Resolution = 1.2 Hz.

Figure 1b Frequency spectrum of Interferometer Signal, with Zero Padding, from Subject (JM). Heart rate = 86 beats per minute. n = 2048 points. Frequency Resolution = 0.15 Hz.


