Spring 1975

AN INTERACTIVE COMPUTER ANALYSIS OF PHONOCARDIOGRAMS

ANTAL ANDRAS SARKADY

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AN INTERACTIVE COMPUTER ANALYSIS
OF PHONOCARDIOGRAMS

by

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B.S., University of New Hampshire, 1965
M.S., University of New Hampshire, 1967

A DISSERTATION

Submitted to the University of New Hampshire
In Partial Fulfillment of
The Requirements for the Degree of

Doctor of Philosophy
In Engineering
Graduate School
June, 1975
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ACKNOWLEDGMENTS

As is usually the case in research of this type, many people have made significant contributions to make this work possible. Foremost among these is my wife, Joyce and my children, Lynne and Kenneth, who made many personal sacrifices during the course of this study. In addition, I would like to thank Dr. Roberta Williams of Children's Hospital, Boston, Massachusetts for suggesting and supporting the aortic stenosis study, Ms. Judith Keymont for her assistance in selecting and recording phonocardiogram data, Dr. Ronald R. Clark, University of New Hampshire, for his advice and guidance throughout the study, Dr. Edward L. Chupp for making the University of New Hampshire Space Science Center computer facilities available for this project, and Mr. Ernie Nichols for helpful suggestions and aid in the development of the FFT subroutine.
# TABLE OF CONTENTS

LIST OF TABLES ............................................. viii
LIST OF ILLUSTRATIONS ................................. x
ABSTRACT ................................................ xii
INTRODUCTION ......................................... 1

I. PHYSIOLOGY OF THE NORMAL AND ABNORMAL HEART ... 3
   FUNCTION AND OPERATION OF THE HEART .............. 3
   STRUCTURE OF THE HEART .............................. 5
   ARTERIAL BLOOD FLOW .................................. 6
   MECHANISM OF THE NORMAL AORTIC VALVE ............. 7
   AORTIC STENOSIS ...................................... 10
   MECHANISM OF THE STENOSED AORTIC VALVE .......... 12
   PATHOPHYSIOLOGICAL DESCRIPTION OF VALVAR AORTIC STENOSIS ............................................. 14
   ESTIMATING THE SEVERITY OF AORTIC STENOSIS .... 16

II. THE PHONOCARDIOGRAM SIGNAL ..................... 18
   STETHOSCOPIC AUSCULTATION .......................... 18
   THE PHONOCARDIOGRAM ................................ 20
   HEART SOUNDS IN THE PHONOCARDIOGRAM SIGNAL ..... 21
   HEMODYNAMIC CORRELATION OF HEART SOUNDS ....... 21
      First Heart Sound - S<sub>1</sub> ................... 21
      Second Heart Sound - S<sub>2</sub> ................ 23
      Third Heart Sound - S<sub>3</sub> .................. 25
      Fourth Heart Sound - S<sub>4</sub> ................. 25
   ABNORMALITIES OF HEART SOUNDS IN VALVAR AORTIC STENOSIS ............................................. 26
      Intensity Changes of S<sub>2</sub> .................. 26
      Variations in the Splitting Interval of S<sub>2</sub>  26
      Ejection Clicks ................................. 27
   HEART MURMURS ........................................ 30
   THE ORIGIN OF CARIOVASCULAR VIBRATION ENERGY .. 31
   AREAS OF AUSCULTATION .............................. 32
   TRANSMISSION CHARACTERISTICS OF THE HUMAN THORAX ......................................................... 35
   INDIRECT CAROTIC PULSE RECORDING .................. 36
   PHONOCARDIOGRAM SIGNAL FEATURES ................. 37
   PHONOCARDIOGRAM IDENTIFICATION FEATURES OF VALVAR AORTIC STENOSIS .................................. 38
TABLE OF CONTENTS (Continued)

DIFFERENTIAL DIAGNOSIS OF VALVAR AORTIC STENOSIS .......................................... 39
MAJOR PHONOCARDIOGRAM SIGNAL PROCESSING WORK DONE BY OTHERS ................. 41

III. DESCRIPTION OF THE EXPERIMENT .................................................. 44
APPROACH TO THE PROBLEM ......................................................... 44
SELECTION OF RECORDING SITES AND TIMING DATA ........................................ 46
ESTIMATION OF THE RECORDING TIME DURATION ........................................ 46
EXPERIMENT ORGANIZATION AND BLOCK DIAGRAM ........................................ 48
ANALOG DATA ACQUISITION AND RECORDING EQUIPMENT ........................................ 49
RECORDING AND CALIBRATION PROCEDURES ........................................ 51
  Differential Phono-Channel Delay .................................................. 55
  Push Down Test ........................................................................ 55
ANALOG-TO-DIGITAL CONVERSION .................................................. 55
SELECTION OF EQUIVALENT CARDIOCYCLES ........................................ 57
  Equivalent Data Ensembles ....................................................... 60
PASS 2-3 DATA ........................................................................ 62
  Pas: 2-3 Data Tape Formats ....................................................... 63

IV. SIGNAL PROCESSING TECHNIQUES .................................................. 69
TIME DOMAIN SAMPLING ............................................................... 69
  Point Sampling ........................................................................ 73
FREQUENCY DOMAIN SAMPLING .................................................. 75
DISCRETE FOURIER TRANSFORM .................................................... 77
  The Fast Fourier Transform (FFT) .................................................. 79
DESCRIPTION OF A STOCHASTIC PROCESS .......................................... 82
  Definition of a Stochastic Process .................................................. 82
  Moments of a Stochastic Process .................................................. 83
  A Stationary Stochastic Process .................................................... 84
POWER SPECTRUM ANALYSIS .......................................................... 84
  The Power Spectral Density of a Deterministic Signal ......................... 85
  Power Spectral Density of a Stochastic Process .................................. 86
  The Discrete Power Spectral Density Estimate ..................................... 87
  Bartlett's Smoothing Procedure .................................................... 88
  Bartlett's Spectral Window .......................................................... 89
  Variance of Smoothed Spectral Estimators ........................................ 92
  Confidence Interval for the Smoothed Spectrum .................................... 93
  Bandwidth of a Spectral Window ................................................... 95
  Summary of Bartlett Window Properties .......................................... 96
TABLE OF CONTENTS (Continued)

Computation of the Discrete Power Spectral Estimate ..................................... 97
Explanations of the Plot Labels .................. 99

ENVELOPE ANALYSIS ............................... 99

The Hilbert Transform .......................... 101
The Analytic Signal .......................... 102
Envelope, Phase and Frequency of the Phonocardiogram Signal ....................... 103
Envelope of Heart Sounds and Clicks .......... 104
Envelope of the Murmur Signal ............... 106
Computation of the Discrete Envelopogram Estimate ....................................... 107

V. RESULTS ......................................... 111

DESCRIPTION OF THE PATIENT DATA SET ............ 112
ENSEMBLE-AVERAGED ENVELOGRAMS ............. 113
ENSEMBLE-AVERAGED WAVELETS ..................... 120
ESTIMATING THE SEVERITY OF AORTIC STENOSIS USING MURMUR POWER SPECTRAL ANALYSIS ............ 124

VI. DISCUSSION OF RESULTS ...................... 135

ADVANTAGES OF ENSEMBLE AVERAGING .......... 135
DISCUSSION OF V.A.S. SEVERITY ESTIMATES .......... 136
SUGGESTIONS FOR FURTHER STUDY ............... 138

BIBLIOGRAPHY .......................................... 141

APPENDIX I ............................................ 149

COMPUTER PROGRAM ANALOG READ BINARY DUMP ........ 155
COMPUTER PROGRAM - DASFFT ....................... 160

APPENDIX II

DESCRIPTION OF THE INTERACTIVE ANALYSIS ........ 175
INTERACTIVE ANALYSIS PROGRAM - AUTOFREQ .......... 177
COMPUTER ANALYSIS PROGRAMS EMPLOYED FOR SEVERITY ANALYSIS ESTIMATES ........ 188
DESCRIPTION OF THE FIRST-PASS SEVERITY ANALYSIS PROGRAM, PANAL ........ 189
MAIN PROGRAM PANAL .......................... 192
SUBROUTINE FANAL ............................... 194
DESCRIPTION OF THE SECOND-PASS SEVERITY ANALYSIS PROGRAM, PPAVER ............ 206
TABLE OF CONTENTS (Continued)

MAIN PROGRAM PPAVER ............. 207
ERROR ANALYSIS OF f .................. 215
LEAST SQUARE REGRESSION LINE AND
CORRELATION COEFFICIENT CALCULATIONS .... 218
LIST OF TABLES

1. LIMITS OF P.S.E.G. AND AORTIC VALVE AREA IN V.A.S. ................. 17
2. A TYPICAL ANALOG RECORD SEQUENCE .................. 54
3. PASS 1 DIGITAL DATA TAPE RECORD FORMAT .......... 58
4. PASS 1 DIGITAL DATA TAPE FILE FORMAT .......... 59
5. PASS 2-3 DIGITAL DATA TAPE RECORD FORMAT .... 65
6. PASS 2-3 DIGITAL DATA TAPE FILE FORMAT .... 67
7. SUMMARY OF BARTLETT WINDOW PROPERTIES .......... 97
8. THIRTEEN CATHETERIZED VALVAR AORTIC STENOSIS PATIENTS DATA ............... 113
9. PERSONAL DATA FOR NORMAL AND CLINICALLY DIAGNOSED VALVAR AORTIC STENOSIS PATIENTS ... 114
10. BANDWIDTH AND FIRST MOMENT OF MEAN POWER SPECTRUM COMPUTED FROM INSPIRATION, EXPIRATION AND CAROTID DATA FILES AT 2ND R.I. .......... 130
11. SUMMARY OF MURMUR SPECTRUM ANALYSIS AT 2ND R.I. FOR THE CATH. V.A.S. PATIENTS .......... 131
13. PREDICTED MEAN P.S.E.G. AND STANDARD DEVIATION FOR THE CLINICALLY DIAGNOSED VALVAR AORTIC STENOSIS PATIENTS .......................... 140
1, AI. ACOUSTICAL COMPARISON OF MICROPHONE 2 VERSUS MICROPHONE 1 ................. 152
2, AI. PATIENT HEADER FORMAT ............... 153
3, AI. DATA HEADER FORMAT ............... 154

viii
 List of Tables (Continued)

1, All. Correlation study between P.S.E.G. and 50%F at the 2nd R.I. on inspiration, expiration and carotid data for the twelve catheterized valvar aortic stenosis patients ........................................... 219

2, All. Correlation study between P.S.E.G. and  at the 2nd R.I. on inspiration, expiration and carotid data for the twelve catheterized valvar aortic stenosis patients ........................................... 220

3, All. Correlation study between P.S.E.G. and  at the 2nd R.I. on inspiration data for the twelve catheterized valvar aortic stenosis patients ........................................... 221

4, All. Correlation study between P.S.E.G. and  at the 2nd R.I. on expiration data for the twelve catheterized valvar aortic stenosis patients. ........................................... 222
**LIST OF ILLUSTRATIONS**

1. Normal Heart of a Child ..................... 4
2. The Normal Aortic Valve ...................... 8
3. Normal and Aortic Stenosed Hearts ........... 11
4. Cross-sectional Views of Normal and Stenosed Valves ................................. 13
5. Spectrum of Chest Vibrations and Threshold of Hearing ................................. 19
6. A Typical Normal Cardiac Cycle ............... 22
7. A Typical V.A.S. Phonocardiogram Cycle ....... 28
8. Primary Auscultation Areas .................... 34
9. Block Diagram of the Experiment .......................... 50
10. Measured Frequency Response of the Electronics System with AM Recording Mode .... 52
11. Measured Frequency Response of the Electronics System with FM Recording Mode .... 53
12. Five Typical Equivalent Phonocardiogram Records .................................. 61
13. A Typical Aligned Four-Channel Cardiocyte Record .................................... 64
14. Pulse and Impulse Sampling of $s(t)$ and Corresponding Amplitude Spectra ........... 72
15. A Sampled Amplitude Spectrum and the Corresponding Periodically Extended $s(t)$ .......... 76
16. A Typical V.A.S. Murmur Power Spectrum Estimate .................................. 100
17. Envelogram Estimate of Fig. 7 ................. 109
18. Averaged V.A.S. Envelogram for Inspiration .................................. 116
19. Averaged V.A.S. Envelogram for Expiration .................................. 117
20. A Typical V.A.S. Carotid Pulse ................ 119
LIST OF ILLUSTRATIONS (Continued)

21. A Typical Normal Second Heart Sound ............... 122

22. The Aligned Averaged Aortic Component of Fig. 21 ............... 123

23. A Typical Aortic Ejection Click ................. 125

24. A Typical Aligned Averaged Aortic Ejection Click ............... 126

25. A Typical Averaged V.A.S. Murmur Power Spectrum ......................... 128

26. Scatter Diagram for the Catheterized V.A.S. Patients at the 2nd. R.I. for Inspiration + Expiration + Carotid Data ............... 133

1,Al. Microphone Placement on the Chest ............... 150

2,Al. Amplitude Response Curve of a Type 53616 Microphone ............... 151
ABSTRACT

AN INTERACTIVE COMPUTER ANALYSIS
OF PHONOCARDIOGRAMS

by

Antal A. Sarkady

Computerized phonocardiogram analysis techniques were developed to aid in the positive diagnosis of systolic heart diseases and these techniques were applied to noninvasively assess the severity of valvar aortic stenosis. Signal processing algorithms were incorporated into an interactive analysis program used to study heart sounds and murmurs in the time and frequency domains. The algorithms are applicable to several heart diseases, but this study was conducted on six normal patients, thirteen catheterized, and four clinically-diagnosed valvar aortic stenosis patients.

For each patient, phonocardiogram data (30-1200Hz range) from four listening sites, along with an ECG, respiration, and carotid pulse, were recorded for approximately 100 seconds. A typical patient data set consists of seven data files; two mid-inspiration, two mid-expiration, two carotid and one calibration file.

As a starting point of the interactive analysis branch, a normalized ensemble-averaged envelogram is
computed and plotted for each file. From these plots, maximum precordial intensity areas, respiration affects, murmur shape, and the timing of clicks, murmurs and sounds are identified or measured. Using the measured onset times and durations, murmur, click, and heart sound signals are gated and separately studied in the time and frequency domains.

The severity of valvar aortic stenosis is estimated noninvasively from a gated and ensemble-averaged phonocardiogram murmur power spectrum. The averaged spectrum is computed from several cardiocycles (typically 40-50 records) recorded from the second right intercostal space. Ensemble averaging is essential in this analysis to reduce spectrum variance and to obtain consistent results. A high degree of correlation exists (correlation coefficient = 0.96) between the peak systolic ejection gradient measured by cardiac catheterization, and the calculated first moment of the mean murmur spectrum.

A Varian 620/I 16 bits/word minicomputer was used for this study. The computer was equipped with a 12K word memory, two seven-track digital tape recorders, a graphics terminal, an analog multiplexer, and an analog-to-digital converter.
INTRODUCTION

The computerized phonocardiogram analysis techniques presented in this dissertation are applicable to many systolic heart diseases found in a wide age group. However, children and adolescents four to twenty years of age were selected for this study for the following reasons. A large portion of heart diseases are congenital or can be traced to a minor cardiac disorder occurring in early life; consequently, early detection and correction are necessary for a long and active adult life. In addition, innocent murmurs are extremely common in children and adolescents, occurring in approximately 50 percent of these subjects [36]. Therefore, a need exists for an accurate and rapid screening instrument. The analysis techniques presented here can be adapted in the design of such an instrument. Finally, children are relatively free from arterial diseases such as arteriosclerosis and may serve as a ready standard for a large number of heart diseases.

In order to assess the merits of the computerized phonocardiogram analysis techniques, the study of valvar aortic stenosis was suggested by Dr. Roberta Williams of Children's Hospital, Boston, Massachusetts. Her proposal was an excellent and challenging choice for several reasons.

Valvar aortic stenosis is a frequently detected disease representing approximately three to six percent of
the total heart diseases found in children [10]. Severity of the disease requires frequent assessment, particularly in moderate and severe cases, since for these patients sudden death is a distinct possibility. Accurate assessment of the severity of this disease is presently possible only by catheterization, an invasive surgical procedure requiring three days of hospital care. It is clear that a definite need exists for an accurate, noninvasive technique to assess the severity of valvar aortic stenosis; such a technique is presented in this dissertation. Finally, the valvar aortic stenosis murmur is produced by a "turbulent jet" [1] where similar jets are found in several other heart diseases (pulmonary stenosis, ventricular septal defect, atrial septal defect, etc.). Consequently, this anomaly can be considered as a representative prototype of several "noisy systolic murmurs" and it may be possible for this analysis technique to be extended to these heart diseases as well.
CHAPTER I

PHYSIOLOGY OF THE NORMAL AND ABNORMAL HEART

FUNCTION AND OPERATION OF THE HEART

The function of the heart is to pump oxygenated blood to all parts of the body. It is readily visualized as two serially-connected dual-chamber pumps, activated by a common electrical pacemaker through conduction bands [28]. The two pumps are similar in size but the left side is a considerably higher-pressured system than the right side. A full scale drawing of a normal child's heart and the connecting great vessels is shown in Fig. 1. Pumping action of the heart is described with the aid of this diagram.

Oxygen-poor blood (blue blood) is pooled in the right atrium (RA) and enters the right ventricle (RV) through the tricuspid valve (TV). The right ventricle pumps the blood through the pulmonary valve (PV) into the small capillaries of the lungs where it becomes enriched with oxygen. The oxygenated blood (red blood) is pooled in the left atrium and enters the left ventricle (LV) through the mitral valve (MV).

The left ventricle pumps the red blood through the aortic valve (AV) to the aorta (AO) where it is distributed by smaller arteries to the rest of the body. The circulation path is completed when the blue blood is returned to the
AO-aorta, AV-aortic valve, IVC-inferior vena cava
LA-left atrium, LV-left ventricle, MPA, main pulmonary artery
MV-mitral valve, PV-pulmonary valve, RV-right atrium
RV-right ventricle, SVC-superior vena cava
TR-tricuspid valve, FB-femoral bifurcation

Fig. 1. Normal heart of a child
right atrium via the inferior vena cava (IVC) and the superior vena cava (SVC).

The pumping cycles of the two sides of the heart are nearly synchronous. A cardiocycle is divided into systolic and diastolic phases, at which times the ventricular muscles are contracted and relaxed respectively. In the early part of the systole, the ventricle is at a constant volume, while during the latter part, blood is being pumped from it. In the early part of the diastole, the ventricle is at a constant volume, while during the latter part, blood is being pooled in it.

Functions of the atria are to assure an adequate blood supply to the ventricle during the filling phase and to assist in the filling by contracting at the end of the diastolic phase. This is often referred to as "topping off" the ventricle.

All of the heart valves are operated by the blood flow; nearly zero pressure drop occurs across the valves during forward flow and they are closed by reverse flow.

STRUCTURE OF THE HEART

A dense connective tissue forms a fibrous "skeleton" of the heart surrounding the valves. The atria, ventricles and arterial trunks are firmly attached to this "skeleton" [1].

The ventricles are composed of sheets of spiralling, tightly-bound, myocardial fibers which thicken near the apex. The wall of the left ventricle is considerably thicker
than that of the right ventricle. Capillaries connected to the coronary arteries supply blood to the heart muscle at a rate ten to twenty times higher than to the skeletal muscle. This high nourishment rate is required to support the mechanical work performed by the ventricles.

**ARTERIAL BLOOD FLOW**

The outstanding feature of arterial blood flow is its pulsatile character. During the early systole, blood is suddenly ejected into the ascending aorta. The ventricle has insufficient energy to overcome the inertia of the long column of blood in the arteries; consequently, the blood tends to pile up in the distended ascending aorta, producing a sudden, local pressure increase. A pressure wave propagates down the descending aorta with a velocity of 4-5 m/sec [1]. This velocity is ten to twenty times greater than the flow velocity of the blood [2] and is a function of the physical properties of the vessel wall and the blood.

The advancing pressure wave is reflected by the peripheral structures (primarily at the femoral bifurcation) producing a reflected wave traveling back toward the heart. The observed pressure wave at any point in the aorta is the superposition of the forward pressure wave and the reflected wave. As the aortic valve closes at the end of the systole, drainage from the aorta and arteries into the arterioles continues, transforming the highly pulsatile flow into a more continuous, steady flow. Dispersion of the pulse
waveform during its travel is one of the characteristics of the vascular system.

A detailed analysis of pulsatile blood flow in distensible arteries is given in a book edited by Attinger [3]. A recent computer model of the left ventricle and the aorta is presented by Watts [4]. He models the aorta as a tapered, electrical delay line and studies the pressure pulse propagation produced by an impaired left ventricle. Watt's model, however, is valid only in the 0-20 Hz frequency range.

MECHANISM OF THE NORMAL AORTIC VALVE

The aortic valve is composed of three cusps of equal size attached around the circumference of the valve orifice. In children and adolescents, the cusps are thin, elastic membranes which thicken later in life. A considerable overlap in the cusps' area assures a tight closure; when open, it forms a triangular orifice which has a smaller cross-sectional area than the aorta. This opening however, is sufficiently large to have a negligibly small pressure drop across the open valve and to have laminar blood flow through the valve. Behind the aortic valve cusps are three cavities, the sinuses of valsalva [29], shown in Fig. 2. Left and right coronary circulation originates from two of these sinuses through small openings called coronary ostia. The sinuses perform an important role in the closing mechanism of the valve. If a valve leaflet comes in contact with the coronary ostia, the rapidly falling coronary pressure and
Fig. 2. The normal aortic valve
the increasing aortic pressure would seal the cusp against the wall of the aorta; space provided by the sinus prevents this from happening.

Bellhouse's [5,6,7] experiments with leaflet-type model valves demonstrated that vortices trapped in the sinuses provided a fluid mechanical valve control and aided systolic coronary circulation. In the model valve, the cusps presented negligible obstruction to the accelerating fluid flow during the opening phase. Thrown open, the cusps aligned themselves with the flow, and stagnation points were formed at the sinus ridges along with intense vortices inside the sinuses. During the early and mid-systole, the cusps were positioned so that their tips were slightly projected in the sinuses. The stagnation points, acting as high pressure sources, contributed to the systolic coronary circulation. During the end of the systole in the de-acceleration phase, the ventricular pressure fell below the sinus pressure and the cusps started to close. Streamlines were spread downstream and the cusps drifted to a three-quarter closed position; the valve was fully closed by a small amount of reverse flow. Bellhouse, et al. [7] measured four percent regurgitation in the model valves during the closing phase. During the entire systole the flow was laminar and no sign of turbulence was reported.
AORTIC STENOSIS

Aortic stenosis is defined as an obstruction to blood flow between the aorta and the left ventricle. Depending upon the location of the obstruction, it is divided into three major classes. Obstruction produced by an impaired valve is called valvar aortic stenosis, while obstruction above or below the valve is referred to as supravalvar aortic stenosis or subvalvar aortic stenosis respectively. Subaortic stenosis is usually further subdivided into discrete and idiopathic classes. The four types of stenosis, along with a normal heart, are shown in Fig. 3. Note that discrete subvalvar obstruction is produced by a fibrous band located below the valve, whereas supravalvar and idiopathic subaortic stenoses are produced by deformation of the aorta and ventricle respectively. Sub- and supravalvar stenoses are infrequent, while valvar stenosis is a common anomaly occurring in three to six percent of patients with congenital cardiovascular defects [10].

Valvar aortic stenosis may be acquired during the course of a disease, but in children it is most often due to congenital fusion of the cusps [12]. When all three cusps are fused near the valve root, valve motion is impaired, but the cusps can function as three independent units. This valve anomaly is called tricuspid valvar aortic stenosis. When the cusps are fused in such a way that they
Fig. 3. Normal and aortic stenosed hearts
function as two independent units, the term bicuspid valvar aortic stenosis is used. Occasionally, in congenital deformation, the valve may become a single, semi-rigid perforated membrane acting as an obstruction rather than as a valve, presenting the same cross-sectional area for both flow directions. This anomaly is rare and its auscultatory features are distinct from tricuspid and bicuspid aortic stenoses [11]. Cross-sectional views of the three valve anomalies and of a normal valve for open and closed conditions are shown in Fig. 4.

The most common forms of aortic stenosis in children are the bicuspid and tricuspid types; the valves are seldom if ever calcified [10]. Calcification in humans begins at age 13-14 and damaged valves tend to accumulate calcium past this age. Consequently, even mild early valve impairment may lead to calcified aortic stenosis in adult life [12].

MECHANISM OF THE STENOSED AORTIC VALVE

A marked change in fluid flow occurs when the aortic valve area is reduced to approximately less than fifty percent of normal size. At the onset of the ejection phase a turbulent jet is formed in the ascending aorta and persists throughout the systole. Presence of the jet in the aorta is routinely observed in angiographic studies [9, 13, 31] and is considered to be a prime distinguishing feature in discriminating between valvar and subvalvar aortic
Fig. 4. Cross-sectional views of normal and stenosed valves
stenosis [11]. In the laboratory, turbulent flow of fluids in tubes and vessels is observed when the Reynolds number exceeds a critical value of $970 \pm 80$ [1].

Bellhouse, et al. [8] simulated valvar aortic stenosis by glueing the leaflets of the model valve together, reducing the valvar area by fifty percent. Under these conditions, instead of laminar flow in the systole, a turbulent jet formed at the valve and no vortices were observed in the sinuses. Pressure at the coronary ostia was slightly lower, indicating mild impairment of systolic coronary circulation and becoming more significant at a higher degree of stenosis. During the closing phase, the amount of reverse flow was only slightly more than that for the normal valve since the stenosed valve was never fully open.

PATHOPHYSIOLOGICAL DESCRIPTION OF VALVAR AORTIC STENOSIS

When the aortic valve area is reduced from the normal range of 2.5-3.5 cm$^2$ to a critical range of 0.5-1.0 cm$^2$, compensatory mechanisms fail and the following physiological symptoms develop: a marked increase in flow impedance [30], a marked left ventricle pressure increase accompanied by a slow rise in the aortic pressure wave, and a pressure drop across the valve. Peak pressure drop across the valve may exceed 100 mm. Hg in severe stenosis. Cardiac output remains nearly the same at rest but is reduced during exercise,
indicating that the left ventricle relies on cardiac reserve to handle the overload. The overstressed ventricle responds by gradually increasing muscle mass [10], commonly observed in angiography [31]. The increased muscle mass and wall tension greatly increase oxygen consumption of the ventricle at the time when coronary circulation is seriously impaired. Impairment is produced partially by the increased and prolonged intramural blood pressure and partially by the reduced systolic sinus pressure [12]. When oxygen demand exceeds the ability of the coronary blood flow to provide oxygen, myocardial ischemia and angina pectoris result [10,12]. Contractility of the oxygen-starved cardiac muscle is reduced and congestive heart failure, syncope, or angina pectoris develops. At this stage the history of patient survival averages two, three, and five years, depending on the symptoms, where ten to fifteen percent die suddenly [32] if corrective surgery is not performed. In most instances the surgery is a valvarlaremy, but in some cases, particularly in older individuals, replacement of the impaired valve with a prosthetic valve is involved.

It is important to emphasize that the human heart tolerates mild aortic stenosis well, and not until the aortic valve area is reduced to less than fifty percent of normal, do clinical symptoms develop [12]. Surgery is required only in severe cases.
ESTIMATING THE SEVERITY OF AORTIC STENOSIS

Vector ECG and phonocardiography are considered to be adequate noninvasive diagnostic techniques for the identification of aortic stenosis; however, estimating the degree of stenosis has been poor with these techniques.

The most reliable invasive techniques for assessing the degree of stenosis are considered to be internal pressure measurements by cardiac catheterization, and simultaneous blood flow studies of X-ray motion pictures, known as angiocardiography. In these methods, access to the left ventricle is gained through hazardous routes, either by a transseptal needle [14] or by a retrograde arterial route past the aortic valve [15]. If the transseptal needle (catheter with a needle tip) is used, it is inserted into the femoral vein and advanced into the right atrium. The interatrial septum is punctured and the catheter is advanced into the left atrium and left ventricle. Proper positioning of the needle prior to puncturing is one of the more hazardous aspects of this procedure.

Retrograde arterial catheterization is usually performed through the femoral artery or the bronchial artery. This procedure often involves some degree of arterial trauma and is occasionally difficult to perform in children.

After the catheter is placed into the left ventricle by one of the foregoing routes, oxygen saturation and pressure measurements are taken. An X-ray absorbing dye is
injected and angiocardiographic studies are performed. Next, the catheter is withdrawn and pressure measurements are performed in the ascending aorta. The peak systolic pressure drop (referred to as peak systolic ejection gradient, P.S.E.G.) across the aortic valve is determined and the valve area is calculated from Gorlin's formula [16]. The degree of stenosis is determined on the basis of these measurements and is classified as mild, moderate, or severe according to the limits [11] listed in Table 1.

It is clear that cardiac catheterization is an accurate diagnostic technique; however, it is a surgical procedure requiring three days of hospitalization and is not a clinical diagnostic tool.

**TABLE 1**

**LIMITS OF P.S.E.G. AND AORTIC VALVE AREA IN V.A.S.**

<table>
<thead>
<tr>
<th>Degree of Stenosis</th>
<th>Peak Valvar Pressure Drop P.S.E.G. in mm. Hg</th>
<th>Value Area cm²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>10 - 40</td>
<td>1.5 - 0.8</td>
</tr>
<tr>
<td>Moderate</td>
<td>40 - 80</td>
<td>0.9 - 0.6</td>
</tr>
<tr>
<td>Severe</td>
<td>&gt; 80</td>
<td>&lt; 0.6</td>
</tr>
<tr>
<td>Surgery Recommended</td>
<td>&gt; 110</td>
<td>&lt; 0.5</td>
</tr>
</tbody>
</table>
CHAPTER II

THE PHONOCARDIOGRAM SIGNAL

In this chapter the normal and abnormal phonocardiogram signal waveforms are discussed and the various signal components are correlated with hemodynamic events. In addition, production and transmission of vibrational energy is described. Finally, diagnostic signal features of aortic stenosis are tabulated and the differential diagnosis of the disease is presented.

STETHOSCOPIC AUSCULTATION

Vibrations in the 1-750 Hz frequency range are commonly observed on the surface of the human chest. A representative power spectrum of the vibrations measured in normal subjects, along with the mean threshold of hearing, are given by [17] and shown in Fig. 5. Note that stethoscopic auscultation is limited to the 40-750 Hz range and that most of the vibration energy is below this range.

In the audible range, the human ear and stethoscope is an extremely sensitive detector and assisted by the brain, forms an adaptive filter; however, it is a time variant, nonquantitative, ausculatory system. Perhaps the most serious problem with stethoscopic auscultation is the lack of data storage and retrieval features which often leads to
Fig. 5. Spectrum of chest vibrations and threshold of hearing.
subjective diagnosis. These shortcomings were demonstrated by recent tests performed on physicians [18].

THE PHONOCARDIOGRAM

The phonocardiogram is an intensity versus time display by a high-frequency chart recorder of the audible vibrations observed on the human chest by a microphone. In principle, phonocardiography is a clinically-quantitative diagnostic technique; however, lack of amplitude calibration and nonstandardization of the recording equipment render this technique semi-quantitative; direct waveform comparison among clinical recordings is difficult. Still, a great deal of quantitative timing information has been gained by phonocardiography and it offers permanent data storage and display features.

The crystal microphones which are most often used in clinical phonocardiography have a relatively flat, frequency response curve in the 40-750 Hz range. Within these bounds the acoustical frequency region of interest can be selected by a band-pass filter. The filter characteristics are not standardized in phonocardiography, but most clinics use "mid-frequency filtration" [21] or "stethoscopic filtration" [33,34]. "Mid-frequency filtration" is produced by a filter with a flat frequency response function in the approximate band-pass range of 120-500 Hz and a roll-off of 6 db/octave outside this range. "Stethoscopic filtration" is similar to "mid-frequency filtration" with the notable exception
that the band-pass is modified to produce a response at the filter output in the 120-500 Hz range which resembles the acoustical response of the human ear (see Fig. 5).

In this experiment "stethoscopic filtration" is employed with a slight low-frequency accentuation. This filter setting produces good sensitivity over a wide frequency range while essential identification features of the time series are preserved.

HEART SOUNDS IN THE PHONOCARDIOGRAM SIGNAL

Typical normal phonocardiogram (PCG) findings in time correlation with ECG, aortic pressure, left ventricular pressure, and left atrial pressure waves are shown in Fig. 6. Note the presence of four distinct groups of vibrations (marked $S_1, \ldots, S_4$) in the phonocardiogram record. These are called heart sounds. Characteristics of these sounds will now be described and correlated with hemodynamic events.

Duration of the systole on a phonocardiogram is defined as the period from the onset of $S_1$ to the onset of $S_2$, and duration of the diastole is from the onset of $S_2$ to the onset of the next $S_1$.

HEMODYNAMIC CORRELATION OF HEART SOUNDS

First Heart Sound - $S_1$. Onset of the first heart sound occurs at the beginning of the systole following the ECG Q wave by approximately 10-20 ms. The entire event lasts for an average of 100-120 ms. It is generally
Fig. 6. A typical normal cardiac cycle
recognized that the first heart sound has four components [20,21] as shown in Fig. 6. The chronological order of these is as follows: The first component is a small, low-frequency (≈ 30-50 Hz) initial vibration which coincides with and is produced by contraction of the left ventricular muscle. The second component consists of a large, high-frequency (80-200 Hz) vibration and is caused by abrupt closure of the mitral valve. The third component follows mitral valve closure by 30 ms. and is also a high-frequency (≈ 80-200 Hz) vibration. It is suspected that this component is produced by rapid ejection of blood into the great vessels, but some investigators contribute it to closure of the tricuspid valve [21]. The fourth component is a small, low-frequency (40-80 Hz) vibration produced by acceleration of blood in the great vessels.

**Second Heart Sound - S₂.** There is general agreement that the second heart sound is caused by closure of the aortic and pulmonic valves. The vibration produced is in the 70-200 Hz range and persists for about 100 ms. This sound is often "split" into aortic (A₂) and pulmonic (P₂) components (see Fig. 6). In normal subjects the splitting sequence is such that A₂ precedes P₂ by 10-20 ms. upon expiration. For any one individual the splitting is not constant, but increases by 6-10 ms. from expiration to inspiration.
The physiologic reasons for increased inspiratory splitting described by Tavel [21] are as follows: During inspiration the blood is pooled in the lungs causing a pressure decrease in the main pulmonary artery and incomplete filling of the left ventricle. The reduced pressure delays pulmonary valve closure, $P_2$, and incomplete filling causes aortic valve closure, $A_2$, to occur early. Thus, both events contribute to inspiratory widening, a respiratory effect which is an important discriminatory feature used to identify $A_2$ and $P_2$.

The onset of $A_2$ was believed to be correlated with the left ventricular pressure change, called the dicrotic notch or dicrotic incisure, (see Fig. 6). More recently, Piemme, et al. [23] demonstrated that the closing sound was delayed from the dicrotic notch by 20-30 ms, occurring in coincidence with maximum reverse blood flow. In addition, the presence of a low-frequency (30-40 Hz) component preceding the closing sound was observed. Its onset occurred in coincidence with the dicrotic notch and with the abrupt slope change of the forward blood flow curve. Piemme attributed this early component to vibration of the cardiohemic system, produced by rapid relaxation of the left ventricle and consequent deacceleration of the blood. These experiments were performed on dogs with implanted transducers of limited frequency response (0-40 Hz), and were significant in providing accurate measurements in vivo of the aortic valve closing time.
**Third Heart Sound - S₃.** The third heart sound is often observed, particularly in children, during the early rapid ventricular filling phase. This low-frequency (20-70 Hz) vibration occurs on the average of 150 ms. after A₂ and has a duration of 40-50 ms. Its origin remains evasive, but most investigators believe that it is caused by vibration of the rapidly elongating left ventricular walls excited by the incoming blood flow. Since the third heart sound is not produced by valve closure its presence is not considered to be clinically significant in children.

**Fourth Heart Sound - S₄.** The fourth heart sound has frequency characteristics similar to the third sound and may occur during the late diastolic filling phase. If observed, it usually precedes S₁ by 70-100 ms. and has a duration of 30-50 ms. This sound is most likely produced by vibration of the ventricle walls excited by rapid inflow of blood produced by atrial contractions. During this cardiac phase the atrium acts as a secondary pump "topping off" the ventricles [1].

The fourth heart sound is often observed in children and is considered to be a normal phonocardiographic finding, disappearing in young adult life. Therefore its presence is not considered to be clinically significant.
ABNORMALITIES OF HEART SOUNDS IN VALVAR AORTIC STENOSIS

Intensity Changes of $S_2$. Comparison of the intensity and tonal qualities of heart sounds taken at the same listening area are affected by extracardiac as well as by cardiac factors. Some prime examples of the former are thickness of the chest wall, pulmonary emphysema, fever, chest deformity and pericardial fluid. Examples of cardiac factors causing an intensity increase of $S_2$ are increased rate of valve closure [24] and stiffening of the valve cusps. An intensity decrease of $S_2$ is commonly caused by a reduced force of closure (reduced pressure gradient) across the aortic or pulmonic valve, and by calcification of valves (calcified aortic or pulmonic stenosis). Children's cusps are supple and noncalcified and thus, highly mobile [11]. Therefore, in children with congenital valvar aortic stenosis $A_2$ is of normal intensity.

Variations in the Splitting Interval of $S_2$. An abnormally wide splitting on expiration, but otherwise normal ($A_2, P_2$) sequence, is caused by delayed pulmonic or early aortic valve closure. This anomaly is often observed in pulmonic stenosis, mitral stenosis and ventricular septal defects.

The reversed or paradoxical splitting sequence ($P_2, A_2$) occurs when aortic valve closure is delayed, causing a splitting interval which decreases from expiration to
inspiration. This condition is caused by left branch block, patent ductus arteriosus and severe aortic stenosis [19]. While paradoxical splitting occurs in severe aortic stenosis, it has not been found to be useful in estimating the degree of stenosis. Bache, et al. [35] reported that a prolonged left ventricular ejection time (LVET) existed in valvar aortic stenosis, but poor correlation was observed between LVET and the calculated valve area.

**Ejection Clicks.** A short, high-frequency (80-200 Hz) vibration may follow \( S_1 \); this extra sound may originate from the right or left side of the heart. The former is referred to as pulmonic click [21] and is associated with pulmonary valvar stenosis, pulmonary hypertension and conditions which increase the right ventricular output (e.g., A.S.D. and V.S.D.). When origin of the sound is from the left side, it is referred to as aortic click and is observed in almost all cases of congenital valvar aortic stenosis [11,21]. Since this form of stenosis is common, and other heart diseases seldom if ever produce an aortic click, its presence in children is considered a prime diagnostic feature of congenital valvar aortic stenosis [11,21]. Later in life, with gradual calcification of the valve, intensity of the click is reduced and its absence signifies severe calcified aortic stenosis. A typical phonocardiogram cycle (i.e., ECG Q-Q interval) containing an aortic ejection click is shown in Fig. 7. This cycle was acquired from a valvar
Fig. 7. A typical V.A.S. phonocardiogram cycle
aortic stenosis patient (Edward D.) at the second right intercostal space (2nd R.I.) during inspiration.

In pulmonary valvar stenosis, the pulmonic ejection click is of decreasing intensity or even disappears during inspiration and occurs earlier than normal [26]. The degree of prematurity has been found to be correlated with the severity of pulmonary stenosis [21]. In comparison, intensity and onset of the aortic click are independent of respiration, and onset time is not related to severity. While the onset time is not clinically useful, constant intensity of this click is an important feature and is used to identify aortic and pulmonic clicks.

Opinion on the origin of the aortic ejection click has been divided. Some investigators [21] state that it is a root event produced by acceleration of blood into the aorta and occurs at the onset of the pressure rise in the indirect carotid pulse. (Thus, more appropriately, it can be called an accentuated component of S\textsubscript{1}). Others report that the click is independent of S\textsubscript{1}, valvar in origin and is produced when the valve is fully domed and stressed by the ejected blood. Recent detailed intracardiac sound and angiogram studies on normal and valvar aortic stenosis subjects demonstrated, that while both mechanisms could produce sound [27,13], the aortic click is valvar in origin. The click produced by the stenosed valve always occurred in coincidence with the anacrotic notch. The time interval from the click to the rise of the aortic pressure pulse is
approximately 24 ms. This time interval is defined as valve mobility and correlates poorly with the degree of stenosis in the wide age group of patients in Epstein's studies [27].

HEART MURMURS

Heart murmurs are relatively long-duration vibrations which may occur in any part of the cardiac cycle. Murmurs are classified into several, not necessarily mutually exclusive, groups. The most important classifications are listed below and are described according to:

1. their physiological properties; innocent or organic.
2. their frequency content; high-pitched, low-pitched, musical or harsh.
3. their intensity envelope; diamond-shaped, crescendo, or descrescendo.
4. their time of occurrence in the cardiac cycle; systolic, diastolic, or continuous.

To further define the time of a murmur's occurrence, the prefixes early, mid, late and holo are often used.

A typical cardiocycle containing a diamond-shaped systolic murmur is shown in Fig. 7. This type of murmur is commonly observed in valvar aortic stenosis. During auscultation, intensity of a murmur is graded on a subjective scale of 1 (very faint) to 6 (loudest possible).

The mere presence of a murmur does not imply the presence of heart disease or a heart disorder. Innocent
murmurs (those not associated with significant heart disease) are common auscultatory findings and occur in approximately fifty percent of normal children [36]. The timing, location, intensity pattern shape and most importantly, the accompanying heart sound abnormalities, determine the presence and type of significant heart disease [18,21,36,37].

THE ORIGIN OF CARDIOVASCULAR VIBRATION ENERGY

A thorough study of the origin of cardiovascular vibration energy can be divided into three parts:

1. Study of the hemodynamic event which causes the heart to vibrate.
2. Modeling of the vibrating system and study of the production of vibrational energy.
3. Study of the propagation modes of vibrational energy and transmission properties of the human thorax.

There has been general agreement among physicians for many decades that $S_1$ and $S_2$ are caused by closing of the valves. Time correlation of heart sounds with major hemodynamic events are well established and well reported in literature [20,22,23,24,25].

It is generally accepted that harsh non-musical murmurs are produced by a turbulent jet of blood flowing through a small orifice [1,18,37]. Examples of such murmurs are aortic stenosis, pulmonic stenosis, ventricular septal
defects, etc. Musical murmurs, those with tonal qualities, are produced by other processes. Vortex shedding, periodic wake, and flitter are the mechanisms proposed by Bruns [39] and Rushmer [18] to explain the origin of musical murmurs.

Detailed theories concerning the production mechanisms of cardiovascular vibration energy have been studied by many [38,39,40,41,42] and the turbulent murmur problem is extensively modeled by Yellin and Bellhouse [43,7,8].

A highly intuitive cardiac model is described by Rushmer [1]. In this model the blood, heart walls, and heart valves are considered as one vibrating "cardiohemic" system, where heart sounds are caused by acceleration or deacceleration of the blood. This non-mathematical model is quite successful in predicting the time of occurrence of normal heart sounds, but fails to account for the wave shapes of the sounds.

AREAS OF AUSCULTATION

Murmurs produced by various anomalies and heart diseases have definite, well established intensity radiation patterns on the chest. The point of maximum intensity and the radiation pattern are two important discriminatory diagnostic features in cardiology.

Conventionally, the chest is divided into four areas, referred to as the aortic (A), pulmonic (P), right ventricular or tricuspid (T), and left ventricular or mitral (M) auscultation areas. The locations of these areas are shown
in Fig. 8. The areas are named after the heart sounds and murmurs which are best observed at these locations [51].

Vibrations originating from the aorta (i.e., aortic stenosis murmur, aortic ejection click, and the aortic component of the second heart sound) are usually best observed at the aortic area, or more specifically, at the second right intercostal space near the sternum border (2nd R.I.S.). Vibrations originating from the main pulmonary artery (i.e., pulmonic ejection click, pulmonary stenosis murmur, and pulmonary component of the second heart sound) are well transmitted to the pulmonary listening area at the second left intercostal space near the sternum border (2nd L.I.S.). While the aortic component of the second heart sound and the aortic ejection click tend to be maximum at the aortic area, they are well transmitted to other listening areas, particularly to the left ventricular area or apex. In contrast, the pulmonary component of the second heart sound is highly localized to the pulmonary area and seldom if ever is observed at the 2nd R.I.S. and apex. Predictable transmission characteristics of the second heart sound are most useful in identifying aortic and pulmonic components of the second heart sound.

Vibrations originating from the left ventricle (i.e., mitral closing sounds, third heart sounds, mitral stenosis murmur, etc.) are best observed at the mitral area or apex, whereas vibrations from the right heart (tricuspid closing
Fig. 8. Primary auscultation areas
sound, tricuspid stenosis murmur, etc.) are generally loudest at the tricuspid area.

As expected, these primary auscultation sites are located on the chest where the left and right ventricles and great vessels are closest to the surface.

TRANSMISSION CHARACTERISTICS OF THE HUMAN THORAX

The human body is an anisotropic, nonhomogeneous, acoustical medium where vibration energy propagates in several modes [45,46].

In the heart and arterial walls vibration energy propagates as shear waves [46] with a velocity of 4-5 m/sec. [45]. Shear waves are attenuated at 20 db/10cm. at 100 Hz. Additional relevant data obtained in vivo measurements are given in [45,46,50,53].

Energy is conducted as compressional waves in bone and tissue with velocities of 3400 m/sec. and 1490 m/sec. [45,53] respectively. Bone conducts sound energy well in a wide frequency range, where in tissue it is attenuated at an approximate rate of 10 db/10cm. at 90 Hz [45,52]. Additional relevant data is available in the literature [44,45,47,48,49].

Faber, et al. [47] suggested that vibrational energy emerges at the primary auscultation sites and spreads to nearby locations as surface waves. Surface wave velocity on the human chest is approximately 15 m/sec. at 100 Hz and
increases approximately with the square root of the frequency [47]. However, these waves are localized since they are attenuated at a rate of 27 db/10 cm. at 100 Hz [45].

From the above discussion we may conclude that the microphones must be located between the ribs and as close to the sources as possible. This choice minimizes the affect of multiple conduction paths and produces maximum signal intensities. In valvar aortic stenosis the murmur, ejection click, and A2 are observed with maximum intensity at the 2nd R.I.S. The arch of the aorta is only 2-3 cm. away from this site as shown in Fig. 8. For the above reasons, the 2nd R.I.S. is chosen for the study of this disease.

INDIRECT CAROTID PULSE RECORDING

The carotid arteries are major vessels directly connected to the aorta and easily accessible at the neck. The recording of the carotid artery wall displacement versus time in the 0.2-20 Hz frequency band is referred to as the indirect carotid pulse recording, or in short, the carotid pulse [21]. This pulse shape closely resembles the wave-shape measured in the ascending aorta, but is delayed by 20-30 ms. In addition, the high-frequency components (dicrotic notch) are considerably attenuated. When the proper time delay correction is applied, the upstroke of the carotid pulse and the dicrotic notch occur in coincidence with the onset of the ventricular ejection and aortic
valve closure. Consequently, the carotid pulse is useful in identifying the aortic ejection click and the closing sound in the phonocardiogram tracings.

In valvar aortic stenosis the carotid pulse may show a "slow" upstroke, a prolonged left ventricular ejection time, and pressure fluctuation or trill. While these signs are usually present in patients with an aortic valve pressure gradient greater than 40-50 mm. Hg, a more precise classification with this method has not been possible [21].

PHONOCARDIOGRAM SIGNAL FEATURES

Phonocardiographic diagnosis of heart diseases must be based on rugged signal features which are statistically reliable and are defined by Levine [54] as features "whose presence are not changed and whose character are not greatly altered by normal variation in the image of a character in a given category".

Selection of general and rugged signal features is highly empirical and requires access to a complete data set, that is, a data set that contains all heart diseases. Cardiologists through years of experience have found the following rugged phonocardiogram signal features relevant to the diagnosis of heart diseases [1,18,21,36].

1. Presence of murmurs.
2. Presence of systolic click or other abnormal sounds.
3. Timing of murmurs and sounds.
4. Location of maximum intensity points and transmission paths of murmurs, clicks, and sounds on the chest.

5. Shape of murmur envelope.

6. Peak intensity of sounds, clicks, and murmurs.

7. Frequency content of murmurs.

It is important to note that correct diagnosis is not reached by considering features from one of the categories listed above. Rather, a combination of features from the entire group must be jointly interpreted; a weighted sum of the features leads to proper diagnosis.

**PHONOCARDIOGRAM IDENTIFICATION FEATURES OF VALVAR AORTIC STENOSIS**

The following phonocardiogram features are considered to be the rugged identification features of congenital valvar aortic stenosis in children [21].

1. A diamond-shaped, systolic ejection murmur is present and is usually loudest at the aortic listening area. The murmur must end before onset of the aortic component of the second heart sound.

2. The murmur is introduced by a constant intensity, aortic ejection click which is usually loudest at the aortic listening area or at the apex.
In addition, the following features are often observed in valvar aortic stenosis, particularly in moderate and severe cases.

3. The left ventricular ejection period is prolonged, producing a paradoxically-split second heart sound.

4. The carotid pulse may show a slow upstroke, prolonged peak time, a flattening of the dicrotic notch, and superimposed vibrations or a trill in the systole.

DIFFERENTIAL DIAGNOSIS OF VALVAR AORTIC STENOSIS

The following innocent systolic murmurs have one or more signal features similar to valvar aortic stenosis [36].

1. Still's murmur.

2. Innocent pulmonic murmur.

3. Supraclavicular atrial bruit.

4. Innocent late systolic murmur.

5. Innocent cardio-respiratory murmur.

These innocent systolic murmurs have typical diamond-shaped envelopes but are never introduced by an ejection click. Consequently, the presence of the aortic ejection click can be used to discriminate between valvar aortic stenosis and innocent systolic murmurs.

In children the following common heart anomalies have phonocardiogram signal features similar to valvar aortic
stenosis [21, 36].

1. Pulmonic stenosis.
2. Mild ventricular septal defect.
3. Moderate to severe atrial septal defect.
4. Tetrology of Fallot.

All of these anomalies have typical diamond-shaped, ejection-type systolic murmurs which may be introduced by an ejection click. However, they can be differentiated from valvar aortic stenosis by observing the following basic differences:

In pulmonic stenosis:

a. The pulmonary ejection click is variable; that is, intensity and onset of the click are functions of respiration.

b. The pulmonic ejection murmur is generally loudest at the pulmonic listening area. Its intensity is often a function of respiration, and it may continue to the onset of the pulmonic component of the second heart sound.

In mild ventricular septal defect:

a. The murmur is a harsh, short-duration, ejection-type, where the location of the maximum intensity point on the chest overlaps the location of the intracardiac location of the shunt, generally at the third left intercostal area or lower.
In moderate to severe atrial septal defect:

a. Abnormal first and second heart sounds are present.

b. A pulmonic-systolic ejection murmur is present related to the large pulmonary blood flow.

In Tetrology of Fallot:

a. The ejection-type murmur in mild to moderate cases is not introduced by an ejection click and consequently, can be easily discriminated.

b. In severe cases the short-duration ejection murmur is introduced by an ejection click making discrimination difficult on the basis of phonocardiogram features alone, but is easily identified by other clinical observations such as cyanosis and low oxygen content of the blood.

MAJOR PHONOCARDIOGRAM SIGNAL PROCESSING WORK DONE BY OTHERS

The following investigators have recently made significant contributions in the field of phonocardiogram signal processing:

Cambron [82] studies 111 patients with mitral stenosis, mitral insufficiency or normal phonocardiograms and
uses pattern recognition techniques, such as the nearest-neighbor method, to identify these conditions in the frequency domain.

Vocker [83] employs an adaptive filter to select the epoch of the first and second heart sounds. He was able to update the filter to follow the epoch variation of the second sound caused by breathing.

Stephens [84] analyzes the first heart sound with bandpass filters and establishes frequency patterns useful in the identification of myocardial infarction.

In the work of Perry, et al. [85], analog as well as digital techniques are considered. In the digital analysis, 2.5 sec. recordings are made from four listening areas and successive threshold levels are used to identify sounds and murmurs. A band of filters is used to obtain the cardiogram energy spectra. A diagnostic decision process is employed to identify common heart diseases.

Frome and Frederickson [86] select several first and second heart sound waveforms, convert them into discrete data segments, and compute averaged first and second heart sound power spectra using the FFT algorithm. The authors suggest that the intensity of the computed spectra can be used to monitor the depth of anesthesia during surgery.

Gerbarg, et al. [87] make an indepth study on the use of computers to identify innocent heart murmurs. Identification is based primarily on timing of the innocent
systolic murmur.

Townes, et al. [88] compare the signal features of several cycles of innocent and stenosed bruits using zero crossing, statistical, and power spectrum analysis techniques. The investigators conclude that the number of major peaks in the power spectra of stenosed bruits exceeds the number found in innocent bruits, and use this signal feature to differentiate the two kinds.
CHAPTER III

DESCRIPTION OF THE EXPERIMENT

APPROACH TO THE PROBLEM

A brief examination of abnormal phonocardiogram records reveals that the time series consists of repetitive cardiocycles. Each cycle is composed of deterministic wavelets (heart sounds) and amplitude-modulated, bandwidth-limited, random signals (murmurs). The term wavelet is used here to indicate a portion of the phonocardiogram time series which is associated with a single hemodynamic event, such as aortic ejection click.

The cardiologist examining a phonocardiogram time series selects a typical inspiration cardiocycle and an expiration cardiocycle free of artifacts, measures the diagnostic signal parameters, and derives a partial diagnosis on the basis of these two cycles [11]. The measured parameters obtained from a single cardiocycle are often statistically unreliable due to added noise and other random artifacts. The statistical errors cannot be reduced since the data is in an unsuitable form. Thus, the single, biggest disadvantage of this type of time series display is that it is unsuited for computer processing, particularly for power spectrum analysis.
The need for frequency information prompted some investigators [55,56] to complement the time series display with online power spectrum analysis display. While online spectrum analysis is useful in identifying and describing musical murmurs, such as the "sea gull" murmur, on the basis of a single estimate [55], high statistical variance prevents accurate descriptions of noiselike murmurs. Since most organic murmurs are of this type, the above statement applies to the majority of heart diseases.

At the start of this research it was felt that by properly averaging equivalent cardiocycles (those which are produced under identical hemodynamic conditions) in the time, envelope and frequency domains, statistically reliable decision parameters could be obtained. With the use of these reliable parameters, the diagnosis of systolic heart diseases can become more accurate, and assessment of the severity of valvar aortic stenosis by computer analysis may now become possible.

This study was conducted on fourteen catheterized and four clinically diagnosed valvar aortic stenosis patients. In addition, six normal patients were included in the study to facilitate the identification of valvar aortic stenosis signal features. The complete patient set is described in Chapter V.

A small 16 bits/word mini-computer with approximately 12-16K words memory capacity, equipped with digital
tape recorders, a multiplexer, an analog-to-digital converter, and a graphics terminal, was used for this study. The multichannel time series was digitized and the large volume of data required was stored on digital magnetic tapes. With the aid of the graphics terminal the operator could interact with the computer, quickly reviewing and interpreting the processed data. In questionable cases, additional analysis could be requested. Quick turn-around time is of course the most essential characteristic of such an interactive analysis system.

SELECTION OF RECORDING SITES AND TIMING DATA

In this study phonocardiogram data from all four classical listening sites (2nd R.I.S., 2nd L.I.S., 4th L.I.S. and apex) were acquired. With this choice of listening sites, an adequate transmission pattern can be obtained and all heart diseases that can be diagnosed by phonocardiography can be adequately analyzed. Additionally, ECG, carotid pulse and respiration were recorded to facilitate the timing and identification of heart sounds and ejection clicks, and to observe changes in the phonocardiogram signal induced by respiration.

ESTIMATION OF THE RECORDING TIME DURATION

Clearly, the confidence limits of each analyzed point will be determined by the type of analysis, smoothing,
and number of cardiocycles included in the analysis. The effects of this will be discussed in Chapter IV. Prior to analysis, the recording time interval must be selected to be consistent with the goals of the analysis, as well as with other considerations. An example of the latter is: can a young child maintain quiet and steady respiration for the duration of the recording interval? Another relevant question may be asked: how reproducible is the measurement on a weekly or monthly basis? It is useless to reduce the short range statistical errors to 1 percent when the unexplainable human variables limit the monthly reproducibility of the measurement to 100 percent.

The repetitive nature of the phonocardiogram makes it possible to estimate the number of cardiocycles required to obtain a desired short range statistical error. Each cardiocycle can be represented as a sample function of a finite population. The task is to estimate the population mean with a desired error \( d \), on the basis of \( N \) samples and with a particular confidence level. Assuming that the population is approximately normally distributed with a mean of \( \eta \) and a standard deviation of \( \sigma \), the number of samples required is given by the equation below [57],

\[
N = \frac{Z^2 \sigma^2}{d^2}
\]

where \( Z \) is the confidence constant; for a 95 percent confidence level \( Z = 1.96 \). Selecting a 10 percent short range
measurement error as a realistic goal with a 95 percent confidence level, and assuming that the population variance is approximately one-half the population mean, these assumptions give

\[ d = \frac{n}{10} \]
\[ \sigma = \frac{n}{2} \]
\[ Z = 1.96 \]

and the corresponding sample size is \( N = 100 \) cardiocycles. Assuming that 0.9 sec. equals the average cardiocycle period, this yields a recording time duration \( T = 90 \) seconds. During data recording, it was observed that a patient could be maintained in a statistical equilibrium for these time durations, and repeated measurements taken on the same patient indicated that an acceptable 10 percent monthly reproducibility was possible.

EXPERIMENT ORGANIZATION AND BLOCK DIAGRAM

In this study the data "handling" was conveniently divided into four steps: analog data acquisition, analog-to-digital conversion, data selection, and data analysis. During the first step, four channels of analog data were acquired and recorded by an analog tape recorder. In the second step, analog data was converted into a continuous stream of digital records. During the third step, equivalent cardiocycles were selected and arranged in equivalent
data files. In the final step, files were analyzed, diagnosis signal features were identified, and the severity of aortic stenosis was assessed.

A complete block diagram of the experiment is shown in Fig. 9.

The first three procedures will be described in this chapter while the next two chapters will be devoted to data analysis.

ANALOG DATA ACQUISITION AND RECORDING EQUIPMENT

The analog data acquisition and recording equipment, as shown in Fig. 9, was located in the Cardiology Department at Children's hospital and an analog tape recorder was used for data storage.

The transducer-amplifier display equipment used in the study was a commercial heart sound monitoring system manufactured by Cambridge Instrument Company, Inc. The crystal microphones were Cambridge type 53616 "adult size" and were secured to the chest by suction as shown in Fig. 1, AI (Appendix I). The manufacturer's acoustical calibration curve is shown in Fig. 2, AI and the measured differential error between the two microphones is given in Table 1, AI. The phonocardiogram amplifier-filters were Cambridge type 72352 with the filter switch set to the "L" position.
Fig. 9. Block diagram of the experiment
At the start of this project the exact phonocardiogram bandwidth was unknown. To obtain good frequency response above 1KHz, the first group of clinically-diagnosed patients (Analog Tapes No. 1 and No. 2) were AM recorded. During the data conversion and initial power spectrum analysis it became obvious that, even for the severe aortic stenosis case, 95 percent of the phonocardiogram signal energy is concentrated below 400 Hz, and therefore high-frequency response was not required. To improve the low-frequency response and the signal-to-noise ratio, subsequent analog tapes were recorded in the FM mode. With the exception of one valvar aortic stenosis patient, all catheterized patients were recorded in the FM mode. The measured frequency response curves of the two phono-channels are shown in Figs. 10 and 11. Here the Cambridge amplifier inputs were taken as the system input and the multiplexer input was taken as the system output. Measurements were performed with a constant-intensity sinusoidal source. Note that the two recording modes have almost identical "frequency response curves" in the 50-1000Hz range, but that below 50Hz the AM recording provides more attenuation.

RECORDING AND CALIBRATION PROCEDURES

Prior to data recording the patient was introduced to the equipment and was assured that no physical pain would be involved with this test. Since emotional strain affects the heart and blood flow rates, this step was
Fig. 10. Measured frequency response of electronics system with A.M. recording mode.
Fig. 11. Measured frequency response of electronic system with F.M. recording mode
required to obtain consistent phonocardiogram recordings and assured that the data was recorded under steady-state conditions. Before the actual recording was begun the four listening sites were briefly tested and the recorder gains were set to the maximum allowable levels (i.e. no limiting) at the maximum intensity sites. These settings were maintained throughout the recording. While being recorded the patients were in a supine position and respiration was at a normal, steady rhythm. A typical analog patient recording sequency is shown in Table 2.

**TABLE 2**

A TYPICAL ANALOG RECORD SEQUENCE

<table>
<thead>
<tr>
<th>Recording Time in sec.</th>
<th>Channel 1</th>
<th>Channel 2</th>
<th>Channel 3</th>
<th>Channel 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>20</td>
<td>Phono 1 2nd L.I.</td>
<td>Phono 2 Apex</td>
<td>Carotid Pulse</td>
<td>ECG Lead 2</td>
</tr>
<tr>
<td>90</td>
<td>Phono 1 2nd L.I.</td>
<td>Phono 2 Apex</td>
<td>Respiration</td>
<td>ECG Lead 2</td>
</tr>
<tr>
<td>20</td>
<td>Phono 1 2nd R.I.</td>
<td>Phono 2 4th L.I.</td>
<td>Carotid Pulse</td>
<td>ECG Lead 2</td>
</tr>
<tr>
<td>90</td>
<td>Phono 1 2nd R.I.</td>
<td>Phono 2 4th L.I.</td>
<td>Respiration</td>
<td>ECG Lead 2</td>
</tr>
<tr>
<td>10</td>
<td>400 Hz Tone Cal.</td>
<td>Background Noise</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>10</td>
<td>Background Noise</td>
<td>400 Hz Tone Cal.</td>
<td>---</td>
<td>---</td>
</tr>
</tbody>
</table>
Note that the phonocardiogram channels were calibrated following each patient's recording by a 400 Hz constant-intensity acoustical source. The sound intensity at the calibrator aperture was approximately $10^{-14}$ watts/cm$^2$. This calibration signal was used later in the data analysis for normalization and made possible the direct comparison of processed data among the patients.

**Differential Phono-Channel Delay.** The total differential system delay between the two phono-channels was measured by on-off keying a common acoustical source placed an equal distance from the two microphones, and by observing the corresponding differential time delay at the graphics terminal. The measured differential time delay was less than 400 µsec. or less than one sample time increment.

**Push Down Test.** The phonocardiogram "signal sense" (sign relation between signal polarity and chest displacement) was determined by placing a rubber sheet over the microphone and applying a downward displacement with a rubber tipped pencil.

This test indicates that an outward chest displacement corresponds to a negative signal level, whereas an inward displacement corresponds to a positive signal level in the phonocardiogram time series as observed on the graphics terminal.

**ANALOG-TO-DIGITAL CONVERSION**

During analog-to-digital conversion, the four
channels of analog data were time multiplexed and digitized by an 8-bit analog-to-digital converter. The choice of an 8-bit converter offers better than 1 percent quantitization accuracy (considered adequate for clinical measurements) and an efficient digital data format (i.e., with a 16 bits/word computer and an 8 bits/sample, two samples/word can be stored). The phonocardiogram signal was sampled at a 2.5 kHz rate, whereas the ECG, carotid pulse and respiration signals were sampled at a 625 Hz rate. The phonocardiogram and timing signals are bandwidth-limited functions with highest significant frequency components of 400 Hz and 60 Hz respectively. It is clear from the above that the selected sampling rates are more than three times the Nyquist sampling rates, and consequently the analog signals are completely defined by their digital sample sequence. A detailed mathematical description of the sampling process is presented in Chapter IV.

Continuous data storage was accomplished by the use of two 4K word computer memory buffers. The digital samples were sequentially stored in one of the buffers; when this buffer was full, data continued to be stored in the second buffer while the contents of the full buffer was transferred to digital magnetic tape by way of the DMA data path. Repetitive use of this process allowed continuous conversion.

The converted data is free of gaps because the read-out time is shorter than the read-in time, and the buffers can be switched within one sample time. During conversion,
the high degree of time correlation between data channels was maintained. The computer subroutine used for sampling is given in Appendix I.

Each buffered data block stored on the magnetic tape comprised a data record and records were separated by inter-record gaps. Digital data processed in this manner is referred to as Pass 1 data and the record format is shown in Table 3. Digital data of each patient is arranged into five data files and each data file is preceded by an alphanumeric header file describing it. Each header contains the patient's name, hospital number, and transducer locations. In addition, the first header contains other essential patient parameters and diagnostic information. The digital tape file format is shown in Table 4, and typical patient header files are shown in Tables 2, AI and 3, AI.

SELECTION OF EQUIVALENT CARDIOCYCLES

A detailed examination of the Pass 1 time series revealed that wavelets in the systole for properly selected phonocardiogram cardiocycles exhibited remarkable time coherence. This coherence existed within a Pass 1 data file for cardiocycles of the same respiration phase whose Q-Q interval variations were within 10 percent. For these selected cardiocycles, the ejection click and first and second heart sounds were very reproducible in both onset time and waveshape, with almost all of the Q-Q time deviation occurring in the diastole. The onset jitter (epoch
### TABLE 3
PASS 1 DIGITAL DATA TAPE RECORD FORMAT

<table>
<thead>
<tr>
<th>Sample Index</th>
<th>PASS 1</th>
<th>RECORD NO.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Phono 1</td>
<td>Phono 2</td>
</tr>
<tr>
<td>2</td>
<td>Phono 1</td>
<td>Phono 2</td>
</tr>
<tr>
<td>3200</td>
<td>Phono 1</td>
<td>Phono 2</td>
</tr>
<tr>
<td>1</td>
<td>Car. or Resp.</td>
<td>ECG</td>
</tr>
<tr>
<td>4</td>
<td>Car. or Resp.</td>
<td>ECG</td>
</tr>
<tr>
<td>3200</td>
<td>Car. or Resp.</td>
<td>ECG</td>
</tr>
</tbody>
</table>

**RECORD GAP**

<table>
<thead>
<tr>
<th>PASS 1</th>
<th>RECORD NO.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phono 1</td>
<td>Phono 2</td>
</tr>
</tbody>
</table>
TABLE 4
PASS 1 DIGITAL DATA TAPE FILE FORMAT

<table>
<thead>
<tr>
<th>File No.</th>
<th>ALPHA-NUMERIC PATIENT HEADER</th>
<th>F.G.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Phono 1/Phono 2</td>
<td>F.G.</td>
</tr>
<tr>
<td></td>
<td>Tone Cal</td>
<td>(FILE GAP)</td>
</tr>
<tr>
<td></td>
<td>Binary Data Recs.</td>
<td>F.G.</td>
</tr>
<tr>
<td>2</td>
<td>ALPHA-NUM. HEADER FOR NEXT FILE</td>
<td>F.G.</td>
</tr>
<tr>
<td>3</td>
<td>Phono 1 = 2L.I.</td>
<td>F.G.</td>
</tr>
<tr>
<td></td>
<td>Phono 2 = Apex</td>
<td>One Patient Data Block</td>
</tr>
<tr>
<td></td>
<td>Pulse = Car.</td>
<td>F.G.</td>
</tr>
<tr>
<td></td>
<td>ECG = Lead 2</td>
<td>F.G.</td>
</tr>
<tr>
<td></td>
<td>Binary Data Recs.</td>
<td>F.G.</td>
</tr>
<tr>
<td>4</td>
<td>ALPHA-NUM. HEADER FOR NEXT FILE</td>
<td>F.G.</td>
</tr>
<tr>
<td>5</td>
<td>Phono 1 = 2L.I.</td>
<td>F.G.</td>
</tr>
<tr>
<td></td>
<td>Phono 2 = Apex</td>
<td>F.G.</td>
</tr>
<tr>
<td></td>
<td>Respiration</td>
<td>F.G.</td>
</tr>
<tr>
<td></td>
<td>ECG = Lead 2</td>
<td>F.G.</td>
</tr>
<tr>
<td></td>
<td>Binary Data Recs.</td>
<td>F.G.</td>
</tr>
<tr>
<td>6</td>
<td>ALPHA-NUM. HEADER FOR NEXT FILE</td>
<td>F.G.</td>
</tr>
<tr>
<td>7</td>
<td>Phono 1 = 2R.I.</td>
<td>F.G.</td>
</tr>
<tr>
<td></td>
<td>Phono 2 = 4L.I.</td>
<td>F.G.</td>
</tr>
<tr>
<td></td>
<td>Pulse = Car.</td>
<td>F.G.</td>
</tr>
<tr>
<td></td>
<td>ECG = Lead 2</td>
<td>F.G.</td>
</tr>
<tr>
<td></td>
<td>Binary Data Recs.</td>
<td>F.G.</td>
</tr>
<tr>
<td>8</td>
<td>ALPHA-NUM. HEADER FOR NEXT FILE</td>
<td>F.G.</td>
</tr>
<tr>
<td>9</td>
<td>Phono 1 = 2R.I.</td>
<td>F.G.</td>
</tr>
<tr>
<td></td>
<td>Phono 2 = 4L.I.</td>
<td>F.G.</td>
</tr>
<tr>
<td></td>
<td>Respiration</td>
<td>F.G.</td>
</tr>
<tr>
<td></td>
<td>ECG = Lead 2</td>
<td>F.G.</td>
</tr>
<tr>
<td></td>
<td>Binary Data Recs.</td>
<td>F.G.</td>
</tr>
<tr>
<td>10</td>
<td></td>
<td>F.G.</td>
</tr>
</tbody>
</table>
jitter) of the wavelets was approximately ± 4 ms, where all onset times were measured from the ECG Q wave. This coherence was observed in all patients included in this study.

As pointed out in Chapter II, in the cardiohemic system the onset time and intensity of the wavelets are functions of the hemodynamics of the heart and the high degree of coherence and reproducibility observed among these selected cycles indicates that these cardiocycles are produced under identical hemodynamic conditions. In conclusion, equivalent cardiocycles at a listening site are defined here as cardiocycles which are produced at the same respiration phase and whose Q-Q interval variations are within 10 percent.

Equivalent Data Ensembles. Clearly, equivalent cycles may be averaged at any respiration phase, but to observe the maximum phonocardiogram signal changes produced by respiration, equivalent cycles are selected at mid-inspiration and mid-expiration.

Mid-inspiration (and mid-expiration) cardiocycles are defined as those cardiocycles where the maximum (and minimum) values of the respiration signal occur at the middle of the cycle. Thus, these cardiocycles are obtained at approximately maximum (and minimum) lung volumes. Fig. 12 shows the systole and early diastole of five mid-inspiration cardiocycles selected as discussed above. Note
Fig. 12. Five typical equivalent phonocardiogram records.
that the onset time and shape of the wavelets are independent of the record length (Q-Q interval).

PASS 2-3 DATA

Equivalent data ensembles (files) are generated with a two-step data reduction process. In the "initial step", equivalent cardiocycles are selected and approximately timed to contain a single Q-Q interval. The data output in this step is referred to as Pass 2 data. During this step the four-channel Pass 1 data is displayed on the graphics terminal; from this display, equivalent Q-Q interval cardiocycle records are selected with the "graphics cursor" and are output to the digital tape recorder. Excessively noisy cycles are omitted at this time.

During the second step the previously selected cardiocycles are aligned and timed to start precisely at the ECG Q wave and the data output in this step is referred to as Pass 3 data. Prior to alignment the ECG waveforms are examined and the largest and most rapidly changing signal feature (R or S wave) is selected as the alignment point. The time interval between the alignment point and the Q wave is measured and defined as the IDQ interval. This interval is stored as a parameter in the "align program" and serves as a common reference within a patient data set. Alignment is accomplished by a computer program which searches for the alignment point (local maximum or minimum) and slides the cardiocycles to the left or right
to cause an alignment about the point mentioned above. To start the aligned records at the ECG Q wave, data points to the left of the IDQ interval are deleted. Selection and timing of the four data channels occur simultaneously, maintaining a time correlation between data channels of 400 µs. Typical aligned cardiocycle records are shown in Fig. 13. As a final check on alignment and timing, ECG records of each file are "stack plotted" and carefully examined. With this two-step process, cardiocycle alignment precision (i.e., Q wave onset jitter) is approximately ± 1.6 ms.

Pass 2-3 Data Tape Formats. Both Pass 2 and Pass 3 data outputs have identical data tape record and file formats as shown in Tables 5 and 6. Note that each data file is preceded by a header file describing it. Each patient "data block" consists of seven data files; two mid-inspiration, two mid-expiration, two carotid, and one calibration file.

Data pertaining to a cardiocycle consists of four records: Phono 1, Phono 2, ECG, and Respiration or Carotid. The first three words of each record are the record length, record number and record identification character or the Pass 1 record number respectively.
Fig. 13. A typical aligned four-channel cardiocycle record
TABLE 5
PASS 2-3 DIGITAL DATA TAPE RECORD FORMAT

<table>
<thead>
<tr>
<th>RECORD LENGTH</th>
<th>15</th>
<th>6</th>
<th>0</th>
<th>BIT No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>PASS 2-3 REC. No.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PASS 1 REC. No.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phono 1 Samp. 1</td>
<td>Phono 1 Samp. 2</td>
<td>Phono 1 Last Samp.</td>
<td>Phono 1 Data Rec.</td>
<td></td>
</tr>
</tbody>
</table>

RECORD GAP

One Cardi-cycle

<table>
<thead>
<tr>
<th>RECORD LENGTH</th>
<th>15</th>
<th>6</th>
<th>0</th>
<th>BIT No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>PASS 2-3 REC. No.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Record P</td>
<td>ID. Char. 2</td>
<td>Phono 2 Samp. 1</td>
<td>Phono 2 Samp.</td>
<td>Phono 2 Last Samp.</td>
</tr>
</tbody>
</table>

RECORD GAP

<table>
<thead>
<tr>
<th>RECORD LENGTH</th>
<th>15</th>
<th>6</th>
<th>0</th>
<th>BIT No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>PASS 2-3 REC. No.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Record E</td>
<td>ID. Char. C</td>
<td>ECG Samp. 1</td>
<td>ECG Samp. 4</td>
<td>ECG Last Samp.</td>
</tr>
</tbody>
</table>
### TABLE 5--Continued

#### RECORD GAP

<table>
<thead>
<tr>
<th>RECORD LENGTH</th>
</tr>
</thead>
<tbody>
<tr>
<td>PASS 2-3 REC. No.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Record R</th>
<th>ID. Char. P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulse/Resp. Samp. 1</td>
<td>Pulse/Resp. Samp. 4</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pulse/Resp. Last Samp.</td>
</tr>
</tbody>
</table>

Pulse/Resp. Data Rec.
#TABLE 6

**PASS 2-3 DIGITAL DATA TAPE FILE FORMAT**

<table>
<thead>
<tr>
<th>ALPHA-NUMERIC PATIENT HEADER</th>
<th>F.G. (FILE GAP)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phono 1/Phono 2</td>
<td>F.G.</td>
</tr>
<tr>
<td>Tone Cal</td>
<td>F.G.</td>
</tr>
<tr>
<td>Binary Data Recs.</td>
<td>F.G.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>ALPHA-NUM. HEADER FOR NEXT FILE</th>
<th>F.G.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phono 1 = 2 L.I.</td>
<td>F.G.</td>
</tr>
<tr>
<td>Phono 2 = Apex</td>
<td>F.G.</td>
</tr>
<tr>
<td>Pulse = Carotid</td>
<td>F.G.</td>
</tr>
<tr>
<td>ECG = Lead 2</td>
<td>F.G.</td>
</tr>
<tr>
<td>Binary Data Recs.</td>
<td>F.G.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>ALPHA-NUM. HEADER FOR NEXT FILE</th>
<th>F.G.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phono 1 = 2 L.I.</td>
<td>F.G.</td>
</tr>
<tr>
<td>Phono 2 = Apex</td>
<td>F.G.</td>
</tr>
<tr>
<td>Inspiration</td>
<td>F.G.</td>
</tr>
<tr>
<td>ECG = Lead 2</td>
<td>F.G.</td>
</tr>
<tr>
<td>Binary Data Recs.</td>
<td>F.G.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>ALPHA-NUM. HEADER FOR NEXT FILE</th>
<th>F.G.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phono 1 = 2 L.I.</td>
<td>F.G.</td>
</tr>
<tr>
<td>Phono 2 = Apex</td>
<td>F.G.</td>
</tr>
<tr>
<td>Expiration</td>
<td>F.G.</td>
</tr>
<tr>
<td>ECG = Lead 2</td>
<td>F.G.</td>
</tr>
<tr>
<td>Binary Data Recs.</td>
<td>F.G.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>ALPHA-NUM. HEADER FOR NEXT FILE</th>
<th>One Patient Data Block</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phono 1 = 2 R.I.</td>
<td>F.G.</td>
</tr>
<tr>
<td>Phono 2 = 4 L.I.</td>
<td>F.G.</td>
</tr>
<tr>
<td>Pulse = Carotid</td>
<td>F.G.</td>
</tr>
<tr>
<td>ECG = Lead 2</td>
<td>F.G.</td>
</tr>
<tr>
<td>Binary Data Recs.</td>
<td>F.G.</td>
</tr>
</tbody>
</table>
### TABLE 6--Continued

<table>
<thead>
<tr>
<th>ALPHA-NUM. HEADER FOR NEXT FILE</th>
<th>F.G.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phono 1 = 2 R.I.</td>
<td></td>
</tr>
<tr>
<td>Phono 2 = 4 L.I.</td>
<td></td>
</tr>
<tr>
<td>Inspiration</td>
<td></td>
</tr>
<tr>
<td>ECG = Lead 2</td>
<td></td>
</tr>
<tr>
<td>Binary Data Recs.</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>ALPHA-NUM. HEADER FOR NEXT FILE</th>
<th>F.G.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phono 1 = 2 R.I.</td>
<td></td>
</tr>
<tr>
<td>Phono 2 = 4 L.I.</td>
<td></td>
</tr>
<tr>
<td>Expiration</td>
<td></td>
</tr>
<tr>
<td>ECG = Lead 2</td>
<td></td>
</tr>
<tr>
<td>Binary Data Recs.</td>
<td></td>
</tr>
</tbody>
</table>
CHAPTER IV

SIGNAL PROCESSING TECHNIQUES

In this chapter phonocardiogram signal processing techniques are described in detail. Time and frequency domain sampling is discussed along with the discrete Fourier transform (DFT) and the fast Fourier transform (FFT). A description of a stochastic process and its power spectrum are presented. The final sections of the chapter are devoted to envelope analysis and a description of the Hilbert transform.

TIME DOMAIN SAMPLING

The sampling process can be represented as amplitude modulation of a discrete carrier by a continuous data function [64]. This modulation process is defined by Eq. 1.

\[ s(t) = \hat{s}(t)p(t) \]  
* Eq. 1

where \[ \hat{s}(t) = \text{sampled data function} \]
\[ s(t) = \text{continuous data function} \]
\[ p(t) = \text{periodic carrier} \]

If \( p(t) \) is a finite pulse duration, unit amplitude, periodic pulse sequence, then the process is called "pulse sampling" and Eq. 1 describes a practical sampling process. If \( p(t) \) is a periodic sequence of unit delta functions, then the process is called "impulse sampling."
Let \( p(t) \) be a finite duration, periodic pulse sequence with

\[ \Delta t = \text{sampling period} \]
\[ T_p = \text{sampling pulse duration} \]

where \( p(t) \) is defined as

\[ p(t) = 1 \quad \text{when} \quad |t| \leq \frac{T_p}{2} \]
\[ p(t) = 0 \quad \text{when} \quad \frac{T_p}{2} < |t| \leq \frac{\Delta t}{2} \]

Since \( p(t) \) is periodic, it can be expanded in the Fourier series as

\[ P(t) = \sum_{k=-\infty}^{\infty} c_k e^{j\frac{2\pi kt}{\Delta t}} \]

where \( c_k \)'s are the Fourier coefficients and \( j = \sqrt{-1} \)

\[ c_k = \frac{1}{\Delta t} \int_{-\frac{T_p}{2}}^{\frac{T_p}{2}} s(t) e^{-j\frac{2\pi kt}{\Delta t}} dt \]

and \( * \)

\[ s(t) = \sum_{k=-\infty}^{\infty} c_k s(t) e^{j\frac{2\pi kt}{\Delta t}} \]

Using the equations given below and denoting the transformed functions with capital letters, the Fourier transform of \( * \) is obtained below.

\[ \int_{\infty}^{\infty} s(t) e^{-j\omega t} dt = S(\omega) \]
\[ \int_{\infty}^{\infty} s(t) e^{j\omega t} dt = S(\omega-a) \]
\[ \hat{S}_p(\omega) = \sum_{k=-\infty}^{\infty} c_k S(\omega - k\omega_s) \]  

Eq. 2

where \( \omega_s = \frac{2\pi}{\Delta t} \) = sampling rate

When impulse sampling is used

\[ p(t) = \sum_{n=-\infty}^{\infty} \delta(t-n\Delta t) \]

where \( \delta(t) = \) unit impulse function.

For this type of sampling the Fourier coefficients in Eq. 2 are

\[ c_k = \frac{1}{\Delta t} \] for all k's

and Eq. 2 reduces to

\[ \hat{S}_{\delta}(\omega) = \frac{1}{\Delta t} \sum_{k=-\infty}^{\infty} S(\omega - k\omega_s) \]  

Eq. 3

The amplitude spectra of \( \hat{S}(\omega) \) along with \( \hat{s}(t) \) for pulse and impulse sampling are given in Fig. 14. From these amplitude plots and from Eqs. 2 and 3, the following properties can be shown:

1. Sampling in the time domain produces a repetitive spectrum in the frequency domain. When impulse sampling is used, the spectrum becomes a periodic extension of \( \frac{1}{\Delta t} S(\omega) \).

2. If \( s(t) \) is bandwidth-limited to \( B \) (i.e., \( S(\omega) = 0 \) for \( |\omega| > B \)) and the sampling rate \( \omega_s \) is \( \omega_s \geq 2B \), then \( S(\omega) \) can be recovered from \( \hat{S}(\omega) \) by low-pass filtering. Consequently, \( s(t) \) can be uniquely determined from \( \hat{s}(t) \).

If \( \omega_s < 2B \), then the adjacent "lobes" of \( \hat{S}(\omega) \) are overlapping
Fig. 14. Pulse and impulse sampling of $s(t)$ and corresponding amplitude spectra
as shown in Fig. 14 and \( S(\omega) \) cannot be recovered from \( \hat{S}(\omega) \). This latter condition is referred to as "aliasing" [76], while statement 2 is referred to as the sampling theorem [64,65,66,71].

**Point Sampling.** Let \( S(\omega) \) be the Fourier transform of a signal \( s(t) \), then

\[
s(t) = \frac{1}{2\pi} \int_{-\infty}^{\infty} S(\omega) e^{i\omega t} d\omega
\]

From the above equation a discrete sequence \( \{s_n\} \) is obtained by substituting discrete values for \( t \) into \( s(t) \). This form of sampling will be referred to as point sampling. It is shown by Cooley et al. [63] that point sampling \( s(t) \) produces a periodic Fourier spectrum which is the superposition of the shifted spectra of \( S(\omega) \). This periodic property is outlined below.

Point sampling \( s(t) \) with a sampling rate of \( F \) produces time samples at the intervals of \( n\Delta t = \frac{n}{F} \), \( n = 0, \pm 1, \pm 2, \ldots \), where

\[
s(n\Delta t) = \frac{1}{2\pi} \int_{-\infty}^{\infty} S(\omega) e^{i\omega n/F} d\omega
\]

Recalling that \( e^{i\omega n/F} \) is a periodic function of \( \omega \) with a period \( 2\pi F \), the above equation is rewritten as

\[
s(n\Delta t) = \frac{1}{2\pi} \sum_{k=-\infty}^{\infty} S(\omega) e^{i\omega n/F} d\omega
\]

Assuming well behaved functions and making the substitution \( \omega = u + 2\pi k F \) (recalling that \( e^{i2\pi kn} = 1 \) for all integers \( k \) and \( n \)), then the above equation
is expressed as

\[ s(n\Delta t) = \frac{1}{2\pi} \int_{0}^{2\pi F} S_p(\omega) e^{j\omega n F} \, d\omega \tag{Eq. 4} \]

where \( S_p(\omega) = \sum_{k=-\infty}^{\infty} S(\omega + k2\pi F) \) \( \tag{Eq. 5} \)

and \( \omega \) is replaced by \( \omega \).

Comparison of Eqs. 3 and 5 reveals that impulse and point sampling are identical within a constant of \( \frac{1}{\Delta t} \).

Additionally, if the signal \( s(t) \) is bandwidth-limited to \( B \) (i.e., \( S(\omega) = 0 \) for \( |\omega| > B \)) and if the sampling rate \( F \geq \frac{2B}{2\pi} \), then the periodic function \( S_p(\omega) \) is an unaliased (nonoverlapping) extension of \( S(\omega) \) [63,66]. Since \( S_p(\omega) \) is a periodic function of \( \omega \) with period \( 2\pi F \), it can be represented by Fourier series as

\[ S_p(\omega) = \sum_{n=-\infty}^{\infty} A_n e^{-j\omega n F} \tag{Eq. 6} \]

where \( A_n = \frac{1}{2\pi F} \int_{0}^{2\pi F} S_p(\omega) e^{-j\omega n F} \, d\omega \)

Comparing Eq. 4 with the Eq. above reveals that the Fourier coefficients \( A_n \)'s can be expressed in terms of the sample values as

\[ A_n = \frac{s(n\Delta t)}{F} \]

Substituting these Fourier coefficients into Eq. 6 we obtain

\[ S_p(\omega) = \frac{1}{F} \sum_{n=-\infty}^{\infty} s(n\Delta t) e^{-j\omega n F} \tag{Eq. 7} \]

It is clear from the above that the periodic function \( S_p(\omega) \) is completely defined by the sample points \( s(n\Delta t) \).
FREQUENCY DOMAIN SAMPLING

Let $s(t)$ be a signal waveform defined on the interval of $|t| < T/2$ and zero for $|t| > T/2$, where $T$ is the record length. The Fourier transform of this signal is defined below.

$$S(\omega) = \int_{-T/2}^{T/2} s(t) e^{-j\omega t} \, dt \quad \text{Eq. 8}$$

Expanding $s(t)$ in Fourier series on the interval of $-T/2$ to $T/2$, we obtain a periodic function $s_p(t)$ given by

$$s_p(t) = \sum_{k=-\infty}^{\infty} c_k e^{j\frac{2\pi kt}{T}} \quad \text{Eq. 9}$$

where

$$c_k = \frac{1}{T} \int_{-T/2}^{T/2} s(t) e^{-j\frac{2\pi kt}{T}} \, dt \quad \text{Eq. 10}$$

and comparing Eqs. 8 and 10, we obtain

$$c_k = \frac{S(\frac{2\pi k}{T})}{T} \quad \text{Eq. 11}$$

Note that the Fourier coefficients $c_k$'s are determined from the sample values of $S(\omega)$ and consequently, define the periodic function $s_p(t)$. The above result can be restated as point sampling of $S(\omega)$ at $\frac{2\pi k}{T}$ angular frequency intervals corresponding to a periodic extension of $s(t)$ in the time domain $[60, 63, 66, 77]$ as shown in Fig. 15.
Fig. 15. A sampled amplitude spectrum and the corresponding periodically extended $s(t)$
DISCRETE FOURIER TRANSFORM

To compute Fourier transforms with a digital computer, one must consider a finite number of discrete samples in the time as well as in the frequency domain.

Suppose that a finite-duration, bandwidth-limited function \( g(t) \) can be represented by a sequence of \( N \) equally-spaced samples in the time domain. The sequence is denoted by \( \{g(n\Delta t)\} \) where

\[
n = \text{time sample index and} \\
\Delta t = \text{sample time interval.}
\]

Similarly, let \( G(\omega) \) (Fourier transform of \( g(t) \)) be represented by a sequence of \( N \) equally-spaced samples in the frequency domain where the sequence is denoted by \( \{G(k\Omega)\} \) and

\[
k = \text{frequency sample index and} \\
\Omega = \frac{2\pi}{N\Delta t} = \text{sample frequency interval.}
\]

Consistent with the above discussion, the discrete Fourier transform (DFT) of the sequence \( \{g(n\Delta t)\} \) is defined as the sequence \( \{G(k\Omega)\} \), where each component of \( \{G(k\Omega)\} \) is computed from Eq. 12 [58,60].

\[
G(k\Omega) = \frac{1}{N} \sum_{n=0}^{N-1} g(n\Delta t) e^{-j\Omega\Delta tnk} \quad \text{Eq. 12}
\]

The original time sequence \( \{g(n\Delta t)\} \) can be recovered by the inverse discrete Fourier transform (IDFT) where each component of \( \{g(n\Delta t)\} \) is computed from Eq. 13.
g(nΔt) = \sum_{k=0}^{N-1} G(kΩ) e^{jΩΔtk} \quad \text{Eq. 13}

The discrete Fourier transform pair is often referred to as the finite Fourier transform pair and within a constant, is equal to the sampled version of the periodically-extended continuous functions g(t) and G(ω) [58,60,61,62,63,75]. The periodic property of the DFT is a clear consequence of time and frequency domain sampling. Derivation of the DFT from the continuous Fourier transform is outlined below.

Consider a finite-duration and bandwidth-limited signal s(t), where s(t) = 0 for |t| ≥ T/2 and its Fourier transform S(ω) = 0 for |ω| ≥ B. Point sampling of s(t) with a sampling rate ω_s ≥ 2B produces a finite, discrete sequence in the time domain and an unaliased, continuous periodic extension of S(ω) in the frequency domain [58,60,61,63] as described in the previous section. This periodic function S_p(ω) is defined by Eq. 7. If we point sample S_p(ω) by replacing ω with kΩ, Eq. 7 can be expressed as

\[ S_p(kΩ) = \frac{T}{N} \sum_{n=-\infty}^{\infty} s(nΔt) e^{-j\frac{2πnk}{N}} \quad \text{Eq. 14} \]

where N = \frac{T}{Δt} = number of sample points per record length T. Note that time and frequency sampling yields N term periodic discrete sequences in the frequency, as well as in the time domain, as shown in Figs. 14 and 15 [58,60,61,63]. We can express Eq. 14 as a finite sum by recognizing that s(nΔt)
and $e^{-j2\pi nk/N}$ are periodic in $n$ with period $N$ where any arbitrary integer $n$ can be expressed as

$$n = rN + n_o$$

where $r =$ integer and

$$n_o = n \mod N.$$

$$S_p(k\Omega) = T(\frac{1}{N} \sum_{n=0}^{N-1} s_p(n\Delta t) e^{-j\frac{2\pi nk}{N}}) \tag{Eq. 15}$$

where $s_p(n\Delta t) = \sum_{\xi=-\infty}^{\infty} s(n\Delta t + 2\xi T) \tag{Eq. 16}$

The righthand side of Eq. 15 is the record length $T$ times the DFT of the signal $s(t)$, while the lefthand side is the point sampled and periodically-extended $S(\omega)$.

The Fast Fourier Transform (FFT). The fast Fourier transform (FFT) introduced by Cooley and Tukey [59,63] is an efficient computational algorithm used to compute discrete Fourier transform pairs. A brief examination of Eqs. 12 and 13 reveals that for a complex sequence, $N^2$ complex operations (multiplications and additions) are required to compute the DFT or IDFT from the definitions. In comparison, the FFT algorithm requires approximately $\frac{3N}{2} \log_2 N$ complex operations and at $N = 1024$, it offers a factor of approximately 200 computational savings [60,62,63]. Using Cooley and Tukey's notation, the FFT algorithm used to compute IDFT involves evaluating the complex sum given below.
\[ X(n) = \sum_{k=0}^{N-1} A(k) Q^{kn} \]  
\text{Eq. 17}

for \( n = 0, 1, \ldots, N-1 \) and \( Q = e^{j \frac{2\pi}{N}} \)

Note that the DFT defined by Eq. 12 can be expressed as

\[ S(k\Omega) = \frac{1}{N} \left\{ \sum_{n=0}^{N-1} s(n\Delta t) Q^{kn} \right\}^{*} \]  
\text{Eq. 18}

where * denotes conjugation. A comparison of Eqs. 17 and 18 reveals that they differ only by a constant and by conjugations; therefore, the same algorithm can be used to compute forward as well as inverse DFT's.

When the sequence length \( N \) is equal to the powers of 2, for example \( N = 8 \), then it is convenient to represent both \( n \) and \( k \) as a binary number; that is, for \( n = 0, 1, \ldots, 7 \) and \( k = 0, 1, \ldots, 7 \) we can write

\[ n = 4n_{2} + 2n_{1} + n_{0} \]
\[ k = 4k_{2} + 2k_{1} + k_{0} \]

where \( n_{0}, n_{1}, n_{2}, k_{0}, k_{1} \) and \( k_{2} \) can take on values of 0 or 1 only. Substituting their values into Eq. 17 and omitting \( \Delta t \) and \( \Omega \) for notational clarity, we may obtain

\[ X(n_{2},n_{1},n_{0}) = \sum_{k_{0}=0}^{1} \sum_{k_{1}=0}^{1} \sum_{k_{2}=0}^{1} A(k_{2},k_{1},k_{0}) Q_{T} \]

where

\[ Q_{T} = Q^{(4n_{2}+2n_{1}+n_{0})(4k_{2}+2k_{1}+k_{0})} \]
Completing the products of exponents and noting that \( Q^{k+n} = Q^k \cdot Q^n \), it is apparent that some of the product terms reduce to unity by the periodic property of the exponential function (i.e., \( Q^{m8} = 1 \) where \( m \) is an integer). This leads to

\[
X(n_2,n_1,n_o) = \frac{1}{\sum_{k_0=0}^{1} \sum_{k_1=0}^{1} \sum_{k_2=0}^{1}} A(k_2,k_1,k_0) Q^{y_2} Q^{y_1} Q^{y_o} \quad \text{Eq. 19}
\]

\[
A_1(n_o,k_1,k_0) \quad \text{1st stage}
\]

\[
A_2(n_o,n_1,k_0) \quad \text{2nd stage}
\]

\[
A_3(n_o,n_1,n_2) \quad \text{3rd stage}
\]

\[
X(n_2,n_1,n_o)
\]

where \( y_2 = 4n_o k_2 \quad y_1 = 2k_1 (2n_1+n_o) \quad y_o = k_o (4n_2+2n_1+n_o) \)

Note that the computation of \( X(n_2,n_1,n_o) \) involves successive computation of \( A_1, A_2 \) and \( A_3 \), each containing 8 complex terms where the last step is a simple reordering operation. It is clear that each new complex term to be computed requires only the previous set of terms; consequently, the same storage area can be shared in computations of stages. The FFT formula, Eq. 19, shows 48 complex computations; however, note that the first multiplication in each sum involves multiplication by +1 and \( Q^0 = -Q^4, Q^1 = -Q^5 \), etc. When each
of these time saving steps is accounted for, the number of operations is reduced to $\frac{3}{2} N\log_2 N$ complex operations.

A 1024 complex number FFT program (DAS FFT), using the computational steps outlined above, is given in Appendix I. This program was developed by E. Nichols, M. Stern and the author and is written in the Varian 620/I assembly language.

DESCRIPTION OF A STOCHASTIC PROCESS

In the following analysis the phonocardiogram murmur signals obtained over a short time duration are approximated as a finite stationary stochastic process. This approximation is particularly reasonable for a "flat" envelope murmur and for ejection murmurs where the ensemble elements are composed of short records centered about the peak intensity of the murmur.

Definition of a Stochastic Process. When a random time series data record is analyzed, it can be regarded as one of the many data records which may have occurred. Representation of such a process is accomplished by associating with each point of time $t$ in the range of $(-\infty \leq t \leq \infty)$, a random variable $X(t)$ which has a sample space $\{-\infty \leq X(t) \leq \infty\}$ and a corresponding probability density function (pdf), $f(x)$. Consequently, a time series can be described as an ordered set of random variables $\{X(t)\}$ defined on $(-\infty \leq t \leq \infty)$ for a continuous time series and an ordered set of random variables $\{X_t\}, t = 0, 1, 2, \ldots$, for a discrete time series.
The ordered set of random variables is called a stochastic process \([67,68,70]\) and provides a probabilistic description of the physical process as it changes with time. The double infinite set of time functions defined on this sample space is called an ensemble \([67]\).

**Moments of a Stochastic Process.** At any point in time we can define the univariant moments of a stochastic process by

\[
E[(X(t))^k] = \int_{-\infty}^{\infty} x^k f(x) dx \tag{20}
\]

and the bivariant moments by

\[
E[(X(t_1))^k (X(t_2))^n] = \int_{-\infty}^{\infty} \int_{-\infty}^{\infty} x_1^k x_2^n f(x_1, x_2) dx_1 dx_2 \tag{21}
\]

where \(f(x) = \text{probability density function at time } t\)

\(f(x_1, x_2) = \text{joint probability density function at time } t_1, t_2\)

The most important univariant moments are the mean function \(E[X(t)]\) and the second central moment, or variance function \(\text{Var}[X(t)]\). These moments are defined by the equations given below.

\[
E[X(t)] = \eta(t) = \int_{-\infty}^{\infty} x f(x) dx \tag{22}
\]

\[
\text{Var}[X(t)] = \sigma^2(t) = \int_{-\infty}^{\infty} (x-\eta(t))^2 f(x) dx \tag{23}
\]
In addition we may define the autocorrelation function (acf), \( R(t_1, t_2) \) and the autocovariance function (acvf), \( C(t_1, t_2) \) by

\[
R(t_1, t_2) = E[X(t_1)X(t_2)] = \int_{-\infty}^{\infty} x_1 x_2 f(x_1, x_2) \, dx_1 \, dx_2 \quad \text{Eq. 24}
\]

\[
C(t_1, t_2) = E[(X(t_1) - \eta(t_1))(X(t_2) - \eta(t_2))] \quad \text{Eq. 25}
\]

where \( \eta(t_i) \) = the mean of \( X(t_i) \) at time \( t_i \).

A Stationary Stochastic Process. In general, the properties of a stochastic process are time dependent. The assumption is often made that the process has reached a steady equilibrium in the sense that the statistical properties of the series are independent of absolute time. In this case the process is called stationary or strictly stationary \([67, 68, 70]\). The minimum requirement for this to hold is that the pdf, \( f(x) \) and the joint pdf of the process, \( f(x_1, x_2, \ldots, x_n) \) be independent of absolute time. The clear consequences of the stationary requirement are that the mean \( E[X] \) and the variance \( \text{Var}[X] \) are constant, and in addition, the autocorrelation and covariance functions are independent of absolute time and are functions of the lag variable \( \tau = t_1 - t_2 \).

POWER SPECTRUM ANALYSIS

In this section power spectrum analysis is introduced to study the power distribution of abnormal phonocardiograms as the function of frequency. Later, in
Chapter V the signal features of the murmur power spectrum are examined and correlated to the severity of valvar aortic stenosis.

The **Power Spectral Density of a Deterministic Signal**. The true power spectral density $B(f)$ of a continuous deterministic signal $s(t)$ is defined by

$$B(f) = \lim_{T \to \infty} B_i(f)$$  \hspace{1cm} \text{Eq. 26}$$

where $B_i(f)$ = power spectral density estimate and is derived from the Fourier transform of a signal of $T$ seconds duration by the equation given below.

$$B_i(f) = \frac{1}{T} |S_i(f)|^2$$  \hspace{1cm} \text{Eq. 27}$$

For a deterministic signal, as the record length $T$ approaches infinity, $B_i(f)$ converges "smoothly" to the theoretical spectrum $B(f)$ in the sense that for all values of $f$, the error $B(f) - B_i(f)$ approaches zero as $T$ approaches infinity [67]. It is shown by Papouilis, Jenkins and Watts; Davenport and Root [66,67,68], that for the power spectral density of a stochastic signal, the above definition cannot be applied. The basic difference between the Fourier analysis of a stochastic signal and a deterministic signal is that for the former, the variance of $B_i(f)$ does not approach zero as the record length $T$ approaches infinity. Consequently, $B_i(f)$ does not approach smoothly to $B(f)$ with increasing record length.

More general definitions for the true power spectral density are given below and apply to deterministic as well as to stationary stochastic processes. The power spectral density $B(f)$ can be defined from the autocorrelation function $R(\tau)$ as

$$B(f) = \int_{-\infty}^{\infty} R(\tau) e^{-j2\pi f \tau} d\tau$$  \hspace{1cm} \text{Eq. 28}$$

If the sample functions of the process are real, then the autocorrelation function $R(\tau)$ is real and is an even function of $\tau$; consequently, $B(f)$ is real and is an even function of $f$. Since $B(f)$ and $R(\tau)$ are Fourier transform pairs, we gain an insight to $B(f)$ by examining the equation given below at $\tau = 0$.

$$E[s(t+\tau)s(t)] = R(\tau) = \int_{-\infty}^{\infty} B(f)e^{j\omega \tau} df$$  \hspace{1cm} \text{Eq. 29}$$

$$R(0) = E[s^2(t)] = \text{Var}[s(t)] + (E[s(t)])^2 = \int_{-\infty}^{\infty} B(f) df$$  \hspace{1cm} \text{Eq. 30}$$

It is clear from Eq. 30 that $B(f)$ is a positive real-valued function which describes how the total signal power is distributed in frequency.

It is shown by Papoulis [66,70] that the true power spectral density $B(f)$ of a stationary random process can be alternatively defined by the equation given below.

$$B(f) = \lim_{T \to \infty} E[B_i(f)]$$  \hspace{1cm} \text{Eq. 31}$$
where ensemble averaging of the power spectral density estimates is required to reduce spectrum variance.

Prior to development of the FFT algorithm, power spectral density estimates were most often computed from the autocorrelation function using Eq. 28 since it was the fastest method available. With the advent of the FFT algorithm, the second expression, given by Eq. 31, is now most often used. This method is the faster of the two and therefore was employed to compute power spectral density estimates in this study.

The Discrete Power Spectral Density Estimate. To take advantage of the computational speed of the digital computer and the FFT algorithm, the discrete power spectral density function, $B_i(k\Delta f)$ is defined by replacing $f$ by $k\Delta f$ in Eq. 27 giving

$$B_i(k\Delta f) = \frac{1}{T} |S_i(k\Delta f)|^2$$  

Eq. 32

Using Eq. 15 to compute $S_i(k\Delta f)$, the above equation is written for $0 \leq k \leq \frac{N}{2} - 1$ as

$$B_i(k\Delta f) = T \left[ \frac{1}{N} \sum_{n=0}^{N-1} s(n\Delta t) e^{-j2\pi nk} \right]^2$$  

Eq. 33

$$B_i(k\Delta f) = \frac{\Delta t}{N} \left[ R_{\text{eal}}^2(k\Delta f) + I_m^2(k\Delta f) \right]$$

where $R_{\text{eal}}(k\Delta f)$ and $I_m(k\Delta f)$ are the real and imaginary components of $B_i(k\Delta f)$. Note that $B_i(k\Delta f)$ is an even function of $f$; therefore, Eq. 33 also provides values for negative frequency components.
It is shown by Jenkins and Watts [67] that for a white Gaussian random signal with zero mean, \( B_i(k\Delta f) \) is chi-square distributed. The mean \( E[B_i(f_k)] \) and variance \( \text{Var}[B_i(f_k)] \) of \( B_i(k\Delta f) \) are given in terms of the variance of the signal \( \text{Var}[s] \) as

\[
E[B_i(f_k)] = (\Delta t)\text{Var}[s] = B(k\Delta f) \quad \text{Eq. 34}
\]

\[
\text{Var}[B_i(f_k)] = \{(\Delta t)\text{Var}[s]\}^2 = B^2(k\Delta f) \quad \text{Eq. 35}
\]

It is clear from the above that \( E[B_i(f_k)] \) and \( \text{Var}[B_i(f_k)] \) are independent of record length \( T \) and consequently a \( B_i(f_k) \) estimate will not converge to the theoretical power spectral density with increasing record length. It is important to note that even if the signal is not Normally distributed, the random variables \( \text{Re}a_i(k\Delta f) \) and \( \text{Im}_m(k\Delta f) \) for \( N > 30 \) computed from the DFT have nearly a Normal distribution as can be shown by the central limit theorem. Therefore, the distribution of \( B_i(k\Delta f) \) will be very nearly chi-square.

Bartlett's Smoothing Procedure. The variance of spectral estimates can be reduced by spectral smoothing. One of the first smoothing procedures was introduced by Bartlett [81]. The procedure involves splitting up the random time series into \( m \) sub-series where each has a record length \( M \). The spectral estimate for each sub-series is computed, and an averaged spectrum for \( 0 \leq k \leq \frac{N}{2} - 1 \) is calculated according to the equation given below.

\[
\overline{B}(k\Delta f) = \frac{1}{m} \sum_{i=1}^{m} B_i(k\Delta f) \quad \text{Eq. 36}
\]
When the signal $s(t)$ is white noise, the sub-series are independent and the mean $E[\tilde{B}(f_k)]$ and variance $\text{Var}[\tilde{B}(f_k)]$ of $\tilde{B}(k\Delta f)$ are defined by the sample statistics [67,79]. If the sample size $m$ is greater than 30, then the sampling distribution is asymptotically normal [79] and the mean and variance of $\tilde{B}(f_k)$ are related to the mean and variance of $B_i(f_k)$ by equations given below.

$$E[\tilde{B}(f_k)] = E[B_i(f_k)]$$

and

$$\text{Var}[\tilde{B}(f_k)] = \frac{\text{Var}[B_i(f_k)]}{m}$$

From the above it can be concluded that the $\text{Var}[\tilde{B}(f_k)]$ is inversely proportional to the number of sub-series (records) averaged, $m$, while the relative error, defined below, is inversely proportional to $\sqrt{m}$.

$$\frac{\sqrt{\text{Var}[\tilde{B}(f_k)]}}{E[\tilde{B}(f_k)]} = \frac{1}{\sqrt{m}} \quad \text{Eq. 37}$$

Clearly, this smoothing procedure can be applied to non-sequential but independent random records such as phonocardiogram murmur signals.

**Bartlett's Spectral Window.** The Bartlett smoothing procedure outlined in the previous section now will be re-examined and its effect on spectral resolution and bias will be described.

Since the Fourier transform is a linear operation, the smoothed estimate $\tilde{B}(f)$ can be expressed in terms of the averaged autocorrelation function $\tilde{R}(\tau)$ as
For the \(i^{\text{th}}\) sub-series we may find the autocorrelation estimate for \(\tau \geq 0\) by

\[
R_i(\tau) = \frac{1}{M} \int_{(i-1)M}^{iM-\tau} s(t)s(t+\tau)dt \quad \text{Eq. 39}
\]

where \(\bar{R}(\tau) = \frac{1}{m} \sum_{i=1}^{m} R_i(\tau)\)

It is shown by Jenkins and Watts, and Richards [67,71] that \(\bar{R}(\tau)\) is given by

\[
\bar{R}(\tau) = \frac{T-m|\tau|}{T-|\tau|} \left(\frac{1}{T}\right) \int_{0}^{T-|\tau|} s(t)s(t+\tau)dt \quad \text{Eq. 40}
\]

where \(\bar{R}(\tau) = 0\) for \(|\tau| > M\).

Examination of Eq. 40 reveals that the segmented averaging described earlier is statistically equivalent to multiplying the original autocorrelation function by a "window" function \(w(\tau)\) where

\[
w(\tau) = \frac{T-m|\tau|}{T-|\tau|} \quad \text{when} \quad |\tau| \leq M
\]

\[
w(\tau) = 0 \quad \text{when} \quad |\tau| > M
\]

When \(M \ll T\), then the denominator of \(w(\tau)\) is approximately equal to \(T\). The corresponding window (Bartlett lag window) is given below.
\[ w_B(\tau) = T - \frac{|\tau|}{M} \quad \text{when } |\tau| \leq M \quad \text{Eq. 41} \]
\[ w_B(\tau) = 0 \quad \text{when } |\tau| > M \]

Substituting the windowed autocorrelation function into Eq. 38 and assuming that the number of segments \( m \) approaches infinity, we obtain the smoothed spectrum estimate as

\[ \bar{B}(f) = \int_{-\infty}^{\infty} w_B(\tau)R(\tau) e^{-j2\pi ft} d\tau \quad \text{Eq. 42} \]

Recalling that the product in the lag domain is convolution (denoted by \( * \)) in the frequency domain, the above equation can be expressed as

\[ \bar{B}(f) = W_B(f) * B(f) = \int_{-\infty}^{\infty} W_B(x)B(f-x)dx \quad \text{Eq. 43} \]

and the corresponding Bartlett spectral window is given below.

\[ W_B(f) = M \left( \frac{\sin(\pi fM)}{\pi fM} \right)^2 \quad \text{Eq. 44} \]

It is clear from Eq. 43 that the estimate \( \bar{B}(f) \) is a biased estimator of the true spectral density \( B(f) \), where the bias \( B_{\text{bias}}(f) \) is defined below.

\[ B_{\text{bias}}(f) = \bar{B}(f) - B(f) \quad \text{Eq. 45} \]

For an arbitrary spectrum containing spectral peaks, the bias will be zero only if the window width \( M \) approaches infinity.
It has been shown in the previous section that for a random signal with finite record length \( T \), the spectrum variance is reduced by decreasing the sub-series length \( M \). However, for the Bartlett window \( W_B(f) \), the first zero crossing occurs at \( 1/M \) and a small \( M \) implies smoothing over a wider range of frequencies. Consequently, with this estimation, one is forced to compromise between variance reduction and spectrum bias.

Several windows are commonly used in power spectrum spectroscopy, most often these are the Bartlett, Tukey, or Parzen windows \([67,69,72,74]\). These offer various degrees of compromise between bias and variance. However, all the windows must satisfy the following conditions \([67]\) in the lag domain.

1. \( w(0) = 1 \)
2. \( w(\tau) = w(-\tau) \)
3. \( w(\tau) = 0 \) for \( |\tau| > M \)

The Bartlett window is used in this study because it offers computational simplicity and good reduction of variance at a moderate bias.

**Variance of Smoothed Spectral Estimators.** To investigate the statistical error of a smoothed spectral estimator \( \bar{B}(f) \), the variance of this function is defined below. It is shown by Jenkins and Watts \([67]\), that for any bandwidth-limited, normal stochastic signal \( s(t) \), the variance of \( B_i(f) \) is
Similarly, for a smoothed spectral estimator \( \overline{B}(f) \) (used to estimate \( B(f) \)) the mean and variance are

\[
E[\overline{B}(f)] \approx B(f) \quad \text{Eq. 47}
\]

\[
\text{Var}[\overline{B}(f)] \approx \frac{B^2(f)}{T} \int_{-\infty}^{\infty} w^2(\tau) d\tau = \frac{B^2(f)I}{T} \quad \text{Eq. 48}
\]

For the Bartlett window

\[
I = \int_{-M}^{M} (1 - \frac{1}{M}\tau)^2 d\tau = \frac{2}{3} M \quad \text{Eq. 49}
\]

and consequently,

\[
\text{Var}[\overline{B}(f)] \approx \frac{B^2(f)}{T} \left( \frac{2}{3} M \right) \quad \text{Eq. 50}
\]

We may define a reduction factor

\[
\frac{I}{T} = \frac{\text{Var}[\overline{B}(f)]}{B^2(f)}
\]

which compares the variance of a smoothed spectrum versus the variance of the estimate. For the Bartlett window this ratio is expressed as

\[
\frac{I}{T} = .667 \left( \frac{M}{T^2} \right) \quad \text{Eq. 51}
\]

**Confidence Interval for the Smoothed Spectrum.** When \( \overline{B}(f) \) is computed from a finite number of phonocardiogram records, the precise value of \( B(f) \) cannot be predicted. However, for this case it is possible to define a confidence
region where $B(f)$ is found with a specified probability or confidence level. It is shown by Jenkins and Watts [67] that for a smoothed spectrum, the probability density distribution function of $\bar{B}(f)$ can be approximated by $a\chi^2$ where

$$\chi^2 = \text{chi-square distribution}$$

$$a \approx \frac{E[\bar{B}(f)]}{\nu}$$

and $\nu \approx \frac{2(E[\bar{B}(f)])^2}{Var[\bar{B}(f)]}$ = degree of freedom

Substituting from the previous section for the mean and variance of the Bartlett window (Eqs. 47 and 50), $\nu = 3 \frac{T}{M}$.

Knowing that the distribution is chi-square, the confidence interval for $B(f)$ at each value of $\bar{B}(f)$ is obtained from the probabilistic equation given below.

$$Pr(\chi_L < B(f) < \chi_H) = 1 - \alpha$$

The percent confidence level is $100(1-\alpha)$ for the limits given below.

$$\chi_L = \frac{\nu}{x(1-\frac{\alpha}{2})} \bar{B}(f)$$

$$\chi_H = \frac{\nu}{x \frac{\alpha}{2}} \bar{B}(f)$$

For a given confidence level, the confidence limits are usually evaluated from chi-square tables or charts [67,79]. As the degree of freedom approaches infinity, the chi-square distribution approaches the Normal distribution. Consequently,
for \( v > 30 \), the confidence limits can be closely approximated from the Normal distribution and the spectrum is estimated with a desired confidence level by the equation below.

\[
B(f) = \overline{B}(f) + z\sqrt{\text{Var}[B(f)]} \tag{Eq. 52}
\]

In the above equation \( z \) is a confidence parameter; for 99.73\% confidence level, \( z = 3.00 \), for a 95\% confidence level, \( z = 1.96 \), and for a 68.27\% confidence level, \( z = 1 \). For the Bartlett window, Eq. 52 can be expressed as

\[
B(f) \approx \overline{B}(f)(1 + z \frac{\sqrt{2/3}}{\sqrt{m}}) \tag{Eq. 53}
\]

where \( m \) is the number of sub-intervals or records averaged.

**Bandwidth of a Spectral Window.** It has been shown in the previous sections that the variance, bias and resolution of \( \overline{B}(f) \) is determined by the shape and width of the spectral window \( W(f) \). In this section the equivalent bandwidth of a spectral window is defined which determines the above-mentioned properties of the spectrum. Consider a rectangular spectral window with a bandwidth \( h \) defined by the equation given below.

\[
W_R(f) = \frac{1}{h} \quad \text{for} \quad -\frac{h}{2} \leq f \leq \frac{h}{2}
\]

The total AC power or variance within this window is given below.

\[
\text{Var}[\overline{B}(f)] \approx \frac{B^2(f)}{T} \cdot \frac{1}{h}
\]
For a nonrectangular spectral window, we define its bandwidth \( b \) as the width of a rectangular window which gives the same variance or AC power [67].

\[
\text{Var} [\overline{B}(f)] \approx \frac{B^2(f)}{T} \cdot \frac{1}{b} = \frac{B^2(f)}{T} \frac{1}{\int_{-\infty}^{\infty} w^2(\tau) d\tau}
\]

This definition is sometimes referred to as equivalent bandwidth and spectrum smoothing occurs within it. Consistent with the above discussion, the bandwidth of an arbitrary window is defined [67] as

\[
b = \frac{1}{\int_{-\infty}^{\infty} w^2(\tau) d\tau} = \frac{1}{\int_{-\infty}^{\infty} W^2(f) df} = \frac{1}{T}
\]

and the bandwidth for the Bartlett spectral window is given below.

\[
b_B = \frac{3}{2M}
\]

**Summary of Bartlett Window Properties.** The essential properties of the Bartlett window described in this section are summarized in the table below.
TABLE 7
SUMMARY OF BARTLETT WINDOW PROPERTIES

<table>
<thead>
<tr>
<th>Spectral Window $W(f)$</th>
<th>Variance Ratio $I/T$</th>
<th>Degree of Freedom $3M$</th>
<th>Bandwidth $\frac{3}{2M}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$M(\sin(\pi fM))^2$</td>
<td>$\frac{2M}{3T}$</td>
<td>$\frac{3T}{M}$</td>
<td>$\frac{3}{2M}$</td>
</tr>
</tbody>
</table>


A discrete power spectral function is defined below in order to study the power distribution of a phonocardiogram signal as a function of frequency. More specifically, the discrete power spectral estimate $P_i(k\Delta f)$ of a signal for $0 \leq k \leq \frac{N}{2} - 1$ is defined as

$$P_i(k\Delta f) = \frac{B_i(k\Delta f)}{T} \quad \text{Eq. 56}$$

Note that $B_i(k\Delta f)$ is a power density function while $P_i(k\Delta f)$ is a power function and each harmonic term $k\Delta f$ represents the signal power in a bandwidth $\frac{1}{T}$ centered at $k\Delta f$. With this definition, the power spectrum is merely the square of the magnitude of the discrete Fourier spectrum as defined by Eq. 12 and the total signal power is computed from the equation given below.

$$P_s = \sum_{k=-\frac{N}{2}+1}^{\frac{N}{2}-1} P_i(k\Delta f) \quad \text{Eq. 57}$$
The power spectral estimate of a phonocardiogram signal is computed here by using the steps outlined below.

1. Select the required signal in the time domain using the rectangular Bartlett data window.
2. Remove the DC bias introduced by the analog-to-digital converter.
3. Divide each signal amplitude by the rms value of the calibration signal. This normalization step removes microphone and other gain setting errors.
4. Compute the complex DFT spectrum as defined by Eq. 12 using an N = 1024 fixed length FFT algorithm. When the time series record length is < 1024, the additional buffer values are set equal to zero. The effect of lengthening the time sequence by extra zeros is merely interpolation in the frequency domain [58].
5. Compute the single-sided (0 ≤ k ≤ 511) power spectral estimate as prescribed by Eq. 56. That is, for each harmonic integer k, find the sum of the squared real and squared imaginary components of the DFT spectrum.

The power spectral estimates were computed either interactively using the program "AUTOFREQ" or in batch mode using the subroutine FANAL. The programs, along with their descriptions, are given in Appendix I. A typical V.A.S.
murmur power spectral estimate smoothed by a 100 ms. Bartlett window is shown in Fig. 16. Explanations of the plot labels are given in the next section, while the analysis and interpretations of the spectrum are given in Chapter V. 

Explanations of the Plot Labels. An explanation of the plot labels is as follows: the first and second lines contain the patient's first name, identification number, respiration phase, and microphone location. The respiration phases are inspiration, expiration, and minspiration (mixed inspiration and expiration). On the third line, T is the plotting time interval measured in ms. from the Q wave of the ECG signal. On the fifth line, N is the number of records averaged and R is the last record number acquired for analysis. The remaining letters describe the channel number, type of analysis performed (TIM. = time, PWS = power spectrum, ENV. = envelopogram), and the analysis sampling rate (DEF. = 2.5 KHz, SUM. = 1.25 KHz). The vertical scales are normalized intensity scales. Prior to computations, the PCG records are amplitude-normalized by the rms value of the appropriate calibration records. This step removes microphone and other gain setting errors, allowing direct data comparison among patients.

ENVELOPE ANALYSIS

In this section a real-valued function, called an envelopogram, is derived from the phonocardiogram signal by complex demodulation. The envelopogram is essentially a
Fig. 16. A typical V.A.S. murmur power spectrum estimate
high-resolution intensity plot of the phonocardiogram signal and is used to locate the epochs, measure durations, and estimate the intensity of phonocardiogram wavelets and murmurs.

In the development of an envelogram, the Hilbert transform and the analytic signal representation of the phonocardiogram signal are required. These are defined and their properties explored in the two sections that follow.

The Hilbert Transform. Let \( s(t) \) be a real signal with a Fourier transform \( S(\omega) \). The Hilbert transform [66,70,73,80] of \( s(t) \), denoted by \( \hat{s}(t) \) is defined by the convolution (denoted by \( * \)) integral given below.

\[
\hat{s}(t) = s(t) * \left( \frac{1}{\pi t} \right) = P \left[ \frac{1}{\pi} \int_{-\infty}^{\infty} \frac{s(\lambda)}{t-\lambda} d\lambda \right]
\]

Eq. 58

where \( P \) stands for the Cauchy principal value of the integral. The Fourier transform of \( \frac{1}{\pi t} \), [66,73] is

\[
\int_{-\infty}^{\infty} \frac{1}{\pi t} e^{-j\omega t} dt = -j \text{sgn}(\omega)
\]

where \( \text{sgn}(\omega) = 1 \) when \( \omega > 0 \)
\( \text{sgn}(\omega) = 0 \) when \( \omega = 0 \)
\( \text{sgn}(\omega) = -1 \) when \( \omega < 0 \)

The Fourier transform of \( \hat{s}(t) \), denoted by \( \hat{S}(\omega) \), can be computed in the frequency domain from the equation given below
by recalling that convolution in the time domain is the product in the frequency domain.

\[ \hat{\mathbf{S}}(\omega) = -j \text{sgn}(\omega) S(\omega) \]  
Eq. 59

It is clear from the above that \( \hat{S}(t) \) is produced by shifting the phase of \( s(t) \) by \(-90^\circ\) for \( \omega > 0 \) and \(+90^\circ\) for \( \omega < 0 \). The singular case when \( f = 0 \) is covered by defining \( \text{sgn}(0) = 0 \). It is shown below that if \( s(t) \) is real, then \( \hat{S}(t) \) is also real. The necessary and sufficient condition for a signal to be real [66,73] is as follows:

\[ S(-\omega) = \hat{S}(\omega) \]

Applying the above definition to Eq. 59 and realizing that \( \text{sgn}(\omega) \) is an odd function of \( \omega \), gives

\[ \hat{\mathbf{S}}^*(\omega) = \hat{S}(-\omega) \]

and consequently \( \hat{S}(t) \) is real. This property of \( \hat{S}(t) \) is required when the analytic signal representation \( s(t) \) is developed.

The Analytic Signal. Using the Hilbert transform, the analytic signal [66,70,73,80,89] denoted by \( z(t) \) is defined as

\[ z(t) = s(t) + j\hat{s}(t) \]  
Eq. 60

Since \( s(t) \) and \( \hat{s}(t) \) are real signals, it is clear from the definition that \( z(t) \) is complex; consequently, \( Z^*(\omega) \) is not equal to \( Z(-\omega) \). In fact \( Z(\omega) \) is a signal which contains only positive frequency components (i.e., \( Z(\omega) \) is an upper
single-sideband signal). To show this, one merely needs to take the Fourier transform of Eq. 60 and substitute Eq. 59 for $S(\omega)$ resulting in

$$Z(\omega) = S(\omega) [1 + \text{sgn}(\omega)] \quad \text{Eq. 61}$$

Recalling the definition of $\text{sgn}(\omega)$, the above equation can be rewritten as

$$Z(\omega) = 2S(\omega) \quad \text{for } \omega > 0$$
$$Z(\omega) = S(\omega) \quad \text{for } \omega = 0 \quad \text{Eq. 62}$$
$$Z(\omega) = 0 \quad \text{for } \omega < 0$$

Envelope, Phase and Frequency of the Phonocardiogram Signal. Let $s(t)$ be the phonocardiogram signal and $z(t)$ be the corresponding analytic signal of $s(t)$. The envelope of the signal $e(t)$ is defined here as

$$e(t) = |z(t)| = [s^2(t) + s^2(t)]^{1/2} \quad \text{Eq. 63}$$

while the phase $\theta_s(t)$ and frequency $\omega_s(t)$ of the signal $s(t)$ are defined as

$$\theta_s(t) = \arctan \left( \frac{s(t)}{s(t)} \right) = \text{ph}(z(t)) \quad \text{Eq. 64}$$
$$\omega_s(t) = \frac{d\theta_s(t)}{dt} \quad \text{Eq. 65}$$

The envelope, phase and frequency as defined above coincide with the normally used descriptors of narrow band signals [73,80]. The envelope is of particular interest in phonocardiogram signal analysis since it can be employed to
time narrow band wavelets and to identify murmur intensity patterns.

Envelope of Heart Sounds and Clicks. In this study heart sounds and clicks are represented as the modulated signal given below.

\[ s(t) = a(t) \cos(\omega_c t + \phi(t)) \]  

Eq. 66

where \( a(t) \) = modulating signal envelope 
\( \omega_c \) = carrier or mean frequency 
and \( \phi(t) \) = phase deviation

It will be shown that if the bandwidth of \( a(t) \cos \phi(t) \) and \( a(t) \sin \phi(t) \) are less than \( \omega_c \), then \( a(t) \) is equal to the magnitude of the analytic signal. To show this, one must investigate the Hilbert transform of the products of two functions. It is shown by Bennett [73] that if \( f(t) \) and \( g(t) \) are low-pass and high-pass signals respectively (i.e., \( f(t) \) and \( g(t) \) are two real functions with nonoverlapping Fourier spectra and the spectrum of \( f(t) \) is confined below the spectrum of \( g(t) \)), then

\[ H[f(t)g(t)] = f(t)H[g(t)] \]  

Eq. 67

where \( H \) is the Hilbert transform operator. In addition, we must recall that

\[ H[\sin(\omega_c t)] = -\cos(\omega_c t) \]

and \[ H[\cos(\omega_c t)] = \sin(\omega_c t) \]

Using the trigonometric identities, Eq. 66 is expressed as
\[ s(t) = a(t)(\cos(\phi(t)))\cos\omega_c t - a(t)(\sin(\phi(t)))\sin\omega_c t \]

Requiring that \( a(t)\cos(\phi(t)) \) and \( a(t)\sin(\phi(t)) \) be low-pass signals with bandwidths below \( \omega_c \), we obtain

\[ \hat{s}(t) = a(t)[(\cos(\phi(t)))\sin\omega_c t + (\sin(\phi(t)))\cos\omega_c t] \]

\[ \hat{s}(t) = a(t)\sin(\omega_c t + \phi(t)) \quad \text{Eq. 68} \]

The corresponding analytic signal is

\[ z(t) = s(t) + j\hat{s}(t) \]

\[ z(t) = a(t)[\cos(\omega_c t + \phi(t)) + j\sin(\omega_c t + \phi(t))] \quad \text{Eq. 69} \]

\[ z(t) = a(t)e^{j\omega_c t + \phi(t)} \]

Taking the magnitude of the above equation gives the desired result.

\[ |z(t)| = e(t) = a(t) \]

For a general modulated signal in the form of Eq. 66, but with overlapping frequency spectra between the carrier and \( a(t)\cos(\phi(t)) \) or \( a(t)\sin(\phi(t)) \), the Hilbert transform of this signal is given by Rihaczek [80] as

\[ \hat{s}(t) = a(t)\sin(\omega_c t + \phi(t)) + K(t) \quad \text{Eq. 70} \]

where the significance of the correction terms \( K(t) \) diminish as the essential frequencies in \( a(t) \) and \( \phi(t) \) decrease compared to \( \omega_c \). It is clear from the above that the envelope of this signal derived from \( z(t) \) is not equal to \( a(t) \);
however, in most cases the error is reasonably small [90].
It is shown by Rubin and DiFranco [90], that for a wide-band
signal defined by Eq. 66, the rms error between \( z(t) \) and
\( a(t) \) is a function of the percent bandwidth, where the
percent bandwidth is defined as 100% times the modulating
signal bandwidth divided by \( f_c \). For a Gaussian pulse
envelope at 50% bandwidth, the error is approximately 2%.

Envelope of the Murmur Signal. In this section it
will be shown that the analytic signal can be used to find
the intensity patterns of heart murmurs. When \( s(t) \) is a
wide-band signal with a bandwidth approximately equal to \( \omega_s \),
the modulating signal \( a(t) \) as defined by Eq. 66 loses its
meaning [80]. However, the envelope \( e(t) \) as defined by
Eq. 63 can still be used to identify a slowly changing modu­
lating signal. To demonstrate this, let \( v(t) \) be a positive-
valued, high-pass random Gaussian signal where the Fourier
transform \( V(f) = 0 \) for \( 500 \text{ Hz} \leq |f| \leq 30 \text{ Hz} \). We define the
murmur signal \( s(t) \) as the product of these two signals.

\[
s(t) = m(t)v(t)
\]

and the Hilbert transform of this product as defined by
Eq. 67 is

\[
\Delta s(t) = m(t)\Delta v(t)
\]

and the corresponding analytic signal is

\[
z(t) = m(t)(v(t)+j\Delta v(t))
\]

and

\[
|z(t)| = e(t) = m(t)|v(t)+j\Delta v(t)|
\]

Eq. 71
Note that Eq. 71 is the product of two positive-valued functions. Since \( v(t) \) is a bandwidth-limited random signal, the ensemble average of \( e(t) \) will produce

\[
E[e(t)] = m(t)K
\]

Eq. 72

where \( K \) is a constant approximately equal to one and therefore, the murmur intensity envelope shape is preserved in this analysis.

**Computation of the Discrete Envelogram Estimate.**

The discrete envelope of a phonocardiogram cycle, as defined by the equation given below, is referred to as the envelopogram estimate.

\[
|\text{IDFT}\{Z(k\Delta f)\}| = |\text{IDFT}\{S(k\Delta f)(1+\text{sgn}(k\Delta f))\}|
\]

Eq. 73

where \( \text{IDFT} \) denotes the inverse discrete Fourier transform operation. The envelopogram estimate is a high-resolution intensity plot of the phonocardiogram signal and is rapidly computed in the frequency domain using Eq. 73 and the FFT algorithm as outlined below.

1. Input the sample points of a phonocardiogram cycle to the real buffer of the FFT subroutine. The number of data points must be less than or equal to 1024.
2. Remove the DC bias introduced by the analog-to-digital converter.
3. Divide each signal amplitude by the rms value of the calibration signal. This normalization
step removes microphone and other gain setting errors.

4. Compute the complex DFT spectrum as defined by Eq. 12 using an N = 1024 fixed-length FFT algorithm. When the phonocardiogram cycle length is less than 1024, the additional buffer values are set equal to zero.

5. Set all negative frequency terms equal to zero (i.e., all terms for \(512 \leq k \leq 1023\)).

6. Multiply the positive frequency terms by 2 (i.e., all terms for \(1 \leq k \leq 511\)). Note that the DC value \(k = 0\) remains unchanged.

7. Take the 1024 point inverse discrete Fourier transform using the FFT algorithm.

8. Compute the magnitude of the complex function obtained in Step 7 (i.e., for each integer, find the square root of the sum of squared real and squared imaginary components). The resulting real-valued function is the envelogram estimate of a phonocardiogram cycle.

A typical phonocardiogram cycle and its corresponding envelogram estimate are shown in Figs. 7 and 17. Note that the large, narrow-band wavelet (aortic ejection click) occurring at approximately 90-113 ms. is demodulated and represented on the envelogram as a single pulse, where its value is equal to the intensity of the wavelet. Similarly, wavelets in the 350-440 ms. time range (\(s_2\)) are demodulated.
Fig. 17. Envelopogram estimate of Fig. 7
and separated into four major components. The demodulation process resembles the absolute value function for the wide-band random signals occurring in the 120-350 ms. and 440-800 ms. time ranges.
CHAPTER V

RESULTS

In this chapter a complete data set is described and the analysis results are presented in two parts. The first part contains averaged envelogram and phonocardiogram plots and their descriptions. These plots aid in the positive diagnosis of systolic heart diseases and condense the large volume of phonocardiogram time series data into a single display. The plots can be used for rapid identification and accurate timing of phonocardiogram events. It is demonstrated that the essential aortic identification signal features are preserved or enhanced in these displays.

In the second part, selected segments of aortic ejection murmurs are gated and an average power spectrum is computed. From this spectrum, quantitative murmur diagnosis parameters (first spectral moment and bandwidth) are defined and computed. For the thirteen catheterized patients, correlation studies between the calculated power spectrum parameters and the peak systolic ejection gradient (P.S.E.G. measured by catheterization) are presented. These studies indicate that good correlation exists between the first spectral moment and the P.S.E.G. and that this noninvasive technique is useful in assessing the severity of aortic stenosis.
DESCRIPTION OF THE PATIENT DATA SET

Fourteen catheterized and four clinically-diagnosed aortic stenosis patients were recorded for this study. One of the catheterized patients, Raymond S., Hosp. #80-62-02, was later omitted from the analysis when post-operative diagnosis indicated that his disease was severe congenital deformation of the aorta rather than valvar aortic stenosis. All the catheterized patients had either mild or no aortic regurgitation. For the catheterized patients, the personal data, the diagnosis, the degree of aortic regurgitation (Aortic Regurg), and the peak systolic ejection gradient (P.S.E.G.), (obtained from Children’s Hospital catheterization data charts) are tabulated in Table 8.

In addition, six normal patients were included in this study to facilitate the identification of aortic stenosis signal features. Personal data for the normal and clinically-diagnosed (uncatheterized) aortic stenosis patients are given in Table 9.

Prior to data recording, the chest wall thickness of each patient was classified as thin, medium, or thick, gauged by the following criteria: thin-walled if the ribs were clearly visible, medium-walled if the ribs were not distinguishable but had no appreciable fatty deposit, and thick-walled if the ribs were covered by a fatty layer. All of the patients included in the study had no chest deformities and all had normal body temperatures.
## TABLE 8

### THIRTEEN CATHETERIZED VALVAR AORTIC STENOSIS PATIENTS DATA

<table>
<thead>
<tr>
<th>Name</th>
<th>Hosp. #</th>
<th>Age/Sex</th>
<th>Chest Wall</th>
<th>Cath. Diagnosis</th>
<th>Aortic Regurg.</th>
<th>P.S.E.G. mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Roger F.</td>
<td>47-99-27</td>
<td>16 M</td>
<td>Thick</td>
<td>Mod. V.A.S.</td>
<td>---</td>
<td>75</td>
</tr>
<tr>
<td>Tommy K.</td>
<td>63-77-80</td>
<td>10 M</td>
<td>Thin</td>
<td>Mild V.A.S.</td>
<td>---</td>
<td>9-18</td>
</tr>
<tr>
<td>Donald G.</td>
<td>62-12-80</td>
<td>10 M</td>
<td>Thin</td>
<td>Triv. V.A.S.</td>
<td>Mild</td>
<td>16</td>
</tr>
<tr>
<td>Natalie K.</td>
<td>70-89-05</td>
<td>8 F</td>
<td>Med.</td>
<td>Mild V.A.S.</td>
<td>Triv.</td>
<td>23</td>
</tr>
<tr>
<td>Bryan K.</td>
<td>60-91-88</td>
<td>14 M</td>
<td>Thin</td>
<td>Mild V.A.S.</td>
<td>---</td>
<td>39</td>
</tr>
<tr>
<td>Robert M.</td>
<td>53-91-59</td>
<td>19 M</td>
<td>Med.</td>
<td>Mild-Mod. V.A.S.</td>
<td>Mild</td>
<td>45</td>
</tr>
<tr>
<td>Elizabeth R.</td>
<td>55-01-61</td>
<td>12 F</td>
<td>Thin</td>
<td>Mod. V.A.S.</td>
<td>---</td>
<td>45</td>
</tr>
<tr>
<td>Rudolph B.</td>
<td>68-97-78</td>
<td>9 M</td>
<td>Thin-Med.</td>
<td>Mod. V.A.S.</td>
<td>---</td>
<td>45</td>
</tr>
<tr>
<td>Mark M.</td>
<td>68-95-48</td>
<td>10 M</td>
<td>Thin</td>
<td>Triv. V.A.S.</td>
<td>Triv.</td>
<td>6-8</td>
</tr>
<tr>
<td>Jonathan F.</td>
<td>64-87-14</td>
<td>9 M</td>
<td>Thin</td>
<td>Triv. V.A.S.</td>
<td>Triv.</td>
<td>5-9</td>
</tr>
</tbody>
</table>
TABLE 9

PERSONAL DATA FOR NORMAL AND CLINICALLY DIAGNOSED VALVAR AORTIC STENOSIS PATIENTS

<table>
<thead>
<tr>
<th>Clinically-Diagnosed Valvar Aortic Stenosis Patients</th>
<th></th>
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</thead>
<tbody>
<tr>
<td>Name</td>
<td>Hospital #</td>
<td>Age</td>
<td>Sex</td>
<td>Chest Wall</td>
</tr>
<tr>
<td>Edward D.</td>
<td>57-03-63</td>
<td>14</td>
<td>M</td>
<td>Thin</td>
</tr>
<tr>
<td>John B.</td>
<td>58-29-30</td>
<td>9</td>
<td>M</td>
<td>Thick</td>
</tr>
<tr>
<td>John R.</td>
<td>66-12-34</td>
<td>7</td>
<td>M</td>
<td>Med.</td>
</tr>
<tr>
<td>Donald D.</td>
<td>79-41-95</td>
<td>15</td>
<td>M</td>
<td>Thin</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Normal Patients</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Name</td>
<td>Hospital #</td>
<td>Age</td>
<td>Sex</td>
<td>Chest Wall</td>
</tr>
<tr>
<td>Kenneth S.</td>
<td>---</td>
<td>10</td>
<td>M</td>
<td>Thin</td>
</tr>
<tr>
<td>Sherry C.</td>
<td>---</td>
<td>13</td>
<td>F</td>
<td>Med.</td>
</tr>
<tr>
<td>Steven C.</td>
<td>---</td>
<td>10</td>
<td>M</td>
<td>Thin</td>
</tr>
<tr>
<td>Cameron C.</td>
<td>---</td>
<td>15</td>
<td>M</td>
<td>Med.</td>
</tr>
<tr>
<td>Lynne S.</td>
<td>---</td>
<td>13</td>
<td>F</td>
<td>Thin</td>
</tr>
<tr>
<td>Sheldon W.</td>
<td>---</td>
<td>10</td>
<td>M</td>
<td>Thin</td>
</tr>
</tbody>
</table>
ENSEMBLE-AVERAGED ENVELOGRAMS

As the initial step of the interactive diagnostic analysis, an ensemble-averaged envelogram was computed for each data file as defined below.

\[ \bar{e}(n\Delta t) = \frac{1}{N_{\text{REC}}} \sum_{i=1}^{N_{\text{REC}}} e_i(n\Delta t) \]

where \( N_{\text{REC}} = \text{Number of records averaged} \)

\( e_i(n\Delta t) = \text{value of the } i^{\text{th}} \text{ envelogram estimate at time } n\Delta t \)

and \( \bar{e}(n\Delta t) = \text{value of the averaged envelogram at time } n\Delta t \).

The computed ensemble-averaged envelograms were plotted on the graphics terminal and hard copies were generated. The plots were examined and the maximum murmur intensity site was noted. For nearly all of the aortic stenosis patients studied, the maximum intensity site was at the 2nd R.I. space. The interactive diagnosis program and its description are given in Appendix II.

Ensemble-averaged envelogram plots at the 2nd R.I. for inspiration and expiration are shown in Figs. 18 and 19. A comparison of Figs. 17 and 18 reveals that in the averaged envelogram, "fine wavelet structures" are preserved while murmur intensity variance is reduced by approximately a factor of three. Note that good correlation exists between Figs. 18 and 19 in the 113-177 ms. time interval.
Fig. 18. Averaged V.A.S. envelopogram for inspiration
Fig. 19. Averaged V.A.S. envelopogram for expiration
Consequently, these peaks are probably not produced by random intensity fluctuations, but are more likely produced by murmur amplitude modulation. This interpretation is supported by the following observations: (1) the pressure fluctuations (trill) occur in the carotid pulse waveform during the 113-177 ms. time interval as shown in Fig. 20 and correlate with the intensity fluctuation observed in their corresponding envelogram, and (2) the gated averaged power spectrum of this region does not contain strong line structures, indicating that the signal is random.

The large intensity increase in the 89-113 ms. range shown in Fig. 18 is identified as an aortic ejection click. Identification is based on the following observations:

1. It correlates well with the upstroke of the carotid pulse.
2. Maximum intensity occurs at the 2nd R.I.
3. Intensity and onset time of the event are not affected by respiration.

Observation 1 suggests that the intensity increase is an ejection click, where 2 and 3 suggest aortic origin.

The intensity change in the 358-448 ms. range shown in Fig. 18 is identified as the aortic component of the second heart sound. Identification is supported by the following observations:

1. It radiates well to all four listening areas, particularly to the 2nd R.I. and to the apex.
Fig. 20. A typical V.A.S. carotid pulse
2. The entire wavelet occurs earlier upon inspiration than upon expiration.

The intensity increase in the 130-358 ms. range shown in Fig. 18 is identified as an aortic ejection murmur. Its timing and diamond shape imply that the murmur is ejection type and its maximum intensity location suggests aortic origin.

ENSEMBLE-AVERAGED WAVELETS

Heart sounds and murmurs are identified and accurately timed from the averaged envelogram plots. Knowing the time of occurrence, wavelets of special interest can be gated and an ensemble-averaged wavelet (averaged signal waveform) can be computed as defined by the equation given below.

$$\tilde{s}(n\Delta t) = \frac{1}{N_{\text{REC}}} \sum_{i=1}^{N_{\text{REC}}} s_i(n\Delta t)$$

where \( s_i(n\Delta t) \) = signal intensity of \( i^{th} \) estimate at time \( n\Delta t \)

and \( \tilde{s}(n\Delta t) \) = averaged signal intensity at time \( n\Delta t \).

As pointed out in Chapter III, wavelets within an equivalent ensemble are highly reproducible in both time and waveshape. The approximate onset time jitter of wavelets is 4 ms. measured from the ECG Q wave while the difference in mean onset time from inspiration to expiration is approximately 16 ms. In addition, waveshapes of the aortic wavelets (i.e., aortic ejection click and A2) are
independent of respiration. The onset time jitter of an aortic wavelet can be removed by searching for and locating a particular signal feature (local maximum or minimum) within a narrow time window. Thus, wavelets can be aligned prior to averaging.

Aligned averaging is equivalent to time domain filtering where superimposed signals can be separated, and reliable average wavelet waveforms can be obtained. When two wavelets with similar frequency characteristics have nearly identical onset times, large segments of their waveforms are superimposed. Due to overlapping frequency components, separation by frequency filtering is not possible. Examples of such events are the aortic-pulmonic components of the second heart sound and the first heart sound ejection click. The onset time jitter of these wavelets tends to be independent, since they are generated by different physical events. Alignment of the early event and ensemble averaging cause enhancement of the early component and suppression of the later event. This effect is shown in Figs. 21 and 22. A typical, single, normal second heart sound of Sherry C., consisting of aortic (373-400 ms.) and pulmonic (406-430 ms.) components is shown in Fig. 21, with the aligned and averaged aortic wavelet shown in Fig. 22. Prior to averaging, alignment was performed on the aortic component. Note that in Fig. 22 the pulmonic component (second wavelet) is suppressed, while the aortic component is preserved.
Fig. 21. A typical normal second heart sound
Fig. 22. The aligned averaged aortic component of Fig. 21
Similarly, aligned averaging can be used to separate wavelets (e.g., mid and late systolic clicks, ejection clicks, etc.) from superimposed random signals (e.g., murmurs). To demonstrate this, a 100 ms. time segment containing the click of Edward D. is gated, and the aligned-averaged time waveform is computed. Note that a high-frequency component is evident in the single record (95-117 ms.), Fig. 23, but that an additional low-frequency component observed in the averaged plot (115-135 ms.), Fig. 24, is obscured by the murmur.

ESTIMATING THE SEVERITY OF AORTIC STENOSIS USING MURMUR POWER SPECTRAL ANALYSIS

As the first step of the severity assessment analysis, averaged envelopgram plots at the 2nd R.I. space are examined, and a 100 ms. "rectangular data window" is chosen centered around the peak intensity of the systolic ejection murmur. Since the duration of the systole is usually 300-400 ms., this data window encompasses approximately one-third of the murmur signal. During this time interval the murmur is at a reasonably constant intensity and is approximated as a stationary random process. In addition, this data window is sufficiently delayed from the first heart sound and the ejection click; consequently, these signals contribute a negligible amount to the total signal intensity. Using the selected data window, the required murmur signals are gated at the 2nd R.I. and an averaged power spectrum is computed.
Fig. 23. A typical aortic ejection click
Fig. 24. A typical aligned averaged aortic ejection click
The averaged murmur power spectrum of Edward D., computed from inspiration and expiration data files as described in Chapter IV, is shown in Fig. 25. The spectral resolution provided by the rectangular data window (computed by Eq. 55) is 15 Hz. The confidence interval of the averaged spectrum (computed by Eq. 53) at $Z = 1$ (68.27% confidence level) is $\pm 0.16 \hat{B}(f)$. Comparison of the averaged murmur power spectrum (Fig. 25) with a spectral estimate (Fig. 16), reveals that the estimate is inherently random and unreproducible, while the averaged spectrum obtained from twenty-four estimates converges to a spectral shape which appears to contain four major peaks.

For each averaged power spectrum, the first spectral moment $\hat{f}$ (AVE. FRQ.) and the spectrum bandwidths ($%F$) at 10% area increments are computed. The first spectral moment $\hat{f}$ is defined by the equation given below,

$$\hat{f} = \frac{\sum_{k=0}^{511} (k\Delta f) \hat{P}(k\Delta f)}{\sum_{k=0}^{511} \hat{P}(k\Delta f)}$$

where $\hat{P}(k\Delta f) = \text{average } k^{th} \text{ spectral component}$

$$\hat{P}(k\Delta f) = \frac{1}{N.\text{REC}} \sum_{i=1}^{N.\text{REC}} P_i(k\Delta f)$$

$\Delta f = 1.2207 \text{ Hz}$

and $N.\text{REC} = \text{number of records averaged}$
Fig. 25. A typical averaged V.A.S. murmur power spectrum
and the spectrum bandwidth is defined as the frequency increment centered about $\tilde{f}$, which includes a specified fraction of the total spectral area. These parameters, calculated at the 2nd R.I. from inspiration, expiration and carotid data files for Natalie K., are given in Table 10. Note that in addition, the table contains the maximum spectral magnitude times $10^4$ (M.M*10K), the frequency of the maximum magnitude (FM.MAG), the total area of the spectrum times 100 (AREA100), the number of records averaged (N.REC), the analysis performed (ANAL), and the start and end times for the spectral window (S.TIM & E.TIM). The severity analysis computer programs and their descriptions are given in Appendix II.

The murmur power spectrum analysis results, along with the personal and catheterization data for the thirteen catheterized aortic stenosis patients, are summarized in Table 11. These results were obtained at the 2nd R.I. from inspiration, expiration, and carotid data files as described above. The table contains the first spectral moment ($\tilde{f}$), the estimated standard deviation of the first moment ($\sigma_{\tilde{f}}$), the spectral bandwidth at 50% total area (50%F), the total number of records averaged (N.REC), and the murmur signal-to-diastolic-noise ratio (S/N). The signal-to-noise ratio was estimated from the averaged envelopogram plots, where the peak murmur intensity is defined as the signal (S) and the mean intensity in the diastole is defined as the noise (N). The analysis for $\sigma_{\tilde{f}}$ is given in Appendix II.
TABLE 10

BANDWIDTH AND FIRST MOMENT OF MEAN POWER SPECTRUM
COMPUTED FROM INSPIRATION, EXPIRATION AND
CAROTID DATA FILES AT 2ND. R.I.

NATALIE K  70 89 05  PHON01:  2ND. R.I.
N. REC= 60  S. TIM=160.00MS.  E. TIM=260.00MS.  SAMP.= 1.22Hz.  ANAL.=PWS.
AVE. FRQ= 124.55Hz.  F.M.MAG= 91.55Hz.  AREA100= 13.45  M.M*10K= 14.06
10%F  20%F  30%F  40%F  50%F  60%F  70%F  80%F
22.0Hz.  41.5Hz.  58.6Hz.  75.7Hz.  95.2Hz.  114.7Hz.  136.7Hz.  161.1Hz.
## TABLE 11

**SUMMARY OF MURMUR SPECTRUM ANALYSIS AT 2ND. R.I. FOR THE CATH. V.A.S. PATIENTS**

<table>
<thead>
<tr>
<th>Name</th>
<th>Hosp. #</th>
<th>Chest Wall</th>
<th>P.S.E.G. mm Hg</th>
<th>$f$ In Hz</th>
<th>$G_f$ In Hz</th>
<th>50% F In Hz</th>
<th>N.Rec.</th>
<th>S/N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Roger F.</td>
<td>47-99-27</td>
<td>Thick</td>
<td>75</td>
<td>134.98</td>
<td>1.52</td>
<td>100.1</td>
<td>54</td>
<td>20</td>
</tr>
<tr>
<td>Tommy K.</td>
<td>63-77-80</td>
<td>Thin</td>
<td>9-18</td>
<td>85.79</td>
<td>2.26</td>
<td>78.1</td>
<td>32</td>
<td>10</td>
</tr>
<tr>
<td>Donald G.</td>
<td>62-12-80</td>
<td>Thin</td>
<td>16</td>
<td>89.70</td>
<td>2.00</td>
<td>61.0</td>
<td>40</td>
<td>11</td>
</tr>
<tr>
<td>Natalie K.</td>
<td>70-89-05</td>
<td>Med.</td>
<td>23</td>
<td>124.53</td>
<td>1.48</td>
<td>95.2</td>
<td>60</td>
<td>10</td>
</tr>
<tr>
<td>Bryan K.</td>
<td>60-91-88</td>
<td>Thin</td>
<td>39</td>
<td>127.97</td>
<td>1.62</td>
<td>73.2</td>
<td>49</td>
<td>15</td>
</tr>
<tr>
<td>Robert M.</td>
<td>53-91-59</td>
<td>Med.</td>
<td>42-45</td>
<td>124.53</td>
<td>1.58</td>
<td>90.3</td>
<td>53</td>
<td>12</td>
</tr>
<tr>
<td>Elizabeth R.</td>
<td>55-01-61</td>
<td>Thin-Med.</td>
<td>45</td>
<td>147.77</td>
<td>1.54</td>
<td>80.6</td>
<td>48</td>
<td>26</td>
</tr>
<tr>
<td>Rudolph B.</td>
<td>68-97-78</td>
<td>Thin-Med.</td>
<td>45</td>
<td>142.86</td>
<td>1.43</td>
<td>97.7</td>
<td>58</td>
<td>12</td>
</tr>
<tr>
<td>Richard F.</td>
<td>57-53-27</td>
<td>Med.</td>
<td>61-68</td>
<td>168.56</td>
<td>1.38</td>
<td>78.1</td>
<td>54</td>
<td>24</td>
</tr>
<tr>
<td>Jean S.</td>
<td>58-79-24</td>
<td>Med.</td>
<td>70-90</td>
<td>201.84</td>
<td>1.70</td>
<td>68.4</td>
<td>30</td>
<td>13</td>
</tr>
<tr>
<td>Mark M.</td>
<td>68-95-48</td>
<td>Thin</td>
<td>6-8</td>
<td>95.89</td>
<td>1.71</td>
<td>78.1</td>
<td>53</td>
<td>10</td>
</tr>
<tr>
<td>Jonathan F.</td>
<td>64-87-14</td>
<td>Thin</td>
<td>5-9</td>
<td>93.12</td>
<td>2.06</td>
<td>36.6</td>
<td>37</td>
<td>11</td>
</tr>
<tr>
<td>Barry F.</td>
<td>60-50-48</td>
<td>Med.</td>
<td>16-24</td>
<td>104.15</td>
<td>2.08</td>
<td>56.2</td>
<td>34</td>
<td>10</td>
</tr>
</tbody>
</table>
The correlation coefficient between the peak systolic ejection gradient (P.S.E.G.) and the 50% bandwidth is equal to .32. Calculations are given in Table 1,AII. The correlation coefficient between the P.S.E.G. and the first moment of the mean power spectrum based on all thirteen subjects is .89 and the corresponding scatter diagram is given in Fig. 26. Careful examination reveals that a single point belonging to Roger F., Hosp. #47-99-27, exhibits a lower frequency moment than expected. Since this patient was the only one with a thick chest wall, the observed spectral moment difference may possibly be due to the increased chest wall thickness. The correlation coefficient between the P.S.E.G. and the first moment of the mean power spectrum $\tilde{f}$ based on the twelve thin-medium chest walled patients is .96. The corresponding least square regression line calculated for the twelve points is shown in Fig. 26. Calculations are given in Table 2,AII.

To investigate the affect of respiration on the correlation between P.S.E.G. and $\tilde{f}$, separate power spectral moments for inspiration and expiration were also computed. The corresponding calculations are given in Tables 3,AII and 4,AII. The resulting correlation coefficients for the twelve thin-medium chest walled patients were .95 for inspiration and .96 for expiration. Results of the correlation studies are summarized in Table 12.
Fig. 26. Scatter diagram for the catheterized V.A.S. patients at the 2nd. R.I. for inspiration + expiration + carotid data.
TABLE 12

SUMMARY OF CORRELATION STUDY BETWEEN P.S.E.G. AND MEAN MURMUR POWER SPECTRUM PARAMETERS, CALCULATED AT THE 2ND. R.I. FOR THE CATH. V.A.S. PATIENTS

<table>
<thead>
<tr>
<th>Chest Wall Thickness and Data Files Av. at 2nd. R.I.</th>
<th>Corr. Coeff. between P.S.E.G./\bar{e}</th>
<th>Corr. Coeff. between P.S.E.G./50% F</th>
</tr>
</thead>
<tbody>
<tr>
<td>For 1 Thick + 12 (Med.-Thin) Inspir. + Expir. + Car.</td>
<td>.89</td>
<td>---</td>
</tr>
<tr>
<td>For 12 (Thin-Med.) Inspir. + Expir. + Car.</td>
<td>.96</td>
<td>.32</td>
</tr>
<tr>
<td>For 12 (Thin-Med.) Inspir.</td>
<td>.95</td>
<td>---</td>
</tr>
<tr>
<td>For 12 (Thin-Med.) Expir.</td>
<td>.96</td>
<td>---</td>
</tr>
</tbody>
</table>
CHAPTER VI

DISCUSSION OF RESULTS

ADVANTAGES OF ENSEMBLE AVERAGING

As pointed out earlier in Chapter IV, the envelogram and power spectral estimates derived from a single phonocardiogram cycle are statistically unreliable and consequently, are unsuitable for the positive identification of phonocardiogram signal features. In Chapter V ensemble averaging of estimates was introduced to reduce the variance and the averaged plots were interpreted. Ensemble averaging of estimates offers the following advantages:

1. Reduces the variance of power spectral and envelogram estimates by approximately a factor of $\sqrt{N}$. Ensemble averaging is particularly required to smooth power spectra and to obtain consistent severity estimates.

2. Improves the detection sensitivity and timing of heart murmurs in the envelograms by approximately a factor of $\sqrt{N}$.

3. Unsynchronized respiratory and other external noise events are approximately evenly distributed and appear as a constant bias in the averaged envelograms.
The murmur detection sensitivity of ensemble-averaged envelograms was clearly demonstrated in the case of a normal patient, Lynne S., where prior to recording, no systolic murmur was detected by a cardiologist using auscultation techniques. However, the averaged envelogram computed from fourteen equivalent cardiocycles indicated a late systolic murmur of a grade 1-2 level, and was later confirmed by a second careful ausculatory examination. The high murmur detection sensitivity makes this technique particularly attractive in the assessment and study of mild regurgitant-type murmurs which occur in mild prolapsed mitral valves and in mild aortic and pulmonic insufficiency.

Unsynchronized noise smoothing (advantage 3) is especially useful in the detection of mild heart murmurs in infants and young children. The detection of these murmurs is, at best, difficult with the usual ausculatory and phonocardiogram techniques due to large respiratory and body background noise.

DISCUSSION OF V.A.S. SEVERITY ESTIMATES

The accurate noninvasive assessment of the severity of valvar aortic stenosis is an important clinical problem, and presently, is possible only by cardiac catheterization (an invasive surgical procedure which requires three days of hospital care). In contrast, the severity estimation procedure outlined in Chapter V is a completely noninvasive
technique where the measurements and data analysis are performed within minutes. For this technique to gain wide acceptance in clinical cardiology, it is suggested that it first be employed to follow the case history of catheterized valvar aortic stenosis patients, thus eliminating additional catheterizations while increasing the cardiologist's confidence in the technique.

Estimating the severity of valvar aortic stenosis from the murmur power spectrum has been tried unsuccessfully by several investigators, notably by Jacobs et al. and McKusick [17,56]. The basic difference between the technique employed by these investigators and that presented in Chapter V is as follows. The estimation parameters for the former were obtained from a power spectral estimate computed from a single murmur signal, while for the latter, these parameters were computed from an averaged power spectrum computed from 30-50 murmur signals. The accurate severity estimation of valvar aortic stenosis is made possible by the ensemble averaging of spectral estimates.

For the twelve thin-medium chest walled patients discussed earlier in Chapter V, excellent correlation exists between the peak systolic ejection gradient and the first spectral moment of the mean murmur spectrum. The correlation can be clearly observed from the scattering diagram, Fig. 26, and from Table 12. These results, however, do not appear to apply to thick chest walled patients where additional fatty deposits can produce high-frequency attenuation.
This observation is implied by the lower f of the single thick chest walled patient, Roger F.

The linear least square regression line fitted to the twelve thin-medium chest walled patients shown in Fig. 26 was used to estimate the severity of the four clinically-diagnosed V.A.S. patients. The corresponding predicted peak systolic ejection gradients are tabulated with other pertinent data in Table 13. From the calculations these patients are classified as having mild to moderate valvar aortic stenosis.

During the severity estimation it may be well remembered that while Fig. 26 can be used to estimate the severity of thin-medium chest walled patients, prior to the use of this plot the diagnosis of valvar aortic stenosis must be established as outlined in Chapter V.

SUGGESTIONS FOR FURTHER STUDY

A significant improvement in the positive diagnosis of heart disease by phonocardiogram signals can be made if echo phonocardiograms were to be included as an extra time series data channel in this analysis. This signal could be used to improve the timing of the aortic ejection click and could possibly be used to measure chest wall thickness between the listening site and the aortic valve cusps. The measured chest wall thickness could in turn be incorporated
to improve severity estimates and to extend the correlation results to include thick chest walled patients.

The envelogram and power spectral analysis techniques employed in this study could be adopted in a computerized phonocardiogram diagnostic system.
TABLE 13

PREDICTED MEAN P.S.E.G. AND STANDARD DEVIATION FOR THE CLINICALLY DIAGNOSED VALVAR AORTIC STENOSIS PATIENTS

<table>
<thead>
<tr>
<th>Name</th>
<th>Hosp. #</th>
<th>Chest Wall</th>
<th>N. Rec.</th>
<th>S/N</th>
<th>$\sigma$ ( \bar{f} ) In Hz</th>
<th>$\bar{f}$ In Hz</th>
<th>Predicted Mean P.S.E.G. In Hg mm</th>
<th>Predicted $\sigma_{P.S.E.G.}$ In Hg mm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Edward D.</td>
<td>57-03-63</td>
<td>Thin</td>
<td>31</td>
<td>15</td>
<td>1.98</td>
<td>138.8</td>
<td>42</td>
<td>1.25</td>
</tr>
<tr>
<td>John B.</td>
<td>58-29-30</td>
<td>Thick</td>
<td>54</td>
<td>14</td>
<td>1.47</td>
<td>147.1</td>
<td>&gt; 47</td>
<td>---</td>
</tr>
<tr>
<td>John R.</td>
<td>66-12-34</td>
<td>Med.</td>
<td>38</td>
<td>8</td>
<td>2.0</td>
<td>100.8</td>
<td>18</td>
<td>1.26</td>
</tr>
<tr>
<td>Donald D.</td>
<td>79-41-95</td>
<td>Thin</td>
<td>40</td>
<td>8</td>
<td>1.85</td>
<td>117.5</td>
<td>28</td>
<td>1.17</td>
</tr>
</tbody>
</table>
BIBLIOGRAPHY


11. Private Communication with Roberta Williams, M.D.


APPENDIX I
CHEST MICROPHONE ADULT SIZE
TYPE: 53616

TO AMPLIFIER

SUCTION BALL

Fig. 1, AI. Microphone placement on the chest
Fig. 2, AI. Amplitude response curve of a type 53616 microphone
TABLE 1, AI

ACOUSTICAL COMPARISON OF MICROPHONE 2 VERSUS MICROPHONE 1

<table>
<thead>
<tr>
<th>Frequency of Tone Generator</th>
<th>Relative Error* of Mic. 2 vs. Mic. 1</th>
</tr>
</thead>
<tbody>
<tr>
<td>125 Hz</td>
<td>- 19.1%</td>
</tr>
<tr>
<td>250 Hz</td>
<td>- 6.15%</td>
</tr>
<tr>
<td>500 Hz</td>
<td>0.0%</td>
</tr>
<tr>
<td>1000 Hz</td>
<td>- 10.8%</td>
</tr>
<tr>
<td>2000 Hz</td>
<td>+ 19.2%</td>
</tr>
</tbody>
</table>

*The relative error of microphone 2 \( E_2(f) \) is defined as

\[
E_2(f) = \left( \frac{\tilde{R}_2(f) - \tilde{R}_1(f)}{\tilde{R}_1(f)} \right) \times 100\%
\]

where \( \tilde{R}_1(f) = \frac{R_1(f)}{R_1(500)} \) = Normalized voltage response of microphone 1 to a constant intensity acoustical source. Reference response is chosen at 500 Hz.
TABLE 2, AI

PATIENT HEADER FORMAT

Name/Hosp. # :
Exam Date :
Diagnosis :

Sex :
Age :
Chest Wall :
Chest Deformity:
Fever :

Catheter Data Available ?:

Analog Tape #, Record # :
Comments on Analog Tape :
Pass 1 Conversion Date :

Next File:
Phono 1: Phono 2:
ECG Lead: Pulse/Resp:
TABLE 3, AI

DATA HEADER FORMAT

Name/Hosp. #: 
Next File: Phono 1: Phono 2: 
ECG Lead: Pulse/Resp:
1 *  
2 * MOD16  
3 * ANALOG READ BINARI DUMP  
4 * A. SARKADY 4/8/73  
5 * TEM1 = MODUL 4 COUNT INI VAL = 077777  
6 * TEM2 = MODUL 3200 COUNTER INI VAL = 071600  
7 * P1B = STAR ADD OF IN BUF  
8 * P2B = STAR ADD OF OUT BUF  
9 * B REG = ADD OF SOUND DATA  
10 * EIGHT M. S. B. = PHONO 1  
11 * EIGHT L. S. B. = PHONO 2  
12 * X REG = ADD OF PULSE AND E.C.G. DATA INI = 010640  
13 * EIGHT M. S. B. = PULSE DATA  
14 * EIGHT L. S. B. = E.C.G. DATA  
15 * SEN SW1 UP = STOP AND CONT WITH READ WHEN DOWN  
16 * SEN SW2 UP = STOP AND PULL FILE GAP  
17 *  
18 * INIT TEM1, TEM2, OTEM, ITEM, XREG, BREG, BIC  
19 *  
20 *  
21 *  
22 * NAME ANAL  
23 *  
24 *  
25 P1B EXT  
26 P2B EXT  
27 *  
28 ANAL ENTR  
29 STX SAVX  
30 STB SAVB  
31 ENT RDF  
32 TZA  
33 IAR  
34 STA TEM3 PASS1 REC COUNT  
35 LDAI 077777  
36 STA TEM1  
37 LDAI 071600  
38 STA TEM2  
39 LDAI P2B  
40 STA OTEM  
41 LDBI P1B  
42 STB ITEM  
43 LDAI P1B  
44 ADDI 3200  
45 TAX INI X REG
46 *
47 JOY SEN 0050, SEN1
48 NOP
49 JMP JOY
50 *
51 *
52 * MAIN PROG
53 *
54 SEN1 CIA 050
55 SEN 0150, G01 PULSE AND RESP DATA
56 NOP
57 JMP +-3
58 G01 INR TEM1
59 JOF PE
60 *
61 SEN 0250, *+5
62 NOP
63 JMP +-3
64 CIA 050
65 *
66 SEN3 SEN 0350, G02 SOUND 1 D
67 NOP
68 JMP +-3
69 G02 CIA 050
70 LRLA 8
71 SEN 0050, G03 SOUND 2 D
72 NOP
73 JMP +-3
74 G03 INA 050
75 STA 0, 2 R=2
76 IBR
77 INR TEM2
78 JOF B1C
79 JMP SEN1+1
80 *
81 *
82 * READ PULSE AND E.C.G. DATA
83 *
84 PE ROF
85 CIA 050
86  LRLA  8
87  SEN  0250, G04
88  NOP
89  JMP  $-3
90  G04  INA  050
91  STA  0, 1  X=1
92  IXR
93  LDAI  077774  INI TAMP 1 MOD 4
94  STA  TEM1
95  JMP  SEN3
96  *
97  * BIC BUSSY ?, ERR MES, SWAP POINT, BIC WRITE
98  *
99  BIC  ROF
100  SEN  0210, CON  MTU READY
101  JMP  BB
102  CON  SEN  021, BS  BIC ABNORM STOP
103  SEN  020, SWAP  BIC, NOT BUSSY
104  JMP  BB
105  SWAP  LDA  ITEM
106  LDB  OTEM
107  STA  OTEM
108  STB  ITEM
109  EXC  021  INIT BIC
110  DAR
111  OAR  020  BIC INI ADD
112  *
113  TAX
114  LDA  TEM3
115  STA  0, 1
116  TXA
117  *
118  ADDI  4000
119  OAR  021  BIC FINAL ADD
120  *
121  LDA  ITEM
122  ADDI  3200
123  TAX
124  LDAI  071600  INI TEM2
125  DATA  0104110
126  STA  TEM2
127  *
128 *
129  EXC  020  ACT BIC
130  EXC  0210  WRITE MTOO
131  JSS1  HALT
132  JSS2  FILE
133  INR  TEM3
134  JMP  G01

135 *
136 *
137 * ERROR ROUTINE
138 *
139  BS  LDAI  'SB'
140  JMP  OCH
141  BB  LDAI  'BB'
142  JMP  OCH
143  OCH  ROF
144  SEN  0101, ++5
145  NOP
146  JMP  *-3
147  OAR  01
148  JOF  HALT
149  SOF
150  LRLA  8
151  JMP  OCH+1
152  HALT  HLT
153  JMP  ENT

154 *
155  FILE  SEN  0210, FG  MTU READY
156  NOP
157  JMP  *-3
158  FG  EXC  0410
159  LDX  SAVX
160  LDB  SAVB
161  JMP*  ANAL

162 *
163  TEM3  DATA  0
164 *
ENTRY NAMES
000000 R ANAL

EXTERNAL NAMES
000024 E P1B 000016 E P2B

SYMBOLS
000000 R ANAL
000125 R CON
000043 R GO1
000217 R HALT
000241 R OTEM
000236 R SAVB
000133 R SWAP

000176 R BB 000120 R BIC 000172 R BS
000003 R ENT 000227 R FG 000222 R FILE
000061 R GO2 000070 R GO3 000110 R GO4
000242 R ITEM 000030 R JOY 000202 R OCH
000024 E P1B 000016 E P2B 000100 R PE
000235 R SAVX 000035 R SEN1 000054 R SEN3
000237 R TEM1 000240 R TEM2 000234 R TEM3
1  ** DFSFT**  N MAX 1054  F. MICHOLZI, GERMANI 8-5-73
2  * * *
3  4  ** COMPLEX **
4  5  ** FAST FOURIER TRANSFORM **
5  6  ** FOR INTEGER VALUES **
6  7  **
7  8  ** CALL DFSFT(LOST,N) **
8  9  ** TO INITIAlIZE COS TABLE **
9  10  ** DIMENSION (DISTR(3,3)) **
10  11  ** CALL FFTREAL(IMG,NSHFTX) **
11  12  ** TO TAKE TRANSFORM **
12  13  ** NSHFTX **
13  14  ** ON INPUT 0 < X 1000  = X DFT **
14  15  ** ON OUTPUT  SCALE FACTOR OF SHIFTS **
15  16  **
16  17  **
17  18  ** MORE  DFSFT,FFT,DFSFT **
18  19  **
19  20  **
20  21  **
21  22  **
22  23  **
23  24  **
24  25  **
25  26  **
26  27  ** FTTFS Nsh **
27  28  **
28  29  **
29  30  **
30  31  **
31  32  **
32  33  **
33  34  **
34  35  **
35  36  **
36  37  **
37  38  **
38  39  **
39  40  **
40  41  **
41  42  **
42  43  **
43  44  **
44  45  **
45  46  **
46  47  **
47  48  **
48  49  **
49  50  **
50  51  **
51
52 *  
53 M1  LOAD RWORK INITIAL POINTS  
54 STA REAL  
55 ADD SFRR  
56 STA RMULT  
57 LOAD TWORK  
58 STA IP66  
59 ADD SFRR  
60 STA MULT  
61 *  
62 *  
63 T2A INITIALIZE  
64 SUB M64 INDEX  
65 STA INDX POINTER  
66 *  
67 M27 T2A EXIT WHEN TERMS  
68 SUB SFRR WITH COMMON MULTIPLIER  
69 STA EXIT HAVE BEEN PROCESSED  
70 *  
71 JMPN INDEX  

72 *  
73 * DETERMINE SIN/COS TERMS  
74 * RETURN WITH ARGUMENT IN A  
75 *  
76 RDF RESET INDICATOR  
77 TAB  
78 MINUS OR OR  

79 ADD NOX CORP H/4+1  
80 INDX  
81 XOP SOF SOF IF OR/4=H/4  
82 JGP M22 SKIP IF OR=H/4  
83 MINUS OR OR DO IF OR/4=H/4  
84 MINUS OR OR  

85 ADD NOX  
86 JMP M22+1  
87 M22 TRG  
88 ADD TABLE  
89 TAB  
90 TGA  
91 ADD TABLE  
92 TAB  
93 LDX 0, X  
94 LOAD ETRN
25  JAP   M221   JMP IF DEF
26  MINUS  XR, XR  NO IF DEF
27  M221  STX   SIN
28  LDG   0.3
29  JOF   R23  JMP IF N(0)=X(0)=N(2)
30  M23  STI   XR  NO IF N(0)<X(0)<N(2)
31  TZA   TZA  FORM TERMS
32  LIBFS* TMULT
33  MUL   SIN
34  STA   RTERM
35  TZA   RTERM
36  LIBFS* RMULT
37  MUL   COS
38  SUB   RTERM
39  STA   RTERM
40  RTERM
41  TZA   RTERM
42  LIBFS* RMULT
43  MUL   COS
44  STA   TTERM
45  TZA   TTERM
46  LIBFS* TMULT
47  MUL   OVFPO
48  ADD   TTERM
49  STA   TTERM
50  RTERM
51  JQFM   OVFPO  IF OVF, CORRECT/RESET
52  RTERM
53  STBFS* RMULT
54  STARS  REAL
55 
136  M5  LDX  IMAG
137    LDA  O,  X
138    SUB  ITERM
139    TFR
140    LDA  O,  X
141    ADD  ITERM
142    JGFEM  OVFLO
143  REMS  STRF#  THMULT
144  STGF#  IMAG

145 *
146    INR  REAL  INCREMENT
147    INR  RMULT  POINTERS
148    INR  IMAG
149    INR  THMULT
150 *
151    INR  EXIT
152    LDA  EXIT  SKIP IF ALL TERMS
153    JAN  MS  WITH COMMON MULT DONE

154 *
155    LDA  RMULT
156    ERA  PASSC
157    JAZ  FIN  ALL DONE THIS STAGE?
158    JMP  NOFNM  NO!

159 *
160 *
161  OVFLO  ENTR#  OVERFLOW FIXUP
162    INRES#  OVMN
163    LDA  RWORK
164    JMPM  SHIFT
165    LDA  TWORK
166    JMPM  SHIFT
167    LDA  RTERM
168    ASRA  01
169    STA  RTERM
170    LDA  ITERM
171    ASRA  01
172    STA  ITERM
173    LDAF  OVFLO
174    ERAI  REM4!
175    JA7  M4
176    LDAF  OVFLO
177  ERA1  REM5
178  JNZ  N5
179  JMP  M3.

180 *
181  SOF  SET  INDICATOR
182 *
183  SHIFT  ENTR  SHIF ALL  DATA
184  TAK  RIGHT  ONE  BIT
185  ADD  N
186  STA  DUN
187  SHT  LDR  O,  x
188  ASPP  01
189  STR  O,  x
190  IXR
191  TYA
192  ERA  DUN
193  JNZ*  SHIFT
194  JMP  SHT

195 *
196  DUN  BSS  1
197 *
198  NOFIN  LDA  REAL  UPDATE  START
199  ADD  SEPR  OF  BUTTERFLY
200  STA  REAL
201  ADD  SEPR
202  STA  RMULT
203  LDA  IMAG
204  ADD  SEPR
205  STA  IMAG
206  ADD  SEPR
207  STA  INMULT
208  JMP  M2

209 *
210  FIN  LDA  SEPR  DIV  SEPR  BY  2
211  ASRA  01
212  JAZ  S1  STAGES  ALL  DONE?
213  STA  SEPR  NO!!!
214  JMP  MJ  START  NEXT  STAGE

215 *
216 *
217  ROUTINE  TO  GIVE
218  ACCESS  TO  COS  TABLE  OR
219  UNSCRAMBLE  FINAL  VALUES
220 *
221 *
222  INDEX  ENTR  DETERMINE  INDEX
223 | LDA | INDX | FOR REVERSE BIT COUNT
224 | LDB | ND2 |
225 | MINUS | BR, BR |
226 | SKIP | NOP |
227 | ASRRB | 01 |
228 | SKPT | STB | ++?
229 | ADDI | 0 |
230 | JAP | SKIP+1 |
231 | MINUS | BR, BR |
232 | ASLB | 01 |
233 | STB | ++?
234 | ADDI | 0 |
235 | STA | INDX |
236 | JMP* | INDEX |
237 | * |
238 | S1 | TZA | UNSCRAMBLE BIT
239 | SUB | N |
240 | STA | EXIT |
241 | LDA | RWORK |
242 | STA | RR1 |
243 | LDA | IWORK |
244 | STA | I1 |
245 | * |
246 | TZA |
247 | SUB | ND2 |
248 | STA | INDEX |
249 | LDA | SKIPVR |
250 | STAE | SKIP |
251 | * |
252 | S2 | JMPN | INDEX |
253 | TAX |
254 | ADD | RWORK |
255 | STA | RR2 |
256 | TAX |
257 | ADD | IWORK |
258 | STA | I1 |
259 | * |
260 | LDA | RR1 |
261 | SUB | RR2 |
262 | JAP | S3 | IF # DON'T SWITCH
263 | * |
264 * LOADS RR1  SWITCH REALS
265 LOADS RR2
266 STAES RR2
267 STBE RR1
268 STBE RR2
269 * LOADS II1  SWITCH INGGS
270 LOADS II2
271 STAES II2
272 STAES II1
273 STBE II1
274 * INR II1  INCR POINTERS
275 INR RR1
276 INR EXIT
277 LDG EXIT
278 JAN 92  GO SWITCH MORE
279 JMP 0  ALL BEEN SWITCHED
280 FFMT BES 0
281 CALL $SE, 3
282 REAL ARRAY ADDRESS
283 INWORK BSS 1
284 INWORK BSS 1
285 OVMUM BSS 1
286 JMP SEET  START TRANSFORM
287 FJEC
288 SEFT LOADS N
289 STG N
290 ASRA 01
291 STA ND7
292 ASRA 01
293 STA ND4
294 LDG TABLE
295 STB TPRK
296 * DETERMINE TABLE VALUE SPACING
297 *
298 TZR
299 TBR
300 MCHI TAX
301 ERGI 256
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<td>* SET UP STORAGE AND DATA</td>
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<td>III EQU INCR</td>
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ENTRY NAMES
000405 R FFTM  000511 R ICSTB  000460 R INFFT
EXTERNAL NAMES
000462 R $SE

SYMBOLS

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APPENDIX II
DESCRIPTION OF THE INTERACTIVE ANALYSIS

An interactive analysis program was used to compute envelograms, power spectra, and time-averaged phonocardiograms for the diagnostic analysis procedure.

Prior to data input, the type of record (PHON1, PHON2, etc.), the data window, the sampling rate and the number of records averaged were selected. A record was deleted from the analysis if the record length (Q-Q interval) was not within an acceptable specified range or if the record number was entered in a delete table. During data input, selected records were aligned (time-shifted to the left or right) by a two-pass process.

In the first pass a specified signal feature (local maximum or minimum) was searched for within a specified time range and the location of the signal feature for each record was tabulated in a table. Using this table, a mean alignment time was computed. The sliding increments necessary to cause alignment about this mean time was computed for each record and stored in the same table. During the second pass, the input data tape was repositioned, the same data was acquired and according to the tabulated correction factors, alignment corrections were made prior to analysis.

An analysis routine was composed of modular computational algorithms (such as FFT, magnitude, IFFT, etc.) and was of a standard type (time, envelope, or power
spectrum) or was specifically created to suit a special need. In either case, the analysis was implemented from a command table containing a sequence of algorithm characters which defined the required analysis routine.

All the analyzed records were added to a 512 floating-point word accumulator buffer. When the specified number of records were analyzed, the average buffer values were computed and plotted on the graphics terminal or output to magnetic tapes. A listing of the interactive analysis program is shown on the following pages.
**PAGE 1**

```plaintext
1 *** AUTOFRED GEN FORT ANALYS VERS#2 3-5-74 A. SARKADY
2 * SAMF AS FREDAN BUT LOADS ANY TIME AND CAL BRANCHES
3 * AND CAN ZERO A TIME INTERVAL
4 *
5 COMMON ID(3), IR1(1024), IM1(1024), IDA(4)
6 COMMON A(512)
7 *
8 DIMENSION INT(6), MAT(12), RMS(2)
9 DIMENSION SAM(4), IH(2), HAD(6), IL(2)
10 DIMENSION IPW(2048), PW(1024)
11 DIMENSION AT(6)
12 DIMENSION MRECS(40)
13 DIMENSION IZ(2)
14 *
15 EQUIVALENCE(ID(1), IPW(1), PW(1))
16 *
17 EXTERNAL ICSTB
18 EXTERNAL NAMBF
19 *
20 DATA RMS/1., 1/
21 DATA SAM/S. E-4, 3. 2E-3, 4. E-4, 1. 6E-3/
22 DATA IH/0, 0/
23 DATA HAD(1)/0, 0/
24 DATA IL/2H1, 2H/
25 DATA AT/4HTIM, 4HANS, 4HPWS, 4HSUM, 4HDEF, 4HABS/
26 DATA LUNO, LUN0/1, 0/
27 DATA MINCOR/1/
28 DATA MAXCOR/2048/
29 DATA INT/0, 818, 0, 1, 0/
30 DATA IZ/0, 0/
31 DATA IBLK/2H/
32 *
33 *
34 *
35 START INTERACTIVE ANALYSIS
36 *
37 ENTER FUNCTION COMMANDS FROM TY
38 *
39 * D=79=OUTPUT ARRAY (A) TO MTO1
40 * PLOT ARRAY (A) =P=80
41 * ENTER INPUT DATA COMMANDS =D=68
42 * ENTER ANALYSIS COMMANDS =C=67
43 * START ANALYSIS =A=65
44 * R=82=ENTER REC# TO BE SKIPPED
45 * M= CALI CURSOR =77
46 * N=78=NEW (INIT OUTPUT REC#, AND MRECS ARRAY)
47 *
48 S=83=STAND ANAL BRANCH
49 G=71=GO BACK TO START ON MTO0
50 J=74=CONTINUE WITH ANALYSIS
```
51 * Z=90=ENTER TIME SEGMENT TO BE ZEROED
52 *
53 *
54 # IDA(4)=0
55 =0
56 CALL HOLLER(8HINIT OR#,8)
57 3 CALL INIBUF(40,MRFC8)
58 =0
59 CALL HOLLER(10HINIT MREC ,10)
60 1 CALL INFFT( I NSTB,1024)
61 CALL CONTAP(LUNO,LUNO,LUNO,LUNO,LUN1)
62 6 CALL BEL
63 2 CALL BEL
64 CALL CHIN(ICOM)
65 *
66 IF(ICOM.EQ.90)GO TO 800
67 IF(ICOM=84)401,150,1
68 401 IF(ICOM.EQ.80)GO TO 15
69 IF(ICOM.EQ.79 )GO TO 20
70 IF(ICOM.EQ.87)GO TO 700
71 IF(ICOM.EQ.78)GO TO 5
72 IF(ICOM.EQ.83)GO TO 600
73 IF(ICOM=76,400,420,402
74 400 IF(ICOM.EQ.68)GO TO 30
75 IF(ICOM.EQ.67)GO TO 34
76 IF(ICOM.EQ.65)GO TO 40
77 IF(ICOM.EQ.71)GO TO 451
78 IF(ICOM.EQ.74)GO TO 42
79 GO TO 1
80 *
81 * Z=90=ENTER TIME SEGMENT TO BE ZEROED
82 *
83 800 IZ(1)=0
84 IZ(2)=0
85 CALL DECIN(IZ(1))
86 CALL DECIN(IZ(2))
87 GO TO 6
88 *
89 * G=71=GO BACK TO START ON MT00
90 *
91 451 J=NRFCONT-1
92 IF(IOFS.EQ.0)J=J+1
93 J=4*J+1
94 CALL CONTAP(1,2HRR,0,J)
95 GO TO 6
96 *
97 * S=83=START STAND ANAL. BRANCHS
98 *
99 * E=69=ENVELOP R,F,D,I,M,V,A
100 * T=84=TIME SERIES R,T,V,A
101 * C=67=CALIBRATION R, C, T, P, V, A
102 * S=83=POWER SPECTRUM R, F, S, V, A
103 *
104 600 =0
105 CALL HOLLER(SH D. TYP=,8)
106 CALL DECIN( INT(1) )
107 =0
108 CALL HOLLER(6HD. AN=,6)
109 CALL BEL
110 CALL CHIN( ICOM )
111 *
112 IF( ICOM. EQ. 67 ) GO TO 610
113 IF( ICOM. EQ. 83 ) GO TO 620
114 IF( ICOM. EQ. 67 ) GO TO 630
115 IF( ICOM. EQ. 83 ) GO TO 640
116 GO TO 6
117 *
118 610 MAT(2)=70
119 MAT(3)=68
120 MAT(4)=73
121 MAT(5)=77
122 MAT(6)=86
123 MAT(7)=65
124 612 INT(5)=50
125 615 MAT(1)=82
126 GO TO 40
127 *
128 620 MAT(2)=84
129 MAT(3)=86
130 MAT(4)=65
131 GO TO 612
132 *
133 630 MAT(2)=67
134 MAT(3)=84
135 MAT(4)=80
136 MAT(5)=86
137 MAT(6)=65
138 INT(5)=1
139 GO TO 615
140 *
141 640 MAT(2)=70
142 MAT(3)=83
143 MAT(4)=86
144 MAT(5)=65
145 GO TO 612
146 *
147 *
148 * R=82=ENTER Q-Q MIN, MAX COR LEN AND REC# TO BE SKIPED
149 *
150 700 MINCOR=1
MAXCOR=2048
CALL DECIN(MINCOR)
CALL DECIN(MAXCOR)
=0
CALL HOLLER(6H REC#=, 6)
CALL INIEUF(40,MRECS)
CALL CHOU(13)
CALL CHOU(10)
=0
CALL HOLLER(4H ,4)
CALL DECIN(J)
IF(J.LE.0)GO TO 6
IF(J.GT.40)GO TO 6
MRECS(J)=J
=J
CALL DECINA
GO TO 702

OUTPUT PROSSESED DATA TO MT01

IDA(1)=W LENGTH IA(2)=#IPUT RECORDS AVERAGED
IDA(3)=REC ID IDA(4)=REC#

IDA(1)=1028
IDA(2)=IREC
IDA(3)=IH(1)
IDA(4)=IDA(4)+1
CALL IDODATA(0,IDA(1),LUN1,IDA(1),IOFS)
GO TO 6

DIV AVER BUF

DO 12 J=1,512
A(J)=A(J)/FLOAT(IREC)
LIMP=LIMP/2
GO TO 6

P=PLOT A

CALL ERAS
NSTAR=1
NEND=512
CALL DECIN(NSTAR)
CALL DECIN(NEND)
CALL PHFAD(INT(1))
=32
CALL BLANKA
IF(IZS.NE.0)GO TO 17
201 CALL DECH4( IBLK, 2H Z, IZ(1) )
202 CALL DECH4( IBLK, IBLK, IZ(2) )
203  17 =0
204 CALL BLANKA
205 CALL DECH4( IBLK, 2H T, INT(2) )
206 CALL DECH4( IBLK, IBLK, INT(3) )
207 CALL CHOU(13)
208 CALL CHOU(10)
209 =30
210 CALL BLANKA
211 CALL DECH4( IBLK, 2H N, IREC )
212 CALL DECH4( IBLK, 2H R, ID(2) )
213 CALL ENPLOT(A, NSTAR, NEND, SAMP, IH(1) )
214 GO TO 6
215 *
216 * CURSOR=MEASURES TIME AND FREQ FROM AVER PLOTS
217 *  T=TIME IN M. SEC RUBOUT =RFT TO 6 OTHERS=FREQ IN I
218 *
219  402 CALL CURSOR( ICHAR, NXP, NYP )
220 NXO=FLOAT( NXP )*SAMP
221 IF( ICHAR. EQ. 84 )NXO=FLOAT( NXP )*SAMP*1000.
222 IF( ICHAR. EQ. 127 )GO TO 6
223 =NXO
224 CALL. DECINA
225 GO TO 402
226 *
227 *
228 * 10*LOG10( A )=4. 34*ALOG( A )
229 *
230  420 GO TO 6
231 *
232 *
233 *
234 * INPUT DATA PARAMETERS
235 *
236 * INT(1)=DATA TYPE 1...4
237 * INT(2)=START TIME IN M. SEC
238 * INT(3)=END TIME IN M. SEC
239 * INT(4)=0=SUM INT(4)=1=DEFOLD
240 * INT(5)=#REC TO BE AVER
241 * INT(6)=EXTRA
242 *
243 *
244  30 INT(4)=0
245 INT(5)=100
246 INT(6)=10
247 =0
248 CALL HOLLER( &HIN PAR, 6)
249 L=1
250  32 IF( L. GE. 6 )GO TO 6
251 CALL DEC INK INT(L))
252 IF(INT(L).LT.0)GO TO 30
253 L=L+1
254 GO TO 32
255 *
256 *
257 * LOAD MAT TABLE
258 *
259 *
260 *
261 34 =0
262 CALL HOLLER(6HAN COM, 6)
263 L=1
264 36 CALL BE
265 CALL CHIN(MAT(L))
266 IF(MAT(L)-65)34, & 38
267 38 L=L+1
268 GO TO 36
269 *
270 *
271 * INPUT DATA ACCORDING TO INT TABLE
272 *
273 *
274 40 CALL INIBUF(1024, A)
275 SAMT=SAM(1)
276 IREC=0
277 NRFCNT=0
278 MINLEN=512
279 IZS=1
280 *
281 IF( INT(1).GE. 3 )SAMT=SAM(2)
282 ISTART=FLOAT(INT(2))/ (SAMT*1000.)
283 IEND=FLOAT(INT(3))/ (SAMT*1000.)
284 RMSV=RMS(1)
285 IF( INT(1).EQ. 2 )RMSV=RMS(2)
286 IF( INT(4).GE. 1 )SAMT=SAMT/2.
287 SAMF=1./(1024.*SAMT)
288 CALL INIBUF(12, HAD)
289 *
290 42 IWC=IEND-ISTART+1
291 CALL INIBUF(2048, IPW)
292 CALL IODATA( INT(1), 2051, LUNO, ID(1), IOFS)
293 IF(IOFS.EQ.0)GO TO 10
294 NRFCNT=NRFCNT+1
295 J=ID(2)
296 IF( J.EQ. MRFCS(J))GO TO 42
297 IF( ID(1).GT. MAXCOR)GO TO 42
298 IF( ID(1).LT. MINCOR)GO TO 42
299 J=ID(1)-ISTART-3
300 IF(IWC.GT. J)IWC=J
IF(IWC, GT, 1024) IWC=1024
CALL LSRBUF(2048, ISTART, IPW)
LWCO=2048-IWC
CALL INIBUF(LWCO, IPW(IWC+1))
*
DEFOLD
*
IF(INT(4), EQ, 0) GO TO 43
CALL DEFOLD(IWC, IR1)
IWC=2*IWC
HAD(2)=AT(5)
GO TO 48
*
SUM
*
CALL SUMBUF(IWC, IR1)
HAD(2)=AT(4)
*
IF(INT(1), EQ, 1) ID(3)=IL(1)
IH(1)=ID(3)
IH(2)=IL(2)
*
TEST COMMAND CHAR
*
V=AVERAGE=86
T=TIME REMAVER=84
S=POWER SPECTRUM=83
R=REMOVE AVERAGE =82
P=PLOT PW ARRAY=80
O=OUTPUT PW ARRAY=79
N=PHASE(IR1, IM1)=78
M=MAGNETUD =77
L=INV FFT =73
H=HAN=72
F=FFT=70
E=ENVELOP=69
D=ANALITIC SIGNAL=68
C=CALIBRATIAN=67
A=ANALYZ=65
*
Z=90=ZERO A SEGMENT OF IR1 ARRAY
*
IC=0
IAV=2
FSCAL=1.
*
IC=IC+1
ICOM=MAT(IC)
351 IF( ICOM .EQ. 90 ) GO TO 850
352 53 IF( ICOM-86 )54, 86, 6
353 54 IF( ICOM-83 )300, 83, 84
354 300 IF( ICOM-82 )55, 82, 82
355 55 IF( ICOM-79 )301, 79, 80
356 301 IF( ICOM-77 )56, 83, 78
357 56 IF( ICOM-72 )57, 72, 73
358 57 IF( ICOM-69 )58, 69, 70
359 58 IF( ICOM-68 )59, 68, 69
360 60 IF( ICOM-65 )6, 59, 67
361 * 59 IREC=IREC+1
362 59 SAMP=SAMP*FLOAT( IAV )
363 CALL CST0P( 127, J )
364 IF( J .EQ. 0 ) GO TO 6
365 IF( IREC .GE. INT(5) ) GO TO 10
366 GO TO 42
367 *
368 * FFT
369 * 70 NSHFT=-1
370 CALL FFTM( IR1, IM1,NSHFT )
371 FSCAL=FLOAT(2**NSHFT)/1024.
372 SAMP=SAMP
373 LIMP=1024
374 GO TO 51
375 * POWER SPECTRUM OR MAGN
376 * 83 CALL ISHUF(1024, IPW )
377 Z=(FSCAL*FSCAL)/(RMSV*RMSV)
378 DO 100 J=1,1024
379 X=IPW(2#J-1)
380 Y=IPW(2#J )
381 POWER(J)=(X*X+Y*Y)*Z
382 IF( ICOM .EQ. 77 ) GO TO 105
383 IAV=1
384 HAD(1)=AT(3)
385 GO TO 51
386 *
387 105 DO 106 J=1,1024
388 PW(J)=SGRT(PW(J))
389 GO TO 51
390 *
391 * TIME FLOATS ARRAY IR1 AND NORMAL BY RMSV AND FSCAL
392 105 DO 180 J=1,1024
393 JNUM=1025-J
394 PW(JNUM)=IR1(JNUM)
PW ( JNUM ) = FW ( JNUM ) * FSCAL / RMSV
HAD ( 1 ) = AT ( 1 )
GO TO 51

* REMOVES AVERAGE VALUE
SUMA = 0.
DO 90 J = 1, IWC
XR = IR ( J )
SUMA = SUMA + XR
SUMA = SUMA / FLOAT ( IWC )
ISUM = SUMA
DO 91 J = 1, IWC
IR ( J ) = IR ( J ) - ISUM
SAMP = SAMT
LIMP = IWC
IF ( IWC . LE. 512 ) IAV = 1
GO TO 51

CALIBRATION = C
RMSC = 0.
DO 96 J = 1, IWC
XR = IR ( J )
RMSC = RMSC + XR * XR
RMSC = RMSC / FLOAT ( IWC )
RMSC = SQRT ( RMSC )
IF ( INT ( 1 ) . EQ. 1 ) RMS ( 1 ) = RMSC
IF ( INT ( 1 ) . EQ. 2 ) RMS ( 2 ) = RMSC
IDA ( 4 ) = 0
SAMP = SAMT
GO TO 51

AVERAGE = V FLOATING POINT ONLY
IAV = 1 = AVER EVERY POINT
IAV = 2 = AVER EVERY 2ND POINT
MINLEN = MIN RFC COR LEN IN AVERAGE

DO 110 J = 1, 512
JDAM = IAV * J
A ( J ) = A ( J ) + FW ( JDAM )
IDA ( 2 ) = IDA ( 2 ) + 1
J = LIMP / 2
IF ( J . LT. MINLEN ) MINLEN = J
GO TO 51
186

PAGE. 10

451 * OUTPUT TO MT01=0
452 *
453 79 GO TO 51
454 *
455 * PLOT =F PW ARRAY ONLY
456 *
457 80 CALL ENPLOT(PW,1,1024,SAMP,IH(1))
458 GO TO 51
459 *
460 *
461 * E=69=ABS VAL
462 *
463 69 DO 320 J=1,1024
464 320 PW(J)=ABS(PW(J))
465 HAD(1)=AT(6)
466 GO TO 51
467 *
468 *
469 * ANALYTIC SIGNAL
470 *
471 68 IR1(1)=IR1(1)/2
472 IM1(1)=IM1(1)/2
473 FSCAL=2.0*FSCAL
474 DO 140 J=513,1024
475 IR1(J)=0
476 140 IM1(J)=0
477 HAD(1)=AT(2)
478 GO TO 51
479 *
480 * HANNING WINDOW
481 *
482 *
483 72 GO TO 51
484 *
485 * INV FFT
486 *
487 73 ISHFT=-1
488 SAMP=SAMT
489 LIMP=IWC
490 DO 500 J=1,1024
491 500 IM1(J)=-IM1(J)
492 CALL FFTM(IR1,IM1,ISHFT)
493 DO 520 J=1,1024
494 520 IM1(J)=-IM1(J)
495 FSCAL=FLOAT(2**ISHFT)*FSCAL
496 GO TO 51
497 *
498 * SYSTEM TEST
499 *
500 150 GO TO 6
501 *  
502 *   PHASE( IR1, IM1 )  
503 *  
504 78   GO TO 51  
505 *  
506 *   Z=90=ZERO A SEGMENT OF IR1  
507 *  
508 850   X=SMAT*1000.  
509 J=IZ(1)-INT(2)  
510 IZS=FLOAT(J)/X+1.  
511 J=IZ(2)-IZ(1)+2  
512 J=FLOAT(J)/X  
513 IF (IZS.LE.1) GO TO 860  
514 IR1(IZS)=IR1(IZS)/2  
515 IZS=IZS+1  
516 860   CALL INIBUF(J, IR1(IZS))  
517 J=IZS+J  
518 IR1(J)=IR1(J)/2  
519 IZS=0  
520   GO TO 51  
521 *  
522 *   END  
523 *  
ENTRY/COMMON BLOCK NAMES  
004353 E   R  
006007 C   COMMON  
000216 E   ICSTB  
000000 E   NAMBF  
001563 E   HOLLER  
004076 E   INIBUF  
000215 E   INFFT  
000523 E   CONTAP  
001572 E   BEL  
001600 E   CHIN  
001526 E   DECIN  
001330 E   CHOU  
001451 E   DECINA  
002041 E   IODATA  
004036 E   $IC  
002174 E   LSRBUF  
00231 E   DEFOLD  
00252 E   SUMBUF  
003700 E   FFTM  
003733 E   $HE  
002616 E   ISHUF  
003254 E   $PC  
004007 E   $OK  
003321 E   SQRT  
003410 E   $HM  
003520 E   ABS
COMPUTER ANALYSIS PROGRAMS EMPLOYED  
FOR SEVERITY ESTIMATES  

While the diagnostic analysis was performed interactively, "number crunching" involved in the aortic stenosis severity estimates was performed in a batch mode. The severity analysis was accomplished by a two-pass process. During the first pass, an analysis was performed on each patient file and a single, averaged power spectrum and envelogram was computed and stored on magnetic tape. During the second pass, the files were either combined to form a single spectrum at a listening site or were analyzed as independent data files. Second-pass analysis consisted of computing and listing the first moment of spectral bandwidths or involved automated plotting of envelograms and spectra on the graphics terminal.

The programs employed in the first pass are PANAL and the subroutine, FANAL, while the second-pass computations were performed by the program PPAVER. The programs and their descriptions follow.
DESCRIPTION OF THE FIRST-PASS SEVERITY
ANALYSIS PROGRAM, PANAL

PANAL = Main program for patient data analysis in batch mode.

Prior to program execution, the following analysis
and data parameters must be entered through the teletype.

1. Analysis parameter: a single teletype character
   E = Envelope analysis
   T = Time analysis
   and S = Power spectrum analysis

2. Data parameters: unsigned integers less than
   5 digits
   INT(1) = Specifies the data type (integers
   1-4)
   If INT(1) = 1 = Phono 1 PCG data
   = 2 = Phono 2 PCG data
   = 3 = ECG data
   = 4 = Respiration or carotid data
   INT(2) = Calibration records start time
            in ms.
   INT(3) = Calibration records end time in ms.
   INT(4) = Sampling rate
   If INT(4) = 0 = 1.25 KHz (SUM)
   If INT(4) = 1 = 2.50 KHz (DEFOLD)
   INT(5) = Spare
   INT(6) = Data record window start time in ms.
   INT(7) = Data record window end time in ms.
INT(8) = Number of patients to be analyzed
INT(9) = Number of records to skip before Phono 2 calibration

Input Data:

Magnetic tape unit: MT00
Data Format: PASS 2 data format

Output Data:

Magnetic tape unit: MT01
Data format: 1024 data words (or 512 floating-point numbers) preceded by 8-word parameter field as given below.

Parameter Words:

#1 = Data type (fixed-point integer)
#2 = Number of records averaged per file (fixed-point integer)
#3 = Start time in ms. (fixed-point integer)
#4 = End time in ms. (fixed-point integer)
#5 and #6 = Sampling rate (floating-point number)
#7 and #8 = 4 alpha numeric numbers (describing analysis performed)
if #7 and #8 = TIM. = Time analysis
if #7 and #8 = ANS. = Envelope analysis derived from analytic signal
if #7 and #8 = PWS. = Power spectrum analysis
1 *** PANAL FORT ANAL 8-17-74 A. SARKADY
2 *
3 *
4 * 1 ENTER
5 *
6 * AN=TYPE OF ANALYSIS REQUIRED
7 *
8 * E=ENVELOPE
9 * T=TIME
10 * S=POWER SPECTRUM
11 *
12 *
13 *
14 * 2 ENTER DATA PARAMETERS
15 *
16 *
17 * INT(1)=DATA TYPE
18 * INT(2)=CAL START TIME
19 * INT(3)=CAL END TIME
20 * INT(4)=SAMP 0=SUM 1=DEFOID
21 * INT(5)=NOT USED
22 * INT(6)=ANAL START TIME
23 * INT(7)=ANAL END TIME
24 * INT(8)=#PATIENT TO ANAL
25 * INT(9)=#REC SKIP BEFORE PH2 CALIB
26 *
27 *
28 * 3 ENTER COMMANDS TO ANALIZ
29 *
30 * A=ANALIZ
31 * C=CHANGE PARAM
32 *
33 *
34 *
35 * COMMON/IBLOCK/INT(17)
36 *
37 10 CALL ERAS
38 CALL BEL
39 =0
40 CALL HOLLER(4H AN=, 4)
41 CALL CHINK ICHAR)
42 CALL CHOU( ICHAR)
43 *
44 * INT(1)=1
45 INT(2)=0
46 INT(3)=818
47 INT(4)=0
48 INT(5)=100
49 INT(6)=0
50 INT(7)=818
51  \text{INT}(\text{8})=8
52  \text{INT}(\text{9})=25
53  *
54  \text{=0}
55  \text{CALL HOLLER(4H DF=, 4 )}
56  \text{DO 15 J=1, 9}
57  \text{CALL CHOU(10)}
58  \text{CALL DECIN(\text{INT}(\text{J}))}
59  \text{=INT(\text{J})}
60  \text{15 CALL DECINA}
61  *
62  \text{20 CALL BEL}
63  \text{CALL BEI}
64  \text{CALL CHIN(\text{J})}
65  \text{IF(\text{J} \geq 87)60, 10, 20}
66  *
67  \text{60 MODE=0}
68  \text{65 NFILE=0}
69  \text{CALL ERAS}
70  \text{CALL CONTAP(MODE, 2HRH, 0, 1 )}
71  \text{MODE=1}
72  \text{J=1}
73  \text{IF(\text{INT}(\text{1}) \geq 2)J=\text{INT}(\text{9})}
74  \text{CALL CONTAP(MODE, 2HRF, 0, \text{J})}
75  \text{INT(2)=0}
76  \text{INT(3)=818}
77  \text{INT(5)=1}
78  \text{CALL FANAL(67, 0 )}
79  \text{CALL CONTAP(MODE, 2HFF, 0, 1 )}
80  *
81  \text{80 INT(2)=\text{INT}(6 )}
82  \text{82 INT(3)=\text{INT}(7 )}
83  \text{83 INT(5)=50}
84  \text{CALL ERAS}
85  \text{CALL CONTAP(MODE, 2HCP, 0, 1 )}
86  \text{CALL FANAL(ICHAR, 1 )}
87  \text{NFILE=NFILE+1}
88  \text{IF(NFILE \text{GE.} 6 )GO TO 90}
89  \text{GO TO 80}
90  *
91  \text{90 INT(8)=INT(8)-1}
92  \text{92 IF(\text{INT}(8) \text{.LE.} 0)GO TO 10}
93  \text{GO TO 65}
94  *
95  \text{END}
ENTRY/COMMON BLOCK NAMES
000420 R
000000 C COMMON
000021 C IBLOCK
EXTERNAL NAMES
SUBROUT FANAL. VERSION #2 FORT 8-23-74 A. SARKADY

GEN FORT ANALYSIS ROUTINE

USE STATE INT(1), INT(5) PARAM IN LABELED COMMON

AND CALL FANAL(MANAL, MOUT)

MOUT = 0, 1 OUTPUT ANALYSIS TO MT01 0=NO 1=YES

MANAL = ANALYSIS PARAM

IF EQUAI TO

67 = C = CALIB

67 = E = ENVELOP

83 = S = POWER SPECTRUM

84 = T = TIME

SEARCH IS DELETED

SUBROUTINE FANAL(MANAL, MOUT)

COMMON/IBLOCK/INT(17)

COMMON ID(3), IR(1024), IM(1024)

COMMON IDA(8)

COMMON A(512)

DIMENSION MAT(9), RMS(2)

DIMENSION SAM(2), IH(2), HAD(5), IL(2)

DIMENSION IPW(2048), PW(1024)

DIMENSION AT(7)

DIMENSION MREC(32)

DIMENSION MCUT(32)

DIMENSION MCOR(15)

DIMENSION DA(2)

EQUIVALENCE(IDA(5), DA(1))

EQUIVALENCE(IR(1), IPW(1), PW(1))

EQUIVALENCE(MREC(1), MCUT(1))

EXTERNAL ICSTB

EXTERNAL NAMBF

DATA RMS/1., 1./

DATA SAM/8. E-4, 3.2F-3/
195

51 DATA IH/O, 0 /
52 DATA HAD( 1 )/0, /
53 DATA IL/2HP1, 2H, /
54 DATA AT/4HTIM, 4HANS, 4HPWS, 4HSUM, 4HDEF, 4HMAX, 4HMIN, /
55 DATA LUNO, LUN1/0, 1 /
56 DATA IBLK/2H /
57 * DATA IZS/0 /
58 DATA IZSA/0 /
59 DATA ICUT/1 /
60 DATA MCOR/O, 2048, 0, 0, 1, 1024, 2, 2, 0, 0, 1, 2, 100, 64 /
61 * DATA IDEAL/200 /
62 DATA LDEAL/700 /
63 * CALL INFFT( ICSTB, 1024 )
64 *
65 *
66 *
67 IF( MANAL. EQ. 65 ) GO TO 635
68 IF( MANAL. EQ. 69 ) GO TO 610
69 IF( MANAL. EQ. 84 ) GO TO 620
70 IF( MANAL. EQ. 67 ) GO TO 630
71 IF( MANAL. EQ. 83 ) GO TO 640
72 IF( MANAL. EQ. 90 ) GO TO 39
73 GO TO 6
74 *
75 600 IF( MANAL. EQ. 65 ) GO TO 635
76 IF( MANAL. EQ. 69 ) GO TO 610
77 IF( MANAL. EQ. 84 ) GO TO 620
78 IF( MANAL. EQ. 67 ) GO TO 630
79 IF( MANAL. EQ. 83 ) GO TO 640
80 IF( MANAL. EQ. 90 ) GO TO 39
81 GO TO 6
82 *
83 610 MAT( 2 ) = 70
84 MAT( 3 ) = 68
85 MAT( 4 ) = 73
86 MAT( 5 ) = 77
87 MAT( 6 ) = 86
88 MAT( 7 ) = 65
89 MAT( 8 ) = 50
90 MAT( 9 ) = 82
91 GO TO 40
92 *
93 620 MAT( 2 ) = 84
94 MAT( 3 ) = 86
95 MAT( 4 ) = 65
101  GO TO 612
102  *
103  630  INT(5)=1
104  INT(4)=0
105  MAT(2)=67
106  632  MAT(3)=80
107  MAT(4)=84
108  MAT(5)=86
109  MAT(6)=65
110  GO TO 615
111  *
112  635  MAT(2)=90
113  GO TO 632
114  *
115  640  MAT(2)=70
116  MAT(3)=83
117  MAT(4)=86
118  MAT(5)=65
119  GO TO 612
120  *
121  *
122  0=81=ENTER FARM
123  P=80=PLOT
124  J=74=CONT WITH ANALYS
125  M=77=MEASUR
126  *
127  =0
128  CALL HOLLER(4HAN= , 4)
129  5  CALL BEL
130  CALL BEL
131  CALL CHIN(J)
132  *
133  *
134  IF(J .EQ. 80) GO TO 15
135  IF(J .EQ. 81) GO TO 700
136  IF(J .EQ. 74) GO TO 44
137  IF(J .EQ. 77) GO TO 402
138  GO TO 6
139  *
140  *
141  0=81=ENTER PARAMETERS
142  *
143  MCOR(1)=MIN REC CORF LENGTH
144  MCOR(2)=MAX REC COR LENGTH
145  MCOR(3)=REC# TO BE DELETED
146  MCOR(4)=REC# TO BE DFLFTED
147  *
148  MCOR(5)=PLOT IR1 START CORE
149  MCOR(6)=PLOT IR1 END CORE
150  MCOR(7)=PLOT IR1 CORF SCIP INCR
151 * MCOR(8)=PLOT GRAIN SKIP IN Crom
152 * MCOR(9)=NOT USED
153 * MCOR(10)=SEARCH START TIME
154 * MCOR(11)=SEARCH END TIME
155 * MCOR(12)=SEL MAX OR MIN 0=MIN 1=MAX
156 * MCOR(13)=PLOT IR1 X SCAL FACTOR
157 * MCOR(14)=PLOT IR1 STACK INCROM
158 * MCOR(15)=PLOT IR1 HORIZ START
159 *
160 700 CALL DECIN(L)
161 IF(L.GT.15)GO TO 6
162 IF(L.LE.0)GO TO 6
163 CALL DECIN(MCOR(L))
164 GO TO 700
165 *
166 F=PLOT A
167 *
168 CALL ERAS
169 NSTAR=1
170 NEND=512
171 CALL DECIN(NSTAR)
172 CALL DECIN(NFND)
173 CALL PHEAD(INT(1))
174 =MCOR(9)
175 CALL FHEAD
176 16 CALL BLANKA
177 CALL DECH4(IBLK, IBLK, INT(2))
178 CALL DECH4(IBLK, IBLK, INT(3))
179 CALL CHOUK(13)
180 CALL CHOUK(10)
181 =30
182 CALL BLANKA
183 CALL DECH4(IDLK, 2H I, INT(2))
184 CALL DECH4(IDLK, IBLK, INT(3))
185 CALL ENPLOT(A, NSTAR, NEND, SAMP, IH(1))
186 GO TO 5
187 *
188 CURSOR=MEASURES TIME AND FREQ FROM AVER PLOTS
189 T=TIME IN M SEC RUBOUT =RET TO 6 OTHERS=FREQ
190 *
191 402 CALL CURSOR(ICHAR, NXP, NYP)
192 NXO=FLOAT(NXP)*SAMP
193 IF(ICHAR.EQ.84)NXO=FLOAT(NXP)*SAMP*1000.
194 IF(ICHAR.EQ.127)GO TO 5
198

PAGE 5

201 =NX0
202 CALL DECINA
203 GO TO 402
204 *
205 *
206 * INPUT DATA ACORDING TO INT TABLE
207 *
208 * INT(1)=DATA TYPE 1...4
209 * INT(2)=START TIME
210 * INT(3)=END TIME IN M. SEC
211 * INT(4)=SEMP 0=SUM 1=DEFOLD
212 * INT(5)=# REC5 IN ANAL.
213 *
214 *
215 *
216 39 IZS=0
217 CALL INIBUF(32, MCUT)
218 ICUT=0
219 GO TO 35
220 *
221 *
222 49 NRECNT=NRECNT+1
223 GO TO 42
224 *
225 40 CALL INIBUF(1024, A)
226 CALL INIBUF(10, HAD)
227 *
228 IZSA=IZS/ICUT
229 *
230 IREC=0
231 35 NRECNT=0
232 *
233 37 SAMT=SAM(1)
234 IF( INT(1), GE. 3 )SAMT=SAM(2)
235 X=SAMT*1000.
236 ISTART=FLOAT( INT(2) )/X
237 IEND=FLOAT( INT(3) )/X
238 RMSV=RMS(1)
239 IF( INT(1), EQ. 2 )RMSV=RMS(2)
240 IF( INT(4), GE. 1 )SAMT=SAMT/2.
241 SAMF=1./(1024.*SAMT)
242 *
243 44 MXSTR=MCOR(15)
244 *
245 42 IWC=IEND-ISTART+1
246 CALL INIBUF(2048, IPW)
247 CALL IODATA( INT(1), 2051, LUNO, ID(1), IOFS)
248 IF( IOFS, EQ. 0 )GO TO 10
249 NRCNNT=NRECNT+1
250 IF( ID(2), EQ. MCOR(3), OR. ID(2), EQ. MCOR(4) )GO TO 42
IF(ID(1), LT. MCOR(1), OR. ID(1), GT. MCOR(2))GO TO 42
J=ID(1)-ISTART-3
IF(IWC. GT. J)IWC=J
IF(IWC. GT. 1024)IWC=1024
CALL SHIFT((2048, ISTART, IPW))
LWCO=2048-IWC
CALL INIBUF(LWCO, IPW(IWC+1))
*
DEFOLED
*
IF(INT(4). EQ. 0)GO TO 43
CALL DEFOLED(IWC, IR1)
IWC=2*IWC
HAD(2)=AT(5)
GO TO 48
*
SUM
*
CALL SUMBUF(IWC, IR1)
HAD(2)=AT(4)
*
CALL SUMBUF(IWC, IR1)
IF(INT(1). EQ. 1)IDC 3 )=IL(1)
IH(1)=IDC 3 )
IH(2)=IL(2)
*
IF( ICOM. EQ. 90 )GO TO 800
*
TEST COMMAND CHAR
V=AVERAGE=86
T=TIME REMAVER=84
S=POWER SPECTRUM=83
R=REMOVE AVERAGE =82
P=80=PLOT IR1 ARRAY
M=MAGNITUD =77
I=INV FFT =73
F=FFT=70
E=69=ERAS ANI PLOT IR1 ARRAY
D=ANALITIC SIGNAL=68
C=CALIBRATIAN=67
A=ANALYZ=65
Z=90=SLIDE REC TO ALINE
*
IC=0
IAV=2
FSCAL=1.
IC = IC + 1
ICOM = MAT(IC)

IF (ICOM = 90) 53, 850, 6
IF (ICOM = 84) 54, 84, 86
IF (ICOM = 82) 55, 87, 83
IF (ICOM = 77) 56, 83, 80
IF (ICOM = 70) 58, 70, 73
IF (ICOM = 68) 50, 68, 69
IF (ICOM = 65) 60

IREC = IREC + 1
SAMF = SAMF * FLOAT(IAV)
IF (IREC .GE. INT(5)) GO TO 10
CALL CSTOPT(127, J)
IF (J .EQ. 0) GO TO 6
GO TO 42

Z = 90 = SCAN TAPE TO ALINF RECORDS
GO TO 42

Z = 90 = SLID EACH REC TO LEFT
GO TO 51

FFT

NSHFT = -1
CALL FFTM(IR1, IM1, NSHFT)
FSCAL = FLOAT(2**NSHFT) / 1024.
SAMP = SAMF
GO TO 51

POWER SPECTRUM OR MAGN

CALL ISHUF(1024, IPW)
X = FSCAL / RMSV
Z = X * X
IAV = 1
IF (ICOM .EQ. 77) IAV = 2
DO 100 N = 1, 512
J = IAV * N
X = IPW(2 * J - 1)
Y = IPW(2 * J)
100 FW(J) = (X * X + Y * Y) * Z
IF (ICOM .EQ. 77) GO TO 105
HAD(1) = AT(3)
GO TO 51
201

351 *  DO 106 N=1,512
352   J=IAV*N
353 106 PW(J)=SORT(PW(J))
355   GO TO 51
356 *
357 *   TIME FLOATS ARRAY IR1 AND NORMAL BY RMSV AND FSCAL
358 *
359 84   DO 180 J=1,1024
360   JNUM=1025-J
361 180 PW(JNUM)=IR1(JNUM)
362 180 PW(JNUM)=PW(JNUM)*FSCAL/RMSV
363   HAD(1)=AT(1)
364   GO TO 51
365 *
366 *
367 *
368 *   RFMMOVES AVERAGE VALUE
369 *
370 82   SUMA=0.
371  90   DO 90 J=1,IWC
372   XR=IR1(J)
373  90   SUMA=SUMA+XR
374   SUMA=SUMA/FLOAT(IWC)
375   ISUM=SUMA
376  91   DO 91 J=1,IWC
377  91   IR1(J)=IR1(J)-ISUM
378   SAMP=SAMT
379   IF(IWC.LE.512)IAV=1
380   GO TO 51
381 *
382 *   CALIBRATION=C
383 *
384 67   RMSC=0.
385  96   DO 96 J=1,IWC
386   XR=IR1(J)
387  96   RMSC=RMSC+XR*XR
388   RMSC=RMSC/FLOAT(IWC)
389   RMSC=SORT(RMSC)
390   IF(INT(1).EQ.1)RMS(1)=RMSC
391   IF(INT(1).EQ.2)RMS(2)=RMSC
392   IDA(4)=0
393   SAMP=SAMT
394   GO TO 51
395 *
396 *   AVERAGE=V FLOATING POINT ONLY
397 *
398 *   IAV=1=AVER EVERY POINT
399 *   IAV=2=AVER EVERY 2ND POINT
400 *
DO 110 J=1,512
JDAM=IAV*J
A(J)=A(J)+PW(JDAM)
IDA(2)=IDA(2)+1
GO TO 51

E=69=ERAS AND PLOT IR1 ARRAY
F=80=PLOT IR1 ARRAY
PLOT RANGE -128,+128

CALL ERAS
GO TO 51

D=68=ANALYTIC SIGNAL
IRD(1)=IRD(1)/2
IMD(1)=IMD(1)/2
FSCAL=2.0*FSCAL
DO 140 J=513,1024
IRD(J)=0
IMD(J)=0
HAD(1)=AT(2)
GO TO 51

INV FFT
ISHFT=-1
SAMP=SAMT
DO 500 J=1,1024
IMD(J)=-IMD(J)
CALL FFTM(IRD,IMD,ISHFT)
DO 520 J=1,1024
IMD(J)=-IMD(J)
FSCAL=FLOAT(2**ISHFT)*FSCAL
GO TO 51

FUNCTION AT END OF FILE
IF(MANAL.EQ.90)GO TO 451
IF(MOUT.EQ.0)GO TO 900
IDA(1)=INT(1)
IDA(2)=IREC
IDA(3)=INT(2)
IDA(4)=INT(3)
DA(1)=SAMP
DA(2)=HAD(1)
CALL IODATA(0, 1032, 1, IDA(1), J)
CALL ADELAY(IDEAL)
CALL CONTAP(1, ZHWE, 1, 1)
CALL ADELAY(LDEAL)
RETURN

GO BACK ON MTOO

J = NRECNT - 1
IF( IOFS .EQ. 0 ) J = J + 1
J = 4 * J + 1
CALL CONTAP(1, ZHRB, 0, 1)
GO TO 900

END
ENTRY/COMMON BLOCK NAMFS
0003517 R FANAL
006013 C COMMON
000021 C ISBLOCK
EXTERNAL NAMES
000002 E $SE
000156 E ICSTB
000000 E NAMBF
000155 E INFFT
000455 E HOLLER
000463 E BEL
000465 E CHIN
000617 E DECIN
002700 E ERAS
000622 E FHEAD
000630 E FHEAD
000663 E BLANKA
000700 E DECH4
000656 E CHOU
000705 E ENPLOT
000716 E CURSOR
003074 E FLOAT
003077 E $QM
002413 E $IC
000777 E DECINA
001426 E INIBUF
002723 E $HN
002534 E $QN
003205 E IODATA
001412 E SHIFTB
001447 E DEFOLD
001470 E SUMBUF
001730 E CSTOP
003034 E FFTM
003067 E $HE
002013 E ISHUF
002631 E $HM
002475 E $PC
002652 E $OK
003057 E $DO
002542 E SQRT
003230 E ADELAY
003265 E CONTAP
DESCRIPTION OF THE SECOND-PASS SEVERITY ANALYSIS PROGRAM, PPAVER

PPAVER = Prints, plots, and averages analyzed data files.

Prior to program execution, the following command parameters must be entered through teletype as unsigned integers less than 5 digit length.

NFILE = Number of files to average
NPRINT = List the f and %F on the printer
   If NPRINT = 0 = No print
   If NPRINT = 1 = Print
IPLOT = Plot averaged data on the graphics terminal
   If IPLOT = 0 = No plot
   If IPLOT = 1 = Plot
MAXL = Maximum number of lines per page for printing
NOP = Number of tables per page
NSKIP(I) = Skip the I^{th} file (in the modulo 6 file format) from the analysis
   I = 1, 2, ..., 6
   If NSKIP(I) = 0 = Do not skip I^{th} file
   If NSKIP(I) = 1 = Skip I^{th} file
Input Data:

Magnetic tape unit: MTOO
Format: PASS 1 severity analysis output format

Output Data:

Plots on graphics terminal or tables on printer
PPAVER FORTRAN 8-17-74 A. SARKADY

PPAVER=PRINTS AND PLOTS AVERAGED FILES

DATA INPUT FORMAT OUT PUT OF ALINEFREQ

INPUT DATA IDS

IDA(1)=DATA TYPE 1...4
IDA(2)=# OF CARDIO CYCLES/FILE
IDA(3)=START TIME IN M. SEC.
IDA(4)=END TIME IN M. SEC.
DA(1)=SAMPLE PLOT SEMPLE INCREMENT
DA(2)=4 CARD OF ANAL. ID

INPUT COMMAND PARAMETERS

NFILE=# OF FILES TO AVER
NPRINT=PRINT? 0=NO, 1=YES
I PLOT=PLOT? 0=NO, 1=YES
MAXL=MAX PRINT LINE NUM /PAGE
NOP=# OF PLOTS OR PRINT BLOCKS

NSKIPv(1)= SIP FILE IN GROUP AND NO AVERAGE
NSKIPv(1)=0, 1 1=SKIP 0=NO SKIP OF I TH FILE

ICOM=INPUT COMMAND

A=65=INIT AND START ANALYSIS
C=67=CHANGE PARAMETERS
J=74=CONTINUE WITH ANAL DONT INIT

COMMON/TABLE/LINE(50)
COMMON IDA(8), A(512), IAV(8)
COMMON DB(1), AV(512), DE(1)
DIMENSION BW(10)
DIMENSION BL2(11), BL3(8), BL4(18)
DIMENSION IBl K(4), AFM(4), AT(4)
DIMENSION NSKIP(6), DA(2), BMAS(5), ITEM(5)

EXTERNAL NAMBF
EXTERNAL IP2BF

EQUIVALENCE(IDA(5), DA(1))

DATA BLK/4H /

DATA IBLK/2H, 2H, 2H, 2H /

DATA BMAS/4HNFL=, 4HNPR=, 4HIPL=, 4HML=, 4HNOP= /

DATA BL2/4H, 4HREC=, 4H S, 4HTIM=,

A4H E, 4HTIM=, 4H SA, 4HMP=,

A4H AN, 4HAL=, 4H /

DATA BL3/4HAVE, 4HFRO=, 4H. M, 4HMAG=,

A4HAREA, 4H100=, 4HM*, 4H10K= /

DATA BL4/4H 10, 4H%F, 4H 20, 4H%F, 4H 30, 4H%F, 4H 40, 4H%F, 4H 50, 4H%F, 4H 60, 4H%F, 4H 70, 4H%F, 4H 80, 4H%F, 4H 90, 4H%F /

CALL ERAS

CALL BEL

ITEM(1)=1

ITEM(2)=1

ITEM(3)=1

ITEM(4)=50

ITEM(5)=1

CALL HOLLER(4HPAR=, 4)

DO 5 J = 1, 5

CALL DECIN(ITEM(J)) =ITEM(J)

5 CONTINUE

CALL DECINA

CALL CHOU(10)

NFILE=ITEM(1)

NPRINT=ITEM(2)

IPLGT=ITEM(3)

MAXLIN=ITEM(4)

NOP=ITEM(5)

CALL INIBUF(6, NSKIP)

=0

CALL HOLLER(6HNSKIP=, 6)
CALL BEL
DO 7 J=1, 6
CALL DECINC(I)
IF(I.GT.7)GO TO 8
NSKIP(J)=I
7 CONTINUE
* * A=65=INIT AND ANALIZ
* C=67=CHANGE PAR
J=74=CONTINUE WITH ANAL.
* * NOPF=0
? =0
CALL HOLLER(6H ICOM=, 6)
11 CALL BEL
11 CALL BEL
17 CALL CHINC( ICOM)
* IF( ICOM.EQ. 65 )GO TO 10
120 IF( ICOM.EQ. 67 )GO TO 2
121 IF( ICOM.EQ. 74 )GO TO 12
122 GO TO 9
* 13 MODE=0
135 IF( NPRINT.EQ. 0 )GO TO 12
127 CALL PRTLNE(100)
* 128 * 129 *
130 CALL INIBUF(1028, DB(1))
131 NAV=0
132 NOPF=NOPF+1
133 IF( NOPF.GT. NOP )GO TO 9
* 135 DO 60 I=1, NFILE
136 CALL ERAS
137 CALL CONTAP( MODE, 2HRH, 0, 1)
138 IF( MODE.EQ. 1 )GO TO 14
139 CALL BEL
140 CALL CHINC(J)
141 IF( J.EQ. 127 )GO TO 8
142 14 MODE=1
143 CALL INIBUF(1032, IDA(1))
144 CALL IODATA(10, 1032, 0, IDA(1), J)
145 CALL CONTAP(1, 2HFF, 0, 1)
* 147 * 148 *
149 AT(1)=IDA(2)
150 AT(2)=IDA(3)
AT(3)=IDA(4)
AT(4)=DA(1)
BLZ(11)=DA(2)
SAMP=DA(1)

IF(NSKIP(I).NE.0)GO TO 60
IF(NPRINT.EQ.0)GO TO 40

PRINT 1ST LINE

CALL INIBUF(50,LINE)
IF(IDA(1).EQ.2)GO TO 20
CALL ANDR(0,NAMBF,-64,0)
GO TO 25

CALL ADDR(0,NAMBF,-44,0)
CALL ADDR(1,IBLK,-2,0)
CALL ADDR(2,IP2BF,-16,0)

CALL PRTLNEC(35)
CALL PRTLNEC(200)

2ND LINE

CALL INIBUF(50,LINE)
N=0
K=1
DO 30 J=1,4
CALL ADDR(N,BLZ(K),-8,0)
N=N+1
CALL ADDR(N,AT(J),6,2)
N=N+1
K=K+2
CONTINUE

CALL ADDR(N,BLZ(K),-12,0)
CALL PRTLNEC(35)
CALL PRTLNEC(200)
NLINE=NLINE+4

DO 50 J=1,512
AV(J)=AV(J)+A(J)
NAV=NAV+IDA(2)
CONTINUE

ANALYSIS

PAGE 4
CONTINUE

PRD=0.

SUM=0.

X=FLOAT(NAV)

DO 75 J=1,512

AV(J)=AV(J)/X

SUM=SUM+AV(J)

PROD=AV(J)*FLOAT(J)+PROD

CONTINUE

IF(NPRINT.EQ.0)GO TO 120

X=PROD/SUM

JAVER=X

AVF=X*SAMP

AREA=SUM*100.

K=-1

AMAX=-10000.

DO 80 J=1,512

IF( AV(J) . GT. AMAX )K=J

IF( AV(J) . GT. AMAX )AMAX=AV(J)

CONTINUE

FMAX=FLOAT(K)*SAMP

AFM(1)=AVF

AFM(2)=FMAX

AFM(3)=AREA

AFM(4)=AMAX*10000.

FIND BAND WIDTH

DB(1)=0.

DE(1)=0.

LUP=512-JAVER

LLow=JAVER-1

LIM=LUP

IF( LLow . GT. LUP )LIM=LLow

SINC=.1*SUM

K=JAVER

I=JAVER+1

TOTAL=AV(JAVER)

N=1

Y=SINC

DO 90 J=1,LIM

IF( K . LE. 512 )K=K+1

IF( I . GE. 1 )I=I-1

TOTAL=TOTAL+AV(K)+DB(I)

IF( TOTAL . LT. Y )GO TO 90
251  X=K-I+1
252  BW(N)=X*SAMP
253  N=N+1
254  IF(N.GT.9)GO TO 95
255  Y=Y+SINC
256  90 CONTINUE
257 *
258 *
259 * PRINT LINE #3
260 *
261  95 CALL INIBUF(50,LINE)
262  K=1
263  N=0
264  DO 100 J=1,4
265  CALL ADDR(N,BLK,-2,0)
266  N=N+1
267  CALL ADDR(N,BLK3(K),-8,0)
268  N=N+1
269  K=K+2
270  CALL ADDR(N,AFM(J),7,2)
271  N=N+1
272  100 CONTINUE
273  102 CALL PRTLNE(35)
274  CALL PRTLNE(200)
275 *
276 *
277 *
278  103 CALL INIBUF(50,LINE)
279  CALL ADDR(0,BLK(1),-64,0)
280  CALL PRTLNE(35)
281  CALL PRTLNE(200)
282 *
283 *
284 *
285  105 CALL INIBUF(50,LINE)
286 *
287  200 CALL ADDR(0,BLK,-2,0)
288  201 CALL ADDR(1,BW(1),5,1)
289  CALL ADDR(2,BLK,-3,0)
290  202 CALL ADDR(3,BW(2),5,1)
291  CALL ADDR(4,BLK,-3,0)
292  203 CALL ADDR(5,BW(3),5,1)
293  CALL ADDR(6,BLK,-3,0)
294  204 CALL ADDR(7,BW(4),5,1)
295  CALL ADDR(8,BLK,-3,0)
296  205 CALL ADDR(9,BW(5),5,1)
297  CALL ADDR(10,BLK,-3,0)
298  206 CALL ADDR(11,BW(6),5,1)
299  CALL ADDR(12,BLK,-3,0)
300  207 CALL ADDR(13,BW(7),5,1)
CALL ADDR(14, BLK, -3, 0)
CALL ADDR(15, BW(8), 5, 1)

CALL PRTLNE(35)
CALL PRTLNE(200)
CALL PRTLNE(200)
NLINE=NLINE+7
IF(NLINE LE MAXLIN)GO TO 120
NLINE=0
CALL PRTLNE(100)

IF(IPLT. EQ. 0)GO TO 150

CALL AV( ) ARRAY NSTAR=START CORE
NEND=END CORE FOR PLOT ARRAY

CALL ERAS
CALL BEL
NSTAR=1
NEND=512
CALL DECIN(NSTAR)
CALL DECIN(NEND)
CALL PHEAD(IDA(1))

=NFIL-1
CALL FHEAD
34 =34
CALL BLANKA
I=IDA(3)
J=IDA(4)
CALL DECH4(IBLK, 2H T, I)
CALL DECH4(IBLK, IBLK, J)
CALL CHOU(13)
CALL CHOU(10)
=N30
CALL BLANKA
CALL DECH4(IBLK, 2H N, NAV)
CALL DECH4(IBLK, 2H F, NFILE)
CALL ENPLOT(AV, NSTAR, NEND, SAMP, DA(2))

CURSOR
P=80=RE PLOT RUBOUT=G0127= GO TO 9
J=74=RETURN TO ANAL. OTHERS=LABEL PLOT

CALL DAMCUR(I, J)
CALL CHOU(31)
X=SAMP
IF(SAMP. LE. .1)X=X*1000.
IF(ICHAR. EQ. 80)GO TO 130
IF(ICHAR. EQ. 127)GO TO 11
IF(ICHAR. EQ. 74)GO TO 12
I = FLOAT(I) * X

CALL DECINA
GO TO 140

* K =

CALL CSTOP(127, J)

IF(J .EQ. 0) GO TO 9

GO TO 12

END'

ENTRY/COMMON BLOCK NAMES

003031 R
004024 C COMMON
000062 C TABLE

EXTERNAL NAMES

001021 E NAMBF
001035 E IF2BF
002342 E ERAS
002344 E BEL
000426 E HOLLER
002356 E DECINA
002471 E CHOU
002074 E $DO
002130 E INIBUF
000606 E CHIN
002374 E PRTLNE
000645 E CONTAP
000633 E IODATA
001732 E $FC
002266 E ADDR
001774 E $OK
002564 E FLOAT
001347 E $QN
002567 E $QM
002572 E $IC
002503 E $QL
002361 E PHEAD
002367 E FHEAD
002433 E BLANKA
002450 E DECH4
002455 E ENPLOT
002464 E DAMCUR
002603 E CSTOP

SYMBOL TABLE
002623 R 000001
002704 R 000002
002633 R 000004
000002 R BW
002637 R 000012
ERROR ANALYSIS OF $\bar{f}$

The $J^{th}$ spectral component of the mean murmur spectrum is defined as

$$\tilde{P}(J\Omega) = \tilde{P}_J = \frac{1}{L} \sum_{i=1}^{L} P_i(J\Omega)$$

and the first moment of this spectrum $\bar{f}$ as

$$\bar{f} = \frac{\sum_{J=0}^{N} (J\Omega) \tilde{P}_J}{\sum_{J=0}^{N} \tilde{P}_J} = \frac{\Omega M}{S}$$

where

$$M = \sum_{J=0}^{N} J\tilde{P}(J\Omega)$$

$$S = \sum_{J=0}^{N} \tilde{P}(J\Omega)$$

$N = 511$ and $L = N\cdot\text{REC}$

and $\Omega = \frac{1.250K\text{ Hz}}{1024} = 1.2207\text{ Hz}$

Assuming that the random errors of $\tilde{P}_1, \tilde{P}_2, \ldots, \tilde{P}_J$ are independent and uncorrelated, the standard deviation of $\bar{f}$ is found from the equation given below [78].

$$\sigma_{\bar{f}} = \text{standard deviation of } \bar{f}$$
\[
\sigma_{\tilde{f}} = \sqrt{\left(\frac{\partial \tilde{f}}{\partial \tilde{p}_0}\right)^2 \left(\sigma_{\tilde{p}_0}\right)^2 + \left(\frac{\partial \tilde{f}}{\partial \tilde{p}_1}\right)^2 \left(\sigma_{\tilde{p}_1}\right)^2 + \ldots \left(\frac{\partial \tilde{f}}{\partial \tilde{p}_N}\right)^2 \left(\sigma_{\tilde{p}_N}\right)^2}
\]

where the \(J^{th}\) partial derivative is

\[
\frac{\partial \tilde{f}}{\partial \tilde{p}_J} = \frac{\Omega}{S^2} [J S - M]
\]

\[
\left(\frac{\partial \tilde{f}}{\partial \tilde{p}_J}\right)^2 = \frac{\Omega^2}{S^4} [J^2 S^2 - 2 J S M + M^2]
\]

\[
\left(\frac{\partial \tilde{f}}{\partial \tilde{p}_J}\right)^2 = \frac{1}{S^2} \left[\Omega^2 J^2 - 2 J \Omega \left(\frac{\Omega M}{S}\right) + \left(\frac{\Omega M}{S}\right)^2 \right]
\]

Assuming that the spectrum is white and that

\[
\sigma_{\tilde{p}_j} = \sigma_{\tilde{p}_1} = \ldots = \sigma_{\tilde{p}_N} = \sigma = \frac{\sigma_p}{\sqrt{N.\text{REC.}}}
\]

\[
S = N \mu_p
\]

and \(\sigma_p \approx \mu_p, [71]\)

where \(\sigma_p\) = standard deviation of spectral estimates

\(\mu_p\) = mean value of power spectrum

and \(N.\text{REC.}\) = Number of records averaged yields the following equation.
Expressing the sums in closed forms,

\[
\sum_{J=1}^{N} J^2 = \frac{N(N+1)(2N+1)}{6} = \frac{N}{6} [2N^2 + 3N + 1]
\]

\[
\sum_{J=1}^{N} J = \frac{N(N+1)}{2}
\]

Substituting these values in the previous equation,

\[
\sigma_{\bar{f}} = \frac{\sigma_p}{S} \sqrt{\frac{1}{N^2} \left[ (\bar{f})^2 + \Omega^2 \frac{N}{6} (2N^2 + 3N + 1) - (2\Omega \bar{f}) \frac{N(N+1)}{2} \right]}
\]

\[
\sigma_{\bar{f}} = \sqrt{\frac{\bar{f}^2}{N} + \frac{\Omega^2}{6N} \left( \frac{2N^2 + 3N + 1}{N} \right) - \frac{\Omega \bar{f}}{N} \left( \frac{N+1}{2} \right)}
\]

\[
\sigma_{\bar{f}} = \sqrt{\frac{\bar{f}^2}{511} + 254.56 - 1.223 \bar{f}}
\]

The calculated values for \( \sigma_{\bar{f}} \) are summarized in Table 2, Chapter 5.
LEAST SQUARE REGRESSION LINE AND
CORRELATION COEFFICIENT CALCULATIONS

The correlation coefficient and linear least square
estimate of P.S.E.G. from $\tilde{f}$ are calculated from the short
formula [79] as given below where where

\[ N = \text{number of patients} \]
\[ X = \text{independent variable (} \tilde{f} \text{ or 50%F)} \]
\[ Y = \text{dependent variable (P.S.E.G.)} \]
\[ r = \text{correlation coefficient} \]

and $Y_{est} = \text{linear least square estimate of } Y \text{ from } X$

\[
D_1 = N \sum_{i=1}^{N} X_i^2 - \left( \sum_{i=1}^{N} X_i \right)^2
\]

\[
a_0 = \frac{\left( \sum_{i=1}^{N} Y_i \right) \left( \sum_{i=1}^{N} X_i^2 \right) - \left( \sum_{i=1}^{N} X_i \right) \left( \sum_{i=1}^{N} X_i \right) \left( \sum_{i=1}^{N} X_i Y_i \right)}{D_1}
\]

\[
K_1 = N \left( \sum_{i=1}^{N} X_i Y_i \right) - \left( \sum_{i=1}^{N} X_i \right) \left( \sum_{i=1}^{N} Y_i \right)
\]

\[
a_1 = \frac{K_1}{D_1}
\]

\[
Y_{est} = a_0 + a_1 X
\]

\[
D_2 = N \left( \sum_{i=1}^{N} Y_i^2 \right) - \left( \sum_{i=1}^{N} Y_i \right)^2
\]

\[
r = \frac{K_1}{\sqrt{D_1D_2}}
\]
TABLE 1, AII

CORRELATION STUDY BETWEEN P.S.E.G. AND 50%F AT THE 2ND, R.I. ON INSPIRATION, EXPIRATION AND CAROTID DATA FOR THE TWELVE CATHETERIZED VALVAR AORTIC STENOSIS PATIENTS

<table>
<thead>
<tr>
<th>Catheterization and Personal Data</th>
<th>Phono. Data</th>
</tr>
</thead>
<tbody>
<tr>
<td>Name</td>
<td>Hosp. #</td>
</tr>
<tr>
<td>Tommy K.</td>
<td>63-77-80</td>
</tr>
<tr>
<td>Donald G.</td>
<td>62-12-80</td>
</tr>
<tr>
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<td>70-89-65</td>
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<tr>
<td>Robert M.</td>
<td>53-91-59</td>
</tr>
<tr>
<td>Elizabeth R.</td>
<td>55-01-61</td>
</tr>
<tr>
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<td>68-97-78</td>
</tr>
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<td>57-53-27</td>
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<td>58-79-24</td>
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</tr>
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<tr>
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<td>60-50-48</td>
</tr>
</tbody>
</table>

\[ \Sigma Y = 403.5 \quad \Sigma Y^2 = 19,488.75 \quad \Sigma 50\%F = 893.5 \]

\[ \Sigma Y(50\%F) = 31,462.65 \quad \Sigma (50\%F)^2 = 69,813.41 \]

Correlation Coefficient between P.S.E.G. and 50%F = .3217
TABLE 2, AII

CORRELATION STUDY BETWEEN P.S.E.G. AND $\hat{f}$ AT THE 2ND. R.I. ON INSPIRATION, EXPIRATION AND CAROTID DATA FOR THE TWELVE CATHETERIZED VALVAR AORTIC STENOSIS PATIENTS

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Correlation Coefficient between P.S.E.G. and $\hat{f} = .9657$

Least Square Line = (P.S.E.G.)$_{est.} = -46.0 + .634 \hat{f}$
TABLE 3, All

CORRELATION STUDY BETWEEN P.S.E.G. AND $\tilde{f}$ AT THE 2ND. R.I. ON INSPIRATION DATA FOR THE TWELVE CATHETERIZED VALVAR AORTIC STENOSIS PATIENTS

<table>
<thead>
<tr>
<th>Name</th>
<th>Hosp. #</th>
<th>Chest Wall</th>
<th>$Y=P.S.E.G.$ mm Hg</th>
<th>$\tilde{f}$ In Hz</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tommy K.</td>
<td>63-77-80</td>
<td>Thin</td>
<td>9-18</td>
<td>89.66</td>
</tr>
<tr>
<td>Donald G.</td>
<td>62-12-80</td>
<td>Thin</td>
<td>16</td>
<td>94.52</td>
</tr>
<tr>
<td>Natalie K.</td>
<td>70-89-05</td>
<td>Med.</td>
<td>23</td>
<td>126.7</td>
</tr>
<tr>
<td>Bryan K.</td>
<td>60-91-88</td>
<td>Thin</td>
<td>39</td>
<td>122.29</td>
</tr>
<tr>
<td>Robert M.</td>
<td>53-91-59</td>
<td>Med.</td>
<td>42-45</td>
<td>125.49</td>
</tr>
<tr>
<td>Elizabeth R.</td>
<td>55-01-61</td>
<td>Thin</td>
<td>45</td>
<td>149.32</td>
</tr>
<tr>
<td>Rudolph B.</td>
<td>68-97-78</td>
<td>Thin-Med.</td>
<td>45</td>
<td>143.91</td>
</tr>
<tr>
<td>Richard F.</td>
<td>57-53-27</td>
<td>Med.</td>
<td>61-68</td>
<td>168.81</td>
</tr>
<tr>
<td>Jean S.</td>
<td>58-79-24</td>
<td>Med.</td>
<td>70-90</td>
<td>203.8</td>
</tr>
<tr>
<td>Mark M.</td>
<td>68-95-48</td>
<td>Thin</td>
<td>6-8</td>
<td>99.98</td>
</tr>
<tr>
<td>Jonathan F.</td>
<td>64-87-14</td>
<td>Thin</td>
<td>5-9</td>
<td>96.66</td>
</tr>
<tr>
<td>Barry F.</td>
<td>60-50-48</td>
<td>Med.</td>
<td>16-24</td>
<td>106.66</td>
</tr>
</tbody>
</table>

$\Sigma Y = 403.5 \quad \Sigma Y^2 = 19,488.75 \quad \Sigma \tilde{f} = 1,527.8$

$\Sigma \tilde{f}Y = 59,762.24 \quad \Sigma \tilde{f}^2 = 207,481.75$

Correlation Coefficient between P.S.E.G. and $\tilde{f}$ = .9575

Least Square Line = $(P.S.E.G.)_{est.} = -48.74 + .647 \tilde{f}$
TABLE 4, ALL

CORRELATION STUDY BETWEEN P.S.E.G. AND $\tilde{f}$ AT THE 2ND. R.I. ON EXPIRATION DATA FOR THE TWELVE CATHETERIZED VALVAR AORTIC STENOSIS PATIENTS

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<tr>
<td>Barry F.</td>
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</tr>
</tbody>
</table>

$\Sigma Y = 403.5 \quad \Sigma Y^2 = 19,488.75 \quad \Sigma \tilde{f} = 1,481.17$

$\Sigma \tilde{f}Y = 58,417.38 \quad \Sigma \tilde{f}^2 = 196,222.54$

Correlation Coefficient between P.S.E.G. and $\tilde{f} = .9669$

Least Square Line = $(P.S.E.G.)_{est.} = -45.70 + .6427 \tilde{f}$