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A DIGITAL COMPUTER ANALYSIS OF SOME TRANSMISSION LINE CHARACTERISTICS OF THE MAMMALIAN ARTERIAL SYSTEM

DISSERTATION

Presented in Partial Fulfillment of the Requirements for the Degree Doctor of Philosophy in the Graduate School of the Ohio State University

By

George Steve Malindzak, Jr., A.B., M.Sc.

The Ohio State University
1961

Approved by

Ralph W. Stacy
Adviser
Department of Physiology
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INTRODUCTION

The velocity of propagation of pressure disturbances through blood vessels has been of interest to the biologist for at least 150 years. Velocity data have been used to provide information about the elastic characteristics of the arterial wall, for the basis of interpretations and studies of the pulsatile flow of blood, and to provide certain clinical information concerning the state of the arterial vessels, especially in disease and malfunction (4).

The analysis (biological, physiological, and mathematical) of these pressure disturbances has presented many complex problems. The complexities arise mainly from the non-linear behavior of the system components; for example, the elastomeric arterial walls. As a direct result of these complexities, investigators have attempted to study the behavior of models and analogs of the arterial system rather than the system itself (10,44,45,46,47,48,49,50).

The term "model" or "analog" refers to the similarity of relationships or properties without direct identity. Measurements or observations made on one or more such systems, which have been found to be analogous to other systems, may be used to predict the behavior of those systems. The systems need not be analogous in every respect, but only in those which are of interest to the observer or investigator.
Such has been the case with transmission-line theory. Communication engineers have worked out the equations describing the transmission of electrical signals over conducting lines. Other engineering groups have used the very same transmission line equations to describe related phenomena in their own field of special interest. For example, the acoustical engineers have used the transmission line equations to develop acoustic horns, delay lines, and so on. The biologist now looks to the transmission line theory in the hopes of gaining some insight about the functional mechanisms of the arterial transmission system.

Unlike the communications engineer, the biologist does not have a transmission medium of uniform composition. The arterial structure is known to change with each increment of length. The engineer treats his transmission line parameters as being distributed, while the biologist finds it advantageous to treat his as being lumped, due to his lack of knowledge of the behavioral components of the arterial system. The communications engineer usually deals with a neat, clean, faithful signal while the biologist has one that is often bizarre, aperiodical and non-reproducible.

The pressure waves may be expressed mathematically by means of a Fourier series, in which case the complex wave is analyzed and described as a set of sinusoidal harmonic
components. Each component may then be examined independently of the others. Observations or calculations made on individual components may be summed to obtain the effect on the total wave. This concept is referred to as the "superposition principle" and it has been shown (32,41) that this principle can be expected to apply to the arterial system.

The arterial tree consists of a network of branching elastic tubes that carry the blood to the tissues. Each branching point represents a point of discontinuity within the circulatory transmission line. At each of these points of discontinuity, part of the energy being transmitted will pass beyond the point of discontinuity, part will be dissipated as heat at the point of discontinuity, and part will be reflected from the point of discontinuity and be directed back along the transmission medium toward the source.

The ratio of the amount of energy reflected to the amount of energy incident at a junction is referred to as the "reflection coefficient." The reflection coefficient may also be expressed as a function of the characteristic impedance and the terminal impedance of the line (cf. the standard "standing wave ratio" equation). In general, the relationship states that when the line is completely open (corresponding to an infinitely large terminal impedance)
all the energy will be reflected. So also is the case when the line is completely closed (that is, shorted). In the latter case, the terminal impedance is nearly zero, and the angle at which the energy wave is reflected is 180° out of phase with the former case. Terminal impedances varying from zero to infinity produce only lesser amounts of reflection except in the case where the terminal impedance equals the characteristic impedance, in which case none of the energy is reflected and the line is said to be "matched."

When certain transmission line parameters are known, such as the velocity of the traveling wave, it becomes possible to calculate the reflection coefficient. The ability to obtain a reliable and accurate value for the pulse-wave velocity in the arterial system has been no small task for the circulatory physiologist. Such quantities as the foot-to-foot velocity and the apparent phase velocity have been used in the past. These quantities, as will be seen later, are not the true propagation velocity of the pressure wave.

The presence of reflection in any transmission line represents wasted energy; that is, energy not immediately available to do work. The presence of reflections also loads the energy source, imposing a greater work load.
In the circulatory system, reflections in the arterial tree make it necessary for the heart to do more work to maintain the circulatory needs. Reflections also interfere with the measurement of the pulse-wave velocity within the arteries. It is impossible to measure the true pulse-wave velocity without taking into account the distortion of each individual harmonic by reflections.

It would, therefore, be interesting to study the arterial system in terms of its transmission line characteristics. It should be possible to obtain, by means of a Fourier analysis (and certain recently derived equations describing the arterial system in terms of a transmission line), information relating the amount of reflected energy present in the arterial pulse wave to the pulse-wave velocity. It should also be possible to vary the amount of reflected energy present in the pulse wave by altering the terminal impedance pharmacologically and mechanically.

The true pulse-wave velocity uncomplicated by reflection might be determined. This true velocity value could then be used in Moens' equation to obtain true arterial elasticity.

This type of study should also lead to a better understanding of the biophysical characteristics and over-all behavior of the arterial system. The application of the
results of such a study is obvious to the experimental cardiovascular physiologist as well as the clinician. Investigations of the type described herein are necessary if such application is to come about.
A. Early Theoretical Studies

E. H. Weber (51) was probably the first to try to determine the velocity of the pulse wave in man. He attempted to relate the velocity of the pulse wave in the arterial tree with its elastic properties by measuring the difference in time between the stroke of the pulse wave in the facial artery and the anterior tibial artery. The velocity was estimated to be about 7.24 meters per second (velocity = path of pulse wave divided between difference in time).

The first theoretical work on this subject appears to be that of Young (60,61) who published an equation describing the transmission of pulses in a distensible fluid-filled system with an added mass term. The classic wave equation, as it's called, is given as

$$\frac{\partial^2 E}{\partial t^2} = \frac{E h}{2 r \rho} \cdot \frac{\partial^2 E}{\partial z^2}, \quad (1)$$

where $E$ is the radial displacement of the wall, $t$ is the time, $E$ is the modulus of the elasticity of the wall, $h$ is the thickness of the wall, $\rho$ is the density of the fluid, $r$ is the internal radius of the tube, and $z$ is the axial displacement.

This work was carried much further by Meons (38) in 1878. Contemporaneously and independently Young's equation
was solved by Korteweg (25) who elaborated on some earlier work done by Resal (43) on transmission of pulse waves. As a result, the formulation has come to be called the Young-Korteweg wave equation.

The solution of the wave equation is commonly called the Meons-Korteweg formula, for the velocity of wave-transmission in an incompressible, non-viscous liquid, enclosed in a thin-walled elastic tube. The solution takes the form:

$$c^2 = \frac{E h}{\rho r}$$  \hspace{1cm} (2)

where \(c\) = wave velocity, \(E\) = Young's modulus, \(h\) is the wall thickness, \(r\) is the mean radius of the tube, \(\rho\) is the density of the fluid.

Meons suggested that this form of the solution be modified to include a dimensionless, empirical constant \(K\),

$$c^2 = K \frac{E h}{\rho r}$$  \hspace{1cm} (3)

where \(K\) has a value of about 0.9.

In 1886, a formula was derived for the velocity of propagation of waves in elastic tubes filled with an incompressible fluid by W. Weber (52).

$$\chi^2 = \frac{r}{2 a \sigma^2}$$  \hspace{1cm} (4)

where \(\chi\) = velocity of propagation, \(r\) = the radius of the
tube, \( \rho \) = the specific density of the fluid, and \( a \) = the differential quotient, \( \frac{dr}{dp} \) (\( p \) = the pressure).

A few years later, v. Kries (26) pointed out that \( \frac{dr}{dp} \) depends not only on the quality of the wall but also on the radius of the tube. He, therefore, attempted to correct Weber's formulation as follows

\[
\lambda = \frac{\Delta P}{\Delta Q} \cdot \frac{Q}{\rho},
\]

in which \( Q \) = the cross sectional area of the tube, \( \rho \) = the specific density of the liquid and \( \Delta P \) = the increase in pressure which produces an enlargement of the cross section with the value \( \Delta Q \).

The expression \( \frac{\Delta P}{\Delta Q} \) is related to the properties of the wall as

\[
\frac{\Delta P}{\Delta Q} \cdot Q = \frac{E}{2} \frac{h}{r} \frac{h}{\rho},
\]

where \( h \) = the thickness of the wall and \( E \) = the elastic modulus. This relation is practically identical with the Moens formulation, i.e.,

\[
\lambda = \rho \sqrt{\frac{E}{2}} \frac{h}{r} \frac{h}{\rho}.
\]

This formula was severely criticized by Otto Frank (12) because it didn't take into account the longitudinal stretching of the arterial vessel. Consequently, he derived his own formula to take this into account.
His expression takes the following form,

\[ d^2 = \frac{x}{\sigma}, \quad (8) \]

where \( x = \frac{\Delta P}{\Delta V} \cdot V, \Delta P = \) the increase in pressure, \( \Delta V = \) the corresponding increase in volume of the vessel with volume \( V \). This expression was to be extended to apply to the whole air-chamber or "Windkessel."

Frank calls \( x \) the volume elasticity modulus, which theoretically differs from the moduli given by Moens and v. Kries, in that Frank's formulation includes both the transverse and longitudinal modulus of the vessel (14).

It is interesting to note that although Frank emphasized the significance of reflections in the pulse wave, nevertheless did not take them into account in his development of the air-chamber ("Windkessel") theory.

Bramwell and Hill (6) found that the modified Moens equation held for the flow of blood in the arteries, but modified it by replacing the original parameters by the compressibility of the artery which is more easily measured;

\[ P.W.V. = 0.357 \sqrt{\frac{dP}{dV}} \cdot V, \quad (9) \]

where \( dP \) and \( dV \) are the changes in pressure and volume of the vessel, respectively, \( V = \) the volume of the vessel and \( P.W.V. = \) the pulse-wave velocity. It was necessary for
them to slow the wave velocities by using mercury in order to facilitate measurements and verify this relationship with accuracy.

Wiggers (54) in reviewing the state of the art in a monograph, stated that there were several factors basic to the understanding of the pulse in its transmission to the peripheral elastic arteries.

These factors include damping due to the viscous elements of the transport system, occurrence of natural and free vibrations in different regions of the system, summation of incident and reflected waves, and the transmission of vibrations through the system at different velocities.

He further stated that the transformation of the arterial pulse altered by the different velocities with which the waves are transmitted, was not only dependent on the period of vibration but also the volume-elasticity coefficient, $\frac{dp}{dv}$, at the moment the wave was launched.

In 1932, A. Muller (40) derived an equation for the velocity of the pulse wave which held for vessels with thick walls,

$$v^2 = E \cdot \frac{A_w}{A_v},$$

where $v$ = the velocity of the pulse wave, $E$ = the elasticity of the wall of the vessel, $A_w$ = the area of cross-section of the vessel wall, and $A_v$ = the area of the entire cross-section.
of the vessel. This equation holds, under the conditions mentioned, for an advancing wave only.

In 1939, a comparison between the so-called air-chamber and a closed pipe at one end was made by Wezler and Böger (53). A standing wave was alledgedly formed by reflections about one-quarter wave length from the aortic valves. The fundamental oscillation time of the wave was determined by the difference in time between two maxima on a pulse curve from the femoral artery. This analogy was found to be oversimplified and calculations of the fundamental oscillation time physically unfounded.

Hamilton and Dow (16) with special manometers in dogs, studied a series of pressure curves recorded from the aortic arch to the femoral artery. They found that the pulse pressure increased as the manometer probe was moved peripherally, but that the mean pressure remained constant. The changes in the wave form and pressure were ascribed primarily to reflections and changes in the volume-elasticity modulus of the vessel. They postulated the existence of a standing wave, and that the standing waves fused with the original form of the pulse causing a deformation of the original wave form.

They also reported that the velocity of the pulse wave increased considerably as it proceeded to the periphery from the heart. They assumed without giving any physical
explanation that the base points (point at which the curve first starts to rise sharply) of pressure curves measured along the aorta correspond.

Thus, up to about 1940, many fundamental concepts remained obscure, in spite of numerous attempts to treat hemodynamical problems from the physical, physiological, and clinical points of view.

B. Recent Theoretical Studies

Prior to 1940, hemodynamics had received considerable attention from the point of view of the so-called "Windkessel" concept. This theory had been founded on a number of questionable premises and therefore any conclusions or practical applications based on this theory might give misleading results.

Prior to 1940, the greatest single obstacle impeding the understanding of pulse-wave phenomena was the lack of a "central theory" which included the reflection relationships of the wave (42). A considerable amount of work had been done on the analysis of the factors which determine the form of the arterial pulse.

The periodic pressure variations which are observed in the pulse recording yields readily to mathematical analysis (particularly, Fourier analysis). Up to 1940,
very few workers had used this well-developed mathematical method to describe the characteristics of the pulse.

In 1933 Matoba and Kajiura (34) studied the radial pulse curves on 10 healthy and 10 sclerotic subjects by means of Fourier analysis. They found that the higher frequency harmonic components of the pulse curve were greater in magnitude in the sclerotic subjects than in the normal subjects.

Aperia (2) stressed the great uncertainty which is attached to the determination of the velocity of the pulse wave. It was apparent to him that a more exact theory needed to be developed to include the role of reflections in the arterial pulse-wave.

He asserted that since pressure is a function of both distance (x) and time (t), then pressure in the temporal and spatial domain can be represented as P(x,t). If P(x,t) is pressure, then the wave function may be written as (42)

$$P(x,t) = f(x - ct) + g(x + ct),$$  \hspace{1cm} (11)

where f and g are arbitrary functions of the arguments (x-ct) (for the wave traveling in the positive x direction), and (x+ct) (for the wave traveling in the negative x direction), and c is the pulse-wave velocity. The pulse wave can therefore be regarded as the sum of the two wave functions, f and g. The f waves move in the positive direction,
while the g waves move in the negative direction, at the same velocity c.

In order to measure the true pulse-wave velocity of the traveling wave, it would be necessary to measure the time difference between corresponding points on two pressure curves (either f or g) and the distance between the points of measurement.

If the retrograde wave is added victorially to the incident wave, the result is a new wave which may be said to contain retrograde and incident components. The separate measurement of these components is difficult, since at practical measurement distances, the phase and amplitude differences are small, and these changes are a small fraction of the pulse cycle.

In view of these complications, Aperia suggested velocity calculations on the basis of time of occurrence of equal pressure levels in the subclavian and femoral arteries. He was aware of the technical difficulties involved. He further suggested calculations based on the time difference between the appearances of minimal points of the two curves should give the best results. When these did not appear, he suggested using points located 1/4 or 1/5 the way up the ascending limbs of the pulse curves as corresponding points.

Broemser (8), using Fourier analysis, described a
method for calculating the period of Franks fundamental oscillation. The method he used consisted simply of a mathematical analysis of a manometer model which supposedly represented an analog of the arterial system. In terms of his model, it was said that the femoral pulse resembled the central arterial pulse recorded with a "bad manometer." In effect, the model merely described the frequency response of the arterial system. By using this model, it would be possible to evaluate the physical constants (mass, elasticity, and damping) of a particular manometer that would transform an aortic pulse curve into a femoral pulse curve. The conclusion of this analysis was that the damping of the arterial system was predominately viscous in nature.

In 1946 Anderson and Porje (1), subjected Broemser's work to severe scrutiny and found that his assumptions regarding the mechanics of the "manometer" were over-simplified. Specifically, his supposition that the "manometer" had a constant damping factor was incorrect.

On the basis of Broemser's theory, they attempted to evaluate the physical constants (mass, elasticity, and viscous damping) of the "manometer" that would transform the central aortic pulse wave into a femoral pulse wave. They found that it was not possible to explain the transformation on this basis.
Porje (42) used Fourier analysis on sphymograms from intact human subjects for the purpose of studying the effect of coarctation on the pulse form. He found that stenosis caused a considerable increase in the damping of the mean and oscillatory pressure and that it produced abnormal reflections with a tendency to form a standing wave in front of the stenotic site.

He also found that the first three harmonics contained most of the energy of the wave. The pulse wave was strongly reflected, the fundamental harmonic being reflected much more than either the second or third harmonic. The velocities of transmission for the second and third harmonics were almost equal and the fundamental harmonic velocity was found to increase with age.

In a study involving hypertensive individuals, he found that the fundamental harmonic was most dominant, that the reflections were greatly increased, and that the wave velocities were greater than those of the normal individuals studied.

In an attempt to study the influence of viscosity on the pulse wave velocity, Branson (7) was able to show mathematically that the classical equations for viscous flow can describe blood flow in the femoral artery. He derived the Moens equation for the pulse-wave velocity in terms of
the fluid density and the arterial elasticity by using an equation for the conservation of mass.

King (23) developed an equation for waves in elastic tubes and applied it to cylindrical tubes with Hookean walls. The equation development yielded the Moens-Korteweg formula which was found to be inadequate because the wall thickness does not remain constant. When he applied his equation to cylindrical tubes with elastomeric walls, a complicated expression for the pulse-wave velocity resulted. From this complicated expression he found the pulse-wave velocity to be a nearly linear function of the pressure above 120 mm Hg.

Landowno (28) showed that a pulsatile application of pressure was associated with a marked change in the wave velocity. His observations indicated that even with a rapidly applied impact force, the elastic modulus of the artery is not constant, but undergoes continuous variations during a single pulse cycle.

In 1954 (29) he was able to show that simultaneous brachial and radial intra-arterial pressure records demonstrate that the velocity of propagation and the attenuation in amplitude of the wave vary with the frequency of the externally induced sinusoidal pressure waves. He also stated that the alteration of the form of the central arterial pulse curve as it traveled toward the periphery
indicated that there were differences in velocities of propagation, damping and/or reflection of some components, and that the wave velocity and damping were both pressure and frequency dependent.

In 1957 (30,31) he confirmed Porje's (42) harmonic analysis of sphygmograms. He concluded that significant frequency dependent wave reflections occur in the aorta (in coarctation) and were appropriately explained by the hypothesis that the reflected components may contribute significantly to the phase and amplitude of the pressure wave.

Karreman (20,21,22) on the basis of the work of Porje (42), Hamilton and Dow (16), and Hamilton (17) (which suggested that the radius of the arterioles play an important role in the production of reflected waves), derived a relationship between the propagation velocity and the geometric and elastic properties of a branching tube.

He assumed the walls of the tube to exhibit Hookean behavior, which is not true (24). He also assumed the fluid to be incompressible.

The equation he derived is,

\[ c^2 = \frac{D \cdot E}{2 \cdot r \cdot \rho} \]  \hspace{1cm} (12)

where \( c \) is the pulse-wave velocity, \( D \) the wall thickness,
E the modulus of elasticity, $r$ the radius of the tube, and 
\( \rho \) the density of the fluid, for a special case of wave 
propagation of the water-hammer effect. It is easy to rec­
ognize that this equation is identical with the Korteweg 
equation derived in 1828.

Taking into account the branchings and distensible 
nature of the tube, he approximated the pulse-wave velocity 
as
\[
c = \sqrt{\frac{E \cdot h}{R \cdot \rho}} \left(0.8 - 0.03\right) \frac{w}{\sqrt{\mu}}
\]
where \( w \) = angular frequency, \( \mu \) is the kinematic viscosity 
(\( \mu / \rho \), \( \rho \) = density, \( \mu \) = viscosity), \( E \) = elastic modulus, 
\( R \) = radius, and \( h \) = wall thickness.

Later (21) he studied the reflection of pressure waves 
within fluid-filled elastic tubes for the case of a localized 
change in the diameter of the tube. As a result of this 
study, he developed a method for locating a coarctation from 
the measurement of the pressures in front of, and behind it.

Still later (22) he calculated the reflection co-
efficient for a branched tube of changing areas. He stated 
that the amplitude of the reflected component under normal 
physiological conditions is about 4% of the initial energy 
and therefore not detectable.
Morgan and Kiely (39) presented a theoretical analysis of the propagation of pressure waves through a fluid-filled flexible tube. They showed the dependence of the phase velocity and damping factor on the viscosity of the liquid and the internal damping of the tube wall. Their analysis was restricted to tubes with thin walls and to waves of infinitessimal amplitude and wave lengths large compared to the radius of the tube. They further showed that the wave velocity was frequency dependent and that the traveling oscillations were attenuated. These observations lead to the conclusion that the pulse wave was a complex quantity.

Womersley (55,56) stated that if the liquid contained in a tube were viscous, the pressure wave could not be propagated without distortion. Not only is the motion damped, but the wave velocity rises as the frequency increases. For a constant frequency, the wave velocity increases as the viscosity of the liquid decreases, tending to approach asymptotically that of a perfect liquid.

Morgan and Kiely (39) working on the same problem almost simultaneously, worked out approximate solutions for these phenomena: one of these solutions applied to a "small viscosity," using an asymptotic expansion of the Bessel Functions, and the other applied to a "large viscosity," using a power series. They did not, however, seem to appreciate the dependence of the wave velocity on Womersley's
single non-dimensional parameter $\alpha$ (which is to be discussed shortly).

Womersley (55) asserted that in order to accurately determine the wave velocity, it would be necessary to measure the pulse-wave at two points simultaneously or to measure the pulse-wave and its derivative at one point. He derived a frequency equation which determined the pulse-wave velocity in terms of the elastic properties of the tube and in terms of the non-dimensional parameter $\alpha$, which characterizes the motion of fluid in an elastic system and is given by

$$\alpha = R \sqrt{\frac{w}{\gamma}},$$

(14)

where $R$ = radius, $w$ = angular frequency, and $\gamma$ = kinematic viscosity of the fluid. He showed that the variations in the pulse-wave velocity with frequency and viscosity can be expressed as a function of this single parameter $\alpha$.¹

Womersley (57,58) also derived equations relating flow to an oscillating pressure gradient. His method of derivation is somewhat similar to a method used in deriving Poiseuille's Equation.²

¹It is interesting to note that Morgan and Kiely and Womersley, almost simultaneously, concluded that the wave velocity was a complex function and frequency dependent.

²In the case of both Poiseuille and Womersley, it was difficult to provide precise experimental tests of the theory.
This relation cannot be rigorously applied to arteries. Nevertheless, it is probably as useful a practical approximation for oscillatory flow as Poiseuille's Law is for steady flow in blood vessels. The difficulty of application of Womersley's theory lies in the problems of instrumentation for the measurement of oscillatory flow.

Womersley (57, 58) also showed that equations for rigid tube flow also apply to arteries. His detailed mathematical investigations showed that changes in the behavior and properties of the arterial wall had relatively little effect on the pressure-flow relationships of the blood, but that they are important with regard to wave propagation.

Since the pressure pulses on the arterial side are periodic in nature, it was possible for Womersley (57, 58) to describe the pressure in terms of a Fourier Series. He was able to describe the velocity of each harmonic component of the pulse wave and the harmonics of the pressure gradient in the presence of a reflected wave. He pointed out that if reflections were present, the calculation of the phase velocity led to an "apparent phase velocity", the magnitude of which depends upon the magnitude of the phase of the reflected harmonic components. In addition, he stated that it was necessary to take into account the complex damping of both transmitted and reflected components.
in order to calculate the reflection coefficient.

Taylor (46,47) examined the nature of the arterial system by analogy, using telegraph equations applied to an attenuating line. From this basis, he derived an expression for the apparent phase velocity over a finite distance of a line and was able to calculate the modulus of the reflection coefficient in terms of the apparent phase velocity, as a function of the distance from the closed end of the tube.

He also studied the frequency response characteristics of a water-filled tube. From these studies, the input impedance of an occluded tube was calculated as a function of its length for a frequency range of 4-12 cycles per second. True standing waves were not found to be present in the tube, although the pressure moduli exhibited maxima and minima.

Stacy and Giles (44) derived a mathematical model of a simplified hydraulic system with lumped parameters to describe the behavior of the arterial system. This approach involved mathematical operations (using analog computers) on a recorded central pulse to produce a computed peripheral pulse. The computed peripheral pulse was compared with a recorded peripheral pulse and the computer parameters varied until a match between the two was made. The remarkable success of this relatively simple description of the
arterial system made possible a whole host of experimental
designs to study in greater detail the transient and steady-
state characteristics (33) and the pharmacological behavior(19)
of the arterial system. The use of the analog computer in
these studies made possible the application of analytical
procedures heretofore unsuccessful.

Evans (9,10) has developed a set of analytical ex-
pressions based on a peculiar coordinate system for the
purpose of analyzing reflections in pressure waves. He
applied his equations to rubber tube models and arterial
pulse waves, and reported that he was able to calculate
the amount of reflection present in the two types of waves.
He stated that the reflections were greater in the models
than in the blood vessels. The reflected component in
the arterial pulse was found to be no greater than 30% of
the propagated component.

Farrow and Stacy (11) studied the transmission line
characteristics of the arterial system by simultaneously
recording the oscillatory pressure at two points along the
aorta (at the junctions of the left renal artery and the
inferior mesenteric artery), under normal conditions, and
after administration of norepinephrine and acetylcholine.
These recordings were subjected to Fourier analysis on the
IBM 650 Digital Computer, and the apparent phase velocities
and foot-to-foot velocities were measured and compared. Considerable discrepancies were found between the values of these two quantities. Specifically, it was shown that the foot-to-foot velocities provided erroneous information about the true conduction velocity of the wave. Their results strongly suggest that reflected waves are present and that the origin of the site of reflection is located in the peripheral 1/3 of the thigh of the dog.
METHODS AND PROCEDURES

A. Introduction

The studies described herein include the examination of some of the physical factors influencing pulse wave transmission in the aorta.

These studies include detection and quantification of the true pulse-wave velocity in the presence of reflections, and determination of the amount of reflected energy in the pulse wave at any point along the aortic trunk. No attempt has been made to describe thoroughly all the physical factors involved. Rather, the majority of the time and effort was spent on developing a sound mathematical foundation upon which further and more sophisticated investigations might be built.

The methods used herein take advantage of the periodic nature of cardiac ejections and arterial pressure fluctuations. The Fourier analysis technique for studying the harmonic content of periodic phenomena seemed particularly suited for this study because the mathematical equations which underlay these phenomena lends themselves readily to such treatment.

It has long been known that as the arterial pressure wave proceeds peripherally from the heart, it encounters many points of physical discontinuity, at which part of the
energy of the wave is reflected in the retrograde direction. This reflected component may be said to represent the total reflected effect of many points of discontinuity. Therefore, at any time, the total energy of the transmitted arterial pressure wave is the sum of that energy which was imparted to it from the heart (the incident wave) plus that which was reflected from the numerous points of discontinuity along the aortic trunk (the reflected wave).

B. The Equation

Due to the involved nature of the derivation of the primary equations used in these studies, this derivation is presented in Appendix B. For simplicity of presentation, the final equations are presented here in their abbreviated form, with their terms defined. The first of these equations is:

\[
\left( \frac{A_3}{A_1} \right)_T^2 = \frac{1 + \left( \frac{R}{I} \right)^2 + 2 \left( \frac{R}{I} \right) \cos (\alpha - 2B)}{1 + \left( \frac{R}{I} \right)^2 + 2 \left( \frac{R}{I} \right) \cos (\alpha)}, \quad (B.17e)
\]

where,

\[
\left( \frac{A_3}{A_1} \right)_T^2 = \text{The theoretical ratio of the amplitudes of the transmitted wave of a harmonic measured at points } x_3 \text{ and } x_1, \text{ along the arterial channel.}
\]

\[ A_3 = \text{Harmonic amplitude of the pressure wave measured at } x_3 \text{(downstream point).} \]
$A_1 = \text{Harmonic amplitude of the pressure wave measured at } x_1 \text{ (upstream point).}$

This equation (B.17e) describes the ratio of amplitudes of a harmonic measured at two points in the aorta. Another important equation is:

$$\left( \frac{R}{I} \right)^2 = \frac{C^2 - AC + B}{C^2 + AC + B}, \quad (B.12d)$$

the ratio of the reflected energy to the incident energy, or, the reflection coefficient, where,

$R = \text{Harmonic amplitude of the reflected component.}$

$I = \text{Harmonic amplitude of the incident component.}$

$$\Lambda = \frac{2\omega \Delta x \left( \theta_3 - \theta_1 \right)}{(\theta_3 - \theta_1)^2 + \left( \frac{A_3}{A_1} - 1 \right)^2} \quad (B.13)$$

$$B = \frac{\Delta x^2 \omega^2}{(\theta_3 - \theta_1)^2 + \left( \frac{A_3}{A_1} - 1 \right)^2} \quad (B.14)$$

$w = \text{Angular frequency in radians.}$

$\theta_3 = \text{Phase of harmonic amplitude } A_3.$

$\theta_1 = \text{Phase of harmonic amplitude } A_1.$

$\Delta x = \text{Distance between recording sites.}$

$$\lambda = \lambda_1 + \lambda_2 \quad (B.18)$$
\[ \alpha_1 = \tan^{-1} \left[ \frac{\frac{c}{\Delta x} \left( \frac{A_3}{A_1} - 1 \right)}{\omega - \frac{c}{\Delta x} (\theta_3 - \theta_1)} \right] \]
is the angle between
the resultant vector
and the incident vector.*

(B.12)

\[ \alpha_2 = \tan^{-1} \left[ \frac{\frac{c}{\Delta x} \left( \frac{A_3}{A_1} - 1 \right)}{\omega + \frac{c}{\Delta x} (\theta_3 - \theta_1)} \right] \]
is the angle between
the resultant vector
and the reflected vector.*

(B.13)

\[ \beta = \frac{\omega \Delta x}{c} \]
is the angle between the reflected and
incident vector.

This equation\(^{(B.12d)}\) describes the reflection coefficient in
terms of factors which can be derived by Fourier analysis
of two simultaneously recorded pressure pulses.

It should be noted that all one needs in order to use
these equations is the information provided by the Fourier
analysis of the pressure wave form, that is, the harmonic
amplitudes and the phases of two simultaneously recorded
pressure waves at two points along the aorta.

*See Appendix B for a more precise definition, if
required.
It should also be noted that the quantities \( \frac{R}{I} \), \( \left(\frac{R}{I}\right)^2 \), and \( c \) appear in the equations of major import. \( \left(\frac{R}{I}\right) \) and \( \left(\frac{R}{I}\right)^2 \) are functions of \( c \), and therefore, \( \left(\frac{A_3}{A_1}\right)_T^2 \) essentially becomes a function of \( c \). Since \( \left(\frac{A_3}{A_1}\right)_T^2 \) is a theoretical quantity, its practical measurable counterpart serves as a guide to the correct value of \( c \) needed to solve the equation. That is, a value of \( c \) is chosen and a value of \( \left(\frac{A_3}{A_1}\right)_T^2 \) is computed. This computed value is compared with the value measured. If the computed value is different from the measured value, new values of \( c \) are chosen and the process is repeated until \( \left(\frac{A_3}{A_1}\right)_T^2 \) computed is identical with \( \left(\frac{A_3}{A_1}\right)_T^2 \) measured. The value of \( c \) chosen at this point uniquely satisfies the equation for the conditions used to derive it. In addition, the values of \( \left(\frac{R}{I}\right)^2 \) and \( \frac{R}{I} \) become available. These values represent the ratio of the reflected energy to the incident energy, or the amount of reflected energy in the wave, and the reflection coefficient.

C. Animal Preparations

Male and female mongrel dogs, averaging about 15 kilograms in total body weight, were given Nembutal, Abbott (30mg. per Kg. of total body weight). Their tracheas were exposed and cannulated. One femoral vein was cannulated for injections. The left or right common carotid artery was
exposed and cannulated with a dual-lumened catheter of the type illustrated in Figure 1.

![Dual Lumened Catheter](image)

**FIGURE 1**

Dual Lumened Catheter

The catheter was made of 1.0 millimeter diameter steel tubing and was 35 centimeters long from yoke to tip. The peripheral ends of the two lumen were 5 centimeters apart, thereby maintaining a constant distance for measuring the simultaneous pressure fluctuations at two points along the aorta. Male Luer Lock connectors were attached to the central ends of the catheter so that conventional syringes, Sanborn Physiological Pressure Transducers, and Statham Strain-gage Transducers might be fitted easily.

The tip of the catheter was advanced, via the common carotid artery (either the right or the left, depending on which was most convenient), to the junction of the common carotid and the aortic arch. At this point, the catheter was threaded around the aortic arch and advanced peripherally until the tip came to approximately the level of the renal artery.
The central end of the dual-lumened catheter was connected to a Differential Pressure Transducer (Sanborn Model 467B), the output of which consisted of an absolute pressure as a function of time of the upstream point on the catheter, and the differential pressure between the two ends of the catheter.

After all the cannulae were secured in place, Heparin sodium (30 mg) was given to each animal to retard clotting. An additional 5 units/cc was added to the Ringer's solution used to flush out the cannulae and associated channels when clots formed, and to fill the pressure transducers.

In some experiments where it became difficult to thread the catheter via the left common carotid artery, the right side was exposed and used. In most cases this change met with success.

D. Instrumentation

A Statham P-23D pressure transducer was used to measure the absolute pressure as a function of time at the upstream recording site. This transducer was calibrated before and after each set of recordings during an experiment with a mercury and/or an aneroid barometer.

A Sanborn Physiological Differential Pressure Transducer, Model 467B, was used to measure the differential pressure. This transducer was ten times more sensitive than
the Statham P-23D.

Extreme caution was taken to insure that the output over the pressure range 0 to 200 mm. Hg. for the absolute pressure transducer, and 0 to 20 mm. Hg. for the differential pressure transducer, was linear, and that the zero baseline did not shift appreciably between recordings.

The baseline was checked before and after recording each set of tracings. These tracings usually consisted of about one minute of sequential pressure recordings or about 100 pulse curves. If a reasonable discrepancy (5 to 10 mm. Hg. difference) were present between the baseline before and after the recording of a set of pulse curves, steps were taken to correct the fault, the previous set of tracings discarded, and another set of pulse curves recorded under the same conditions.

The phase shift between the two pressure transducers was noted to be less than two degrees. With this relatively small distortion, it was assumed that matching of the two cannulae of the catheter was unnecessary, and that mathematical operations might be performed on each pressure recording, either separately or together without seriously destroying the accuracy of the data.

The tracings (pulse pressure curves) were recorded on a Twin-Viso Sanborn Recorder, which has a frequency response
flat to about 40 cycles per second. The strain-gage amplifiers and the pressure transducers both have a frequency response that is flat to about 300 cycles per second. Thus, the frequency response of the overall system was limited by the 40 cycle per second cutoff of the recorder. This was more than adequate for these experimental needs, since the highest frequency component of interest was 15 to 20 cycles per second.

E. Experimental Procedures

Several types of experiments were performed to determine the effects of various mechanical and pharmacological procedures on the harmonic content, reflected energy content, and the true phase velocity of the transmitted pressure wave in the aorta.

In all the animals used, (15 for this study) after the insertion of the catheter into the aorta, 30 minutes was allowed for a steady-state to be re-established and the traumatic effects to wear off. At this time, a control recording was taken and the zero pressure baseline checked before and after the recording to insure that no DC artifacts were present. After the control recording was taken, the dogs were given norepinephrine intravenously (0.0015 mg./kg.) which produced vasoconstriction. Recordings were made before,
during, and after the injection to insure "capturing" the maximum effect of the drug. The baselines were also checked before and after the recording of each set of pulse curves.

The dogs were then allowed 30 minutes to recover. After that time, another control record was made, and the baselines were checked again. Following the second control recording, the dogs were given acetylcholine intravenously (0.005 mg./kg.) which produced vasodilation. The recordings were also made before, during, and after the injection to insure "capturing" the maximum effect of the drug. The baselines were checked again. The dogs were again allowed 30 minutes to recover, after which a third control record was made and the baselines were checked.

This procedure was done four times for each experimental animal, and the best set of tracings of the four sets was chosen as being representative for that particular animal. Each representative set consisted of three control and two experimental groups of pulse curve recordings. A total of 15 dogs were used for this particular experiment.

F. Record Preparation

The simultaneous tracings of the absolute pressure (upstream) and the differential pressure served as the source of data used for the analysis. These tracings were recorded on thermo-sensitive paper on the Sanborn Twin-Viso Recorder.
From each set of tracings, for each experimental condition, a representative pair of simultaneously recorded pulses (absolute pressure and differential pressure) was selected. A transparency was then made of each pair selected. The transparency was used as a negative in a photographic enlarger, the image of which was projected onto a sheet of recording paper (8 1/2" x 11"), which contained about 40 evenly spaced vertical lines. The outline of the two simultaneously recorded pulses was then traced out onto this paper.

The period of each pulse was normalized to a fixed interval (14.0 centimeters), into which fit 24 evenly spaced intervals. This was accomplished by varying the image size in the enlarger. Each interval represented 1/24 or 15° of the total pulse cycle. This procedure was followed mainly because of the relative ease with which one can perform a graphical analysis of the Fourier coefficients by numerical integration based on a 24-ordinate method.

The ordinate values for each of the 24 intervals were tabulated onto forms designed by the laboratory for standard Fourier analysis. Analytical operations were then performed on these values.

G. Analytical Procedures and Data Presentation

From equation (A.11), (A.13), and (A.15), in Appendix A, rewritten here as follows,
and, $A_n \approx \frac{2}{m} \sum_{k=1}^{m} y_k \cos n\theta_k$. \hspace{1cm} (A.13)

$B_n \approx \frac{2}{m} \sum_{k=1}^{m} y_k \sin n\theta_k$. \hspace{1cm} (A.15)

it is possible to obtain the values of the Fourier coefficients and compute the moduli and phases of the various harmonics of the pulse wave. This procedure was accomplished on each of the simultaneously recorded pairs of curves.

The differential pressure curve Fourier coefficients were then subtracted from the absolute pressure coefficients to obtain a third curve, the absolute pressure curve at the downstream point. Justification for this procedure is obvious when one considers the difficulty involved in matching the two catheters for zero phase shift. This problem is technically much too difficult and cumbersome. Hence, it was assumed that by subtracting a very accurately measured differential pressure pulse from a not-so-accurately measured
absolute pressure pulse (upstream), the resultant pressure pulse (downstream) will have an accuracy no worse than the upstream pulse. In the long run the difference in the curves is all important.

Thus, the Fourier analysis yielded a set of upstream pressure coefficients \((A_1's)\), a set of differential pressure coefficients \((A_2's)\), and a set of downstream pressure coefficients \((A_3's)\). These quantities were then inserted into equation (B.17f) and the non-velocity dependent constants computed, after which the iteration process was done on \(c\).

Due to the sheer number of computations involved in this procedure, some data reduction steps needed to be taken. Therefore, it was assumed that once all the curves (for each experimental condition) were analyzed, they would be averaged in order to represent some experimental mean. The same result could be obtained if just one curve, representing the average response of all the curves were presented for analysis. That is to say, if 10 curves were to be analyzed and averaged, they were averaged before the analysis to obtain one average curve. This single average curve was then analyzed for the Fourier coefficients. The resultant data were identical to that obtained from the average of the Fourier coefficients from each of the 10 curves.
This single mean curve was very easily obtained by simply averaging the sequential ordinates of each of the curves under consideration. When this procedure was completed, there were $2^{14}$ numbers representing the sequential values of the ordinates (1 to $2^{14}$) of an average curve for each experimental condition.

In these studies, five records were selected from the representative set of curves from each animal. These included three controls (one before norepinephrine administration, one after norepinephrine injection and before acetylcholine injection, and one after acetylcholine injection), one during the norepinephrine effect and one during the acetylcholine effect.

The three controls from each animal were averaged to obtain a mean for each animal. Surprising similarity existed among these control records. At this point, there were only three records to represent the experimental data on each animal: (1) averaged controls, (2) norepinephrine, and (3) acetylcholine.

In the 15 dogs studied, all the averaged individual controls were averaged to produce an average control curve for the whole group. All the norepinephrine and acetylcholine records were treated likewise. At this point, three curves were to be analyzed for all 15 dogs: (1) average controls, (2) average norepinephrine, and (3) average acetylcholine.
This procedure fantastically reduces the amount of computation required.¹

From each of the curves, the following information was obtained for each harmonic (cf. equation section, Appendix A, and Appendix B), (1) harmonic number (n), (2) Fourier sine coefficients (Aₙ's), (3) Fourier cosine coefficients (Bₙ's), (4) vector sum of the sine and cosine coefficients (Cₙ's), (5) phase angle (θₙ's), (6) incident vector phase angle (α₁), (7) reflected vector phase angle (α₂), (8) sum of (6) and (7) (β), (9) angle through which the incident and reflected vector moves along x (θ), (10) the true phase velocity (cₙ), (11) ratio of reflected energy to incident energy (R/I)²ₙ, (12) reflection coefficient (R/I)ₙ, (13) error (E), (14) constant (A), (15) constant (B), and (16) Apparent Phase Velocity (APV).

From this information, the following comparisons were made: (1) true phase velocity versus harmonic for controls, norepinephrine, and acetylcholine; (2) apparent phase velocity versus harmonic for controls, norepinephrine, and acetylcholine; (3) (R/I)² and (R/I) versus velocity for each harmonic for each experimental condition, and (4) true phase velocity and apparent phase velocity versus harmonic for each experimental condition.

¹It was calculated that in order to perform all the necessary computation manually in these studies, it would require 48 man-years of time.
H. The Computer Program

In these studies, the IBM 704 General Purpose Digital Computer, housed at the Ohio State University Numerical Computation Laboratory, was programmed to perform most of the arithmetic steps outlined in the previous discussion. The output of the computer was in the form of a printed page from which all the necessary comparison data was extracted.

The FORTRAN compiler language was used to program the machine in view of its facility in handling the arithmetic concepts used in these studies.

A flow chart diagram of the program is presented in Appendix C, which is followed by a list of terms used in the program. The program, as used here, was designed to compute the Fourier coefficients and phase angles of each of the curves presented for analysis for as many harmonics as is desired. These computed quantities were inserted into equation (B.17f) from Appendix B, and by a specific instruction the computer selected a starting value for $c$, and computed values for $\alpha_1$, $\alpha_2$, $\alpha$, $\beta$, $(R/I)^2$, $(R/I)$, $E$, $A$, $B$, and APV. After these quantities were computed, they were printed out on a single line.

This procedure was followed for each curve to be analyzed, for each of 5 harmonics per curve. The initial value of $c$ was set at 20 centimeters per second. For each subsequent
computation, the value of c was increased by increments of 20 centimeters per second. The computations stopped after the value of c reached 2500 centimeters per second. The initial and final values for c were fed to the machine as a separate instruction before each run, so that the starting and ending point could be changed as desired without revising the whole program.

It was decided, a posteriori, that contributions beyond the 5th harmonic were insignificant. Therefore, the true phase velocity calculations and the harmonic data beyond the 5th were automatically excluded from this presentation.

Since the selection of the correct value of c during the first pass would be fortuitous, it was decided to allow the computer to run through the entire procedure and print out line by line all the computations made over the entire range of c's. Afterwards, the correct value of c was selected by plotting the error (E) between the computed ratio $\left(\frac{A_3}{A_1}\right)^2$ and the theoretical ratio against the values of c used. When this error was zero, the correct value of c obtained, and the associated quantities dependent on c were extracted and used for the various comparisons discussed earlier.
RESULTS

A. Fourier Analysis

Figure 2 shows a typical averaged set of curves of the absolute and differential pressures as a function of time from which the ordinate values were obtained and on which the computations were made. The curves shown represent the average pressure variation of all the control records obtained over one pulse cycle. On the top half of the graph is plotted the absolute pressure as recorded from the upstream and downstream points on the catheter. The curve that rises first represents the upstream absolute pressure, while that which arises later represents the downstream pressure.

The lower curve of Figure 2 represents the differential pressure as measured between the two ends of the catheter tips (5 centimeters apart). The scale is magnified 5 times (with respect to the absolute curves) in order to show the form of the differential pressure curve.

The downstream curve was obtained, not by direct recording, but rather by vectorially adding the differential pressure Fourier components to the upstream pressure Fourier components.

It should be noted that the differential pressure curve does not oscillate above and below zero, but rather about a
mean considerably above zero (4.316 mm.Hg.). This 4.316 mm.Hg. was the DC pressure drop along the 5 centimeter length of artery along which the pressures were being measured.

A dramatic comparison may be made on the curve of Figure 2 of the differences between various velocity indices used by different investigators. The foot-to-foot velocity was calculated to be 1.96 meters per second. The apparent phase velocity was 15.9 meters per second for the first harmonic and the true phase velocity was 16.6 meters per second.

The average period of the data used to construct these curves was 0.4163 seconds and the average frequency 2.539 cycles per second (15.933 radians per second). ¹

Tables 1, 2, and 3 show the results of Fourier analyses on the averaged control, norepinephrine and acetylcholine pressure curves, respectively. Each of the tables is divided into three sets of data; the absolute pressure (upstream), the differential pressure, and the absolute pressure (downstream). The first column of each set lists the harmonic (1 to 5 inclusive), the second column the sine coefficients, the third the cosine coefficients, the fourth the modulus, and the last column the phase (in degrees). The sine

¹Although, in some instances, four significant figures are cited throughout this text, it is felt certain that no more than two of them are significant.
coefficient, cosine coefficient, and modulus data are in units of mm. Hg.

Table 1 shows the Fourier analysis data for the averaged control pressure curves. Attention should be directed to the modulus column for the data on the upstream and downstream pressure curves. The modulus of the first harmonic for the upstream pressure curve was 15.269, while for the downstream pressure curve it was 16.345. The same direction of change is seen in the values of the modulus for the second and third harmonics, i.e., 7.423 to 9.181 (an increase) for the second harmonic, and, 2.305 to 2.682 (an increase) for the third harmonic. For the fourth and fifth harmonics, the direction of the change was reversed, i.e., 1.135 to 1.101 (a decrease) for the fourth harmonic, and 1.862 to 1.774 (a decrease) for the fifth harmonic.

Also, it should be noted that there is a difference in the mean pressure between the two curves. The mean pressure for the upstream curve was 135.409 mm. Hg., while the mean pressure for the downstream curve was 131.487 mm. Hg., corresponding to a pressure drop of 4.316 mm. Hg. This value is not unreasonable, since this would correspond to a flow of about 100 cm.³/second, a viscosity of 0.04 poise, and a radius of about 3 millimeters for the vessel; well within the range of acceptable physiological values.
Table 2 shows the Fourier analysis data on the averaged norepinephrine pressure curves. By a similar comparison, it is shown that the modulus of the first three harmonics for the upstream and downstream pressure curves increases in magnitude, (i.e., from 23.879 to 25.388 for the first harmonic, from 12.158 to 13.700 for the second harmonic, and, from 5.866 to 6.089 for the third harmonic). Similarly, the modulus of the fourth and fifth harmonics for the upstream and downstream pressure curves decreases in magnitude (from 2.620 to 2.324 for the fourth harmonic, and from 2.739 to 2.602 for the fifth harmonic). Also it is seen that the mean pressure of the upstream and downstream curves decreases from 163.120 mm. Hg. to 158.804 mm. Hg., respectively. This corresponds to a drop in mean pressure of 4.316 mm. Hg., across the 5 centimeter distance between the tips of the catheter, which also seems physiologically reasonable.

By a similar comparison, Table 3 (for the averaged acetylcholine curves) shows an increase in the modulus of the first harmonic (13.572 to 14.554), a decrease in the second harmonic (5.808 to 4.627), a decrease in the third harmonic (3.299 to 2.513), a decrease in the fourth harmonic (3.127 to 2.853), and, a decrease in the fifth harmonic (1.718 to 1.308). The table also shows the mean pressure difference between the upstream and downstream curve to be
3.263 mm. Hg. (the difference between 71.279 mm. Hg. and 68.015 mm. Hg., respectively).

Some striking similarities should be noted between the moduli of each of the sets of upstream and downstream pressure curve data. For example, in Table 1, the upstream pressure curve data shows the first harmonic modulus to be greater than the second, the second three times greater than the third, and so on. It is noticed that each set of moduli exhibit the same trend, i.e., the modulus of the first or fundamental harmonic is much greater in magnitude than either of the other harmonics. This trend is consistent with what is expected in transmission line systems and will be discussed at length later.

Also attention is brought to the difference in the first harmonic moduli values from each of the three tables (1,2,3). For example, the upstream pressure curve moduli exhibited values of 15.269, 23.879, and 13.572, for the control, norepinephrine, and acetylcholine, respectively. It is not surprising to find that the fundamental harmonic for norepinephrine was greater than that for the control, and the fundamental harmonic for acetylcholine was less than that of the control. The values for the mean pressure in each case vary in the same manner [135.409 (control), 163.120 (norepinephrine) and 71.279 (acetylcholine)]. In the discussion:
section, an attempt will be made to explain this trend as well as some of the others mentioned previously.

B. Error Function Analysis

Table 4 shows the variations of \((R/I)^2\), \((R/I)\), and the error \((E)\) as a function of the true phase velocity \((c)\), for the first harmonic of the averaged acetylcholine curves. It will be recalled that equation (B.12d) shows the mathematical relationship of these parameters. It will also be remembered that the error \((E)\), was the difference between the actual \((A_3/A_1)^2\) measured and the theoretical \((A_3/A_1)^2\). The first column of the table contains the true phase velocity in meters per second; the second column, \((R/I)^2\) in percent; the third column, \((R/I)\) in percent; and the last column, the error \((E)\). The range of \(c\) selected for which the associated data were calculated was from 1.0 to 25.0 meters per second in increments of 1.0 meters per second.

Attention is directed to the last column on the right, the Error column. For a phase velocity of 1.0 meter per second, the error was calculated to be \(+0.5119\); for a phase velocity of 2.0 meters per second the error was \(+0.6510 \times 10^{-1}\); for 3.0 meters per second, the error was \(-0.3658 \times 10^{-1}\), until at 25.0 meters per second the error becomes \(-0.1213\).

These data are illustrated in Figure 3. Note that the curve is continuous throughout the selected range of \(c\).
Note also that the zero error phase velocity appears to lie somewhere between 2.0 and 3.0 meters per second. The data on Table 4 clearly supports this observation. That is, at 2.0 meters per second the error was $+0.6510 \times 10^{-1}$, and at 3.0 meters per second it was $-0.3658 \times 10^{-1}$. According to the intermediate value theorem, the function (error) must take on at least one zero value somewhere between 2.0 meters per second and 3.0 meters per second.

Table 5 shows the data tabulated for the values of the error (+ and -) near the vicinity of zero, at 0.2 meter per second increments. Figure 4 presents, in graphic form, the same information.

From Figure 4, it appears that the zero error phase velocity is about 2.5 meters per second. Table 5 shows an error of $+0.9968 \times 10^{-2}$ for a phase velocity of 2.4 meters per second, and an error of $-0.9001 \times 10^{-2}$ for a phase velocity of 2.6 meters per second. Thus again, applying the intermediate value theorem, the zero error phase velocity must lie somewhere between 2.4 and 2.6. The plot (Figure 4) reveals that it is 2.5 meters per second.

This same procedure for determining the zero error phase velocity (the true phase velocity) was carried out on all three averaged curves for each of 5 harmonics per curve. Without the aid of the digital computer, the computations involved in this procedure would have been nearly insurmountable.
C. True Phase Velocity, \((R/I)^2\), and \((R/I)\) Analysis

Table 6 shows a typical tabulation of the true phase velocity \((c)\) in meters per second and the corresponding \((R/I)^2\) and \((R/I)\) values (in percent). Again the selected range of the true phase velocity \((c)\) was 1.0 to 25.0 meters per second. This information is plotted in Figure 5.

Again reference is made to equation (B.12d) from which the table and the plot are derived. It can be seen that when \(c\) is equal to zero, the \((R/I)^2\) value is 1.00, or 100\% also \((R/I) = 1.00\), since \((R/I)\) is the square root of \((R/I)^2\). \((R/I)^2\) is again 1.00 when \(c\) is equal to infinity. What happens to the function between \(c = 1\) and \(c = 25\) is shown in Figure 5 and Table 6.

If equation (B.12d) is differentiated with respect to \(c\), and the resulting differential equation set to zero and solved for \(c\), then the value of \(c\) for the minimum value of \((R/I)^2\) can be obtained. When this operation is performed the value of \(c\) for which \((R/I)^2\) is minimal was \(\sqrt{B}\) \((B = a\ constant\ term\ from\ equation\ B.12d)\). This quantity appears to be of no physiological significance and will be discussed later.

Table 7 presents a summary of the more significant results obtained in the three experiments; the control, the norepinephrine, and the acetylcholine. It contains the values
for the true phase velocity, the apparent phase velocity, \((R/I)^2\) and \((R/I)\) as a function of the harmonics (1 to 5). Figures 6, 7, 8, 9, 10, 11, 12, and 13 are graphical illustrations of the same data.

Figure 6 shows both the true and apparent phase velocity, in meters per second, plotted against the harmonic (1 to 5), for the control pressure curves. There is noted a remarkable correlation between the two plots. Also, the values for the phase velocity for the fundamental harmonic are almost identical in the two cases (16.6 meters per second for the true phase velocity and 15.9 meters per second for the apparent phase velocity). This near identity between the two curves decreases for the second, third, and fourth harmonic, and by the fifth harmonic there is a divergent trend between the two curves.

There appears to be a non-linear decrease in the phase velocities as the harmonic or frequency increases. The values for the true phase velocity (harmonics 1 to 5) were 16.6, 5.4, 4.8, 4.2 and 6.0 meters per second. The corresponding values for the apparent phase velocity were 15.9, 2.2, 1.4, 1.1 and 1.2 meters per second. From Table 7, it should be noted that the foot-to-foot velocity was 1.96 meters per second.
Figure 7 shows relatively the same behavior for the norepinephrine pressure curve data. That is, the true phase velocity and apparent phase velocity for the first harmonic are very similar (22.9 and 22.0 meters per second, respectively) the values for the second, third, and fourth harmonics decrease gradually in a non-linear fashion, and the fifth harmonic values appear to diverge as in Figure 6 (the control curve data plot). Note that the value obtained for the foot-to-foot velocity was 3.30 meters per second.

Figure 8 is a similar plot for the acetylcholine pressure curve data. However, the behavior is quite different. Although the values for the first harmonic phase velocities are nearly equal (2.5 for the true phase velocity and 2.3 for the apparent phase velocity), the comparison beyond that point is quite different. The true phase velocity for the second harmonic is nearly the same as the first harmonic, namely 2.4 meters per second. The apparent phase velocity for the second harmonic was 1.4 meters per second. The third harmonic difference is even more marked. The value for the true phase velocity rose to 4.5 meters per second; it rose only to 1.8 meters per second for the apparent phase velocity. The true phase velocity for the fourth harmonic was 4.8 meters per second; the apparent phase velocity was 1.3 meters per second. For the fifth harmonic, both
velocity values decrease. The true phase velocity was 3.6 meters per second, and the apparent phase velocity was 0.9 meters per second.

In Figure 9, the apparent phase velocity is plotted against the harmonic for each of the three experimental conditions, (averaged control, averaged norepinephrine, and averaged acetylcholine). Note that for the fundamental harmonic, the norepinephrine phase velocity is greater than the control phase velocity, which is in turn greater than the acetylcholine phase velocity. Specifically, the norepinephrine apparent phase velocity for the first harmonic was 22.9 meters per second; for the control, the apparent phase velocity was 16.6 meters per second; and for acetylcholine it was 2.5 meters per second.

The second harmonic shows the same ordered arrangement of apparent phase velocities for the three experimental conditions mentioned, although the corresponding values for each are much smaller. The apparent phase velocity with norepinephrine was 7.4 meters per second; with the control, 5.4 meters per second; and with acetylcholine, 2.4 meters per second. The values for the apparent phase velocity for the third, fourth, and fifth harmonics were remarkably similar. The contribution of these higher harmonics to the total energy content of the system is generally insignificant and shall not be discussed at length here.
Figure 10 shows the same kind of plot for the true phase velocity as a function of the harmonics. The two plots appear to be almost identical, except that the true phase velocity curve values are a little higher than the apparent phase velocity values. Also, by the fifth harmonic, the two values appear to be diverging rather than converging as in Figure 9. The slightly higher values of the true phase velocity as compared to the apparent phase velocity will be discussed in the next section.

Figures 11, 12, and 13 show plots of the ratio of the reflected to the incident energy \((R/I)^2\) vs. the harmonic (1 to 5), for the control, norepinephrine, and acetylcholine pressure curve data. These plots represent the real significance and importance of these studies. Since it was necessary to obtain an accurate value for the true phase velocity before the energy relationships could be worked out, investigations and research on securing such velocity values by necessity, preceded obtaining the energy relationships.

Figure 11 and Table 7 show that for the first harmonic of the control, 34.09% of the energy originally imparted to the wave by the heart was reflected and was present in the wave at that recording position. For the second harmonic only 12.44% of the incident energy was reflected, for the third harmonic 2.64%, for the fourth harmonic 0.04%, and for the fifth harmonic 0.17%. These data unquestionably point
out that the reflected fundamental harmonic constitutes
the majority of reflected energy that appears in the wave.

Figure 12 shows the same plot for the norepinephrine
pressure curve data. 39.87% of the energy of the first
harmonic was reflected. 6.42% of the second harmonic energy
was reflected. The third harmonic shows that only 0.21%
of the energy was reflected, while for the fourth harmonic
1.33% was reflected. By the fifth harmonic only 0.35% of
the energy was reflected. These results bear out what was
expected, namely, that the effect of norepinephrine causes
greater reflections to occur.

Figure 13 illustrates a similar plot for the acetylcho-
line pressure curve data. This curve shows values of 1.21%
reflection for the first harmonic, 3.22% for the second,
5.96% for the third, 0.57% for the fourth, and 2.79% for
the fifth. It should be pointed out that in this case the
greatest amount of reflection occurs with the third harmonic,
whereas in the previous two plots (Figures 11 and 12), the
greatest amount of reflection occurred with the first harmonic.

Also, a comparison of the moduli of the first harmonic
for all three experimental conditions shows the control
reflected energy to be 34.09% while the corresponding values
for norepinephrine and acetylcholine were 39.87% and 1.21%
respectively. These values appear to be consistent with what
was expected according to present physiological theories.
With regard to all three plots (Figures 11, 12, and 13), it appears that at the higher harmonics, either, the biological transmission line is almost "matched", or the higher harmonics contributed very little to the total energy transfer of the system. An exception occurs with the acetylcholine pressure curve data plot (Figure 13), where it is seen that very little of the first harmonic is reflected. This observation strongly suggests that for the driving frequency of the acetylcholine pressure curve the arterial conductile channel impedance is "matched" by the terminal arteriolar impedance. A more complete discussion follows in the next section.
DISCUSSION

A. Transmission Line Characteristics of the Pressure Pulse

The general purpose of these studies has been to examine some of the physical factors involved in the transmission of the pulse-wave in the aorta. These factors include such things as the true phase velocity (detected and quantified in the presence of reflections) and the amount of reflected energy in the pulse-wave at any point along the aortic trunk. No attempt has been made to describe thoroughly all the physical factors involved in this mechanism. Rather, the majority of the time and effort was spent on developing a sound mathematical foundation upon which further and more sophisticated investigations might be built.

The method used herein takes advantage of the periodic nature of the cardiac ejections and arterial pressure fluctuations. The Fourier analysis technique for studying the harmonic content of periodic phenomena is particularly suited for this method because the mathematical equations which underlie these cardiovascular phenomena lend themselves readily to such mathematical treatment.

It has long been known that as the arterial pressure wave proceeds peripherally from the heart it encounters
many points of physical discontinuity at which part of the energy of the wave is reflected. This reflected energy component, traveling at the same velocity as its incident counterpart but in the opposite direction, fuses with the oncoming wave to give rise to a new, resultant wave.

If the retrograde, or reflected component, from the site of reflection is upright (and assuming the transmitted or incident component is upright), the reflection is said to occur at a phase of $0^\circ$ and the termination to be of a "closed" type. If the reflected component is inverted, the phase of reflection is said to be $180^\circ$ and the termination to be of an "open" type. If the wave does not return at all, the termination is said to be "matched" and no reflection occurs. The relationship between the terminal impedance and the amount of reflection is related to the reflection coefficient.

The reflection coefficient, $r$, is defined as follows,

$$
r = \frac{Z_T - Z_o}{Z_T + Z_o}.
$$

where $Z_o$ is the characteristic impedance of the conducting line and $Z_T$ is the impedance of the terminus or branches. By inspection it can be easily seen that when $Z_T = Z_o$, or when the terminal impedance of the line is equal to the characteristic impedance, $r = 0$. For any other value of $Z_T$ or $Z_o$, the value of $r$ will range between $+1$ and $-1$. 

For example, when the passage is completely occluded \( Z_T = \infty \), then \( r = +1 \). When the passage is completely open \( Z_T = 0 \), then \( r = -1 \).

The reflection coefficient may also be represented as follows,

\[
    r = \frac{A_3}{A_1}
\]

where \( A_1 \) is the amplitude of the advancing or incident wave, and \( A_3 \) the amplitude of the retrograde or reflected wave. This latter expression is the one used in these studies.

When the above expression \[ equation (16) \] is used to determine the reflection coefficient, it must be accompanied by a phase angle which determines whether \( r \) is positive or negative. If the phase angle of reflection is between \( 0^\circ \) and \( 180^\circ \), the sign on \( r \) is taken to be positive and reflection is said to occur in phase. If the phase angle of reflection is between \( 180^\circ \) and \( 360^\circ \), the sign on \( r \) is taken to be negative and reflection is said to occur out of phase. The addition of the reflected components to the incident components along the line is done vectorially, so that not only must the amplitudes of the individual components be considered, but also their respective phases.

If a wave is generated at regular intervals of time and propagated without attenuation along a transmission medium
such as an elastic tube filled with a fluid (the end of which is occluded), the incident and reflected components of the wave fuse together (vectorially) to form a new steady-state oscillatory pattern. At the terminus of the tube the generated (incident) wave is reflected in phase and of equal amplitude (since no attenuation takes place). The generated and reflected wave fuse to produce a new wave with an amplitude twice that of the original wave. At sites removed from the point of reflection the incident and reflected waves cancel each other. This cancelling effect produces new waves progressively smaller in amplitude. At a distance equal to one-quarter wave length of the original wave from the point of reflection no oscillations will be seen. The position within the steady-state oscillatory pattern at which the maximum oscillations occur is referred to as the antinode of the standing wave pattern, while the point at which no oscillations occur is referred to as the node. Along the entire length of the tube, there will appear nodes and antinodes, the number of which will be determined by the driving frequency and the length of the transmitting tube.

If, however, the terminus of the tube is open, the reflected wave will cancel the incident wave at the point of reflection so that at that point no oscillations will occur. At points removed from the site of reflection the incident and reflected waves progressively reinforce themselves.
At a distance equal to one-quarter wave length of the original wave from the point of reflection, maximum oscillation occurs. Thus it is seen that in the "open" type termination, nodes are present at the site of reflection and anti-nodes one-quarter wave length away, while in the "closed" type termination, anti-nodes are present at the site of reflection and nodes one-quarter wave length away.

Maxima and minima in the wave pattern may occur as a result of partial cancellation of the incident and reflected waves. The ratio between the maximum and minimum value of the pattern, because of attenuation, will change as one moves away from the point of reflection.

When reflections are being measured at any point along the arterial tree, the amount of reflections present is dependent on the nature of the arteriolar ending (either "open" or "closed") as well as the distance from the termination. In order to determine whether the termination in the arterial bed is of an "open" or "closed" type, it should be remembered that the pressure oscillations over a one-quarter wave length distance from the termination will increase toward a "closed" type ending and decrease toward an "open" type ending. Since there is less than one-quarter wave length of transmission line to work with for the fundamental harmonic in the dog, (approximately 1/8 wave length), one should not expect to find maxima and minima. However, for the higher harmonics,
it may be possible to detect maxima and minima in the wave pattern, since the minimum length of line required to detect such changes is considerably less than that required for the fundamental.

B. Analysis of Approach

The Fourier representation of a periodic function of time, distance, or both by an infinite number of sinusoidal waves is ideally suited to the cardiovascular pressure pulse. The pressure pulse satisfies each one of the Dirichlet conditions mentioned in Appendix A, with the possible exception of the variation in the period length. This variation is so slight that it is often ignored. If, however, one chooses not to ignore it, steps may be taken to normalize the pulse period to a given spatial length and then average the various times corresponding to the periods of the curves being normalized to obtain an average period for the number of curves being considered. In this way, one obtains a non-variant period in space and time which indeed satisfies the one questionable Dirichlet condition.

In the cardiovascular system, energy is propagated periodically as a pressure wave and as the movement of fluid. Since these phenomena are periodic, they lend themselves readily to the Fourier method of representation. The result of such a representation is a number of moduli or magnitudes which are generally referred to as the "harmonic content" of
The Fourier Series is not the only way to describe and represent a periodic function. Other methods include the exponential representation and the polynomial representation. The exponential and polynomial methods of representing periodic functions have not been considered in these studies. Rather, the Fourier Series method was selected due to the ease of visualization, handling, and representation of the periodic cardiovascular phenomena.

Regardless of what is physically inferred from the Fourier representation, the method is not based on any existing physical concept. It is entirely mathematical in nature and was designed to be used as a tool in solving a particular group of physical problems. A Fourier analysis cannot predict the degree of damping or attenuation of a propagated wave. It can only yield the harmonic content of a given wave or waves. Physical hypotheses must augment this analysis if such method is to be useful to the physicist as well as the physiologist.

In the studies undertaken here, Fourier analysis was used as a tool to derive an expression for the true phase velocity in the aorta in the presence of reflections. The method employed consists essentially of comparing changes in the harmonic magnitudes of two waves measured 5 centimeters apart in the aorta. On the basis of such comparisons, we propose physical theory to describe how the changes occur in
terms of precise measurable physiological quantities.

The equation is derived in Appendix B and shown in its final form in Figure B.6. Although the approximations and assumptions are mentioned in the derivation and in the body of the text, they are discussed here because they are possible points of criticism.

One of the first assumptions made in the derivation states that the channel under consideration is relatively cylindrical. Since the measurements in these studies are made within a 5 centimeter distance, any tapering of the vessels within that distance is small and may be ignored. The channel beyond the point of measurement is involved also: but, since reflections are assumed to occur, the tapering and branching effect contribute to the likelihood of reflections occurring and do not affect the assumption concerning the uniform cylindrical shape.

Also, the assumption that little energy is dissipated as heat within the 5 centimeter length appears equally reasonable. By the same reasoning, the remainder of the arterial channel may dissipate considerably more heat than the 5 centimeter segment. However, it is not considered appreciable or significant enough to consider here.

Two of the most critical approximations in the derivation involve the assumption of the linearity of A(x) and O(x). This
again is not too critical when one considers that within the 5 centimeter segment of a wave length of 1000 centimeters, $A(x)$ and $\theta(x)$ could be extremely non-linear and a linear approximation would still be good. However, for the higher frequencies (20th harmonic and higher), this approximation fails. In the light of the 5th harmonic limit in the analysis, this again should not impair the validity of the derivation.

Another serious approximation is the assumption that $I \cong I'$ and $R \cong R'$. That is, within the 5 centimeter segment, the energy (incident or reflected) entering at either of the two ends of the measurement segment is approximately equal to the energy leaving at the other end. This again does not appear to be too gross an approximation. Steps are now being taken to solve these equations, taking into account possible minute energy losses between recording points. A preliminary inspection shows that the results will hardly be distinguishable from the approximate results now being used.

The velocity of the incident wave is assumed to be the same as the velocity of the reflected wave. Several investigators have expressed doubts about this assumption and believe that the viscous nature of the blood and the viscoelastic elements of the arterial wall are capable of altering the propagated and reflected phase velocity.

If this is truly the case (which appears doubtful at this time), then a new approach will be required to take into
account those non-linear elements. However, it appears that these non-linear elements may change the phase of the incident and reflected wave and thereby make it appear that there is a change in velocity. Until such time as it is clearly shown that the velocities do change, it will be assumed that they are equal and traveling in opposite directions. It is hoped that the method of analysis utilized here, with linear approximations, will be first thoroughly understood before any attempt is made to analyze and describe the cardiovascular system in terms of non-linear elements. These considerations do not invalidate the work presented here, but merely establishes the basis for some criticism of it.

The one factor that the equations used here do not take into account is the attenuation or propagation constant of the wave function. As mentioned earlier, work is now being done to include this parameter which is certainly required for a complete physical description of the system. For these present studies, this parameter is assumed to be non-critical.

All other approximations made throughout the derivations and text are well established and consistent with procedures common to the mathematical and physical sciences.

The expression \[ P(x,t) = \sum_{m=1}^{\infty} I_n \sin \frac{w_n(t-x)}{c_n} + R_n \sin \frac{w_n(t + x + \theta_n)}{c_n} \] (B.1) relating the observed pressure to the incident and reflected
components, is taken from classical transmission line theory. The analogy of voltage to pressure is made and it is hoped that in the future the analogy of current to flow can be included. With the equations as they now exist, the latter relationship is unnecessary.

Little needs to be mentioned concerning the instrumentation and animal preparation other than perhaps a few comments on each. The limiting factor in any harmonic analysis is not the fidelity of the equipment used to reproduce the pulses, for these have been engineered and designed to fulfill the precise needs of the investigator. For example, modern pressure transducers with low volume displacement are at least an order of magnitude superior in quality to the recording transducers used several decades ago. Linear strain-gage amplifiers are now available to fulfill any band-width requirement. The 20 cycle per second requirement needed in these studies is an extremely modest request in the light of the major technological advancements.

The metal dual-lumened catheter offers another possible point of criticism. It may be said that the presence of such an instrument in the aortic blood stream could distort the wave forms and interfere with the normal flow patterns. This is, of course, true. The object of the study was not to measure the absolute value of the parameters, but rather to
measure and ascertain the magnitude of their change when certain procedures were performed. In other words, all parameters were measured with respect to an admitted artificial control situation.

It can be shown mathematically that the averaging of all the curves before the Fourier analysis is done is exactly equivalent to averaging the curves after the analysis is done. Although in these studies the averaging was done by hand, it is a relatively simple matter to include this step as part of the computer program. This data reduction technique reduces the amount of time, money, and effort by a factor of about 20.

The computer program was originally set up to be run on the IBM 704 Digital Computer and was written in 704 FORTRAN language. The problem was later programmed on the IBM 1620 Digital Computer in 1620 FORTRAN language. Although there are significant differences in the two languages, the results turned out to be exactly alike, thus eliminating the possibility of a machine error (an idea which often escapes computer users).

Although the program may seem needlessly lengthy, in that it prints out all the quantities for each c value selected (in these studies, over 10,000 lines of data were printed), it is much more accurate than having the computer select the zero error value of a c and printing out the one line of data.
The reason for this apparent paradox will become clear in the next section.

The program is designed to take any number of curves, to analyze for as many harmonics as is desired, to choose any starting and ending point for c, and to choose the increment by which the true phase velocity is increased each time it goes through a computation cycle. Such an arrangement is highly desirable, for if such changes are required and no provision has been made for them, it becomes necessary to compile a whole new program to include them. The program in its final form is general, yet flexible enough to handle the majority of problems that may arise in connection with this study as well as many related ones.
C. Analysis of Results

1. Determination of c. Attention is now directed to the error function curves and tabulations (Figures 3 and 4, and Tables 4 and 5). There can be no dispute as to where the curves cross the x-axis and the error is zero. Values less than 1 meter per second were not used in the figures shown because the error curve began to oscillate above and below the zero line. Furthermore, as the values of c approach zero, the frequency of oscillation approaches infinity. It was for this reason that the computer was not instructed to seek out a single zero value, else it would find just one for a low value of c and then proceed to the next curve. The chances are that for this particular c, the error curve was still oscillating. At the time this program was being compiled, it was judged that the best procedure would be to have the assumed velocities run through the entire range of possible values. It is now known that if one started at the high-valued end of the c range no difficulties would have been encountered. This was not known at the time the problem was being run. Furthermore, it could not have been possible to know this until detailed analysis, such as presented here, was available.

The variations of \((R/I)^2\) and \((R/I)\) with phase velocity (an example of which is shown in Figure 5 and Table 6) were one
of the first products of this study. Since it appeared that
two values of \( c \) could exist for any \((R/I)^2\) or \((R/I)\) value,
with the exception of the minimum point on the curve (for
which only one value of each exists), it was decided that
there must be a unique value of \( c \) to satisfy the incident
and reflected energy relationship. All attempts to link
the minimum value to such a conclusion proved futile.
Therefore, steps were taken to find another relationship
completely independent of the minimum value. Such steps led
to these studies and the results reported herein.

Again, it is stressed that the actual values of the
\((R/I)\) curve mean little. Since the phase of reflection is not
known, the curve could be either in the first or fourth
quadrants (I is, by convention, taken as being positive).

2. Phase and group velocities. The true phase velo-
cities obtained in these studies are higher than either the
apparent phase velocities or the group velocities. The group
velocities will be essentially discarded in this discussion
because of an inherent error in measuring these velocities:
usually one point is picked as a reference from which to measure
the velocity without regard to the harmonics that are carrying
the most energy. The difference between the true and apparent
phase velocities may well reflect the error of apparent phase
velocity due to reflections. One important aspect of this
difference measurement needs to be pointed out, however.
For the fundamental harmonic in each set of Fourier
analysis data, there is a phase difference between the upstream and downstream wave of about $2^\circ$. (the only exception is in the acetylcholine curves, where the difference is much greater). Since there is about the same order of magnitude of phase shift in the pressure transducers, the values for the velocities obtained for these harmonics are not as accurate as one would desire.

It has often been stated that increased reflections cause an increase in apparent phase velocity \((36,42)\). This is no necessarily true. It was seen in the first part of the discussion section that an increase in the magnitude of reflection may increase or decrease the magnitude of the resultant observed harmonic depending on the phase. Changes in the phase of the harmonic, and therefore changes in the apparent phase velocity, are brought about by changes in the phase at which the reflection occurs. The site at which the measurement is made with respect to the site of reflection is also important. Very probably what happens in questionable cases is that the terminal arterioles either constrict or dilate, causing a change in both the amplitude and phase of reflection. It is therefore not uncommon to find reports of negative phase velocities \((11)\), which could very easily occur with the proper change in phase of reflection.
Again, it is not too surprising to find the true phase velocity increasing and decreasing with norepinephrine and acetylcholine, respectively. These drugs change the extensibility (distensibility) of the arterial wall, which in the case of norepinephrine makes the artery "stiffer," and therefore causes the wave to move faster. In the case of acetylcholine, relaxation of the tension on the artery is effected, causing the wave to move slower. These results are consistent with those obtained by other investigators who have reported on this subject (11,30,37).

3. Reflection coefficients. The \((R/I)\) and \((R/I)^2\) values listed in this paper do not reflect, as percentages, the absolute value of the amount of reflected energy and pressure in the wave. Rather, they represent the ratio between the amount of energy which has been reflected to that amount which was originally imparted to the wave by the heart.

For example, the first harmonic of the controls shows a true phase velocity of 16.6 meters per second and a \((R/I)\) value of 58.39\% (Table 7). By making a few simple calculations (using equations (B.10b) and (B.11b) in Appendix B), it can be shown that the reflected pressure component is equivalent to 8.095 mm. Hg., the incident pressure component is equivalent to 13.86 mm. Hg., and the ratio of the two is 0.5839, or \((R/I)\). The total pressure for that harmonic
is about 16.34 mm. Hg. This means, of course, that the addition of the reflected and incident waves (8.09 and 13.86) does not yield their true sum because they were not in phase. Note that \((R/I)\) does not give the pressure that has been reflected, nor does it give the amount of the reflected energy. In other words, the percentages do not indicate the amount of the total harmonic that is reflected energy or pressure. The only information these percentages indicate is the relationship between the amount of energy and pressure incident to the point of measurement and the amount of that same energy that has been reflected. In order to determine the contribution of the reflected components to the total recorded pressure, additional calculations are needed. This is being planned as a separate study in the near future.

In spite of this lack of additional information it is clear from Table 7 that more of the incident energy is reflected with norepinephrine than in either the control or with acetylcholine. This again is not surprising, but it illustrates one important point. That is, since norepinephrine is predominantly a vasoconstrictor and since there is an increase in the relative amount of reflections in this case, the reflections must be originating from a "closed" type termination. The closing down of the arterioles apparently
causes a greater "mismatch" of the line, causing more reflections to be directed back along the line. It is interesting to compare these results with the control, which appears to have about the same values. This comparison seems to indicate that a "closed" type termination is predominant in the control or steady-state situation.

The acetylcholine data present somewhat of a different picture. Apparently, vasodilation, or an opening of the terminus, brings about a better "match" of the line which produces reflections. This evidence again indicates that a "closed" type termination is predominant in the steady-state situation.

D. Comparative Findings

In the past, emphasis has been placed on trying to relate the pulse-wave velocity to the arterial elasticity. The foot-to-foot velocity has been found to increase as it travels peripherally. This has been taken as evidence of an increase in the modulus of elasticity of the arteries as one proceeds peripherally from the heart (18). If, instead, phase velocities are used, they are found to be greatly affected by the reflections (35). If the effects of the changing wall characteristics and reflections are to be distinguished, the true phase velocity in the absence of reflections must be known.
The foot-to-foot or group velocity method of measurement is the simplest and has been standard for years. However, this method introduces gross inaccuracies that render it unreliable as an index of the wave velocity. Occasionally, a short distance along an artery is used to make the measurement. In this case, distortion errors are not too important and may be neglected. But, as the wave is markedly distorted as it travels toward the periphery, the identity of any single feature on the curve as having correspondence cannot be taken as being obvious. Velocities calculated from curves taken a long distance apart are in considerable error. Frank (13), Wiggers (54), Bramwell and Hill (5) and Hale (15) all made group velocity studies. Our work explains some of the discrepancies noted and adds to the knowledge gained in this earlier work.

It appears physically unsound to expect a single point on a pair of waves to correspond when the individual harmonics of each wave are undergoing untold changes. It is firmly felt that until the mechanism of wave travel is thoroughly understood, one could do little more than identify one curve as being related to another.

In 1957 Landowne (31) conducted a series of experiments in which he tapped the artery externally with a solenoid-operated metal plunger and measured the transmission time of a spike (10 m. seconds duration) superimposed on the pressure
pulse. He obtained values for the pulse-wave velocity of 10 meters per second at 30 mm. Hg. and 30 meters per second at 150 mm. Hg. Thus, Landowne's values are close to those we have observed for true velocity. It is interesting that in Landowne's experiments, the spike traveled faster than the pulse wave. This spike may be assumed free of reflections.

Another index used for measuring the rate of wave travel is the apparent phase velocity. Unlike the foot-to-foot or group velocity, it is based on the rate of change of the phase of the individual harmonics. The expression is given below as

$$ A.P.V. = \left[ \frac{S}{\left( \frac{\theta_n^3 - \theta_n^1}{360} \right) T} \right] $$

where $A.P.V.$ = the apparent phase velocity, $S$ = the distance between the two points of measurement, $T$ = the period, in seconds, of the pulse cycle, $\theta_n^3$ = the phase of the $n$th harmonic of the wave measured at a point $x_3$ along the tube, and $\theta_n^1$ = the phase of the $n$th harmonic of the wave measured at a distance $x_1$ along the tube ($x_3$ further from the heart than $x_1$).

It can be seen that when the phase difference is small the apparent phase velocity is large. When the phase difference is large (i.e., approaches $360^\circ$), the apparent phase
velocity approaches the constant velocity value S/T. Therefore, the apparent phase velocity appears to be very much dependent on the resultant phase of the harmonic components of the wave. A slight change in the phase of reflection may change the apparent phase velocity considerably.

Porje (42) was probably the first to study, in detail, phase velocities of the harmonic components of the pulse wave. He found the apparent phase velocity of the fundamental harmonic to be between 3.3 and 56.2 meters per second, while the second and third harmonic values ranged between 4 and 13 meters per second. The apparent phase velocity for hypertensive individuals was found to be between 10.0 and 32.0 meters per second for the first harmonic, 6.7 and 47.3 for the second harmonic, and 6.0 and 45.5 meters per second for the third harmonic.

Taylor (46,47) using a rubber tube model and a sinusoidal pump as a forcing element with a driving frequency of 10 cps, obtained a phase velocity of 14.1 meters per second. McDonald and Taylor (35) found the apparent phase velocity of the fundamental harmonic, in the dog, to be 3.8 meters per second at the root of the aorta and about 15.0 meters per second in the abdominal aorta. In another study, they found the apparent phase velocity for the first five harmonics to be 17.0, 6.0, 6.0, 5.0, and 5.5, respectively. In still another study, the
corresponding harmonic phase velocities were shown to be 10.0, 5.5, 5.0, 10.0, and 7.0 meters per second.

Farrow and Stacy (11) showed the control values for the apparent phase velocity for the first five harmonics to be 15.24, 6.77, 3.0, 2.77, and 2.28 meters per second. For norepinephrine treated dogs, the first five harmonics showed the apparent phase velocities to be -36.41, 69.85, 5.08, 3.88, and 2.82 meters per second. For acetylcholine treated animals, the corresponding values were 148.35, 11.49, 4.13, 4.02, and 5.00 meters per second. It is doubtful that the fundamental phase velocities of norepinephrine and acetylcholine injected animals are real, since this large deviation can be explained by the shift in the phase of the resultant wave due to the change in peripheral resistance.

The values for the apparent phase velocity obtained in these studies appear to be in close agreement with those obtained by most other investigators who have made similar measurement (11,35,42,46,47). Again, it should be stressed that the apparent phase velocity is not the true conduction velocity of the wave. Rather, it is the velocity at which the wave appears to be traveling, based on the phase of the harmonic components of the wave. Its detection is altered and complicated by reflections. Its experimental value lies in its usefulness in detecting changes in the phase of reflection, and possibly the site of reflection.
Several investigators have reported reflection coefficients based on the harmonic moduli of waves measured at several points along the aorta. Most generally a comparison is made of the ratio of the upstream modulus with the downstream one. Such comparisons are not considered to be physically sound, since the modulus of either the upstream harmonic or the downstream one contains both an incident and reflected component. Further, since the reflection coefficient is the ratio of the reflected component to the incident component of each harmonic, it is first necessary to resolve each component, as such, from the measured resultant wave before the ratio is calculated. This, of course, was not done in such comparisons and therefore the values reported are not considered physically sound.

Womersley (59) on the basis of a relationship he worked out between the reflection coefficient and the ratio of the area of the parent tube and its bifurcations, predicted a reflection coefficient of 14% at the terminal division of the aorta and about 7% at the coeliac axis.

Taylor (46,47) by deriving a relationship between the apparent phase velocities and the reflection coefficients, calculated the reflection coefficient in the femoral vascular bed of the dog to be about 50%. In order to arrive at a definite value, it was necessary to estimate the point of reflection from his impedance measurements.
McDonald and Taylor (36) calculated the reflection coefficient from an approximation of the terminal impedance based on a relationship worked out from the relationship of the input impedance as a function of frequency. It was shown (36,37) that the normal reflection coefficient was about 12%. Vasodilation produced by acetylcholine gave a reflection coefficient of 6%. Vasoconstruction produced by norepinephrine showed a reflection coefficient of 55%.

Although McDonald and Taylor (36) show results in close agreement with what these studies bear out, Karreman (22), predicted the reflections to be about 5% at an aortic bifurcation and therefore too small to detect. His presentation was essentially a theoretical one in which he derived expressions for the reflection coefficient of pressure waves in vessels which have branches.

E. Physiological Significance of the Findings

When reflections occur in any system, a waste of useful energy ensues. The reflected energy in the arterial system becomes unavailable to propel the blood through the capillaries. Rather, it is directed in the retrograde direction and dissipated as heat. Therefore, the amount of work that the heart is required to perform to maintain the normal body needs is in excess of that amount actually required if reflections were absent.
When small amounts of reflection occur, energy transfer to the periphery is nearly optimal and the efficiency of transfer is greatest. To a cardiac patient who is interested in obtaining all the efficiency possible, it would be vital to know under what conditions the heart could do the least amount of work to obtain the greatest amount of efficiency of energy transfer. The key to the solution of this problem is to know what the impedance of the line is and how to "match" it. The key to the impedance determining problem is obtaining a true phase velocity.

For many years now, investigators and clinicians have been using the arterial elasticity as an index to the state of the arterial wall. Arteriosclerosis, for example, increases the thickness and stiffness of the wall material, which produces an increase in the pulse-wave velocity. Therefore, by noting abnormally high values for the pulse-wave velocity, it was possible to relate them to an increase in the elastic modulus of the wall, and hence sclerotic arteries. However, since no satisfactory values have been available for the pulse-wave velocity, this relationship and the conclusions drawn from it have been somewhat questionable. With the values for the true phase velocity obtained in these studies, the elastic modulus might be calculated point for
point along the aorta *in vivo*. This information coupled with the *in vitro* studies would help complete the understanding of the cardiovascular system and aid in the practice of its principles.

There is every reason to believe that the Moens equation can be solved for arterial elasticity, and the results can be accurate if the velocity value used is accurate. We believe that inaccuracies encountered to date were due to velocity errors caused by reflections, and that it may now be possible to obtain accurate arterial elasticity values.

In addition to the use of the digital computer in this study, it is well worthwhile to consider other possible applications. With regard to some of the work planned in the future (specifically, the relationships between the AC and steady-state pressures and flow), much could be done with a digital computer. The useful output of such a study could well be a method for the continuous registration of the cardiac output.

The list of applications as a result of such studies could perhaps be endless. One thing must be kept in mind, however, and that is that these studies and investigations are going to involve vast amounts of computations and processing of data, and the most efficient way to do such processing is with the digital computers. For the on-line uses, smaller computers, which are relatively inexpensive, might be used. Such small computers are now being designed and will soon be within the reach of the average research grant. Regardless
of the acquisition of such a device, one thing is certain. The proposer of such a study is going to need unlimited access to a small, general purpose digital computer. Until such arrangements are made no such sophisticated and detailed study can proceed effectively.

F. Future Experimental Plans

Throughout the body of this text, it has been repeatedly mentioned that certain phases of this study, now incomplete, are planned for the future. Because of the great amount of time required to prepare the data before a computer can operate on it and decipher the computer output into something meaningful, it was impossible to complete all the work necessary for a thorough study. Therefore, the investigation plan was divided into steps.

The first of these steps was designed to seek a value of the true phase velocity and the reflected energy relationships associated with it. This, of course, has been completed and is, in fact, the thesis of the work presented here. The second of these steps was designed to measure the true phase velocity along the entire length of the aorta, and by mechanically occluding and unoccluding the arterial termination, to study the changes, if any, in reflected energy content of the wave and the wave velocity. This step, too, has been
completed, but to include it as part of this dissertation would make this writing much too voluminous.

In the third step of this study it is planned to calculate the phases of the reflected wave. The phases are necessary to determine the sign of the reflection coefficients. Once the phases are obtained, then all the important parameters of the biological transmission line will have been obtained. From these parameters (reflected and incident pressure, phase velocity, and phase of reflection), it would be possible to obtain the true impedance values of the arterial tree. With the acquisition of the impedance values, a true pictorial view of the behavioral characteristics of the arterial system would be available.

Next, it is proposed to test the entire system of equation used in these studies. Without all the above information, it is impossible to test the system thoroughly. For the test phase of the investigation, a whole new system of equations has been developed in which the phase velocities, reflected energies, and reflected phases are examined directly. No iteration process is needed. The instrumentation required in this plan includes elaborate pressure transducers, multi-lumened catheters which eliminate the 2° phase shift limitation of the present ones, and a digital computer.
Once the hypothesis has been confirmed, it is planned to launch a program in which the digital computer may be used on-line to evaluate all the necessary parameters continuously and in real time as the experiment is taking place.

This evaluation, coupled with AC flow theory, should yield a system of ideas, concepts, and theories designed to describe completely the behavior and mechanisms of the arterial system as a transmission line.
SUMMARY

A study of the transmission line characteristics of the arterial system in the dog has been made to determine the true harmonic phase velocities, the reflected energy relationships, and the reflection coefficients. These studies are based on Fourier analysis of two simultaneously measured pressure pulses in the aorta at a distance of 5 centimeters apart. These studies were made on pressure pulses obtained from dogs in a control state, and after given norepinephrine and acetylcholine, intravenously.

A set of mathematical expressions was derived which made possible the calculation of reflection coefficients and a "true velocity." This true velocity was uncomplicated by phase shifts due to the presence of reflections.

The data from the pressure curves obtained from the animals that were given norepinephrine intravenously show an increase in the true phase velocity, reflected energy, and reflection coefficient in comparison to the controls. The data from the acetylcholine curves show a decrease in the same corresponding quantities.

A comparison has been made between the true phase velocity, apparent phase velocity, and the foot-to-foot (group) velocity in the same animal. Since the apparent phase velocity is reflection phase dependent and the group velocity is
correspondent point dependent, it was judged that these two quantities are less accurate than the true phase velocity reported.

In view of the behavior of the resultant harmonic pressure in the artery to vasoconstrictor and vasodilator drugs, it is apparent that the arterial bed has a termination of the "closed" type.

The discussion of the projected continuation of these studies in relation to the clinician and clinical sciences indicate that the use of a small general purpose digital computer would be immeasurably valuable.
CONCLUSIONS

The derivation of the equations used in these studies, appears to be consistent with the classical theory which describes them. There are, however, some physical factors which these equations do not take into account. These factors include the attenuation of the wave and the precise way in which the amplitudes and phases vary with distance from the site of reflection. It is felt that these factors would contribute significantly to the accuracy of the results, but very little to their form. One of the prime objectives here was to establish a sound mathematical form on which to build a more sophisticated research program. On this basis, the exclusion of these factors was deemed justified.

These studies confirm previous observations that peripherally recorded pulses are richer in harmonic content than the simultaneously recorded central ones. This effect has been attributed to the presence of additional reflected energy in peripheral pulse by virtue of its proximity to the site of reflection.

The data from the animals that were given norepinephrine intravenously show a greater amount of reflected energy in the pulse wave, which as been attributed to the vasoconstrictor effect causing a greater "mismatch" of the line. Conversely,
the administration of acetylcholine produces a lesser amount of reflected energy in the wave, which has been attributed to the vasodilator effect producing a better "match" of the line. Normal terminal impedance is considered higher than line impedance.

No definite conclusions can be made about the exact nature of the reflection coefficients, since the phases of reflection have yet to be determined. However, it may be deduced that since the resultant harmonic pressure appears to increase toward the periphery and the site of reflection, a "closed" type of termination prevails, in which case the sign on the reflection coefficient would be positive. There has been no evidence that the termination is any type other than the "closed" one.

Norepinephrine appears to reduce the caliber of the arterioles and thereby increase terminal impedance slightly more than it is in the control state. This suggests that under the control conditions the arterioles are nearly completely closed down. The effect of acetylcholine, on the other hand, suggests that the terminus is being dilated to a considerable extent, almost optimally (note not maximally). This makes for greater efficiency of energy transfer in the peripheral vascular bed.
The true phase velocities, although different from the apparent phase velocities and group velocities, approach values that were expected.

Although these studies have been considered a second approximation to the understanding of the transmission of pressure energy within the artery, it is more correctly a first approximation, in view of the planned research yet to be done. If any of the suggested clinical applications are to ever come about, such investigations must take place.
Figure 2
Typical records of aortic and differential pulses

Average controls

Absolute pressure (mm Hg)

Differential pressure \((X-5)\) (mm Hg)

Time

Foot-to-foot velocity = 196 meters/sec.

Apparent phase velocity = 15.9 meters/sec.

True phase velocity = 16.6 meters/sec.

Period \((T)\) = 0.4163 sec.

Frequency \((F)\) = 2.3359 sec\(^{-1}\)

Angular frequency \((\omega)\) = 15.9334 sec\(^{-1}\)
FIRST HARMONIC

ERROR FUNCTION CURVE
ACETYLCHOLINE-FIRST HARMONIC
EXTENDED RANGE

CROSSOVER POINT
(C = 2.5 M/SEC)

ERROR FUNCTION CURVE
ACETYLCHOLINE-FIRST HARMONIC
LIMITED RANGE

FIGURE 3
ERROR FUNCTION CURVE
ACETYLCHOLINE-FIRST HARMONIC
EXTENDED RANGE

FIGURE 4
ERROR FUNCTION CURVE
ACETYLCHOLINE-FIRST HARMONIC
LIMITED RANGE

VELOCITY - METERS/SECOND

ERROR

ERROR x 10^-1
FIGURE 5
VARIATION OF \((R/I)^2\) AND \((R/I)\)
OVER FULL RANGE OF \(c\) USED
FIRST HARMONIC—CONTROL
FIGURE 6
TRUE AND APPARENT PHASE VELOCITY
VARIATION FOR CONTROL, 5 HARMONICS

FIGURE 7
TRUE AND APPARENT PHASE VELOCITY
VARIATION FOR NOREPINEPHRINE
5 HARMONICS

FIGURE 8
TRUE AND APPARENT PHASE VELOCITY
VARIATION FOR ACETYLCHOLINE
5 HARMONICS
FIGURE 9
APARENT PHASE VELOCITY VARIATION FOR NOREPINEPHRINE, ACETYLCHELINE AND CONTROL, FOR 5 HARMONICS.
- NOREPINEPHRINE
- CONTROL
- ACETYLCHELINE

FIGURE 10
TRUE PHASE VELOCITY VARIATION FOR NOREPINEPHRINE, ACETYLCHELINE AND CONTROL, FOR 5 HARMONICS.
- NOREPINEPHRINE
- CONTROL
- ACETYLCHELINE

VELOCITY-METERS/SECOND
HARMONIC

0 2 4 6 8 10 12 14 16 18 20 22 24

0 2 4 6 8 10 12 14 16 18 20 22 24

0 2 4 6 8 10 12 14 16 18 20 22 24

2 3 4 5

2 3 4 5
Figure 11: Reflected energy for control 5 harmonics.

Figure 12: Reflected energy for norepinephrine 5 harmonics.

Figure 13: Reflected energy for acetylcholine 5 harmonics.

\begin{align*}
\left( R_1 \right)^2 & \quad \text{(Figure 11)} \\
\left( R_1 \right)^2 & \quad \text{(Figure 12)} \\
\left( R_1 \right)^2 & \quad \text{(Figure 13)}
\end{align*}
TABLE 1

Fourier Analysis Data-Average Control

<table>
<thead>
<tr>
<th>Harmonic</th>
<th>Sine Coeff.</th>
<th>Cos Coeff.</th>
<th>Modulus</th>
<th>Phase (degrees)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>14.594</td>
<td>-4.489</td>
<td>15.269</td>
<td>312.90</td>
</tr>
<tr>
<td>2</td>
<td>3.996</td>
<td>-6.255</td>
<td>7.423</td>
<td>302.57</td>
</tr>
<tr>
<td>3</td>
<td>0.101</td>
<td>-2.303</td>
<td>2.305</td>
<td>272.52</td>
</tr>
<tr>
<td>4</td>
<td>0.815</td>
<td>-0.790</td>
<td>1.135</td>
<td>315.90</td>
</tr>
<tr>
<td>5</td>
<td>0.772</td>
<td>-0.695</td>
<td>1.862</td>
<td>294.50</td>
</tr>
</tbody>
</table>

Differential Pressure

Mean = 3.922 mm.Hg.

<table>
<thead>
<tr>
<th>Harmonic</th>
<th>Sine Coeff.</th>
<th>Cos Coeff.</th>
<th>Modulus</th>
<th>Phase (degrees)</th>
</tr>
</thead>
<tbody>
<tr>
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<td>1.092</td>
<td>1.336</td>
<td>125.13</td>
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<tr>
<td>2</td>
<td>2.092</td>
<td>2.727</td>
<td>3.436</td>
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<tr>
<td>3</td>
<td>1.427</td>
<td>0.028</td>
<td>1.427</td>
<td>1.13</td>
</tr>
<tr>
<td>4</td>
<td>0.761</td>
<td>0.310</td>
<td>0.822</td>
<td>22.18</td>
</tr>
<tr>
<td>5</td>
<td>1.145</td>
<td>0.040</td>
<td>1.146</td>
<td>1.99</td>
</tr>
</tbody>
</table>

Absolute Pressure (downstream)

Mean = 131.487 mm.Hg.

<table>
<thead>
<tr>
<th>Harmonic</th>
<th>Sine Coeff.</th>
<th>Cos Coeff.</th>
<th>Modulus</th>
<th>Phase (degrees)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>15.363</td>
<td>-5.581</td>
<td>16.345</td>
<td>340.04</td>
</tr>
<tr>
<td>2</td>
<td>1.905</td>
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<td>9.181</td>
<td>281.97</td>
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<tr>
<td>3</td>
<td>-1.326</td>
<td>-2.331</td>
<td>2.682</td>
<td>240.38</td>
</tr>
<tr>
<td>4</td>
<td>0.053</td>
<td>-1.100</td>
<td>1.101</td>
<td>272.78</td>
</tr>
<tr>
<td>5</td>
<td>-0.373</td>
<td>-1.734</td>
<td>1.774</td>
<td>257.87</td>
</tr>
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</table>
TABLE 2.

Fourier Analysis Data-Average Norepinephrine

<table>
<thead>
<tr>
<th>Harmonic</th>
<th>Sine Coeff.</th>
<th>Cos Coeff.</th>
<th>Modulus</th>
<th>Phase (degrees)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>23.623</td>
<td>-3.492</td>
<td>23.879</td>
<td>351.59</td>
</tr>
<tr>
<td>2</td>
<td>8.544</td>
<td>-8.650</td>
<td>12.158</td>
<td>314.65</td>
</tr>
<tr>
<td>3</td>
<td>1.796</td>
<td>-5.584</td>
<td>5.866</td>
<td>287.83</td>
</tr>
<tr>
<td>4</td>
<td>0.878</td>
<td>-2.469</td>
<td>2.620</td>
<td>289.58</td>
</tr>
<tr>
<td>5</td>
<td>1.387</td>
<td>-2.362</td>
<td>2.739</td>
<td>300.42</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Harmonic</th>
<th>Sine Coeff.</th>
<th>Cos Coeff.</th>
<th>Modulus</th>
<th>Phase (degrees)</th>
</tr>
</thead>
<tbody>
<tr>
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<td>1.212</td>
<td>1.797</td>
<td>137.56</td>
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<tr>
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<td>1.688</td>
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<tr>
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<td>2.517</td>
<td>0.461</td>
<td>2.559</td>
<td>10.39</td>
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<td>1.244</td>
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<td>1.256</td>
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<td>1.129</td>
<td>0.227</td>
<td>1.152</td>
<td>11.39</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Harmonic</th>
<th>Sine Coeff.</th>
<th>Cos Coeff.</th>
<th>Modulus</th>
<th>Phase (degrees)</th>
</tr>
</thead>
<tbody>
<tr>
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<td>25.388</td>
<td>349.32</td>
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<td>2</td>
<td>6.856</td>
<td>-11.861</td>
<td>13.700</td>
<td>300.03</td>
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<td>-0.720</td>
<td>-6.046</td>
<td>6.089</td>
<td>263.21</td>
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<tr>
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<td>0.257</td>
<td>-2.590</td>
<td>2.602</td>
<td>275.68</td>
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</table>
TABLE 3

Fourier Analysis Data-Average Acetylcholine

<table>
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<tr>
<th>Harmonic</th>
<th>Sine Coeff.</th>
<th>Cos Coeff.</th>
<th>Modulus</th>
<th>Phase (degrees)</th>
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<tbody>
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<td>314.58</td>
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<td>0.089</td>
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<td>1.718</td>
<td>272.97</td>
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</table>

Differential Pressure

Mean = 3.263 mm.Hg.

<table>
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<th>Harmonic</th>
<th>Sine Coeff.</th>
<th>Cos Coeff.</th>
<th>Modulus</th>
<th>Phase (degrees)</th>
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<tbody>
<tr>
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<td>4.993</td>
<td>74.39</td>
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<td>0.040</td>
<td>1.540</td>
<td>1.49</td>
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<tr>
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<td>5</td>
<td>1.076</td>
<td>-0.857</td>
<td>1.375</td>
<td>321.45</td>
</tr>
</tbody>
</table>

Absolute Pressure (downstream)

Mean = 68.015 mm.Hg.

<table>
<thead>
<tr>
<th>Harmonic</th>
<th>Sine Coeff.</th>
<th>Cos Coeff.</th>
<th>Modulus</th>
<th>Phase (degrees)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>11.652</td>
<td>-8.721</td>
<td>14.554</td>
<td>323.19</td>
</tr>
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<td>2</td>
<td>0.987</td>
<td>-4.520</td>
<td>4.627</td>
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<td>3</td>
<td>0.776</td>
<td>-2.390</td>
<td>2.513</td>
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</tr>
<tr>
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<td>-2.702</td>
<td>2.853</td>
<td>251.26</td>
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<td>5</td>
<td>-0.987</td>
<td>-0.858</td>
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<td>221.03</td>
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</tbody>
</table>
TABLE 4

Comparison of $(R/I)^2$, $(R/I)$, and the Error (E) with the True Phase Velocity (c)
Average Acetycholine - First Harmonic

<table>
<thead>
<tr>
<th>c (m/sec)</th>
<th>$(R/I)^2$ (%)</th>
<th>$(R/I)$ (%)</th>
<th>Error (E)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.00</td>
<td>17.03</td>
<td>41.26</td>
<td>+0.5119</td>
</tr>
<tr>
<td>2.00</td>
<td>16.64</td>
<td>12.81</td>
<td>+0.6510x10^-1</td>
</tr>
<tr>
<td>3.00</td>
<td>2.60</td>
<td>16.14</td>
<td>-0.3658x10^-1</td>
</tr>
<tr>
<td>4.00</td>
<td>7.96</td>
<td>28.21</td>
<td>-0.7369x10^-1</td>
</tr>
<tr>
<td>5.00</td>
<td>14.24</td>
<td>37.74</td>
<td>-0.9114x10^-1</td>
</tr>
<tr>
<td>6.00</td>
<td>20.35</td>
<td>45.12</td>
<td>-0.1007</td>
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<tr>
<td>7.00</td>
<td>25.97</td>
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<td>-0.1065</td>
</tr>
<tr>
<td>8.00</td>
<td>31.01</td>
<td>55.69</td>
<td>-0.1102</td>
</tr>
<tr>
<td>9.00</td>
<td>35.51</td>
<td>59.59</td>
<td>-0.1128</td>
</tr>
<tr>
<td>10.00</td>
<td>39.52</td>
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<td>-0.1147</td>
</tr>
<tr>
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<td>43.09</td>
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<td>-0.1160</td>
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<td>71.96</td>
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</tr>
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TABLE 5

Comparison of \((R/I)^2\), \((R/I)\), and the Error \((E)\) with the True Phase Velocity \((c)\)
Average Acetylcholine - First Harmonic

<table>
<thead>
<tr>
<th>(c) (m/sec)</th>
<th>((R/I)^2) (%)</th>
<th>((R/I)) (%)</th>
<th>Error ((E))</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.00</td>
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</tr>
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<td>7.30</td>
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<td>4.53</td>
<td>21.27</td>
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<tr>
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<td>-0.7369x10^-1</td>
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<tr>
<td>c (m/sec)</td>
<td>(R/I)^2 (%)</td>
<td>(R/I) (%)</td>
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<td>-------------</td>
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## TABLE 7
Results of True Phase Velocity Analysis

<table>
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<tr>
<th>Curve Ident.</th>
<th>Harmonic</th>
<th>True Phase Velocity (m/sec)</th>
<th>A.P.V. (m/sec)</th>
<th>(R/I)$^2$ (%)</th>
<th>(R/I) (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Controls</td>
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<td>35.27</td>
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<td>foot-to-foot</td>
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<td>4.8</td>
<td>1.4</td>
<td>2.67</td>
<td>16.34</td>
</tr>
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<td>1.2</td>
<td>0.17</td>
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</tr>
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<td>Noradrenaline</td>
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<td>22.9</td>
<td>22.0</td>
<td>39.87</td>
<td>63.15</td>
</tr>
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<td>foot-to-foot</td>
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<td>7.4</td>
<td>3.4</td>
<td>6.42</td>
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</tr>
<tr>
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<td>6.2</td>
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<td>meters/second</td>
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<td>1.21</td>
<td>10.94</td>
</tr>
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<td>2.1</td>
<td>1.4</td>
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<td>4.5</td>
<td>1.8</td>
<td>5.96</td>
<td>24.42</td>
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<td>meters/second</td>
<td>4</td>
<td>4.8</td>
<td>1.3</td>
<td>0.57</td>
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<td>3.6</td>
<td>0.9</td>
<td>2.79</td>
<td>16.71</td>
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</table>
APPENDIX A

GRAPHICAL EVALUATION OF THE FOURIER COEFFICIENTS BY NUMERICAL INTEGRATION
The pulse pressure fluctuations are functions which repeat in form at regular intervals and which therefore are called periodic functions. Alternating currents, mechanical oscillations, pendular motion, and transverse vibrations of an elastic string are also examples of periodic phenomena.

The simplest type of periodic function is a sinusoid of the form,

\[ y = y_m \sin \theta. \]  \hspace{1cm} (A.1)

However, many periodic phenomena, such as pulse pressure changes, cannot be assumed to be this simple in form. Such phenomena are handled conveniently by means of a Fourier Series. It can be shown that periodic functions can, with certain restrictions\(^1\), be expressed by an infinite trigonometric series of the form

\[ f(\theta) = A_0 + (A_1 \cos \theta + B_1 \sin \theta) + (A_2 \cos 2\theta + B_2 \sin 2\theta), \]

\[ + \hspace{1cm} \ldots \ldots \ldots \ldots \]

This is called the Fourier Series and A and B are called the Fourier Coefficients or the modulus of the wave.

---

\(^{1}\)These restrictions are (1) the function must be finite everywhere, (2) the function must have, at most, a finite number of discontinuities in one period, and (3) the function must have a finite number of maxima and minima in one period. These restrictions are called the Dirichlet conditions.
The series can be written more compactly as

$$f(\theta) = A_0 + \sum_{n=1}^{\infty} (A_n \cos n\theta + B_n \sin n\theta), \quad (A.3)$$

or in an equivalent form,

$$f(\theta) = A_0 + \sum_{n=1}^{\infty} C_n \cos (n\theta - \phi_n), \quad (A.4)$$

where

$$C_n = \sqrt{\frac{A_n^2 + B_n^2}{\tan \phi_n = B_n/A_n}}, \quad (A.5)$$

and

The term $A_0$ represents the average value of the function over one period. The term $C_1 \cos (\theta - \phi_1)$ is called the fundamental wave or the first harmonic and has a period of the original function. The $n$th term, $C_n \cos (n\theta - \phi_n)$, is called the $n$th harmonic or the harmonic of order $n$, and has a frequency $n$ times as great as the fundamental wave.

$\theta$ is the angle of a periodic function of time and can be represented by using $\theta = \omega t$, where $\omega = 2\pi$ times the fundamental frequency.

The Fourier expansion is an infinite series, but in practice the higher harmonics are ordinarily of such small magnitude that they can be neglected without serious error. The expansion then reduces to a trigonometric polynomial with comparatively few terms.
To obtain the expressions for the Fourier coefficient $A_n$, multiply equation (A.3) by $\cos m\theta d\theta$, and integrate over the interval 0 to $2\pi$. This yields

$$A_n = 2 \left[ \frac{1}{2\pi} \int_0^{2\pi} f(\theta) \cos n\theta d\theta \right]. \quad (A.7)$$

To obtain the Fourier coefficient $B_n$, multiply equation (A.3) by $\sin m\theta d\theta$, and integrate over the interval 0 to $2\pi$. This yields

$$B_n = 2 \left[ \frac{1}{2\pi} \int_0^{2\pi} f(\theta) \sin n\theta d\theta \right]. \quad (A.8)$$

$A_0$ is merely the average of the value of the function over one period and is represented as

$$A_0 = \frac{1}{2\pi} \int_0^{2\pi} f(\theta) d\theta. \quad (A.9)$$

From a practical viewpoint, equations (A.7), (A.8), and (A.9) are changed to a more useful form for a graphical evaluation of the Fourier coefficients by numerical integration.

This method consists of using finite increments of instead of $d\theta$. The period $2\pi$ ($360^\circ$) is divided into $m$ equal increments of $\Delta \theta$ degrees so that $360^\circ = m \Delta \theta$. The value of $y$ or $f(\theta)$ used for each increment is taken at the intersection of the ordinate and the curve at the time interval $\Delta \theta$ as $y_1, y_2, y_3, \ldots, y_k, \ldots, y_m$. 
Then,

\[ A_0 = \frac{1}{2\pi} \int_0^{2\pi} y \, d\theta \approx \frac{1}{2\pi} \sum_{k=1}^{k=m} y_k \Delta \theta_k, \quad (A.10) \]

or

\[ A_0 \approx \frac{1}{m} \sum_{k=1}^{k=m} y_k, \quad (A.11) \]

remembering that \( 2\pi = m \Delta \theta_k \). Thus, the average value of the wave is determined by adding the values of the ordinates of each increment and dividing by the number of increments.

In evaluating \( A_n \) and \( B_n \), the terms \((y \sin n\theta) \Delta \theta \) and \((y \cos n\theta) \Delta \theta \) also represent areas under the curve. The \( y \) or \( f(\theta) \) must be the ordinate value and \( \theta \) must be the angle at that point. Then,

\[ A_n = 2 \left[ \frac{1}{2\pi} \int_0^{2\pi} y \cos n\theta \, d\theta \right] \approx 2 \left[ \frac{1}{2\pi} \sum_{k=1}^{k=m} y_k \cos n\theta_k \Delta \theta_k \right], \quad (A.12) \]

or,

\[ A_n \approx \frac{2}{m} \sum_{k=1}^{k=m} y_k \cos n\theta_k, \quad (A.13) \]

and,

\[ B_n = 2 \left[ \frac{1}{2\pi} \int_0^{2\pi} y \sin n\theta \, d\theta \right] \approx 2 \left[ \frac{1}{2\pi} \sum_{k=1}^{k=m} y_k \sin n\theta_k \Delta \theta_k \right], \quad (A.14) \]

or,

\[ B_n \approx \frac{2}{m} \sum_{k=1}^{k=m} y_k \sin n\theta_k. \quad (A.15) \]
As the number of increments \( m \) is increased, the accuracy of \( A_0, A_n, \) and \( B_n \) improves, but the amount of work involved increases. Hence, judgment must be used in selecting \( m \). A rule-of-thumb used in this selection is that the number of ordinates used should be at least twice the highest harmonic of interest. Originally, it was planned to analyze for as many as ten harmonics, so that a 24-ordinate scheme was more than adequate. Therefore, in the above equations, \( m = 24 \), for these studies.
APPENDIX B

DERIVATION OF AN ARTERIAL WAVE EQUATION FOR THE DETERMINATION OF THE TRUE HARMONIC PHASE VELOCITY IN THE PRESENCE OF REFLECTED COMPONENTS.
Assume that the arterial system can be represented by a cylindrical elastic tube as follows,

\[ P \xrightarrow{\text{FIGURE B.1}} \]

\[ \text{DIAGRAM OF AN ARTERIAL SEGMENT} \]

and that a disturbance \( P \) is propagated along this tube from left to right (i.e., the positive \( x \) direction). Further assume that there is little attenuation of this disturbance during its propagation and that the conversion of stored energy into heat is slight. That is, the conversion of stored energy into heat over a 5 centimeter length of the channel is insignificant.

If this disturbance is repetitive, unattenuated, continuous, and periodic, and it represents the pressure in the channel, the pressure may be represented as a Fourier Series in the following manner,

\[ P(x,t) = \sum_{n=1}^{\infty} I_n \sin \frac{w_n(t - x)}{c_n} + R_n \sin \frac{w_n(t + x + \Theta_n)}{w_n}, (B.1) \]

where,

- \( P(x,t) \equiv \text{Total pressure as a function of distance and time.} \)
- \( n \equiv \text{Order of the harmonic (i.e., fundamental, 2nd, 3rd, and so on).} \)
- \( I_n \equiv \text{Modulus of an incident harmonic component } n. \)
For simplicity, equation (B.1) is stated in the following form:

\[ P_T = I \sin \left( \frac{w(t - x)}{c} \right) + R \sin \left( \frac{w(t + x + \theta)}{c} \right), \]  

(B.1a)

where \( P_T \) is the total pressure in the channel and the subscripts of the various \( n \) values are understood when referring to a specific harmonic.

Differentiating equation (B.1a) with respect to time, one obtains,

\[ \frac{\partial P_T}{\partial t} = Iw \cos \left( \frac{4(t - x)}{c} \right) + Rw \cos \left( \frac{4(t + x + \theta)}{c} \right). \]  

(B.2)

Differentiating equation (B.1a) with respect to distance, one obtains,

\[ \frac{\partial P_T}{\partial x} = -Iw \cos \left( \frac{4(t - x)}{c} \right) + Rw \cos \left( \frac{4(t + x + \theta)}{c} \right). \]  

(B.3)

Multiplying equation (B.3) by \( c \),

\[ c \frac{\partial P_T}{\partial x} = -Iw \cos \left( \frac{4(t - x)}{c} \right) + Rw \cos \left( \frac{4(t + x + \theta)}{c} \right). \]  

(B.3a)
By adding equation (B.3a) to equation (B.2), one obtains,

$$\frac{\partial P_t}{\partial t} + c \frac{\partial P_t}{\partial x} = 2Rw \cos \omega \left( t + \frac{x}{c} + \theta \right) \quad (B.4)$$

By subtracting equation (B.3a) from equation (B.2), one obtains

$$\frac{\partial P_t}{\partial t} - c \frac{\partial P_t}{\partial x} = 2Rw \cos \omega (t - \frac{x}{c}) \quad (B.5)$$

By examining equations (B.4) and (B.5), it is obvious that by simply adding or subtracting the space derivative times the harmonic velocity to or from the time derivative, there are obtained two equations which uniquely define $I$ and $R$, or, the modulus of the reflected harmonic component and the modulus of the incident harmonic component, respectively.

The foregoing, of course, is purely a theoretical consideration. To be of practical use to the hemodynamical research investigator, it would be necessary to relate these relationships to those phenomena which are observed in the system being studied; in this case, the arterial system.

From the experimental measurements, one obtains a complex periodic, repetitive, and fairly continuous waveform that represents the fluctuation of the arterial pressure as a function of time at two points along the arterial trunk. If these waveforms are subjected to a Fourier analysis, information is obtained relating the amplitudes and the phases of the various harmonic components that constitute these complex
waveforms. Thus, for each wave, one obtains an amplitude and a phase for each harmonic constituent. In short, for a single harmonic, there is an $A_1$ (modulus or amplitude) measured at point $x_1$ (distance along the longitudinal axis), at a phase of $\theta_1$, and an $A_3$ measured at point $x_3$, at a phase of $\theta_3$ (see Figure B.2).

![Figure B.2](image)

**ARTERIAL SEGMENT**

With this in mind, the following approximations are used,

$$P_T = A(x) \sin (\omega t + \Theta(x)), \quad (B.6)$$

where:

$$P_T = \text{Total pressure (same as equation (B.1a)).}$$

$$A(x) \equiv A_1 + \frac{A_3 - A_1}{\Delta x} \cdot x. \quad (B.6a)$$

$$\Theta(x) \equiv \theta_1 + \frac{\Theta_3 - \Theta_1}{\Delta x} \cdot x. \quad (B.6b)$$

$A_1, A_3, \theta_1, \text{ and } \Theta_3$ are defined as stated above. $\Delta x = x_3 - x_1$, or the distance between the two recording sites.

$A(x)$ and $\Theta(x)$ are assumed to progress in a linear fashion as they move along the arterial channel. This assumption appears to be reasonably valid when $\Delta x \ll \lambda$ (wavelength of the wave $= \lambda$). Even if $A(x)$ and $\Theta(x)$ were sinusoidal in
nature, the linear approximation would hold true.

By substituting equations (B.6a) and (B.6b) into equation (B.6),

\[ P_T = \left[ A_1 + \frac{A_3 - A_1}{\Delta x} \right] \sin \left[ \omega t + \Theta_1 + \frac{\Theta_3 - \Theta_1}{\Delta x} \right] \] (B.6c)

If we set \( x_1 = 0 \), and differentiate equation (B.6c) with respect to time at \( x_1 \),

\[ \frac{\partial P_T}{\partial t} = A_1 \omega \cos (\omega t + \Theta_1) \] (B.7)

Differentiating equation (B.6c) with respect to distance is not so simple. However, from the Chain Rule, the following expression is used to approximate the space differential,

\[ \frac{\partial P_T}{\partial x} = \frac{\partial P_T}{\partial A(x)} \cdot \frac{\partial A(x)}{\partial x} + \frac{\partial P_T}{\partial \Theta(x)} \cdot \frac{\partial \Theta(x)}{\partial x} \] (B.8)

Evaluating each of these terms in equation (B.8) separately from equations (B.6) and (B.6c), at \( x = x_1 = 0 \),

\[ \frac{\partial P_T}{\partial A(x)} = 1 \sin (\omega t + \Theta_1) \] (B.8a)

\[ \frac{\partial P_T}{\partial \Theta(x)} = A_1 \cos (\omega t + \Theta_1) \] (B.8b)
and,

$$\frac{\partial A(x)}{\partial x} = \frac{A_3 - A_1}{\Delta x} \quad (B.8c)$$

and,

$$\frac{\partial \Theta(x)}{\partial x} = \frac{\Theta_3 - \Theta_1}{\Delta x} \quad (B.8d)$$

substituting equations (B.8a), (B.8b), (B.8c), and (B.8d) into equation (8),

$$\frac{\partial P_T}{\partial x} = \frac{A_3 - A_1}{\Delta x} \sin(wt + \Theta_1) + A_1 \left( \frac{\Theta_3 - \Theta_1}{\Delta x} \right) \cos(wt + \Theta_1) \quad (B.9)$$

since,

$$\sin(wt + \Theta_1) = \cos\left( wt + \Theta_1 - \frac{\pi}{2} \right)$$

equation (B.9) is restated as follows,

$$\frac{\partial P_T}{\partial x} = \frac{A_3 - A_1}{\Delta x} \cos(wt + \Theta_1 - \frac{\pi}{2}) + A_1 \left( \frac{\Theta_3 - \Theta_1}{\Delta x} \right) \cos(wt + \Theta_1) \quad (B.9a)$$

equations (B.4), (B.5) and (B.7) are rewritten here for simplicity of the discussion to follow.

$$\frac{\partial P_T}{\partial t} + c \frac{\partial P_T}{\partial x} = 2Rw \cos \omega \left( t + \frac{x}{c} + \Theta \right) \quad (B.4)$$
By examining equations (B.9a) and (B.7), it is seen that the precise quantities necessary to satisfy the left-hand members of equations (B.4) and (B.5) are available. In short, the correlation between the theoretical consideration and the observed experimental phenomena has been found.

By substituting equations (B.7) and (B.9a) into the left-hand member of equation (B.4), the following is obtained:

$$\frac{\partial P_t}{\partial t} - c \frac{\partial P_t}{\partial x} = 2\Omega \cos \Omega (t - \frac{x}{c})$$

(B.5)

$$\frac{\partial P_t}{\partial t} = A_1 \Omega \cos (\Omega t + \Theta_1)$$

(B.7)

$$\frac{\partial P_t}{\partial t} + c \frac{\partial P_t}{\partial x} = A_1 \Omega \cos (\Omega t + \Theta_1) + c A_1 (\Theta_2 - \Theta_1) \cos (\Omega t + \Theta_1)$$

$$+ c \left( \frac{A_2 - A_1}{A_1} \right) \cos (\Omega t + \Theta_1, -\frac{\pi}{2}) = 2\Omega \omega \cos \Omega \left( t + \frac{x}{c} + \frac{\Theta}{\Omega} \right)$$

(B.4a)
Rewriting and collecting terms,

\[ \left[ A_i \omega + c A_1 \left( \frac{\Theta_2 - \Theta_1}{\Delta x} \right) \right] \cos(\omega t + \Theta_1) + c \left( \frac{A_2 - A_1}{\Delta x} \right) \cos \left( \omega t + \Theta_1 - \frac{\pi}{2} \right) = \]

(B.4b)

\[ 2 R \omega \cos \omega \left( t + \frac{x}{c} + \frac{\Theta}{c} \right) \]

By the use of orthogonal addition, the following equality is derived,

\[ (2 R \omega)^2 = \left[ A_i \omega + c A_1 \left( \frac{\Theta_2 - \Theta_1}{\Delta x} \right) \right]^2 + c^2 \left[ \frac{A_2 - A_1}{\Delta x} \right]^2 \]  

(B.10)

Similarly, by substituting equation (B.7) and (B.9a) into the left-hand member of equation (B.5), the following is obtained,

\[ \frac{\partial P_T}{\partial t} - c \frac{\partial P_T}{\partial x} = A_i \omega \cos(\omega t + \Theta_1) - c \left( \frac{A_2 - A_1}{\Delta x} \right) \cos \left( \omega t + \Theta_1 - \frac{\pi}{2} \right) \]

- c A_1 \left( \frac{\Theta_2 - \Theta_1}{\Delta x} \right) \cos(\omega t + \Theta_1) = 2 R \omega \cos \omega \left( t - \frac{x}{c} \right) \]

(B.5a)

Rewriting and collecting terms,

\[ \left[ A_i \omega - c A_1 \left( \frac{\Theta_2 - \Theta_1}{\Delta x} \right) \right] \cos(\omega t + \Theta_1) - c \left( \frac{A_2 - A_1}{\Delta x} \right) \cos \left( \omega t + \Theta_1 - \frac{\pi}{2} \right) = \]

\[ 2 R \omega \cos \omega \left( t - \frac{x}{c} \right) \]  

(B.5b)
From the orthogonal addition of equation (B.5b), the following is derived,

\[(2 \pm \omega)^2 = \left[ A_1 \omega - c A_1 \left( \frac{\theta_2 - \theta_1}{\Delta x} \right) \right]^2 + c^2 \left( \frac{A_3 - A_1}{\Delta x} \right)^2 \] (B.11)

From equation (E.4b) and (B.5b), the following vector diagram (Figure B.3) is drawn (N.B.: \( x = x_1 = 0 \)).

By definition,

\[\tan \alpha_1 = \frac{c \left( \frac{A_3 - A_1}{\Delta x} \right)}{A_1 \omega - c A_1 \left( \frac{\theta_2 - \theta_1}{\Delta x} \right)} = \frac{c \left( \frac{A_3}{A_1} - 1 \right)}{\omega - c \left( \frac{\theta_3 - \theta_1}{\Delta x} \right)} \] (B.12)
By inspection of Figure B.3, it appears that the $I_w$ vector is less in magnitude than the $R_w$ vector, which would imply physically that the reflected energy is greater than the incident energy, or, the source from which it was derived. But, by examining the denominator of equations (B.12) and (B.13), it is seen that there is a quantity $(\frac{A_3 - A_1}{\Delta x})$, which, when physically determined, is a negative quantity. This, therefore, reverses the lengths of the drawn vectors, $I_w$ and $R_w$, respectively. That is, $I_w$ will have the length of the drawn $R_w$, and vice versa, but, maintaining their same position in the given coordinate system with respect to each other.

The following conditions are stated for the quadrant placement of angles $\alpha_1$, and $\alpha_2$.

$\alpha_1$ is in the 1st quadrant, if,

1. $\omega - \frac{c}{\Delta x} (\Theta_3 - \Theta_1) > 0$

2. $\frac{c}{\Delta x} \left( \frac{A_3}{A_1} - 1 \right) > 0$
$\alpha_1$ is in the 2nd quadrant, if,

1. $\omega - \frac{c}{\Delta x} (\Theta_3 - \Theta_1) < 0$
2. $\frac{c}{\Delta x} \left( \frac{A_2}{A_1} - 1 \right) > 0$

$\alpha_2$ is in the 3rd quadrant, if,

1. $\omega - \frac{c}{\Delta x} (\Theta_3 - \Theta_1) > 0$
2. $\frac{c}{\Delta x} \left( \frac{A_2}{A_1} - 1 \right) < 0$

$\alpha_3$ is in the 4th quadrant, if,

1. $\omega - \frac{c}{\Delta x} (\Theta_3 - \Theta_1) > 0$
2. $\frac{c}{\Delta x} \left( \frac{A_2}{A_1} - 1 \right) < 0$

$\alpha_4$ is in the 1st quadrant, if,

1. $\omega + \frac{c}{\Delta x} (\Theta_3 - \Theta_1) > 0$
2. $\frac{c}{\Delta x} \left( \frac{A_2}{A_1} - 1 \right) > 0$

$\alpha_5$ is in the 2nd quadrant, if,

1. $\omega + \frac{c}{\Delta x} (\Theta_3 - \Theta_1) < 0$
2. $\frac{c}{\Delta x} \left( \frac{A_2}{A_1} - 1 \right) > 0$
\( a_2 \) is in the 3rd quadrant, if,

1. \( \omega + \frac{c}{\Delta x} (\Theta_3 - \Theta_1) < 0 \)
2. \( \frac{c}{\Delta x} \left( \frac{A_3}{A_1} - 1 \right) < 0 \)

\( a_2 \) is in the 4th quadrant, if,

1. \( \omega + \frac{c}{\Delta x} (\Theta_3 - \Theta_1) > 0 \)
2. \( \frac{c}{\Delta x} \left( \frac{A_3}{A_1} - 1 \right) < 0 \)

Expanding equation (B.10),

\[ 4\omega^2 R^2 = A_1^2 \omega^2 + \frac{2c A_1(\Theta_3 - \Theta_1) A_1 \omega + c^2 A_1^2 (\Theta_3 - \Theta_1)^2 + c^2 (A_3 - A_1)^2}{\Delta x^2} \]  

(B.10a)

rewriting and collecting terms,

\[ 4\omega^2 R^2 = c^2 \left[ \frac{A_1^2 (\Theta_3 - \Theta_1)^2 + (A_3 - A_1)^2}{\Delta x^2} \right] + \left[ \frac{2A_1^2 \omega (\Theta_3 - \Theta_1)}{\Delta x} \right] + A_1^2 \omega^2 \]  

(B.10b)

Expanding equation (B.11),

\[ 4\omega^2 I^2 = A_1^2 \omega^2 - 2c A_1^2 \omega (\Theta_3 - \Theta_1) + \frac{c^2 A_1^2 (\Theta_3 - \Theta_1)^2 + c^2 (A_3 - A_1)^2}{\Delta x^2} \]  

(B.11a)

rewriting and collecting terms,

\[ 4I^2 \omega^2 = c^2 \left[ \frac{A_1^2 (\Theta_3 - \Theta_1)^2 + (A_3 - A_1)^2}{\Delta x^2} \right] - \left[ \frac{2A_1^2 \omega (\Theta_3 - \Theta_1)}{\Delta x} \right] + A_1^2 \omega^2 \]  

(B.11b)
Dividing equation (B.10b) by equation (B.11b),

\[
\frac{4R^2w^2}{4I^2w^2} = \frac{c^2 \left( \frac{A_1^2(\theta_3-\theta_1)^2}{\Delta x^2} + \frac{(A_3-A_1)^2}{\Delta x^2} \right) + \frac{2A_1^2w(\theta_3-\theta_1)}{\Delta x} + A_1w^2}{c^2 \left( \frac{A_1^2(\theta_3-\theta_1)^2}{\Delta x^2} + \frac{(A_3-A_1)^2}{\Delta x^2} \right) - \frac{2A_1^2w(\theta_3-\theta_1)}{\Delta x} + A_1w^2} \tag{B.12}
\]

Dividing both numerator and denominator of equation (B.12) by \(A_1^2\),

\[
\frac{4R^2w^2}{4I^2w^2} = \frac{c^2 \left( \frac{(\theta_3-\theta_1)^2}{\Delta x^2} + \frac{(A_3/A_1-1)^2}{\Delta x^2} \right) + \frac{2w(\theta_3-\theta_1)}{\Delta x} + w^2}{c^2 \left( \frac{(\theta_3-\theta_1)^2}{\Delta x^2} + \frac{(A_3/A_1-1)^2}{\Delta x^2} \right) - \frac{2w(\theta_3-\theta_1)}{\Delta x} + w^2} \tag{B.12a}
\]

Now by dividing equation (B.12a), both numerator and denominator, of the right-hand member, by the coefficient of the \(c^2\) term, and cancel (i.e., divide by

\[
\left[ \frac{(\theta_3-\theta_1)^2}{\Delta x^2} + \frac{(A_3/A_1-1)^2}{\Delta x^2} \right]
\]
Equation (B.12b) is rewritten in the following simplified form,

\[
\left( \frac{R}{I} \right)^2 = \frac{c^2 + c \left[ \frac{2\omega (\theta_3 - \theta_1) \Delta x}{(\theta_3 - \theta_1)^2 + \left( \frac{A_2}{A_1} - 1 \right)^2} \right] + \left[ \frac{\Delta x^2 \omega^2}{(\theta_3 - \theta_1)^2 + \left( \frac{A_2}{A_1} - 1 \right)^2} \right]}{c^2 - c \left[ \frac{2\omega (\theta_3 - \theta_1) \Delta x}{(\theta_3 - \theta_1)^2 + \left( \frac{A_2}{A_1} - 1 \right)^2} \right] + \left[ \frac{\Delta x^2 \omega^2}{(\theta_3 - \theta_1)^2 + \left( \frac{A_2}{A_1} - 1 \right)^2} \right]}
\]  

(B.12b)

where:

\[
A = \frac{2\omega \Delta x (\theta_3 - \theta_1)}{(\theta_3 - \theta_1)^2 + \left( \frac{A_2}{A_1} - 1 \right)^2}
\]  

(B.13)

\[
B = \frac{\Delta x^2 \omega^2}{(\theta_3 - \theta_1)^2 + \left( \frac{A_2}{A_1} - 1 \right)^2}
\]  

(B.14)

Examination of equation (B.12c), shows that \((R/I)^2\) is greater than 1 for all positive values of parameters A and B. However, B is always positive, since all its terms are squared.
C, by definition, is the time rate of change of distance in the positive x direction, and A can either be positive or negative depending on the value of \((\theta_3 - \theta_1)\). Physical measurements show that \((\theta_3 - \theta_1) < 0\), or is negative, which makes A negative. This term then changes the appearance of equation (B.12c) to be

\[
\left(\frac{R}{I}\right)^2 = \frac{c^2 - Ac + B}{c^2 + Ac + B}
\]  

(B.12d)

in which case, the ratio to the left is less than one, as, if it were greater than one, it would be meaningless.

Thus, there is obtained an expression for the ratio of the reflected energy to the incident energy as a function of \(\omega\).

A plot of this function appears as follows,

\[\left(\frac{R}{I}\right)^2\]

where the minimum occurs at the value of \(\pm \sqrt{B}\).
So far, the attempt to express the total pressure as the sum of an infinite number of Fourier expressions in terms of an incident modulus and a reflected modulus, and relate them to the measured amplitude and phase of the observed phenomena, has been successful.

It would be useful, indeed, to be able to relate the quantities which are dependent on the value $c$ to some theoretical measurable quantity. So far a theoretical $(R/I)^2$ is related to $c$; but $c$ is not capable of being measured in the presence of reflections. This function, as was shown above (Figure B.4), gives an infinite number of values of $(R/I)^2$ which is satisfied by a corresponding infinite number of values of $c$. This type of relationship is far from being unique and is therefore, essentially useless, as such.

It was thought that there must be a unique value of $c$ which satisfies the $(R/I)^2$ ratio at a given measurement point along the arterial channel.

Consider the statement proceeding equation (B.7). It is stated that the derivative with respect to time is taken at $x_1$. If the time and space derivative were taken at $x_3$, another system of equations can be derived relating $A_3$ to an $I$ and an $R$, and another vector diagram can be drawn similar to Figure B.5.
Consider Figure B.5, where both vector diagrams are plotted in the same plane.

\[ \phi_{\text{interior}} = 180 - (d_{LM}) \]

\[ \beta \]

is the angle obtained by rotating vector 2Iw to 2Iw' and 2Rw to 2Rw'. Numerically \( \beta = \frac{\omega \Delta x}{C} \) in radians. The vector parallelograms are drawn the same way as Figure B.3, only now more complete.
Consider $\phi$ interior, for the parallelogram of sides $2Iw$ and $2Rw$, and the triangle whose legs are $2Iw$, $2Rw$, and $2A_1w$,

$$\phi_{\text{interior}} = 180 - (\alpha_1 + \alpha_2) = 180 - \alpha_1 - \alpha_2$$

Using the Law of Cosines, for triangle $2Iw$, $2Rw$, and $2A_1w$,

$$(2A_1w)^2 = (2Iw)^2 + (2Rw)^2 - 2(2Iw)(2Rw)\cos(\phi_{\text{interior}}) \quad (B.15)$$

For triangle $2Iw$, $2Rw$, and $2A_2w$,

$$(2A_2w)^2 = (2Iw)^2 + (2Rw)^2 - 2(2Iw)(2Rw)\cos(\phi_{\text{interior}} + 2\beta) \quad (B.16)$$

If $I$ and $I'$, and, $R$ and $R'$ do not change significantly in the short distance $\Delta x$, then, it may be said that $I \approx I'$ and, $R \approx R'$, in which case equation (B.16) may be written as follows

$$(2A_2w)^2 = (2Iw)^2 + (2Rw)^2 - 2(2Iw)(2Rw)\cos(\phi_{\text{interior}} + 2\beta) \quad (B.16a)$$

Dividing equation (B.16a) by equation (B.15), and cancelling,

$$\left(\frac{A_2}{A_1}\right)^2 = \frac{4w^2I^2 + 4w^2R^2 - 8w^2RI \cos(\phi_{\text{interior}} + 2\beta)}{4w^2I^2 + 4w^2R^2 - 8w^2RI \cos(\phi_{\text{interior}})} \quad (B.17)$$
Substituting the value $\phi_{\text{interior}}$ into equation (B.17),

$$\left(\frac{A_3}{A_1}\right) = \frac{4w^2I^2 + 4w^2R^2 - 8w^2RI \cos(180 - \phi_1 - \phi_2 + 2\beta)}{4w^2I^2 + 4w^2R^2 - 8w^2RI \cos(180 - \phi_1 - \phi_2)} \quad (B.17a)$$

Dividing both numerator and denominator of the right-hand member of equation (B.17a) by $4w^2$,

$$\left(\frac{A_3}{A_1}\right) = \frac{I^2 + R^2 - 2RI \cos(180 - \phi_1 - \phi_2 + 2\beta)}{I^2 + R^2 - 2RI \cos(180 - \phi_1 - \phi_2)} \quad (B.17b)$$

Dividing again, both numerator and denominator of the right-hand member of equation (B.17b) by $I^2$,

$$\left(\frac{A_3}{A_1}\right) = \frac{1 + \left(\frac{R}{I}\right)^2 - 2\left(\frac{R}{I}\right) \cos(180 - \phi_1 - \phi_2 + 2\beta)}{1 + \left(\frac{R}{I}\right)^2 - 2\left(\frac{R}{I}\right) \cos(180 - \phi_1 - \phi_2)} \quad (B.17c)$$

Expanding the cosine argument, equation (B.17c) becomes,

$$\left(\frac{A_3}{A_1}\right) = \frac{1 + \left(\frac{R}{I}\right)^2 + 2\left(\frac{R}{I}\right) \cos(\phi_1 + \phi_2 - 2\beta)}{1 + \left(\frac{R}{I}\right)^2 + 2\left(\frac{R}{I}\right) \cos(\phi_1 + \phi_2)} \quad (B.17d)$$

By definition (see Figure B.3),

$$\phi_3 = \phi_1 + \phi_2 \quad (B.18)$$
Substituting equation (B.18) into equation (B.17d),

$$\left( \frac{A_3}{A_1} \right)_T^2 = \frac{1 + (\frac{R}{I})^2 + 2\left( \frac{R}{I} \right) \cos (\alpha - 2\beta)}{1 + (\frac{R}{I})^2 + 2\left( \frac{R}{I} \right) \cos (\alpha)}$$  \hspace{1cm} (B.17e)

$(A_3/A_1)_T^2$ is a theoretical value that could be obtained regardless of the value of c used.

Thus, it is seen that for a given c, one can calculate a ratio $(A_3/A_1)_T^2$. If this value corresponds to the experimentally measured value, then the value of c used is unique and satisfies the necessary conditions of the systems of equations.

If the chosen c does not correspond to the value necessary to satisfy equation (B.17e), the value of $(A_3/A_1)_T^2$ calculated will be different from the one measured.

Therefore, all that is necessary is to find a c, which, when used in equation (B.17e), produces a value of $(A_3/A_1)_T^2$ that corresponds to the $(A_3/A_1)_T^2$ measured. Mathematically, iterative techniques are necessary to perform this task.

Rewriting equation (B.17e) into a form in which the terms of the initial assumptions are recognized, appears as follows (in Figure B.6), as equation (B.17f).
FIGURE B.6

EQUATION B.17f
\[
\begin{align*}
\left[ \begin{array}{c}
\frac{(e^{-g} \gamma \lambda + m)}{3} \\
\frac{(1 - e^{-g} \gamma \lambda)}{3} \\
\frac{c}{x \lambda + 2}
\end{array} \right] & = \frac{1}{2} \cos \left[ \frac{1}{2} \left( \frac{1}{z} \frac{1}{x} \right) \left( \frac{1}{z} \frac{1}{x} \right) \left( \frac{1}{z} \frac{1}{x} \right) \right] \\
& + \frac{1}{2} \cos \left[ \frac{1}{2} \left( \frac{1}{z} \frac{1}{x} \right) \left( \frac{1}{z} \frac{1}{x} \right) \left( \frac{1}{z} \frac{1}{x} \right) \right] \\
& + \frac{1}{2} \cos \left[ \frac{1}{2} \left( \frac{1}{z} \frac{1}{x} \right) \left( \frac{1}{z} \frac{1}{x} \right) \left( \frac{1}{z} \frac{1}{x} \right) \right] \\
& \text{EQUATION (8.11)}
\end{align*}
\]
APPENDIX C

FLOW CHART AND EXPLANATORY NOTES FOR THE TRUE HARMONIC PHASE VELOCITY ANALYSIS FORTRAN COMPUTER PROGRAM
FIGURE C.1

FORTRAN FLOW DIAGRAM 1
FIGURE C.2

FORTRAN FLOW DIAGRAM 2
FIGURE C.3

FORTRAN FLOW DIAGRAM 3
PRINT I, T1, T2, ALPHA, BETA, C, REFL 2, REFL 3, A1, B1, APV

74

75 C < CF NO

76 YES C + FINCG -> C

77 GO TO 53

78 I < K NO

79 YES I+1 -> I

80 Go TO 48 Z+1 -> Z

81 L = LM

82 L + 1 -> L

83 Go TO 5

84 STOP
The following are the definitions and explanations, when necessary, of the symbols used in the foregoing FORTRAN flow diagram.

LM = Maximum number of curve pairs to be analyzed.

DELTAC = Increment by which the pulse-wave velocity \( c \) is increased during the iteration process.

K = Highest harmonic to be sought.

HPI = \( \pi/12 \).

MC = conditional switch (to be -1, 0, or +1).

ID = Curve Identification number.

D(I)'s, D(J)'s = ordinate values for each of the 24 ordinates used.

I, J = fixed point constants.

AO = Mean or average pressure.

Y, Z = Floating point constants.

A(I)'s = Fourier sine coefficients, downstream and differential pulse.

B(I)'s = Fourier cosine coefficients, downstream and differential pulse.

SINF = Sine

COSF = Cosine

SQRTF = Square root

GM(I)'s = vector sum of A(I)'s + B(I)'s, downstream and differential pulse.

ATANF = Arc tangent (in radians).
PHI(I)'s = phase angle, downstream and differential pulse.

ARCT = Arc tangent (in degrees).

AB(I)'s = Fourier sine coefficients, upstream pulse.

BB(I)'s = Fourier cosine coefficients, upstream pulse.

GMB(I)'s = Vector sum of AB(I)'s and BB(I)'s.

PHIB(I)'s = Phase angle, upstream pulse.

W = Angular frequency (in radians).

CI = Initial value for pulse-wave velocity calculations.

CF = Final value for pulse-wave velocity calculations.

DELTAX = Distance between catheter tips (ΔX).

A1 = Constant A

B1 = Constant B

PINCC = DELTAC

APV = Apparent Phase Velocity

REFL2 = (R/I)²

REPL = (R/I)

BETA = β

FN1 = \( \frac{C}{ΔX} \left( \frac{A_3}{A_1} - 1 \right) \)

D1 = W - \( \frac{C}{ΔX} (θ_3 - θ_1) \)

FN2 = FN1 = \( \frac{C}{ΔX} \left( \frac{A_3}{A_1} - 1 \right) \)

D2 = W + \( \frac{C}{ΔX} (θ_3 - θ_1) \)

T1 = \( a_1 \)

T2 = \( a_2 \)
FN, D, T = Temporary storage place for FN1, FN2, D1, D2, T1, and T2 while computations are going on.

\[ \alpha = \alpha_1 + \alpha_2 = \alpha \]

\[ T_3 = \left( \frac{A_3}{A_1} \right)^2 \text{ Theoretical} \]

\[ E = \text{Error} \]


38. Moens, A.I., Die Pulskurve Leiden, cited by D.A. McDonald and M.G. Taylor (36).


I, George Steve Malindzak, Jr., was born in Cleveland, Ohio, January 3, 1933. I received my secondary school education at the Centerville High School, in Centerville, Pennsylvania. I attended undergraduate school at Western Reserve University, where I was granted the Bachelor of Arts degree (cum laude) in Biology, in 1956. In the fall of 1957, I matriculated in the Graduate School at the Ohio State University and was appointed a Research Assistant in the Department of Physiology. From the Ohio State University, I received my Master of Science degree in 1958. My major subject was Physiology. In 1959, I received an appointment as a Research Associate, and Research Fellow in the Department of Physiology. In June, of 1959, I received a pre-doctoral research fellowship from the National Institutes of Health, and held that appointment for one year while completing the requirements for the Doctor of Philosophy degree.