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AORTIC IMPEDANCE PATTERNS
IN CLOSED CHEST DOGS

DISSERTATION

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

By
Michael Keith Cope, B. S.

* * * * *

The Ohio State University
1975

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VITA

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FIELD OF STUDY

Cardiovascular Physiology
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CHAPTER I
INTRODUCTION

Background

Classically, investigators examined the relationship between the heart's pumping action and the circulatory system's acceptance of blood flow by measuring or estimating the peripheral resistance. Peripheral resistance relates the steady pressure to the steady flow. The heart, however, pumps blood in a pulsatile manner and consideration must be given to this periodic input when examining the interaction between the heart and the vasculature. In 1955 McDonald (25) and Womersley (70) applied the concept of impedance to the analysis of heart-circulatory system interaction. The impedance concept relates pulsatile pressure to pulsatile flow.

Impedance is a complex number which consists of a real and an imaginary part. Impedance can be represented by a modulus, or the length of a vector originating from the origin of a coordinate system and a phase angle, which is the angle between the vector and the positive horizontal axis of the coordinate system. The imaginary part of the impedance is found by multiplying the modulus by the sine of the phase angle. The real part of the impedance is obtained by multiplying the modulus by the cosine of the phase angle; this real part has been referred to as the in-phase impedance by O'Rourke and Taylor (48). The in-phase impedance relates the portions of the flow and pressure pulses that are simultaneous rather than the peaks of these pulses which may be occurring at different times in the cycle. The real part
of the impedance is also the part in which oscillatory power is dissipated and, therefore, is significant to the analysis of the cardiovascular system.

In determining the impedance of a system, simultaneous pressure and volume flow pulses are subjected to Fourier analysis. Fourier analysis determines the amplitude and phase angle of the sinusoidal components that make up the complex pressure and volume flow curves. To obtain the modulus, the amplitude of the pressure component of a given frequency is divided by the amplitude of the flow component of that frequency. To obtain the phase angle, the flow component phase angle is subtracted from the pressure component phase angle. When the flow component phase angle is larger than the pressure component phase angle, the phase angle of the impedance is negative, in the converse, the phase angle of the impedance is positive. Similar calculations are conducted to determine the modulus and phase angle of each of the harmonic frequencies present in the system.

The application of Fourier analysis to a waveform requires that the waveform be a steady-state oscillation. There is some concern as to whether the pressure and flow waves in the cardiovascular system satisfy the requirement of steady-state oscillations. However, McDonald (27) stated that the damping present in the cardiovascular system attenuates the oscillations associated with the initiation of a pulse within the period of that pulse. Therefore, before the initiation of a second pulse, the transient oscillations are indetectably small and the system is essentially in steady-state oscillation.

The impedance of the circulatory system is affected by the distensibility of its vessels. Distensibility of the vessels in the cardiovascular system is decreased with distance from the heart
due to changes in the relative amounts of collagen and elastin in the vessel wall. Distensibility is also affected by the smooth muscle present in the vascular wall. When the distensibility is changed, there are concomitant alterations in pulse wave velocity, attenuation, and pressure wave amplification which then lead to changes in impedance and in the oscillatory power requirements for flow in the cardiovascular system.

The impedance of the circulatory system is also affected by wave reflections. Reflection sites occur at branching points, terminations and where there are abrupt changes in vascular distensibility with positive reflections. Pressure waves reflected from these sites are reflected in phase, and flow waves are reflected 180° out of phase. These reflected waves combine with the incident waves at any point in an algebraic manner described by Taylor in 1966 (62). When the position examined has a distance from the reflection site equal to one-fourth the wave length of a frequency, the incident and reflected pressure waves of that frequency are 180° out of phase and thus, at that point, the pressure of the frequency examined is at a minimum. However, since the flow waves are reflected 180° out of phase, the incident and reflected flow waves are in phase at the one-fourth wave length distance and thus flow of the frequency is at a maximum. Therefore, the impedance calculated for this frequency is a minimum and the power necessary for flow at that point is also minimal. The reflection of waves, therefore, plays an important part in determining the impedance at any point in the cardiovascular system.

From McDonald's and Womersley's first work in the ascending aorta, the calculation of impedance was expanded to the pulmonary system (12, 50), the descending aorta (43), and the
femoral bed (47). Taylor (64) used the concept of impedance in examining the relationship between pressure and flow in the ascending aorta. The ascending aorta is the input to the circulatory system so this pressure-flow relationship is termed the input impedance. The impedance calculated at another position in the aorta is the input impedance for the remainder of the system. The input impedance of the ascending aorta has been shown to be different from that of the periphery. Patel, et al. (50), showed that the input impedance of the ascending aorta falls from high values at very low frequencies to a minimum value at about 4 Hz. This value is usually about 4 to 5 percent of the 0 Hz or peripheral resistance value. They also found that the modulus remained fairly constant at higher frequencies with some dips at 8 Hz and 18 Hz. The phase was found to be initially negative moving toward zero and becoming positive beyond the modulus minimum. Patel, et al. also examined the ascending aorta with peripheral vasodilators and vasoconstrictors. However, the large standard error of the values found made precise interpretations difficult. Vasodilation reduced the 0 Hz value and, therefore, the moduli appeared as a greater percentage of this peripheral resistance value. Also, under vasodilation, the phase remained negative and showed very little variation. Vasoconstriction caused modulus minima at 4 to 5 Hz and again at 12 Hz but the intermediate values had large standard errors.

In 1965 Patel, et al. (52) were able to examine three patients undergoing cardiac surgery and to calculate impedance spectra in their ascending aortas. These patients showed similar modulus spectra to those found in the dog, however, their phase spectra showed great irregularities when compared to studies in dogs.
Gabe, et al. (20) studied the impedance spectra in the ascending aorta of three human patients undergoing cardiac catheterization. These patients were examined under both control and vasoconstricted conditions. They found the modulus of the impedance to fall to about 8 to 10 per cent of the peripheral resistance and the phase to go from $-60^\circ$ at the fundamental to about $-10^\circ$ at the fourth harmonic. Above these points, the moduli and phase changed very little. With vasoconstriction the modulus increased at the fundamental frequency, returned to control in the 4 to 8 Hz range and was elevated again above 8 Hz. Cox was not able to demonstrate any changes in the impedance modulus of the ascending aorta with vasoconstriction (14).

Investigators have continued to work on the ascending aorta (1, 37, 41, 44, 45, 46, 48). The modulus spectra found by these investigators are similar to those found by Patel in 1963. Changes in heart rate have been effected by Noble (41) and no alteration from the usual pattern of moduli or phase could be found. In these experiments on conscious dogs, the same modulus and phase patterns were exhibited as shown by Patel in 1963. Those findings lead one to believe that the arterial tree acts as a linear system. Dick (17) also concluded linearity for the arterial system based upon his examination of the system using two pumps operating at different frequencies. He examined the intermodulations of the two pumps with the heart rate and actually found that the system became more linear with vasoconstriction than under control conditions.

O'Rourke and Taylor (48) found the ascending aorta of the dog to have two minima in its modulus spectrum. The first minimum occurred at 2 Hz and the second at 4 to 5 Hz; above 5 Hz the modulus remained relatively constant. In comparing the
impedance pattern in the ascending aorta to that found in the descending aorta, they noticed certain differences. The descending aorta had only one minimum at a low frequency. At twice this frequency the impedance was maximum and at three times this frequency, the impedance again approached a minimum. This pattern is to be expected from a system with a single reflecting site. The ascending aorta impedance spectrum exhibited two minima, however, the second minimum was not at three times the frequency at which the first minimum occurred. O'Rourke (43) believed that the second minimum represented the reflection of a second and shorter arterial system. He proposed a model of the system as an asymmetrical T-tube whose short arm represented the arteries supplying the head, neck and upper limbs, and whose long arm represented the arteries supplying the trunk and lower extremities. The base of the T corresponded to the ascending aorta. A system of this type would have a first minimum at a low frequency due to the long lower arm of the "T". The short arm being approximately one half the length of the lower arm would cause a second minimum at about twice the frequency at which the minimum of the lower arm occurred. Therefore, impedance patterns in the base of the "T" would have two minima, one due to the lower arm and the second due to the shorter upper arm.

O'Rourke and Taylor (48) showed some spectra that were like those found by Patel (50) having a minimum at about 2 Hz and remaining fairly constant thereafter. Nichols and McDonald (37) also reported these two different impedance spectra. The phase values these investigators reported differed due to the orientation of their transducers. O'Rourke and Taylor positioned their pressure transducer centrally to their flow transducer whereas
Nichols and McDonald positioned their pressure transducer distally to their flow transducer.

As a model for peripheral arteries most investigators examined the femoral artery. Randall and Stacy (56) first published data on the femoral artery. Their results are somewhat dubious, however, because of their technique and instrumentation. McDonald and Taylor (29) presented femoral impedance spectra calculated from high speed cinematographic techniques. These spectra had a single minimum at 13.5 Hz. Determination of the reflecting site from their data places the mean reflecting site somewhere below the knee and above the ankle. In 1960 McDonald (26) showed changes in femoral impedance that occurred with vasodilation and vasoconstriction. Vasoconstriction elevated the impedance at lower frequencies but the value at the minimum was reduced due to the peripheral reflection of waves being more complete with constriction. Vasodilation reduced the low frequency impedance values and increased the value at the minimum.

O'Rourke and Taylor (47) studied the impedance of the femoral bed in detail, using constant heart rates achieved by pacing or vagal stimulation. They also showed high impedance values at low frequencies with a decline to a minimum at 10 Hz and a subsequent rise to a maximum at 20 Hz. The phase was initially negative, became positive at 12 Hz and returned to 0 at 18 Hz. These phase angles are generally smaller than those found by other investigators. This finding can be attributed to the positioning of the pressure transducer distal to the flow transducer. They plotted several sets of data with varying heart rates on the same graph and found very little variation in the impedance values. This fact again demonstrated the remarkable linearity of the arterial system. They also examined the system under
vasodilation and vasoconstriction and found vasodilation to reduce the modulus at the lower frequencies and to increase the modulus at the minimum. Vasoconstriction increased the modulus at lower frequencies and reduced the value at the minimum. The effects of vasodilation and vasoconstriction on the phase angle were as follows: dilation caused a reduction in the negative phase angle at low frequencies and a reduction in the largest positive phase angle; however, the frequencies at which crossover occurred from negative to positive or positive to negative remained the same. Vasoconstriction caused an increase in the phase angle, but again the values at which crossover occurred remained unchanged.

Taylor (63) modeled the arterial system as a collection of randomly branching tubes and calculated the input impedance of the system. He showed that the distributed nature of the terminal beds either minimizes or obliterates the formation of antinodes and nodes of impedance due to reflection. He also demonstrated that the role of non-uniform elastic properties in the arterial system is to damp the high frequencies and thus reduce the amplitudes of reflected waves and maintain the input impedance of the ascending aorta low. Taylor (61) also stated that the elastic non-uniformity effectively decouples the heart from the peripheral resistance. Thus, the heart is able to adjust its pumping rate without having to alter its work output drastically. This decoupling is due to the distributed nature of the terminations and the interaction of the reflected waves from the upper and lower reflecting sites in the body.

O'Rourke and Taylor (48) found the in-phase impedance of the ascending aorta above 2 Hz to be only one fiftieth of the peripheral resistance. Thus, an increased heart rate does not require any significant increase in oscillatory power.
O'Rourke (44) also claimed that the impedance spectrum can be regarded as an expression of the power dissipating properties of the vascular system. Attinger (6) demonstrated that the frequencies which move most of the blood have the lower impedance values and, therefore, the aorta presents only small opposition to the pulsatile ejection of blood. The power dissipated in maintaining a pulsatile flow is, therefore, quite low. This power has been found to range from 10 per cent (44) to 20 per cent (14) of the total power dissipated in maintaining flow in the vascular system.

The hydraulic power associated with flow in the vascular system is divided into oscillatory and steady flow components. Each of these components has a potential and a kinetic portion. The total power associated with blood flow is dependent upon heart rate, peripheral resistance, blood pressure and the physical characteristics of the vascular bed.

O'Rourke (44) showed that an increase in heart rate increased the power associated with steady flow; the power associated with oscillatory flow remained essentially unchanged. The ratio of pulsatile power to total power has been used as a measure of the efficiency with which the vascular system accepts blood flow (14). Thus, when the heart rate is increased the ratio of oscillatory power to total power decreases denoting more efficient operation of the vascular system. Wilcox (69) also reported a decreased oscillatory power to total power ratio with increased heart rate.

O'Rourke (44) effected changes in peripheral resistance by infusing isoproterenol in dogs. He found that with this decreased peripheral resistance both the oscillatory and steady flow power were increased. The oscillatory power increased more than the
steady flow power which denotes a decrease in the efficiency of the vascular system. The decreased efficiency caused by isoproterenol was also demonstrated by Cox (14) and Abel (1). However, Abel reported that the oscillatory and steady flow components decreased instead of rising. Wilcox (69) infused isoproterenol and found no difference between the oscillatory power-total power ratio under isoproterenol and control conditions.

O'Rourke (44) caused an increase in blood pressure by infusing noradrenaline and also made observations when there were spontaneous blood pressure changes. He found that the total power was increased by an increase in steady flow power. Thus, the apparent efficiency of the system increased. Cox (14) and Abel (1) found similar increases in efficiency with noradrenaline.

O'Rourke (44) achieved changes in the distensibility by applying lucite ferrules to the brachiocephalic artery and the descending thoracic aorta. This procedure did not appreciably alter the resistance to steady flow. This decreased distensibility resulted in increased wave velocity, increased impedance at all frequencies, increased oscillatory power and either decreased or unchanged total power. The oscillatory power-total power ratio was increased denoting a decreased vascular efficiency with decreased distensibility.

Cox and Pace (16) determined the total power in the ascending aorta, descending thoracic aorta, brachiocephalic artery and left subclavian artery. They considered the ascending aorta power as the input power, and the sum of the power dissipated in the brachiocephalic artery, left subclavian artery and descending thoracic aorta as the output power. Under control conditions, an average of 6.3 per cent of the input power was dissipated in the aortic arch and parts of the brachiocephalic and left subclavian
arteries. The power dissipation of these vascular segments decreased to 2.3 per cent of the input power with vagal stimulation. The decrease was attributed to the reduction in wall viscosity with reduced mean arterial pressure and the reduction in fluid losses associated with a small decrease in fluid velocities. Upon sympathetic stimulation, the power dissipation of these vascular segments increased to 10.1 per cent. This increase was attributed to increased frictional losses in the arterial walls associated with decreased distensibility.

Cox and Pace (16) presented their power information as two separate components; the potential power and the kinetic power. Under control conditions, the kinetic power was 1.7 per cent of total power in the ascending aorta and 2.7 per cent of the total power in the descending thoracic aorta. Vagal stimulation increased the kinetic power of the ascending aorta to 2.9 per cent of total power and decreased the kinetic power of the descending thoracic aorta to 2.2 per cent of total power. Sympathetic stimulation caused the kinetic power in the ascending aorta and descending thoracic aorta to increase to 3.4 per cent and 3.6 per cent of total power, respectively.

Patel, et al. (50) were the first to provide good results of studies conducted on the pulmonary system. They found a modulus minimum at about 4 Hz and a maximum at 8 Hz. The infusion of a vasoconstrictor drug made the maximum at 8 Hz more distinct while vasodilator drugs caused all values above 3 Hz to remain fairly constant. They found the phase angle to be initially negative and then to increase in negativity throughout the frequency range. Bergel and Milnor (10) found the phase spectrum of the pulmonary system to be similar to that of the ascending aorta and, therefore, the findings of Patel, et al. (50) are difficult to explain.
The pulmonary vascular system branches into two similar subdivisions which have similar reflection characteristics. The reflections from these two subdivisions should occur at approximately the same frequency and, therefore, produce an impedance spectrum similar to that found in the descending aorta rather than the spectrum found in the ascending aorta.

The total power that is associated with blood flow in the pulmonary system is dependent upon the heart rate, resistance, blood pressure and the physical characteristics of the vascular bed as was the case for the systemic arterial system. The power in the oscillatory components is higher in the pulmonary artery than in the ascending aorta, it is 30 per cent of total power (33) as compared to 10-20 per cent of total (14) power in the aorta. The power in the kinetic portion is also higher in the pulmonary artery than in the ascending aorta; 7 per cent of total power (33) as opposed to 1.7 per cent (16) in the aorta.

Several different techniques and designs of instrumentation have been used to determine the flow and pressure in the circulatory system. In regard to determinations in the thoracic aorta, there were basically two major approaches: closed-chest preparations and open-chest preparations. With the closed-chest preparation either the differential pressure technique (20) or catheter tipped electromagnetic flow probes (19) have been the methods of choice for determining flow. With the electromagnetic flow probe, pressure was determined by a separate catheter. The differential pressure method for flow simultaneously provides pressure recordings. In all cases where pressure was recorded, it was measured by a fluid filled catheter. With closed-chest approaches, the average vessel diameter was determined either by fluoroscopic techniques or by external measurement post mortem.
With open-chest preparations, the most common means of flow determination was with electromagnetic flow probes that encircled the vessel and prevented diameter changes. Pressures were determined by use of fluid filled catheters. Experimentation in arteries outside of the thorax have used either electromagnetic or pressure differential techniques and fluid filled catheter recordings of pressure.

**Objectives**

The maintenance of blood flow is dependent upon the relationship of the total power available for flow at the input of the vascular system and the impedance of the vascular system. The changes that occur in structure and distensibility along the length of the descending aorta alter the impedance and thus affect the relationship between impedance and power along this vessel.

The objectives of the experiments presented in this manuscript are as follows: (1) Examine the impedance patterns of the descending aorta at 5 cm increments in length, (2) Determine the changes in the amount of potential power associated with pulsatile flow and the oscillatory potential power to total potential power ratio at 5 cm increments of the descending aorta, and (3) Examine the effects of epinephrine and amyl nitrite on impedance patterns, power dissipation characteristics, oscillatory potential power, and oscillatory potential power to total potential power ratio along the aorta.
CHAPTER II

METHODS

Animal Preparation

Experiments were performed on a total of 19 male mongrel dogs weighing from 19-35 kgs. The animals were premedicated by subcutaneous injection of morphine sulfate (2mg/kg) and anesthetized by intravenous injection of sodium pentobarbital (20mg/kg). During the course of the experiments the corneal reflex was used to determine when the animals required supplemental doses of sodium pentobarbital. Great care was taken during the surgical procedure to minimize the loss of blood. Either a Birtcher electro-cautery (Birtcher Corp., Los Angeles, Calif.) or a heat cautery (National E. I. Cautery, Elmhurst, N.Y.) was used to minimize bleeding. After the surgical procedures were completed and prior to the insertion of the instrumentation the animals were heparinized with 3mg/kg of heparin sodium. All animals were intubated with an endotrachial tube.

Instrumentation

The parameters monitored in these experiments were pressure, diameter and flow velocity. These parameters were recorded in both the ascending and descending aorta using catheter tip instruments. The flow velocity and the diameter in the ascending aorta were measured by means of a gauge (Figure 1) mounted on the tip of a 0.25 cm stainless steel catheter which had a 30 degree angle 6 cm behind the tip. Both the flow velocity measuring component and the diameter measuring component of
Fig. 1. -- Ascending Aorta Flow-Diameter Gauge
this instrument used the principle of a linear variable differential transformer as described by Pieper and Paul (54). The flow velocity transducer was located 1 cm in front of the largest cross-section of the diameter gauge braces when they were in the fully opened position. The frequency response of this flow velocity transducer was found to be flat to 30 Hz (53). The diameter gauge of this instrument was similar to that described by Pieper and Paul (54). The frequency response of the instrument was flat to 30 Hz and down only 5 per cent at 40 Hz. However, the version used in the present experiment had five braces spaced at 72 degree intervals around the instrument tip. This modification allowed the force exerted by the braces on the aortic wall to be distributed over a larger area of the vessel. In addition, the rear hinges of the braces were attached to a movable sleeve which itself was attached to a spring. This spring was in turn attached to a teflon sleeve which enabled the braces to be opened or closed as desired. For insertion of this instrument, the braces were collapsed by retracting the teflon sleeve.

The instrument was inserted into the right carotid artery and positioned with the flow transducer approximately 1 cm distal to the aortic valves. The rigid catheter was secured to the dissection table and the teflon sleeve advanced, thus opening the braces in the ascending aorta.

The pressure in the ascending aorta was measured by means of a catheter-tip gauge. The manometer was mounted on the tip of a flexible number seven cardiac catheter (U. S. Catheter Instrument Co., Glens Falls, N.Y.). This miniature manometer used the principle of a linear variable differential transformer and was similar to the kind previously described by Wetterer and Pieper (68). The frequency response of the manometer was flat to
at least 1000 Hz.

This instrument was inserted into the right brachial artery and threaded into the ascending aorta. With the use of a fluoroscope it was positioned approximately 4 cm behind the largest cross-section of the diameter gauge braces when they were opened. As mentioned above, the pressure, flow velocity, and diameter in the descending aorta were also measured by means of a catheter-tip gauge (Figure 2). This instrument incorporated all three measuring devices mounted on the tip of a single flexible number seven USCI cardiac catheter. Hereafter, this instrument will be referred to as a Trimeter. The pressure, flow velocity, and diameter transducers of the Trimeter all used the principle of a linear variable differential transformer, and were of the same type as used in the ascending aorta instruments.

The flow velocity transducer of the Trimeter was located 1 cm in front of the largest cross-section of the diameter gauge when they were in the fully opened position. The manometer of the Trimeter was located 4 cm behind the largest cross-section of the diameter gauge braces when they were in the fully opened position. The flexible catheter of this instrument was marked at 5 cm intervals originating from the largest cross-section of the diameter braces in the fully opened position.

The instrument was inserted into the left femoral artery by collapsing the braces with the fingers and was then positioned in the thoracic aorta about 1 cm below the arch with the aid of a fluoroscope.

The use of catheter-tip instruments for both the ascending and descending aorta precluded the necessity of opening the chest or disturbing the longitudinal or radial tethering of the aorta in any way. Tests conducted by Nicolosi and Pieper (39) assured that this
Fig. 2.--Descending Aorta Flow-Diameter-Pressure Gauge: Trimeter
type of instrumentation did not elicit a smooth muscle response from the aortic wall.

**Recording Equipment**

Data were recorded on a Beckman type S-II Dynograph recorder (Beckman Instruments, Inc., Schiller Park, Ill.). The recorder was equipped with Quartec PMC-1 carrier preamplifiers (Quartec, Inc., Columbus, Ohio). Simultaneous recordings were taken on an Ampex SP-300 instrumentation recorder (Ampex Corporation, Redwood City, Calif.).

**Calibration**

All instruments were electrically balanced and zeroed prior to the experiments. The sensitivities on the recorder were adjusted to assure that all expected data values were within the limits of the recorder. After completion of the experiments, the manometers were calibrated by attaching a calibrated vacuum pump to a side tube of the catheter-tip manometers. The side tube is continuous through the catheter with the back side of the manometer membrane. Application of a negative pressure to the side tube produces an output equal to a positive pressure of the same magnitude applied to the front side of the manometer membrane. The diameter calibrations were accomplished by pulling the diameter gauges through lucite rings of known internal diameters. To calibrate the flowmeters the transducers were subjected to an outflow of the dog's own blood from a calibrated constant velocity pump. All calibration values were recorded on both the Beckman and Ampex recorders.

**Data Collection**

For each experiment data were recorded on both the Beckman and Ampex recorders. The pressure, diameter, and
flow velocity in the descending aorta were recorded at 5 cm intervals by withdrawing the Trimeter down the descending aorta until no fluctuations were discernable in the diameter recordings. The same process was followed for all experimental procedures and hence, the diameter, pressure, and flow velocity data were recorded at the same 5 cm intervals for all control and experimental manipulations. At the completion of the experiment, the dog was bled and the blood was used in the calibration of the flow velocity transducer. The thoracic and abdominal cavities were opened and the descending aorta exposed by reflecting the viscera. The positions of the Trimeter were palpated and major structures in the areas noted (e.g. diaphragm, superior mesenteric artery, renal artery, etc.). Measurements of the wall thickness were taken at each position by inserting a teflon rod of known diameter into the lumen of the vessel and measuring the external diameter of the aorta with calipers. The diameter of the teflon rod was then subtracted from the outside diameter measurements of the vessel and the remainder divided by two to give the wall thickness at that diameter.

At a later time, pressure diameter relationship curves (PDR) for both the ascending and descending aorta were obtained from an oscilloscope by connecting the diameter signal from the Ampex recorder to the horizontal deflection plates and the pressure signal from the Ampex recorder to the vertical deflection plates. Using this technique, the instantaneous relationship of pressure to diameter could be observed and photographed. The oscilloscope used was a Tektronix Type RM 561A equipped with a Type 72 dual trace amplifier and a Type 63 differential amplifier. Photographs were taken with a C-27 Tektronix oscilloscope camera equipped with a polaroid film pack (Tektronix, Inc., Portland, Oregon).
Experimental Drugs

In ten experiments epinephrine was infused into the femoral vein at 20 μg per minute after a 60 μg primer dose. This procedure provided data concerning the effects of a peripheral vasoconstrictor on the impedance patterns of both the ascending and descending aorta. After recovery from the epinephrine, the endotracheal tube of the animal was connected to a plastic bottle containing amyl nitrite. This procedure provided data on the effects of peripheral vasodilation on the impedance patterns of both the ascending and descending aorta.

Linearization of Diameter and Flow Recordings

Due to the non-linearity of both the diameter and flow velocity transducers, the recordings of these parameters were linearized before computation of the volume velocity was undertaken. This linearization was accomplished by an EAI TR48 Analog/Hybrid Computer (Electronic Associates, Inc., West Long Branch, N.J.).

Data Analysis

The data analysis was conducted in two major steps. The first step involved the use of the EAI analog computer. The analog computer received actual recorded data from FM tape, linearized the flow and diameter values, squared the linearized diameter (d), and then multiplied this value by the corresponding linearized flow velocity (v). This calculation produced the volume velocity data $Q = \pi d^2 v / 4$. Because $\pi / 4$ is a constant, it is ignored in the analog calculation of volume velocity at this point. It is, however, added to the calculation of any digital volume velocity.

The second major step of the data analysis used a PDP-12 digital computer (Digital Equipment Corp., Maynard, Mass.). The volume velocity and pressure from either the Trimeter or the
instrument in the ascending aorta were fed into the Analog to Digital converter of the PDP-12 computer. This information was then stored on LINC tape by a Digital Equipment Corporation User Service (DECUS) program entitled ADTAPE. This program stored three seconds each of pressure and volume velocity by interleaving them in one second segments on the LINC tape (1 sec pressure, 1 sec volume velocity, etc.). A second DECUS program entitled ADCON connected the interleaved data into continuous recordings of pressure and flow.

A representative set of pressure and volume velocity curves was visually selected from the data by a DECUS program entitled CATACALE. This set of curves was then expanded or compressed to fill a one second segment of the LINC tape using a program written in FOCAL-12 called $TRANS (see Appendix 1). This program also calculated the heart rate of the segment of data chosen in the CATACALE program. These manipulations were made so that the input to the Fourier analysis program would consist of whole cycles of pressure and volume velocity waves and, therefore, the results of Fourier analysis would be in terms of the fundamental frequency and its harmonics. The Fourier analysis of the pressure and the volume velocity waves was done with a modified DECUS program entitled FRQDI (Modification of FRQDD), which took the one second of LINC tape filled by the $TRANS program and computed the Fourier components for harmonics one through sixty-four and stored these components on the LINC tape.

Calculations of the average pressure and volume velocity of one second segments were done by a program written in FOCAL-12 which was entitled $COPE (see Appendix 2). The values for the average pressure and volume velocity over one second and the corresponding digital tape values were then printed out for use in
the next calculation step.

The calculation of the real and imaginary components of the impedance spectrum was done by a second program written in FOCAL-12 entitled $IMPCALC (see Appendix 3). This program read the sine and cosine components for the pressure and volume velocity from the LINC tape and calculated the magnitude and phase angle for all harmonics from 1 to 40 Hz. This calculation resulted in digital values which were then scaled to actual impedance values using the mean pressure and the mean volume velocity, and the corresponding digital tape values calculated by the $COPE program. The $IMPCALC program also converted the harmonic values into Hz by multiplying the number of the harmonic by the heart rate which was determined by the $TRANS program. This program then stored the actual magnitude and phase for each frequency represented on LINC tape, and printed these values and actual frequencies on a teletype.

The values of modulus and phase for each frequency represented were later plotted in the Bode plot by the FRQDI program.

For the correct calculation of impedance all of the parameters (pressure, diameter, and flow velocity) would have to be measured in the same cross-section of the vessel. Due to mechanical limitations in their construction, our instruments did not occupy the same cross-section of the vessel and, therefore, an error in the calculation of impedance values exists (Figure 3). The close proximity of the instruments precludes any appreciable errors in amplitude of the parameters; however, there was appreciable phase shift. Using the parameters of pressure, diameter, and flow velocity, the correct impedance, $Z$, equals:
Figure 3

Drawing of relationship of the transducers in the Trimeter. v - position of flow velocity measurement; d - position of diameter measurement; p - position of pressure measurement. $\Delta_1$ displacement of flow velocity measurement from diameter measurement. $\Delta_2$ displacement of pressure measurement from diameter measurement. The same spatial relationship also exists with the ascending aorta instrumentation.
Fig. 3. --Drawing of Relationship of the Transducers in the Trimeter
In equation 2.1, \( p, v, \) and \( d \) are complex numbers. Since the position of the diameter gauge was used as the reference position, all pressure and flow values were corrected with respect to this position. Obviously, the correct diameter equals the diameter measured:

\[
d = d_m \tag{2.2}
\]

The flow velocity transducer was proximal to the diameter transducer in the blood stream and, therefore, the corrected velocity equals the velocity measured multiplied by a correction factor for phase shift, i.e.:

\[
v = v_m \cdot e^{-j\phi_1} \tag{2.3}
\]

The pressure transducer was distal to the diameter transducer in the blood stream and the corrected pressure, therefore, is equal to the pressure measured, multiplied by a correction factor for phase shift:

\[
p = p_m \cdot e^{j\phi_2} \tag{2.4}
\]

Substituting 2.2, 2.3, and 2.4 into 2.1:

\[
Z = \frac{4p_m \cdot e^{j\phi_1}}{v_m \cdot e^{-j\phi_2} \cdot d_m^2} \tag{2.5}
\]

Combining the correction factors in 2.5 into a single term:

\[
Z = \frac{4p_m \cdot e^{j(\phi_1 + \phi_2)}}{v d^2_m} \tag{2.6}
\]
The impedance is now expressed in terms of the parameters measured and the correction factors. The impedance calculated from the measured parameters is:

\[
Z_m = \frac{4\pi m}{\pi y m d^2 m}
\]  

(2.7)

Substituting 2.7 into 2.6 gives:

\[
Z = Z_m \cdot e^{j(\phi_1 + \phi_2)}
\]  

(2.8)

The amplitude of the correction factor in 2.8 is 1, therefore, the amplitude of the corrected impedance is equal to the amplitude of the measured impedance:

\[
|Z| = |Z_m| \cdot |e^{j(\phi_2 + \phi_1)}|
\]  

(2.9)

The phase angle of the impedance calculated from the recorded values of pressure and flow velocity is in error and is corrected to the real phase angle \(\phi_z\) as follows:

\[
\phi_z = \phi_m + \phi_1 + \phi_2
\]  

(2.10)

These corrected phase angles are related to the distance between the transducers \(\Delta_1\) and \(\Delta_2\) and the wave length of the frequency being examined:

\[
\phi_1 = (\Delta_1/\lambda_f) \cdot 360
\]  

(2.11)

\[
\phi_2 = (\Delta_2/\lambda_f) \cdot 360
\]  

(2.12)

In this set of equations, the distance between transducers is known but the wave length must be calculated. The relationship between wave length and wave velocity \(c\) is given by the following equation:

\[
\lambda_f = c/f
\]  

(2.13)
Substituting 2.13 into 2/11 and 2.12 gives:

\[ \phi_1 = \Delta_1/c \cdot f \cdot 360 \]  
\[ \phi_2 = \Delta_2/c \cdot f \cdot 360 \]  

\[ (2.14) \quad (2.15) \]

The wave velocity at any point in the aorta is given by Weber's equation (42) as:

\[ c = \sqrt{dp/dr \cdot r/2\rho} \]  

\[ (2.16) \]

where:

- \( dp/dr \) = slope of the pressure vs. radius curve
- \( r \) = radius, averaged over the amplitude of pulsation
- \( \rho \) = density of blood

These values of \( \phi_1 \) and \( \phi_2 \) provide a very good first approximation for the correction of the measured phase angle to the actual phase angle of the impedance. Once these corrections were made, the moduli and phase angles of the impedance were plotted in Bode plots for examination.

Mean potential power was calculated using the mean pressure and mean volume flow values calculated in the $COPE program:

\[ \hat{W}_M = PQ \]  

\[ (2.17) \]

The oscillatory potential power was calculated from the impedance modulus, impedance phase angle, and volume flow calculated for each frequency present by the $IMPCALC program:

\[ \hat{W}_0 = 1/2 \sum_{n=1}^{N} (q_n)^2 z_n \cos \phi_n \]  

\[ (2.18) \]

The total potential power present is then:

\[ \hat{W}_T = \hat{W}_M + \hat{W}_0 \]  

\[ (2.19) \]
The calculation of kinetic power has been omitted from this consideration. The data available did not lend itself to the calculation of kinetic power, nor does kinetic power in the aorta constitute a significant portion of the total power. It rarely ever exceeds 4 per cent of the total power (16).
CHAPTER III

RESULTS

Impedance

Control

In Figures 4 and 5, control modulus and phase angle spectra of the ascending aorta calculated from data at eight and seven different heart rates, respectively, are plotted on the same graph as a function of frequency over the range of 0 to 20 Hz. While these recordings were being taken, the instrument positioned in the descending aorta was withdrawn in 5 cm increments. The number of recordings per figure equals the number of positions examined in the descending aorta of that dog. These two figures illustrate the two different impedance modulus spectra found in this investigation. The magnitudes and phase angles of one complete spectrum are connected by an unbroken line in each figure.

The impedance spectrum presented in Figure 4 has two minima below 10 Hz. The modulus decreases from the high 0 Hz or peripheral resistance value to the first minimum in the 3-4 Hz range then increases to a maximum at approximately 6 Hz before decreasing to a second minimum in the 7-8 Hz range. At frequencies above the second minimum, the modulus oscillates but increases generally. The phase angle increases to a maximum in 5 Hz range, decreases to a minimum in the 7 Hz range and then oscillates as it further increases. The phase angle becomes positive in the 8-10 Hz range. The great scatter in the modulus
Figure 4

Composite plot of eight impedance modulus and phase angle spectra of the ascending aorta vs. frequency under control conditions. The moduli and phase angles of one complete spectrum are connected by the unbroken lines.
Fig. 4.--Impedance Modulus and Phase Angle Spectra of the Ascending Aorta: Control
Figure 5

Composite plot of *seven* impedance modulus and *phase* angle spectra of the ascending aorta vs. frequency under control conditions. The moduli and phase angles of one complete spectrum are connected by the unbroken lines.
Fig. 5. — Impedance Modulus and Phase Angle Spectra of the Ascending Aorta: Control
and phase angle value above 10 Hz in Figure 4 probably can be attributed to the very small values obtained for pressure and flow amplitudes at these frequencies. Biological and transducer noise present in either of the parameters may cause great dispersion due to the small amplitudes.

The impedance spectrum presented in Figure 5 demonstrates a single minimum below 10 Hz. The modulus decreases from the high peripheral resistance value to very small values at all harmonics. The modulus reaches a minimum in the 9-10 Hz range and increases again at frequencies above 10 Hz. The phase angle increases throughout the frequency range becoming positive in the 8-9 Hz range.

Figures 6 and 7 present representative samples of the control modulus and phase angle spectra, respectively, calculated at eight different positions in the descending aorta and plotted as a function of frequency over the range 0-20 Hz. The uppermost spectrum (P1) in each figure was calculated for the descending aorta just below the arch. The next four spectra (P2, P3, P4, and P5) were calculated for positions 5, 10, 15, and 20 cm below the position of the first spectrum. The last of these four spectra (P5) was 3 cm above the diaphragm. The sixth spectrum (P6) was calculated for a position 2 cm below the diaphragm, the seventh spectrum (P7) for a position 2 cm below the renal arteries, and the eighth spectrum for a position 7 cm below the renal arteries.

Table 1 contains the mean pressure, mean flow, impedance at 0 Hz, impedance at the first minimum and frequency of the first minimum for each position under control and experimental conditions.
Figure 6

Plots of the impedance moduli vs. frequency under control conditions. Each plot represents a position in the descending aorta. P1: 1 cm below the aortic arch; P2: 6 cm below the aortic arch; P3: 11 cm below the aortic arch; P4: 16 cm below the aortic arch; P5: 21 cm below the aortic arch and 3 cm above the diaphragm; P6: 2 cm below the diaphragm; P7: 2 cm below the renal arteries; and P8: lowest position measured.
Fig. 6. -- Impedance Modulus Spectra of the Descending Aorta: Control
Figure 7

Plots of the impedance phase angle vs. frequency under control conditions. Each plot represents a position in the descending aorta. P1: 1 cm below the aortic arch; P2: 6 cm below the aortic arch; P3: 11 cm below the aortic arch; P4: 16 cm below the aortic arch; P5: 21 cm below the aortic arch and 3 cm above the diaphragm; P6: 2 cm below the diaphragm; P7: 2 cm below the renal arteries; and P8: lowest position measured.
Fig. 7. -- Impedance Phase Angle Spectra of the Descending Aorta: Control Conditions
Table 1. -- Data are presented for eight positions in the descending aorta of a representative dog under control, epinephrine infusion, control, and amyl nitrite inhalation conditions. The pressure (mm Hg), flow (ml/sec), impedance modulus at 0 Hz (dyne·sec·cm⁻²), impedance at the minimum (dyne·sec·cm⁻²), and frequency of the minimum (Hz), are presented at 5 cm intervals along the descending aorta.

<table>
<thead>
<tr>
<th>Condition</th>
<th>P</th>
<th>Pressure</th>
<th>Flow</th>
<th>Z₀Hz</th>
<th>Zₘin</th>
<th>Eₘin</th>
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<td></td>
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The modulus spectra presented in Figure 6 all decrease from the peripheral resistance value to a minimum and then oscillate through lesser maxima and minima at frequencies above the first minimum frequency. The value of the peripheral resistance increases throughout the length of the aorta as do the moduli values at the first minimum and the frequency of the first minimum, as can be seen from the Control 1 values in Table 1.

The maxima and minima in the phase angle spectra in Figure 7 occur at frequencies near those of the maxima and minima of their respective modulus spectra seen in Figure 6.

Epinephrine

With the prolonged infusion of epinephrine at 20 µg/min, the two different patterns for the impedance modulus of the ascending aorta are still present. In figures 8 and 9, modulus and phase angle spectra under epinephrine infusion from the ascending aorta calculated from data at eight and seven different heart rates, respectively, are plotted on the same graph as a function of frequency over the range 0 to 20 Hz. The moduli and phase angles of one complete spectrum are connected by an unbroken line in each figure.

The impedance spectrum presented in Figure 8 has two minima below 10 Hz. The frequencies at which the minima below 10 Hz occur are slightly below the corresponding frequencies of the control condition. The first minima under epinephrine infusion occurs just below 3 Hz and the second in the 6-7 Hz range. The phase angle increases to a maximum in the 4-5 Hz range, decreases to a minimum in the 5-7 Hz range and becomes positive in the 7-10 Hz range. The great scatter of modulus and phase angle values above 10 Hz in Figure 8 can be attributed to the small pressure and flow amplitudes and biological and transducer
Figure 8

Composite plot of eight impedance modulus and phase angle spectra of the ascending aorta vs. frequency under epinephrine infusion. The moduli and phase angles of one complete spectrum are connected by the unbroken line.
Fig. 8. -- Impedance Modulus and Phase Angle Spectra of the Ascending Aorta: Epinephrine Infusion
Figure 9

Composite plot of seven impedance modulus and phase angle spectra of the ascending aorta vs. frequency under epinephrine infusion. The moduli and phase angles of one complete spectrum are connected by the unbroken line.
Fig. 9. -- Impedance Modulus and Phase Angle Spectra of the Ascending Aorta: Epinephrine Infusion
noise in this frequency range as discussed above.

The impedance spectrum presented in Figure 9 shows a single minimum below 10 Hz. The frequency range in which this minimum occurs under epinephrine infusion is reduced from the control range of 9-10 Hz to the 6-8 Hz range. The phase angle increases throughout the frequency range presented becoming positive between 7 and 9 Hz. The value of the modulus in either of the two patterns is not appreciably changed by the infusion of epinephrine except in the frequency ranges where the minima occur, and at the 0 Hz value. The infusion of epinephrine causes a reduction of the peripheral resistance or 0 Hz impedance due to a relatively greater increase in flow than in pressure.

Figures 10 and 11 present representative samples of the epinephrine infusion modulus and phase angle spectra, respectively, calculated at eight different positions in the descending aorta and plotted as a function of frequency over the range 0-20 Hz. The positions represented in these figures are the same as those represented in Figures 6 and 7.

The modulus spectra shown in Figure 10 all decrease from the peripheral resistance value to a minimum and then oscillate through maxima and minima at frequencies above the frequency of the first minimum. The values of peripheral resistance, modulus at the first minimum, and frequency of the first minimum, increase throughout the length of the aorta as can be seen from the epinephrine values in Table 1. Comparison of control and epinephrine infusion values in Table 1 shows that the peripheral resistance decreased, the modulus at the first minimum increased, and the frequency at the first minimum decreased for each position in the aorta during epinephrine infusion.
Figure 10

Plots of the impedance moduli vs. frequency under epinephrine infusion. Each plot represents a position in the descending aorta. P1: 1 cm below the aortic arch; P2: 6 cm below the aortic arch; P3: 11 cm below the aortic arch; P4: 16 cm below the aortic arch; P5: 21 cm below the aortic arch and 3 cm above the diaphragm; P6: 2 cm below the diaphragm; P7: 2 cm below the renal arteries; and P8: lowest position measured.
Fig. 10. -- Impedance Modulus Spectra of the Descending Aorta: Epinephrine Infusion
Figure 11

Plots of the impedance phase angle vs. frequency under epinephrine infusion. Each plot represents a position in the descending aorta. P1: 1 cm below the aortic arch; P2: 6 cm below the aortic arch; P3: 11 cm below the aortic arch; P4: 16 cm below the aortic arch; P5: 21 cm below the aortic arch and 3 cm above the diaphragm; P6: 2 cm below the diaphragm; P7: 2 cm below the renal arteries; and P8: lowest position measured.
Fig. 11. -- Impedance Phase Angle Spectra of the Descending Aorta: Epinephrine Infusion
Examination of Figures 6 (control) and 10 (epinephrine) shows that the moduli of the first three harmonics in spectra P1, P2, P3, and P4 were not greatly changed by epinephrine infusion. Beginning with spectrum P5, the moduli of the first three harmonics decreased with epinephrine infusion and in spectrum P8 the reduction in modulus value persisted up to the 6-7 Hz range.

The maxima and minima in the phase angle spectra seen in Figure 11 occur at frequencies near the maxima and minima of their respective modulus spectra seen in Figure 10. Comparison of the control and epinephrine phase angle spectra (Figures 7 and 11, respectively,) shows that the phase angle at frequencies up to the 5-7 Hz is less negative at all positions in the descending aorta with epinephrine.

Amyl Nitrite

With the inhalation of amyl nitrite, the two different impedance moduli patterns become essentially the same. In Figures 12 and 13 modulus and phase angle spectra under amyl nitrite infusion from the ascending aorta, calculated from data at eight and seven different heart rates, respectively, are plotted on the same graph as a function of frequency over the range 0 to 20 Hz. The moduli and phase angles of one complete spectrum are connected by an unbroken line in each figure.

The data in Figure 12 were collected from a dog which had previously exhibited a modulus pattern with two minima below 10 Hz. The modulus spectrum under amyl nitrite does not show the distinct maxima and minima that its control spectrum exhibited (Figure 14). Comparison of the modulus spectra in Figures 12 and 14 shows the moduli in the 4-7 Hz range to be decreased to the modulus values found in the 1-4 and 7-9 Hz ranges with amyl nitrite inhalation. The phase angle spectra under amyl nitrite
Figure 12

Composite plot of eight impedance modulus and phase angle spectra of the ascending aorta vs. frequency under amyl nitrite inhalation. The moduli and phase angles of one complete spectrum are connected by the unbroken line.
Fig. 12. Impedance Modulus and Phase Angle Spectra of the Ascending Aorta: Amyl Nitrite Inhalation
Figure 13

Composite plot of seven impedance modulus and phase angle spectra of the ascending aorta vs. frequency under amyl nitrite inhalation. The moduli and phase angles of one complete spectrum are connected by the unbroken lines.
Fig. 13. -- Impedance Modulus and Phase Angle Spectra of the Ascending Aorta: Amyl Nitrite Inhalation
Figure 14

Composite plot of eight impedance modulus and phase angle spectra of the ascending aorta vs. frequency under control conditions. The moduli and phase angles of one complete spectrum are connected by the unbroken lines.
Fig. 14. -- Impedance Modulus and Phase Angle Spectra of the Ascending Aorta: Control
inhalation (Figure 12) did not contain the maximum or minimum that were present in the control spectra (Figure 14) and were associated with the modulus maximum and minimum which occurred below 10 Hz.

The inhalation of amyl nitrite did not alter the modulus spectrum of the dog that exhibited only one minimum in the 0-10 Hz range (Figure 13 - amyl nitrite; Figure 15 - control). Comparison of the phase angle spectra in Figures 13 and 15 does not demonstrate any appreciable changes under amyl nitrite inhalation.

Figures 16 and 17 show representative samples of the amyl nitrite inhalation modulus and phase angle spectra, respectively, calculated at eight different positions in the descending aorta and plotted as a function of frequency over the range 0-20 Hz. The positions represented in these figures are the same as those shown in Figures 6 and 7.

The modulus spectra in Figure 16 all decrease from the peripheral resistance value to a minimum and then oscillate through maxima and minima at higher frequencies. The values of peripheral resistance, modulus at the first minimum, and frequency of the first minimum increase throughout the length of the aorta, as can be seen from the amyl nitrite values in Table 1.

Comparison of Control 2 and amyl nitrite values in Table 1 shows that the peripheral resistance increased at all positions in the descending aorta. The frequency at the first minimum decreased for some positions (P1 and P2) but remained almost the same at other positions (P3, P4, and P6) and was elevated at positions P5, P7, and P8.

Comparison of modulus and phase angle spectra from control (Figures 16 and 17) and amyl nitrite inhalation (Figures 18 and 19)
Figure 15

Composite plot of seven impedance modulus and phase angle spectra of the ascending aorta vs. frequency under control conditions. The moduli and phase angles of one complete spectrum are connected by the unbroken lines.
Fig. 15.--Impedance Modulus and Phase Angle Spectra of the Ascending Aorta: Control
Figure 16

Plots of the impedance moduli vs. frequency under amyl nitrite inhalation. Each plot represents a position in the descending aorta. P1: 1 cm below the aortic arch; P2: 6 cm below the aortic arch; P3: 11 cm below the aortic arch; P4: 16 cm below the aortic arch; P5: 21 cm below the aortic arch and 3 cm above the diaphragm; P6: 2 cm below the diaphragm; P7: 2 cm below the renal arteries; and P8: lowest position measured.
Impedance, dyne-sec cm \( \sim 5 \times 10^5 \),

\[\text{Fig. 16. -- Impedance Modulus Spectra of the Descending Aorta: Amyl Nitrite Inhalation}\]
Figure 17

Plots of the impedance phase angle vs. frequency under amyl nitrite inhalation. Each plot represents a position in the descending aorta. P1: 1 cm below the aortic arch; P2: 6 cm below the aortic arch; P3: 11 cm below the aortic arch; P4: 16 cm below the aortic arch; P5: 21 cm below the aortic arch and 3 cm above the diaphragm; P6: 2 cm below the diaphragm; P7: 2 cm below the renal arteries; and P8: lowest position measured.
Fig. 17. -- Impedance Phase Angle Spectra of the Descending Aorta: Amyl Nitrite Inhalation
Figure 18

Plots of the impedance moduli vs. frequency under control conditions. Each plot represents a position in the descending aorta. P1: 1 cm below the aortic arch; P2: 6 cm below the aortic arch; P3: 11 cm below the aortic arch; P4: 16 cm below the aortic arch; P5: 21 cm below the aortic arch and 3 cm above the diaphragm; P6: 2 cm below the diaphragm; P7: 2 cm below the renal arteries; and P8: lowest position measured.
Fig. 18. - Impedance Modulus Spectra of the Descending Aorta: Control
Figure 19

Plots of the impedance phase angle vs. frequency under control conditions. Each plot represents a position in the descending aorta. P1: 1 cm below the aortic arch; P2: 6 cm below the aortic arch; P3: 11 cm below the aortic arch; P4: 16 cm below the aortic arch; P5: 21 cm below the aortic arch and 3 cm above the diaphragm; P6: 2 cm below the diaphragm; P7: 2 cm below the renal arteries; and P8: lowest position measured.
Fig. 19. -- Impedance Phase Angle Spectra of the Descending Aorta: Control Conditions
showed some obvious differences. However, the high heart rate and these small number of harmonic values present causes some uncertainty in the comparison of amyl nitrite and control spectra.

There was great similarity among most of the descending aorta impedance spectra calculated for this manuscript. One experiment, however, did not conform to patterns found in the rest of the experiments. Figures 20 and 21 present the modulus and phase angles, respectively, for seven different positions along the descending aorta plotted as a function of frequency over the range 0-20 Hz. Spectra P1-P5 were in the thoracic aorta, P6 below the diaphragm and above the renal arteries, and P7 below the renal arteries. Examination of Figures 20 and 21 shows that the modulus and phase spectra calculated for positions in the thoracic aorta contain only minor oscillations and it is not until the point of impedance measurement is below the diaphragm that distinct maxima and minima can be discerned.

Power
Control

Table 2 contains the values of mean power, oscillating power, total power and oscillatory power to total power ratio calculated for different positions along the ascending and descending aorta under control and experimental conditions. The total power and mean power for Control 1 are presented graphically in Figure 22. The difference between the upper or total potential power curve and the lower or mean potential curve is the oscillatory potential power. This figure illustrates the large dissipation rate of potential energy that is present:

1. between the ascending aorta and the descending aorta,
2. between the mid thoracic level and positions 6 to 9 cm above the diaphragm and
3. where the descending aorta passes through the diaphragm and where it gives off the superior mesenteric
Figure 20

Plots of the impedance moduli vs. frequency under control conditions. Each plot represents a position in the descending aorta. P1: 1 cm below the aortic arch; P2: 6 cm below the aortic arch; P3: 11 cm below the aortic arch; P4: 16 cm below the aortic arch; P5: 21 cm below the aortic arch and 2 cm above the diaphragm; P6: 3 cm below the diaphragm; and P7: 4 cm below the renal arteries.
Fig. 20. --Impedance Modulus Spectra of the Descending Aorta: Control
Fig. 21. Impedance Phase Spectra of the Descending Aorta: Control
Table 2. Data are presented for nine areas in the aorta under control, epinephrine infusion, control, and amyl nitrite inhalation conditions. The average oscillatory power in milliwatts (MP), total power in milliwatts (TP), and oscillatory power to total power ratio, as a per cent, are presented with their standard errors for each area under each condition.

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<th>Control 2 ± SE</th>
<th>Amyl Nitrite ± SE</th>
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<td>OP</td>
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<td>MP</td>
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<tr>
<td>OP</td>
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<td>MP</td>
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<td>4.48 ± 1.59</td>
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<td>2.29 ± 0.29</td>
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<tr>
<td>OP</td>
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<td>% OP/TP</td>
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<td>4.61 ± 3.42</td>
<td>2.53 ± 0.01</td>
<td>0.80 ± 0.16</td>
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Figure 22

Plots of the total power and mean power vs. position in the aorta under control conditions. Closed circles - total power and open circles - mean power. Positions in the aorta are as follows: AA: ascending aorta; DA: 1 cm below the aortic arch; 6: 6 cm below the aortic arch; MTA: mid thoracic aorta; 6-9: 6-9 cm above the diaphragm; 1-3: 1-3 cm above the diaphragm; BD: below the diaphragm and above the renal arteries; BR: below the renal arteries; and LO: lowest position measured.
Fig. 22. Plots of Total Power and Mean Power vs. Position in the Aorta under Control Conditions.
artery. The Control 1 values of total pressure, oscillatory pressure, and mean pressure from Table 2 are also expressed in Table 3 as a per cent of the respective ascending aorta value dissipated to the position examined, and only shown graphically in Figure 23. The large reductions noted in Figure 22 also appear in Figure 23 as large percentage dissipations. Also, as can be seen from Figure 23, the percentage of oscillatory power dissipated from the ascending aorta to the descending aorta is the largest of any aortic segment. Figure 24 presents the total power percentages from Table 2 for both Control 1 and prolonged epinephrine infusion. The oscillatory power to total power ratio under control conditions can be seen to decrease to the mid thoracic area, rise in the next segment and then decrease again to the lowest portion of the aorta examined.

Epinephrine

Figure 25 graphically presents the total power and mean power values from Table 2 for epinephrine infusion. These values, as well as the oscillatory power values, are larger under epinephrine conditions than the control values at all positions in the aorta. Figure 26 presents the data on dissipation from Table 3. Comparison of Figures 23 and 26 showed that epinephrine infusion: (1) reduced the percentage of total, mean, and oscillatory power dissipated from ascending aorta to descending aorta, (2) increased the percentage of total, mean, and oscillatory power dissipated from a position 6 cm below the aortic and to the mid thoracic region, and (3) increased the percentage of total and mean powers dissipated across the renal arteries. The ratio of oscillatory to total power under epinephrine infusion and control conditions are presented in Figure 24. This ratio for epinephrine infusion is larger than the control ratio for the ascending aorta and
Table 3. Data are presented for the per cent reduction from ascending aorta levels of oscillatory power (OP), mean power (MP), and total power (TP) at eight areas in the aorta under control, epinephrine infusion, control and amyl nitrite inhalation conditions.

<table>
<thead>
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<td>TP 42.4</td>
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Figure 23

Plots of the percentage reduction from ascending aorta values for total, mean, and oscillatory power vs. position in the aorta for control conditions. Closed circles - total power, open circles - mean power, and open squares - oscillatory power. Positions in the aorta are as follows: AA: ascending aorta; DA: 1 cm below the aortic arch; 6: 6 cm below the aortic arch; MTA: mid thoracic aorta; 6-9: 6-9 cm above the diaphragm; 1-3: 1-3 cm above the diaphragm; BD: below the diaphragm and above the renal arteries; BR: below the renal arteries; and LO: lowest position.
Fig. 23. -- Plots of the Percentage Reduction from Ascending Aorta Values for Total, Mean, and Oscillatory Power vs. Position in the Aorta for Control Conditions
Figure 24

Plots of oscillatory power to total power ratio vs. position in the aorta under control and epinephrine infusion conditions. Closed circles - control conditions and open circles - epinephrine infusion. Positions are as follows: AA: ascending aorta; DA: 1 cm below the aortic arch; 6: 6 cm below the aortic arch; MTA: mid thoracic aorta; 6-9: 6-9 cm above the diaphragm; 1-3: 1-3 cm above the diaphragm; BD: below the diaphragm and above the renal arteries; BR: below the renal arteries; and LO: lowest position measured.
Fig. 24. --Plots of Oscillatory Power to Total Power Ratio vs. Position in the Aorta under Control and Epinephrine Infusion Conditions.
Figure 25

Plots of total power and mean power vs. position in the aorta under epinephrine infusion. Closed circles - total power and open circles - mean power. Positions in the aorta are as follows: AA: ascending aorta; DA: 1 cm below the aortic arch; 6: 6 cm below the aortic arch; MTA: mid thoracic aorta; 6-9: 6-9 cm above the diaphragm; 1-3: 1-3 cm above the diaphragm; BD: below the diaphragm and above the renal arteries; BR: below the renal arteries; and LO: lowest position measured.
Fig. 25. -- Plots of Total Power and Mean Power vs. Position in the Aorta under Epinephrine Infusion
Figure 26

Plots of the percentage reduction from ascending aorta values for total, mean, and oscillatory power vs. position in the aorta for epinephrine infusion. Closed circles - total power, open circles - mean power, and open squares - oscillatory power. Positions in the aorta are as follows: AA: ascending aorta; DA: 1 cm below the aortic arch; 6: 6 cm below the aortic arch; MTA: mid thoracic aorta; 6-9: 6-9 cm above the diaphragm; 1-3: 1-3 cm above the diaphragm; BD: below the diaphragm and above the renal arteries; BR: below the renal arteries; and LO: lowest position measured.
Fig. 26. -- Plots of the Percentage Reduction from Ascending Aorta Values for Total, Mean and Oscillatory Power vs. Position in the Aorta for Epinephrine Infusion
descending aorta to the mid thoracic region. The oscillatory power to total power ratio is also larger with epinephrine than the control ratio at positions 1-3 cm above the diaphragm, below the diaphragm, below the renal arteries, and the lowest position examined.

Amyl Nitrite

Figures 27 and 28 present the values from Table 2 on total and mean power for amyl nitrite inhalation and its control, respectively. Both the total and mean powers at all positions in the aorta were reduced from control values with amyl nitrite inhalation. The data on total, mean, and oscillatory power dissipation for amyl nitrite inhalation and its control are presented in Table 3 and graphically in Figures 29 and 30, respectively. Inhalation of amyl nitrite increased the percentage of total and mean power dissipated from ascending aorta to descending aorta compared to the control value. The segment of descending aorta from 1 cm below the arch to 6 cm below the arch showed an increase in total and mean power dissipation with amyl nitrite but the same segment under control conditions showed a decrease in total and mean power dissipation. At the descending aorta position 1-3 cm above the diaphragm, the total and mean power dissipations for amyl nitrite and control conditions are approximately the same and remain comparable along the remainder of the aorta measured. The percentage of oscillatory power dissipated from ascending aorta to descending aorta is approximately the same for control and amyl nitrite inhalation conditions. The percentage of oscillatory power dissipated in the thoracic aorta was larger under amyl nitrite than under control conditions. The percentage of oscillatory power dissipated in the abdominal aorta was smaller under amyl nitrite than under control conditions.
Figure 27

Plots of the total power and mean power vs. position in the aorta under amyl nitrite inhalation. Closed circles - total power and open circles - amyl nitrite inhalation. Positions in the aorta are as follows: AA: ascending aorta; DA: 1 cm below the aortic arch; 6: 6 cm below the aortic arch; MTA: mid thoracic aorta; 6-9: 6-9 cm above the diaphragm; 1-3: 1-3 cm above the diaphragm; BD: below the diaphragm and above the renal arteries; BR: below the renal arteries; and LO: lowest position measured.
Fig. 27.--Plots of Total Power and Mean Power vs. Position in the Aorta under Amyl Nitrite Inhalation
Figure 28

Plots of the total power and mean power vs. position in the aorta under control conditions. Closed circles - total power and open circles - mean power. Positions are as follows: AA: ascending aorta; DA: 1 cm below the aortic arch; 6: 6 cm below the aortic arch; MTA: mid thoracic aorta; 6-9: 6-9 cm above the diaphragm; 1-3: 1-3 cm above the diaphragm; BD: below the diaphragm and above the renal arteries; BR: below the renal arteries; and LO: lowest position measured.
Fig. 28.--Plots of Total Power and Mean Power vs. Position in the Aorta under Control Conditions.
Figure 29

Plots of the percentage reduction from ascending aorta values for total, mean, and oscillatory power vs. position in the aorta for amyl nitrite inhalation. Closed circles - total power, open circles - mean power and open squares - oscillatory power. Positions in the aorta are as follows: AA: ascending aorta; DA: 1 cm below the aortic arch; 6: 6 cm below the aortic arch; MTA: mid thoracic aorta; 6-9: 6-9 cm above the diaphragm; 1-3: 1-3 cm above the diaphragm; BD: below the diaphragm and above the renal arteries; BR: below the renal arteries; and LO: lowest position measured.
Fig. 29.--Plots of the Percentage Reduction from Ascending Aorta Values for Total, Mean, and Oscillatory Power vs. Position in the Aorta for Amyl Nitrite Inhalation.
Figure 30

Plots of the percentage reduction from ascending aorta values for total, mean, and oscillatory power vs. position in the aorta for control conditions. Closed circles - total power, open circles - mean power and open squares - oscillatory power. Positions in the aorta are as follows: AA: ascending aorta; DA: 1 cm below the aortic arch; 6: 6 cm below the aortic arch; MTA: mid thoracic aorta; 6-9: 6-9 cm above the diaphragm; 1-3: 1-3 cm above the diaphragm; BD: below the diaphragm and above the renal arteries; BR: below the renal arteries; and LO: lowest position measured.
Fig. 30. --Plots of the Percentage Reduction from Ascending Aorta Values for Total, Mean, and Oscillatory Power vs. Position in the Aorta for Control Conditions
The oscillatory power to total power ratio from Table 2 for amyl nitrite and its control condition are presented in Figure 31. This ratio under amyl nitrite was appreciably different from its control values only in the lower half of the thoracic and the abdominal aorta. At these positions, amyl nitrite reduced the ratio from its control values.
Figure 31

Plots of oscillatory power to total power ratio vs. position in the aorta under control and amyl nitrite inhalation conditions. Closed circles - control conditions and open circles - amyl nitrite inhalation. Positions are as follows: AA: ascending aorta; DA: 1 cm below the aortic arch; 6: 6 cm below the aortic arch; MTA: mid thoracic aorta; 6-9: 6-9 cm above the diaphragm; 1-3: 1-3 cm above the diaphragm; BD: below the diaphragm and above the renal arteries; BR: below the renal arteries; and LO: lowest position measured.
Fig. 31. -- Plots of Oscillatory Power to Total Power Ratio vs. Position in the Aorta under Control and Amyl Nitrite Inhalation Conditions.
CHAPTER IV
DISCUSSION

Impedance
Control

Examination of the figures containing data obtained from the ascending aorta (Figures 4, 5, 8, 9, 12, 13, 14 and 15) showed that the modulus and phase angle values obtained under similar physiological conditions within any single animal fell on or near the same line. These graphs each contained impedance spectra calculated at several different heart rates; thus the frequencies and wave forms of the input were varied. If the arterial system were linear or nearly linear, variations of input frequency and wave form would not appreciably alter the output of the system measured in terms of impedance, which was the case with these experiments. Noble (41), Dick (17) and Bergel and Milnor (11) examined the ascending aorta to determine its linearity and concluded that the system was fairly linear.

The impedance spectra obtained here are similar to those described by Attinger (5) and Patel, et al. (52). Attinger used the differential pressure method to determine flow, while Patel, et al. used electromagnetic flow meters.

The impedance modulus spectrum containing a single broad minimum was also found by Patel, et al. (50) while O'Rourke and Taylor (48) showed the impedance modulus pattern containing two distinct minima. Both types of patterns have been repeated by Nichols and McDonald (27). The existence of one or the other of
these two patterns may be dependent upon the state of the vasculature of the animal. Changes in vascular conditions would alter the apparent distances to the reflecting sites in either the upper or lower or both sections of the vascular system. If the two reflection distances changed such that they became equal or close to equal, the frequencies at which the two different impedance minima associated with these reflection sites occurred would also be equal or close; thus the minima would blend together. In Figures 12 and 14, it is shown that the prolonged inhalation of amyl nitrite caused a spectrum with two minima to be transformed to one with a single minimum. O'Rourke and Taylor (48) also showed the disappearance of distinct minima in the impedance pattern with the infusion of vasodilator drugs.

The pattern of phase angle spectra found in the ascending aorta showed decreasing negativity at frequencies above the fundamental frequency. The spectra became positive between 9 and 10 Hz and increased in positivity above 10 Hz. Attinger (6), McDonald (27), and O'Rourke and Taylor (48) reported very similar patterns from their investigations.

Examination of the descending aorta at 5 cm intervals showed that this system undergoes a smooth change in its impedance spectrum throughout the length of the thoracic aorta (Figures 6 and 7). As the aorta passes through the diaphragm, the impedance modulus increases disproportionately from position to position. The impedance spectrum also exhibits a sudden step change as the diaphragm is traversed. However, these disproportionate increases in impedance are still smooth transitions. Other investigators examined the descending aorta and the peripheral vasculature but recordings from the descending aorta were made at only one or two positions (45) while the primary
emphasis has been focused on the femoral artery (26, 29, 47 and 56). In contrast, investigations presented in this paper provided a stepwise examination of the descending aorta from below the arch to above the iliac bifurcation.

In the analysis of the data from these experiments, it was found that many phase angle values exceeded $\pm 90^\circ$. Gessner (21), however, showed that a system which is passive cannot have phase angles in excess of $\pm 90^\circ$. Since longitudinal displacement of the transducers was corrected for during analysis, phase values in excess of $\pm 90^\circ$ would not be attributed to instrument error. However, some of these extreme values occurred at frequencies above 10 Hz and, therefore, may be attributed to biological noise, instrument noise, and the very small amplitudes in this frequency range. There were, however, phase values present in the frequency range 0-10 Hz which exceeded $\pm 90^\circ$ and cannot be explained by noise.

One possible explanation for these extreme phase values may lie in the design of the flow transducer. This transducer measures the axial flow velocity of the vessel. Thus when the pressure gradient reverses causing the average flow velocity to reverse also, the transducer may still be measuring an unreversed axial flow velocity for a period of time. Note that the axial flow has a greater kinetic energy than the flow along the wall; therefore, it takes longer to change its direction. This possibility becomes less likely in light of the non-dimensional number $\alpha$ as discussed by McDonald (27). This number is calculated from the dimensions of the vessel and the flow velocity in that vessel:

$$\alpha = \frac{R}{\sqrt{\omega/v}} \quad (4.1)$$
where: \( R \) = average vessel radius \\
\( \omega \) = angular frequency \\
\( v \) = flow velocity

As \( \alpha \) increases the flow profile becomes flatter. The \( \alpha \) calculated for the descending aorta of the dog is 10 or greater, indicating a flow profile which is quite blunt. Therefore, the axial velocity would not be greatly different from the average velocity over the cross-sectional area. Nerem, et al. (35), and Seed and Woods (59) also showed the velocity profile of the aorta to be quite flat by direct measurement techniques. It is, therefore, concluded that phase values greater than \(+90^\circ\) constitute a true finding for the descending aorta.

It is interesting to speculate why the flow should precede the pressure so greatly in the aorta. On theoretical grounds, the aorta can exhibit phase angle values greater than \(+90^\circ\) only if energy is actively put into the system by a mechanism other than the heart's pumping action.

Nicolosi and Pieper (39) have driven the aorta of intact animals with a sinusoidal pump at frequencies from 0.07 Hz - 0.75 Hz. They were not able to elicit a hysteresis effect from the aorta on a plot of pressure vs. diameter. Note that the frequency of 0.75 Hz was close to the normal heart rate of the animals examined. On the other hand, these same recordings showed hysteresis like loops being formed with each cardiac cycle, the pressure rising before the diameter in systole and falling before the diameter during diastole.

These loops during the cardiac cycle remained quite consistent and of appreciable size with each heart cycle. The absence of hysteresis loops during the pump cycle and the presence of loops during the cardiac cycle suggest that the cardiogenic loops may not be due to delayed compliance as postulated by Alexander (2).
With delayed compliance, hysteresis loops of the excised blood vessels become smaller with successive cycles until the inflation and deflation portions of the loops are quite close to each other.

One might speculate that the cardiogenic loops could be obtained if the aortic smooth muscle contracted rhythmically with each heart beat. Systolic stiffening of the aorta would cause the pressure to rise faster than the diameter during systole, whereas, with diastolic relaxation the pressure would fall faster than the diameter during diastole. Thus the aortic smooth muscle might actively generate the cardiogenic loops. Although there is at present no other evidence for aortic smooth muscle rhythmicity, our extreme phase angles would tend to warrant further investigation in this direction.

**Epinephrine**

Administration of epinephrine did not alter the number of minima present in the modulus spectrum of the ascending aorta below 10 Hz, nor did it alter the modulus level over the whole spectrum except at the 0 Hz value (Figures 4 and 8, 5 and 9). The frequency at which the minima (um) occurred was, however, lower under epinephrine than under control conditions. The lower frequency for modulus minima would indicate increase in the distance to the point of reflection.

The oscillations in phase angle spectra of the ascending aorta and the frequency at which the phase angle became positive occurred at lower frequency values under epinephrine infusion than under control conditions. These oscillations and zero crossover occurred at frequencies similar to those at which the modulus spectra showed maxima and minima and, therefore, would be expected to shift in frequency proportionately with those modulus maxima and minima.
Table 4. Data are presented for different positions in the descending aorta of three dogs under control, epinephrine infusion, control, and amyl nitrite inhalation conditions. The pressure range in mm Hg, mean pressure in mm Hg, and diameter range in cm are presented for each position under each condition.

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<th>Epinephrine</th>
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<td>142</td>
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<td>159.4-107.6</td>
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<td>154.8-97.2</td>
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<td>171.9-109.3</td>
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<td>140</td>
<td>118.2</td>
<td>103.4</td>
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<tr>
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<td>1.61-1.50</td>
<td>177.3-112.3</td>
<td>1.68-1.50</td>
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<td>135</td>
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<td>140</td>
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<td>106.4</td>
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<td>103.4</td>
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<td>153.3-94.5</td>
<td>1.43-1.30</td>
<td>140.3-99.0</td>
<td>1.39-1.30</td>
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<td>125</td>
<td>127.0</td>
<td>102.5</td>
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<tr>
<td>P6 140.0-99.3</td>
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<td>147.7-104.9</td>
<td>1.30-1.24</td>
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<td>131.8</td>
<td>102.8</td>
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</tr>
<tr>
<td>P7 135.3-96.6</td>
<td>1.13-1.10</td>
<td>174.3-94.5</td>
<td>1.19-1.13</td>
<td>146.2-99.0</td>
<td>1.16-1.12</td>
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<td>131</td>
<td>125</td>
<td>130.0</td>
<td>97.5</td>
<td></td>
</tr>
<tr>
<td>P8 147.7-98.1</td>
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<td>183.2-96.0</td>
<td>1.21-1.15</td>
<td>152.1-97.5</td>
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<td>126</td>
<td>136</td>
<td>127</td>
<td>132.9</td>
<td>99.9</td>
<td></td>
</tr>
</tbody>
</table>
McDonald (27) and O'Rourke and Taylor (48) have reported that either norepinephrine infusion or an increase in mean blood pressure caused the ascending aorta to exhibit: (1) increases in the low frequency modulus values, (2) increases in the frequency at which minima occurred, and (3) a shift of the zero crossover toward higher frequencies.

Epinephrine infusion reduced the impedance modulus at 0 Hz for each position along the descending aorta because of relatively larger increases in flow than in pressure. The frequency at which the minima occurred was also reduced with epinephrine at each position indicating a more distant reflecting site. The low frequency modulus values were reduced from control with epinephrine for positions in the lower thoracic aorta and abdominal aorta but not appreciably changed from control for positions in the upper thoracic aorta. The depression of impedance found with epinephrine in the abdominal aorta may be due to the increase in diameter of the descending aorta which occurred with prolonged epinephrine infusion (Table 4). Table 4 presents the pressure and diameter ranges and mean pressure values for three experiments for positions along the descending aorta under control conditions during epinephrine infusion and with amyl nitrite inhalation. It is seen from this table that epinephrine infusion is associated with a consistent increase in the diameter of the descending aorta.

Examination of the pressure and diameter data in Table 4 shows that the pressure diameter relationship curve is shifted to the right under epinephrine infusion. This shift of the pressure diameter relationship curve to the right indicates that the aortic smooth muscle is responsible for the diameter change. If the pressure was responsible for the diameter change, the curve would not be shifted to the right but rather be moved to a different position on
the same curve.

Epinephrine infusion also reduced the phase angle values at each position along the descending aorta for frequencies in the 0-5.7 Hz range. Thus the flow in the descending aorta led the pressure by fewer degrees with epinephrine infusion than with control conditions. This fact could result from an aorta which was stiffer due to an increased load placed on the collagen fibers as a result of an increased aortic diameter brought about by epinephrine infusion. Burton (10) has suggested that at large diameters the collagen fibers primarily determine the distensibility of the aorta.

Amyl Nitrite

Prolonged inhalation of amyl nitrite caused the double minima impedance spectrum of the ascending aorta to be changed to one with only one single broad minimum. O'Rourke and Taylor (48) also showed the disappearance of the two minima with vasodilator drugs. The loss of this distinct two minima may have been caused by changes in the two reflection sites of the vascular system as discussed before. The phase angle spectra that were associated with the two minima in the modulus pattern of the ascending aorta was also altered with amyl nitrite inhalation. Under control conditions, the phase angle spectra contained a maximum and minimum associated with the first maxima and second minimum of the modulus spectra, respectively. Under amyl nitrite, the maximum was not present in the spectrum. The slope of the phase angle vs. frequency curve was also reduced with amyl nitrite inhalation over the 0-10 Hz range.

Determination of impedance changes in the descending aorta with amyl nitrite inhalation was difficult at best. The large increase in heart rate with amyl nitrite resulted in large gaps in the impedance spectrum where no intermediate frequency values were available.
Therefore, connection of the values which did exist could have resulted in erroneous interpretation of the spectra.

**Power**

**Control**

The total power values for the ascending aorta calculated in these experiments were greater than the values reported by others (14, 16, 44) in spite of our omission of the kinetic components. These kinetic components are small in comparison to the total and are almost entirely oscillatory in nature. The addition of the kinetic power values to our values would not appreciably alter the total power. It would, however, increase our oscillatory power values and thus bring the oscillatory power to total power ratio into closer agreement with the ratios reported by others (14, 16, 44) for the ascending aorta.

The equations used to calculate power in these experiments involve either the flow and pressure, or flow and impedance of the system. It was for this reason that when significant reductions in flow occurred, such as between the ascending and descending aorta (Figure 32) the power dissipation calculated for this segment was large (Figure 23). The flow reduction was also associated with a decrease in the diameter of the aorta (Table 5). This decrease in diameter was such that the velocity of flow did not change appreciably along this segment. The total power decrease that occurred in the aortic segment between mid thoracic levels and 6-9 cm above the diaphragm (Figure 22) was also accompanied by a flow reduction along this segment (Figure 32). Comparison of the per cent total power vs. position in the thoracic aorta curve (Figure 23) and the per cent flow reduction curve (Figure 33) showed that the flow and total power reductions are of the same magnitude and this reduction in total power values was, therefore,
Figure 32

Plot of the average blood flow vs. position in the aorta under control conditions. Positions in the aorta are as follows: AA: ascending aorta; DA: 1 cm below the aortic arch; 6: 6 cm below the aortic arch; MTA: mid thoracic aorta; 6-9: 6-9 cm above the diaphragm; 1-3: 1-3 cm above the diaphragm; BD: below the diaphragm and above the renal arteries; BR: below the renal arteries; and LO: lowest position measured.
Fig. 32: --Plot of Average Blood Flow vs. Position in the Aorta under Control Conditions
Table 5. -- Data are presented for nine areas in the aorta under control, epinephrine infusion, control, and amyl nitrite inhalation conditions. The average diameter in cm and standard error for the ascending aorta (AA), 1 cm below the aortic arch (DA), 6 cm below the aortic arch (6), mid thoracic aorta (MTA), 6-9 cm above the diaphragm (6-9), 1-3 cm above the diaphragm (1-3), below the diaphragm and above the renal arteries (BD), below the renal arteries (BR), and the lowest position measured (LO) are presented for each condition.

<table>
<thead>
<tr>
<th>Area</th>
<th>Control 1±SE</th>
<th>Epinephrine+SE</th>
<th>Control 2±SE</th>
<th>Amyl Nitrite+SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>AA</td>
<td>2.59±0.01</td>
<td>2.55±0.01</td>
<td>2.43±0.01</td>
<td>2.38±0.01</td>
</tr>
<tr>
<td>DA</td>
<td>1.61±0.08</td>
<td>1.72±0.13</td>
<td>1.59±0.08</td>
<td>1.60±0.09</td>
</tr>
<tr>
<td>6</td>
<td>1.50±0.08</td>
<td>1.57±0.11</td>
<td>1.51±0.05</td>
<td>1.44±0.08</td>
</tr>
<tr>
<td>MTA</td>
<td>1.38±0.04</td>
<td>1.44±0.06</td>
<td>1.34±0.04</td>
<td>1.30±0.06</td>
</tr>
<tr>
<td>6-9</td>
<td>1.24±0.08</td>
<td>1.27±0.10</td>
<td>1.20±0.07</td>
<td>1.20±0.11</td>
</tr>
<tr>
<td>1-3</td>
<td>1.23±0.06</td>
<td>1.28±0.09</td>
<td>1.19±0.06</td>
<td>1.20±0.04</td>
</tr>
<tr>
<td>BD</td>
<td>0.97±0.12</td>
<td>1.03±0.13</td>
<td>1.01±0.15</td>
<td>1.05±0.17</td>
</tr>
<tr>
<td>BR</td>
<td>0.90±0.09</td>
<td>0.94±0.09</td>
<td>0.86±0.09</td>
<td>0.88±0.09</td>
</tr>
<tr>
<td>LO</td>
<td>0.85±0.05</td>
<td>0.90±0.07</td>
<td>0.82±0.06</td>
<td>0.85±0.08</td>
</tr>
</tbody>
</table>
Figure 33

Plot of the percent reduction from ascending aorta blood flow vs. position in the aorta under control conditions. Positions in the aorta are as follows: AA: ascending aorta; DA: 1 cm below the aortic arch; 6: 6 cm below the aortic arch; MTA: mid thoracic aorta; 6-9: 6-9 cm above the diaphragm; 1-3: 1-3 cm above the diaphragm; BD: below the diaphragm and above the renal arteries; BR: below the renal arteries; and LO: lowest position measured.
Fig. 33. --Plot of the Per Cent Reduction from Ascending Aorta Blood Flow vs. Position in the Aorta under Control Conditions
attributable to the reduction in flow. This finding is not surprising since we are looking at a smaller and smaller peripheral bed as the catheter is withdrawn along the aorta.

The large rate of energy dissipation (power) that occurred as the aorta passed through the diaphragm (Figure 22) was also associated with a large reduction in flow (Figure 32) and diameter (Table 5). However, the small fluctuations of total power across the renal arteries and the lowest segment of the thoracic aorta (Figure 23) were not associated with a flow reduction (Figure 33) and with only small diameter changes (Table 5). These energy losses were probably the result of the drop in pressure along this segment.

Since the calculation of oscillatory power is dependent upon the square of the flow terms and the impedance (Equation 2.18), a change in either of these will result in changes in both the oscillatory power and oscillatory power to total power ratio. Consequently, the large changes in flow that occur between the ascending and descending aorta result in large changes in oscillatory power. Since the flow components are squared, changes in the magnitude of the flow terms have a greater effect on the oscillatory power than changes in impedance. This fact is particularly apparent in this aortic segment and is the reason for the decrease in the oscillatory power to total power ratio. The oscillatory power and oscillatory power to total power ratio decreased along the descending aorta to the mid thoracic region due to decreases in both the flow and impedance. The decrease in impedance was due mainly to the increase of the phase angle between pressure and flow thus causing the cosine to decrease. In the aortic segment below the mid thoracic level, a significant increase in oscillatory power occurred. Across this same segment there was a decreased flow
and an increased impedance. The increased impedance would result in an increased oscillatory power while the decreased flow should cause the power to decrease. These findings are paradoxical and for the present unexplainable.

Following the increase that occurred at the mid thoracic level for oscillatory power and oscillatory power to total power ratio these values decreased again with distance along the aorta. The large increases in impedance that occurred across the diaphragm and renal arteries were accompanied by reductions in flow that occurred across these segments resulting in decreased oscillatory power and oscillatory power to total power ratio.

Epinephrine

With epinephrine infusion the total rate of energy dissipation (power) in the cardiovascular system was increased by elevation of both the mean flow and mean pressure values. In order to analyze these changes in the system, the per cent in flow (Figures 33 and 34) and per cent change in power (Figures 23 and 26) for control and epinephrine infusion were examined.

Epinephrine resulted in less energy dissipation from ascending to descending aorta than under control conditions. The lower percentage dissipation in this segment is attributable to the smaller per cent reduction in flow that occurred across this segment with epinephrine.

The rate of energy dissipation in the segment of aorta above the mid thoracic range was considerably increased by epinephrine infusion. The reason for this greater drop in power was a large change in blood flow along that aortic segment. The aortic segment below the mid thoracic aorta also showed reductions in flow and thus power. These reductions were smaller under epinephrine than they were under control conditions.
Figure 34

Plot of the per cent reduction from ascending aorta blood flow vs. position in the aorta under epinephrine infusion. Positions in the aorta are as follows: AA: ascending aorta; DA: 1 cm below the aortic arch; 6: 6 cm below the aortic arch; MTA: mid thoracic aorta; 6-9: 6-9 cm above the diaphragm; 1-3: 1-3 cm above the diaphragm; BD: below the diaphragm and above the renal arteries; BR: below the renal arteries; and LO: lowest position measured.
Fig. 34. --Plot of the Per Cent Reduction from Ascending Aorta Blood Flow vs. Position in the Aorta under Epinephrine Infusion.
Epinephrine infusion increased the percentages of power loss as the aorta passed the diaphragm and the renal arteries as compared to control conditions. These larger power losses could also be accounted for by larger flow reductions.

The infusion of epinephrine did not alter the percentage of ascending aorta flow measured at the lowest position in the abdominal aorta. Epinephrine, however, increased the flow into the descending aorta and into those vascular beds draining the descending aorta from its origin to the lowest position measured. In other words, the flow distribution was altered by epinephrine. A larger percentage went to the upper portions of the thorax, the superior mesenteric artery, and the renal arteries and smaller percentages went to the head and upper extremities and the branches of the lower thoracic aorta. The loss in total power closely paralleled the changes in flow; however, the percentage of the ascending aorta's total power dissipated at the lowest aortic position measured was smaller under epinephrine infusion than under control conditions.

The increase in oscillatory power was relatively larger than the increase in total power when epinephrine was infused. The fact that there was a greater increase in oscillatory power than total power resulted in the oscillatory power to total power ratio at all positions in the aorta being higher with epinephrine infusion than under control conditions.

The increase in oscillatory power and thus oscillatory power to total power ratio that occurred under control conditions across the aortic segment below the mid thoracic area was less pronounced with epinephrine. However, the last segment in the thoracic aorta also exhibited an increased oscillatory power drop and oscillatory power to total power ratio. These findings cannot
be adequately explained at this time.

A third increase in the oscillatory power to total power ratio occurred as the aorta passed through the diaphragm. The oscillatory power in this segment was reduced but the total power was reduced proportionately more resulting in the increased oscillatory power to total power ratio. The smaller change in oscillatory power with epinephrine resulted from a smaller change in impedance across this segment. The increase in impedance offset the increase in flow and caused a smaller change in the oscillatory power across this segment with epinephrine infusion.

The decreased oscillatory power to total power ratio that occurred below the renal arteries resulted from a larger reduction of oscillatory power than total power. The oscillatory power decrease across the renal arteries was greater with epinephrine than under control conditions due to the greater reduction in flow that occurred in this segment with epinephrine.

Amyl Nitrite

Examination of Figures 27 and 28 showed that the total power is reduced from control levels at all positions in the aorta by inhalation of amyl nitrite. This reduction was the result of relative decrease in mean pressure for all positions and a relatively greater reduction of flow at some positions. The total flow into the ascending aorta was not appreciably changed from control by amyl nitrite (Figures 36 and 37), however, the reduced mean pressure (Table 4) resulted in less total energy available for flow. The flow into the descending aorta was below the control value as were the flow values for all positions in the upper and middle thoracic aorta. These reductions in flow and pressure (Table 4) under amyl nitrite resulted in a reduced loss of total power along the upper and middle thoracic aorta. The flow at any
Figure 35

Plot of the average blood flow vs. position in the aorta under epinephrine infusion. Positions in the aorta are as follows: AA: ascending aorta; DA: 1 cm below the aortic arch; 6: 6 cm below the aortic arch; MTA: mid thoracic aorta; 6-9: 6-9 cm above the diaphragm; 1-3: 1-3 cm above the diaphragm; BD: below the diaphragm and above the renal arteries; BR: below the renal arteries; and LO: lowest position measured.
Fig. 35. -- Plot of Average Blood Flow vs. Position in the Aorta under Epinephrine Infusion
Figure 36

Plot of average blood flow vs. position in the aorta under control conditions. Positions in the aorta are as follows: AA: ascending aorta; DA: 1 cm below the aortic arch; 6: 6 cm below the aortic arch; MTA: mid thoracic aorta; 6-9: 6-9 cm above the diaphragm; 1-3: 1-3 cm above the diaphragm; BD: below the diaphragm and above the renal arteries; BR: below the renal arteries; and LO: lowest position measured.
Fig. 36.--Plot of Average Blood Flow vs. Position in the Aorta under Control Conditions
Figure 37

Plot of the average blood flow vs. position in the aorta under amyl nitrite inhalation. Positions in the aorta are as follows: AA: ascending aorta; DA: 1 cm below the aortic arch; 6: 6 cm below the aortic arch; MTA: mid thoracic aorta; 6-9: 6-9 cm above the diaphragm; 1-3: 1-3 cm above the diaphragm; BD: below the diaphragm and above the renal arteries; BR: below the renal arteries; and LO: lowest position measured.
Fig. 37. -- Plot of Average Blood Flow vs. Position in the Aorta under Amyl Nitrite Inhalation
position along the lower thoracic aorta and abdominal aorta was not appreciably changed (Figures 36 and 37) from control by amyl nitrite, thus the reduced pressure was the major reason for less power being present in these segments.

The percentage of total power in the ascending aorta that was dissipated along the whole length of the aorta to the lowest position examined was not altered from control by amyl nitrite (Figures 29 and 30). There was, however, a redistribution of the flow and the power dissipation changed parallel with the flow. The percentage dissipation from ascending to descending aorta was increased due to a decreased flow while the percentages dissipated in the upper thoracic aorta appeared to vary. The fluctuation in the total power curve (Figure 25) and thus per cent dissipation curve (Figure 30) in the upper thoracic aorta under control conditions may have been the result of variations in flow and pressure along this segment (Figure 38). Since the recordings at different positions were taken at different times variations in mean pressures may have occurred between recordings.

From the mid thoracic area to the lower thoracic area the smaller decline in power dissipation resulted from smaller flow losses. Only slight changes in power dissipation from control levels are seen with amyl nitrite in the lower thoracic and abdominal aorta. This fact is explained by the lack of major changes in flow distribution in these areas.

Since the calculation of oscillatory power has the impedance of the vessel as one of its major determinants, the deviation in impedance from control is a major cause of changes in oscillatory power. Due to the great difficulty in determining impedance changes under amyl nitrite conditions as explained earlier, no firm explanation of oscillatory power changes and oscillatory power to total power ratio changes with amyl nitrite can be derived from the data.
Figure 38

Plot of the per cent reduction from ascending aorta blood flow vs. position in the aorta under control conditions. Positions in the aorta are as follows: AA: ascending aorta; DA: 1 cm below the aortic arch; 6: 6 cm below the aortic arch; MTA: mid thoracic aorta; 6-9: 6-9 cm above the diaphragm; 1-3: 1-3 cm above the diaphragm; BD: below the diaphragm and above the renal arteries; BR: below the renal arteries; and LO: lowest position measured.
Fig. 38. -- Plot of the Per Cent Reduction from Ascending Aorta Blood Flow vs. Position in the Aorta under Control Conditions
Figure 39

Plot of the per cent reduction from ascending aorta blood flow vs. position in the aorta under amyl nitrite inhalation. Positions in the aorta are as follows: AA: ascending aorta; DA: 1 cm below the aortic arch; 6: 6 cm below the aortic arch; MTA: mid thoracic aorta; 6-9: 6-9 cm above the diaphragm; 1-3: 1-3 cm above the diaphragm; BD: below the diaphragm and above the renal arteries; BR: below the renal arteries; and LO: lowest position measured.
Fig. 39. --Plot of the Per Cent Reduction from Ascending Aorta Blood Flow vs. Position in the Aorta under Amyl Nitrite Inhalation
Table 6. Data are presented for nine areas in the aorta under control, epinephrine infusion, control, and amyl nitrite inhalation conditions. The average blood flow in ml/sec and standard error for each of the areas are presented for each condition. The areas are as described for Figure 5.

<table>
<thead>
<tr>
<th>Area</th>
<th>Control ±SE</th>
<th>Epinephrine ±SE</th>
<th>Control 2 ±SE</th>
<th>Amyl Nitrite ±SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>AA</td>
<td>82.43 ± 3.15</td>
<td>93.48 ± 4.51</td>
<td>59.00 ± 2.12</td>
<td>57.22 ± 1.92</td>
</tr>
<tr>
<td>DA</td>
<td>56.67 ± 6.74</td>
<td>80.50 ± 19.50</td>
<td>57.67 ± 7.62</td>
<td>50.00 ± 3.61</td>
</tr>
<tr>
<td>6</td>
<td>56.00 ± 10.79</td>
<td>78.00 ± 13.58</td>
<td>56.33 ± 10.04</td>
<td>46.00 ± 3.05</td>
</tr>
<tr>
<td>MTA</td>
<td>53.50 ± 4.57</td>
<td>63.25 ± 7.04</td>
<td>48.00 ± 6.19</td>
<td>43.75 ± 3.92</td>
</tr>
<tr>
<td>6-9</td>
<td>43.33 ± 4.26</td>
<td>62.33 ± 10.93</td>
<td>43.67 ± 2.73</td>
<td>42.00 ± 1.00</td>
</tr>
<tr>
<td>1-3</td>
<td>43.67 ± 4.10</td>
<td>61.33 ± 12.12</td>
<td>40.00 ± 6.81</td>
<td>40.67 ± 6.69</td>
</tr>
<tr>
<td>BD</td>
<td>26.50 ± 7.50</td>
<td>38.50 ± 13.50</td>
<td>31.00 ± 7.00</td>
<td>32.00 ± 6.00</td>
</tr>
<tr>
<td>BR</td>
<td>26.67 ± 8.19</td>
<td>30.33 ± 4.70</td>
<td>26.67 ± 4.06</td>
<td>24.00 ± 2.31</td>
</tr>
<tr>
<td>LO</td>
<td>27.50 ± 5.50</td>
<td>30.50 ± 5.50</td>
<td>24.50 ± 1.50</td>
<td>23.00 ± 5.00</td>
</tr>
</tbody>
</table>
Table 7.—Data are presented for the per cent reduction from ascending aorta levels of blood flow at eight positions in the aorta under control, epinephrine infusion, control and amyl nitrite inhalation conditions.

<table>
<thead>
<tr>
<th>Position</th>
<th>Control 1</th>
<th>Epinephrine</th>
<th>Control 2</th>
<th>Amyl Nitrite</th>
</tr>
</thead>
<tbody>
<tr>
<td>Descending Aorta</td>
<td>31.25</td>
<td>13.89</td>
<td>2.25</td>
<td>12.62</td>
</tr>
<tr>
<td>6 cm Below Aortic Arch</td>
<td>32.06</td>
<td>16.56</td>
<td></td>
<td>19.61</td>
</tr>
<tr>
<td>Mid Thoracic Aorta</td>
<td>35.10</td>
<td>32.34</td>
<td>18.64</td>
<td>23.54</td>
</tr>
<tr>
<td>6-9 cm Above Diaphragm</td>
<td>47.43</td>
<td>32.25</td>
<td>25.98</td>
<td>26.60</td>
</tr>
<tr>
<td>1-3 cm Above Diaphragm</td>
<td>47.02</td>
<td>34.39</td>
<td>32.20</td>
<td>28.92</td>
</tr>
<tr>
<td>Below Diaphragm and Above Renal Arteries</td>
<td>67.85</td>
<td>58.81</td>
<td>47.46</td>
<td>44.08</td>
</tr>
<tr>
<td>Below Renal Arteries</td>
<td>67.65</td>
<td>67.55</td>
<td>54.80</td>
<td>58.06</td>
</tr>
<tr>
<td>Lowest Position</td>
<td>66.64</td>
<td>67.37</td>
<td>58.47</td>
<td>59.80</td>
</tr>
</tbody>
</table>
CHAPTER V
CONCLUSIONS

The impedance modulus was found to change smoothly from position to position along the length of the descending aorta investigated. The magnitude of these changes was increased for the aortic segments as the diaphragm was crossed and for those aortic segments below the diaphragm.

The ascending aorta exhibited an impedance modulus pattern which had either a broad flat minimum or two distinct minima below 10 Hz. The presence of either of these patterns appeared to depend upon the state of the vascular beds as amyl nitrite inhalation resulted in a change of the double minima pattern to a broad flat minimum pattern.

The infusion of epinephrine caused the frequency at which the minima occurred to become less and also decreased the peripheral resistance values. Epinephrine had no other effects on the impedance of the ascending aorta. The impedance moduli of the lower thoracic and abdominal aorta were reduced with epinephrine infusion.

The power dissipation of the aorta was found to be proportional to flow changes. The efficiency with which the aorta accepted flow was found to vary along the descending aorta. The infusion of epinephrine increased the flow and the power dissipation at all positions in the aorta. Epinephrine also resulted in a redistribution of the flow and power dissipation characteristics along the aorta. The efficiency with which the aorta accepted oscillatory flow was also found to be reduced with epinephrine infusion.
APPENDIX I

01.10 O S;O C
01.20 T !;A "BLOCK NUMBER", B1
01.40 T !;"STARTING LOC ENDING LOC", !
01.50 T !;A "", S1
01.60 T "";A "", S2
01.70 T !;A "TRANSFER LOC", B2
01.71 S X=FX()
01.72 D 1.82
01.73 D 1.83
01.74 D 1.84
01.80 D 5;D 6
01.81 G 1.85
01.82 T !;"DATA TRANSFERRED FROM BLOCK #", %3, B1
01.83 T " TO BLOCK #", %3, B2
01.84 T " HEART RATE IS", %3.02, 500/(S2-S1), "CYCLES/SECOND", !
01.85 L O, F1, I, #000, 1
01.90 F N=O, 511; D 2
01.95 L C, F1
01.98 G

02.10 S K=FITR(N*(S2-S1)/512); S H=256*B1+K+S1; S M=256*B2+N
02.20 S F1(M)=FO(H)+(FO(H+1)-FO(H))*(N*(S2-S1)/512-K)

05.10 S X=FITR(B1/100); S Y=FITR((B1-X*100)/10); S Z=FITR(B1-X *
*100-Y*10)
05.20 S B1=Z+Y*8+X*64

06.10 S X=FITR(B2/100); S Y=FITR((B2-X*100)/10); S Z=FITR(B2-X *
*100-Y*10)
06.20 S B2=Z+Y*8+X=64
APPENDIX II

01. 10 L O, F0, I, #002, 1
01. 13 S A=0
01. 14 O S;O C;T !, "";A "FILTER FACTOR", O
01. 15 F I=0, 511;D 2
01. 20 S HM=A/512;S A=0
01. 25 F I=512, 1023;D 2
01. 30 S ZN=A*O/512;S A=0
01. 35 L C, F0
01. 36 L O, F0, I, #011, 1
01. 40 F I=0, 511;D 2
01. 45 S XP=A/512;S A=0
01. 50 F I=512, 1023;D 2
01. 55 S UV=A*O/512;S A=0
01. 60 L C, F0
01. 65 O S;O C;T !, "TYPE L O, F0, I, #BLK, 1;G 3. 10", !
01. 70 Q
02. 10 S A=A+F0(I)
03. 10 O C;T !, "MAX PRESSURE MAX LIN FLOW MAX DIAMETER", !
03. 15 T "# OF BLOCKS/RUN STARTING BLK # FOR ANALYSIS", !
03. 20 T !;A "", P
03. 22 T "";A "", V
03. 24 T "";A "", D
03. 26 T "";A "", B
03. 28 T "";A "", N
03. 35 G 5. 10
03. 37 O S;O C;T "";A "APPARENT ZERO FLOW PRINTER LISTING*
  WATCH*YOU MIGHT NEED TO SUGGEST IF NO ZERO FLOW
  REALLY OCCURRED", C
03. 38 S A=0
03. 50 F I=0, 511;D 2
03. 55 S X=FX(); T !, "DATA AT BLOCK ", %3. 0, N, !
03. 57 S A=A-512*HM
03. 60 T %4. 01, (A*P)/(512*(XP-HM)), "
  AVERAGE
  PRESSURE", !
03. 62 S P=A/512
03.65 S A=0
03.67 F I=(256*B), (256*B+511); D 2
03.70 T %4.01, (A-C*512)*D*D*V*O. 7856/(512*(UV-ZN)), "AVERAGE FLOW", !
03.72 S V=(A/512)-C
03.73 T !!!, %4.01, P, "TAPE VALUE", %4.01, V, "TAPE VALUE", !
03.75 G 1. 65

05.10 S X=FX(); S M=0; T !
05.20 F I=(256*B), (256*B+511); D 6
05.30 G 3.37

06.10 T %3, F0(I); S M=M+1
06.20 I (M-24)6.30, 6.40, 6.40
06.30 R
06.40 S M=0; T !
APPENDIX III

01.01 C THIS IS $IMPCALC
01.03 O S;O C;A "TYPE BLOCK ", P2
01.05 S X=FX();T !," F MAG PHASE PRES FLOW", !
01.07 T "-------------------------- DATA AT
B O L K ",%3, P2, !
01.12 O S;T !;A "HEART RATE", P2
01.15 T "ACTUAL AV PRES ACTUAL AV FLOW", !
01.16 T "AV PRES ON TAPE AV FLOW ON TAPE", !
01.17 T !;A " ", T1
01.18 T " ";A " ", T2
01.19 T !;A " ", Y
01.20 T " ";A " ", Z
01.21 T !;A "COR FAC FOR PRES", H1;T !;A "COR FAC FOR FLOW", H2
01.22 S X=FX()
01.23 S N=0;S W=0
01.24 T %3, 02, N, " ",%5, 01, T l*1332. 8/T2, " ",%4, N, " ", T1, " ", T
2, !
01.25 S N=1;S PI=3.1416;D 2
01.30 S P1=P;D 3
01.40 F N=2, 40/P2;D 2;D 3
01.42 S F0(512)=900;S F0(1024)=450;S F0(1025)=-450
01.45 F I=W, 511;S F0(I+512)=0
01.46 F I=W, 511;S F0(I+1024)=0
01.48 T "", !
01.50 L C, FO
01.60 O S;O C;T ""TYPE L O, F0, I#BLK, 1;G
01.70 Q

02.20 S M1=FSQT(F0(383+N) 2+F0(447+N) 2)
02.30 S M2=FSQT(F0(127+N) 2+F0(191+N) 2)
02.32 I (M2)2. 33, 2. 33, 2. 35
02.33 T I, "ZERO FLOW*INFINITE IMPED", !
02.34 S M=10;G 2. 40
02.35 S M=M1/M2
02.40 S C0=F0(383+N);S S0=F0(447+N);D 4
02.45 S P=P0
02. 60 S C0=F0(127+N); S S0=F0(191+N); D 4
02. 65 S P=P-P0; S P=P*(180/P1); R

03. 10 I (P-180)3. 15, 3. 20, 3. 20
03. 15 I (P+180)3. 30, 3. 50, 3. 50
03. 20 S P=P-360; G 3. 50
03. 30 S P=P+360+P; G 3. 50
03. 54 S K=N*P2*12-FITR(N*P2*12)
03. 58 I (K-. 5)3. 62, 3. 70
03. 62 S K=FITR(N*P2*12)
03. 66 S F0(K+512)=M*T1*Z*H21332. 8/T2*Y*H1*5; S F0(K+1024)=P*5
03. 68 F I=W, K-1; S F0(I+512)=0; S F0(I+1024)=0
03. 69 S W=K+1; R
03. 70 S K=FITR(N*P2*12)+1; G 3. 66

04. 09 I (C0)4. 10, 4. 20, 4. 30
04. 10 S C0=-C0
04. 11 I (S0)4. 12, 4. 15, 4. 17
04. 12 S S0=-S0
04. 13 S P0=PI+FATN(S)/C0); R
04. 15 S P0=PI; R
04. 17 S P0=PI-FATN(S0/C0); R
04. 20 I (S0)4. 21, 4. 24, 4. 27
04. 21 S P0=3*PI/s; R
04. 24 S P0=0; R
04. 27 S P0=PI/2; R
04. 30 I (S0)4. 31, 4. 34
04. 31 S S0=-S0
04. 32 S P0=2*PI-FATN(S0/C0); R
04. 34 S P0=FATN(S0/C0); R
BIBLIOGRAPHY


43. O'Rourke, M. F. Pressure and flow waves in systemic arteries and the anatomical design of the arterial system. J. Appl. Physiol. 23(2): 139, 1967.


