MASSACHUSETTS INSTITUTE OF TECHNOLOGY

Electrical and Mechanical Engineering Departments

6.022J/2.792J: Quantitative Physiology: Organ Transport Systems

PART I: Cardiovascular Physiology

A. FUNCTION OF THE CIRCULATION

Transport: oxygen/CO_2; nutrients; waste products; heat

Communication: Hormones

Heat Exchanger

Protection: Clotting mechanisms; WBC's, anti-bodies

B. COMPONENTS OF THE C.V. SYSTEM

Pumps, conduits, exchangers, reservoirs, fluid medium

Division into high-pressure delivery system and low-pressure capacitance system (See Figure 1).

Organization of components (See Figure 2).

Typical dimensions, velocities, continuity equation (See Figure 3, Table 1).

Perfusion and O_2 uptake of representative organs (See Figure 4.

Typical Normal Values (See Table 2).

C. FUNCTIONAL ANATOMY OF THE HEART

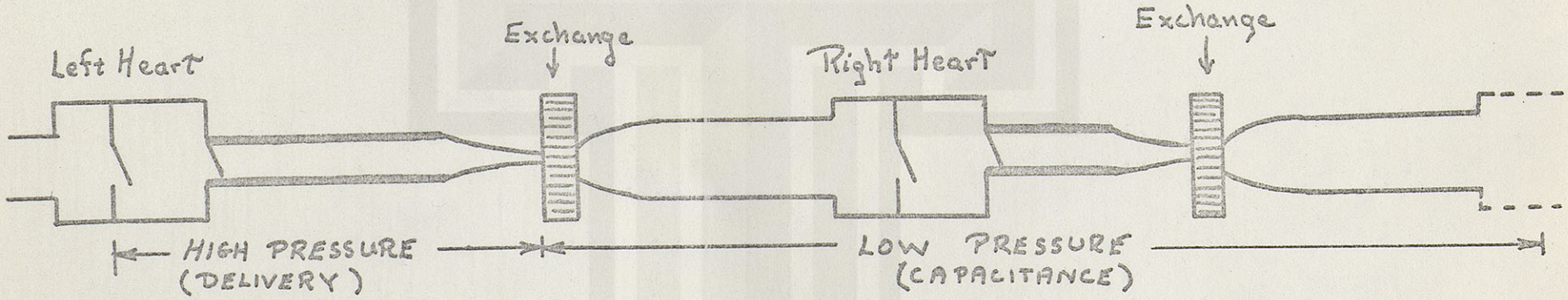
Anatomical Landmarks

Conduction System

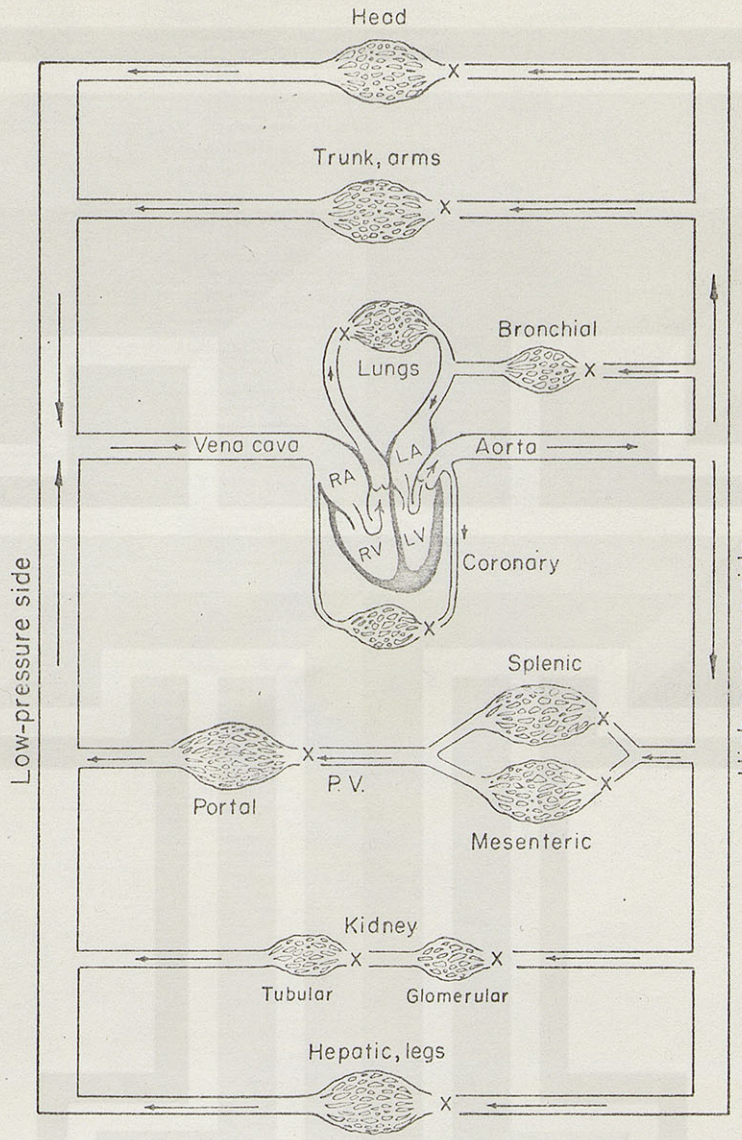
Cardiac Cycle and function of valves (See Figure 5).

Heart Sounds.

FIG. 1 SCHEMA OF THE CIRCULATION



FLOW RATE 5–30 L/min.



Arrangement of the parallel routes by which the circulation passes from the aorta to the ve cava. Representatives of the different categories of route discussed in the text are indicated. The ×'s in cate the location of control points where arterioles may control the flow. *RA*, right atrium; *LA*, left atriu *RV*, right ventricle; *LV*, left ventricle; *PV*, portal vein. (From Green, H. D.: Circulation: Physical prin ples, in Glasser, O. [ed.]: *Medical Physics*, Vol. 1 [Chicago: The Year Book Publishers, Inc., 1949], p. 21 Original illustration kindly furnished by H. D. Green.)

Figure 2

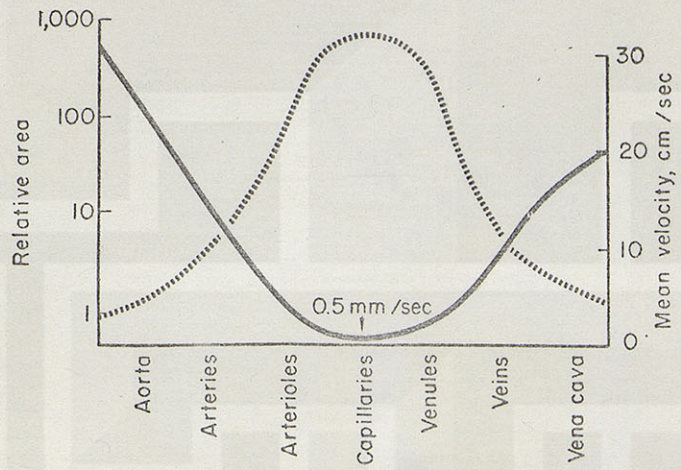
TABLE 1

TABLE 1—GEOMETRY OF MESENTERIC VASCULAR BED OF THE DOG*

KIND OF VESSEL	DIAMETER (Mm)	No.	TOTAL CROSS-SECTIONAL AREA (Cm^2)	LENGTH (Cm)	TOTAL VOLUME (Cm^3)	
Aorta	10	1	0.8	40	30	
Large arteries	3	40	3.0	20	60	
Main artery branches	1	600	5.0	10	50	
Terminal branches	0.6	1,800	5.0	1	25	
Arterioles	0.02	40,000,000	125	0.2	25	
Capillaries	0.008	1,200,000,000	600	0.1	60	
Venules	0.03	80,000,000	570	0.2	110	
Terminal veins	1.5	1,600	30	1	30	
Main venous branches	2.4	600	27	10	270	740
Large veins	6.0	40	11	20	220	
Vena cava	12.5	1	1.2	40	50	
					930	

*Data of F. Mall.

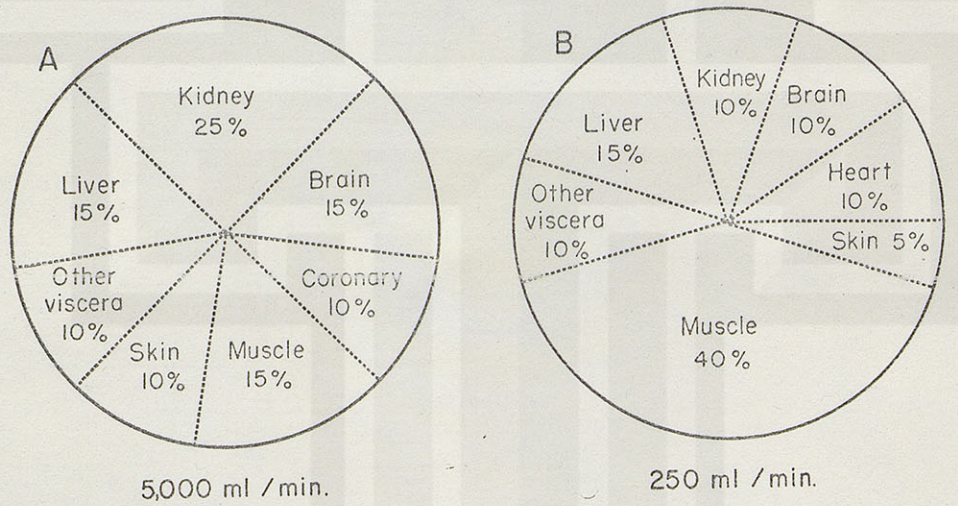
Figure 3



Schematic graph showing *broken line*, the changes in relative total cross-sectional area (on a logarithmic scale) of the vascular bed; *solid line*, the mean velocity in the different categories of vessel.

Figure 4

Fig. 4 Estimated distributions of cardiac output (A) and oxygen consumption (B) to different organs of the body in a man at rest. The estimates are very rough, from data taken from many sources and not very consistent. The kidney is greatly overperfused; the muscles, underperfused. In exercise the proportion of blood flow to muscle increases enormously, as it does for skin in hot environments.



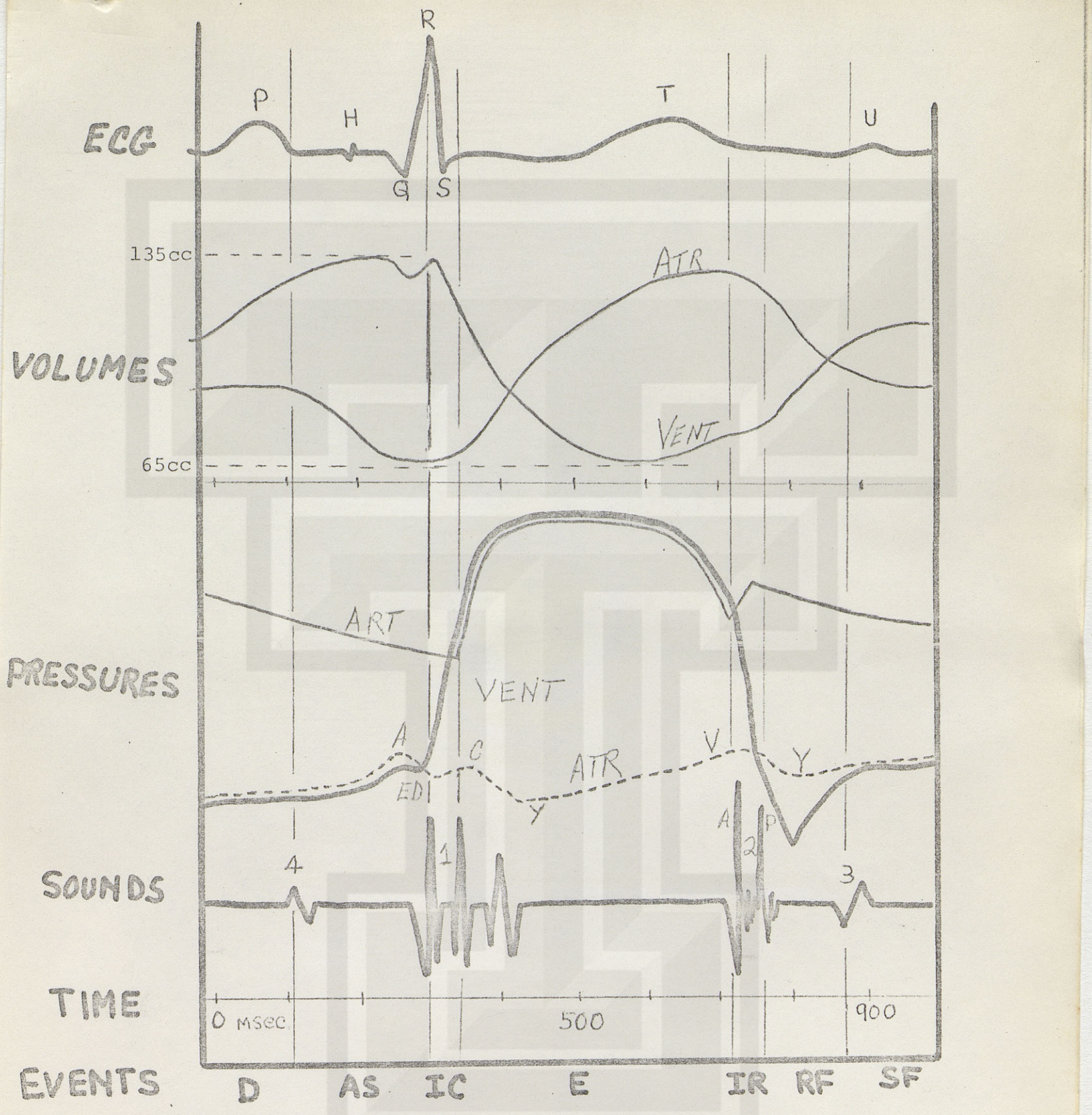


FIGURE 5 - THE CARDIAC CYCLE

TABLE 2

Representative Values for Human Circulation

Cardiac Output:	5 Liters/min. (resting) 15-25 Liters/min. (exercise)	
Heart Rate:	60-80 beats/min. (resting) 120-160 beats/min. (exercise)	
Stroke Volume	70cc. (resting) 160cc. (exercise)	
Pressures:	Aortic Phasic	120/80 mmHg.
	Mean	100 mmHg.
	Pulmonary Artery	25/10
	Mean	15
	Venous Mean	5
	Intrathoracic	-5
	1 mmHg. = 1330 dynes/cm^2	
Dimensions: (diameters)	Aorta	2.5 cm.
	Medium Artery	0.5 cm.
	Arteriole	30 - 60 μ
	Capillary	8 μ
	Vein (Medium)	0.5 cm.
	Vena Cava	3.0 cm.
	Red Blood Cell	7 μ
Velocities: (approximate)	100 cm/sec. peak in aorta 0.5-1 mm/sec. in capillaries 20 cm/sec. in vena cava	
Viscosities:	Water = 1.0 centipoise Plasma 1.5 centipoise Whole blood 4.0 centipoise	
	1 centipoise = 10^{-2} dyne-sec/cm.^{2}	
Resistance:	Total Pulmonary	150 dyne-sec. cm.$^{-5}$
	Systemic	1500 dyne-sec. cm.$^{-5}$

MASSACHUSETTS INSTITUTE OF TECHNOLOGY
Electrical and Mechanical Engineering Departments

6.022J/2.792J: Quantitative Physiology: Organ Transport Systems

PROBLEM SET #1

Assigned: February 7, 1974
Due: February 14, 1974

Problem 1:

I. Figure 1A shows the pressure waveforms in the left and right ventricles, the aorta, and pulmonary artery. (Note the two ordinate scales). The cardiac output was determined to be 5.0 liters per minute. Using the simple Windkessel model for the circulation:

a) Calculate the mean power dissipation in the peripheral circulation in watts. Do the same for the pulmonary circulation. What is the total average power dissipation? (Note: 1 dyne-cm/sec. = 10^{-7} watts; 1 mmHg = 1330 dynes/cm^2).

b) Estimate values for the resistance and capacitance in the Windkessel model, and calculate the time constant, $\tau = RC$.

c) Construct the pressure-volume loop (i.e. work diagram) for the left ventricle. Label the four major phases of the contraction sequence.

d) Using the pressure-volume loop, estimate the average power output of the left ventricle. Assume an end-diastolic volume of 130 cc. How does it compare to the power dissipated peripherally?

II. Figure 1B shows the left ventricular and aortic pressures from a patient with aortic stenosis. The cardiac output is again 5 liters/min.

a) Estimate the average power dissipation in the systemic circulation in watts.

b) Construct the pressure-volume loop for the left ventricle, assuming an end diastolic volume of 150cc.

c) Estimate the average power output from the left ventricle, and compare this with the power dissipated peripherally. Comment on the discrepency.

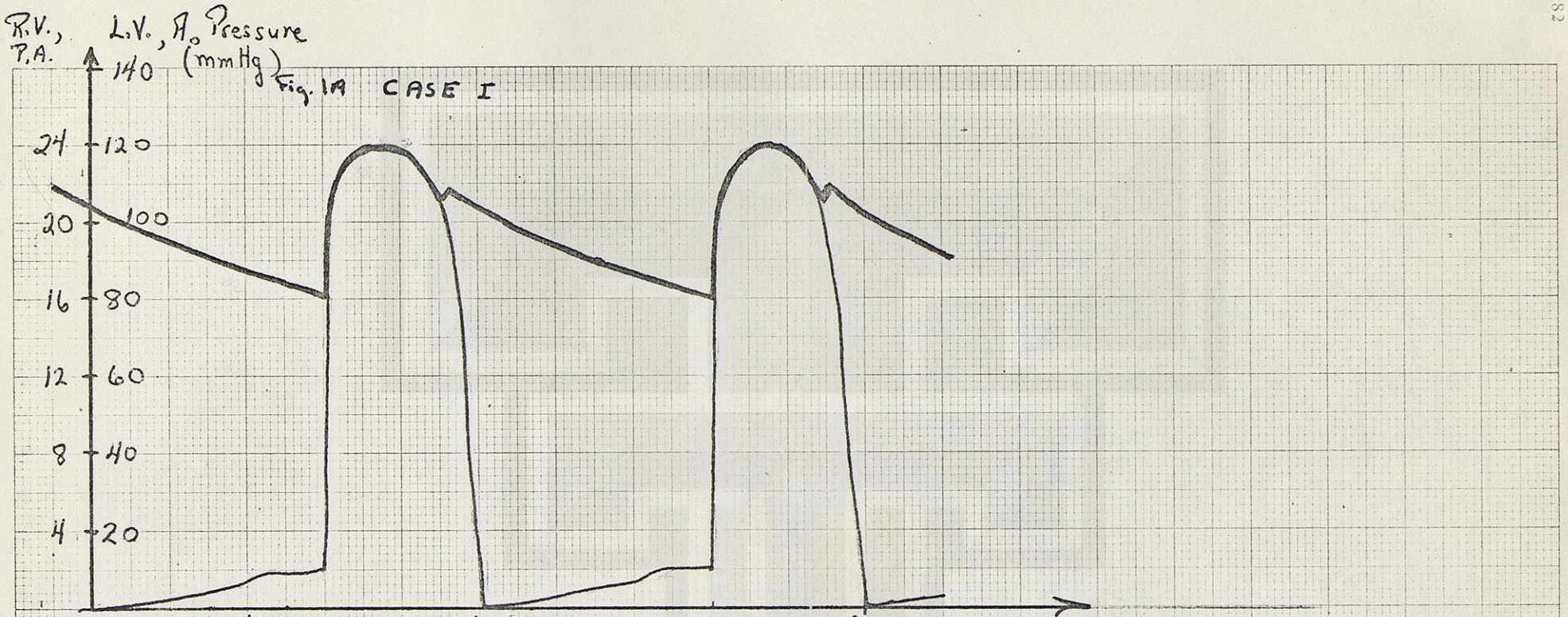


Fig. 1A CASE I

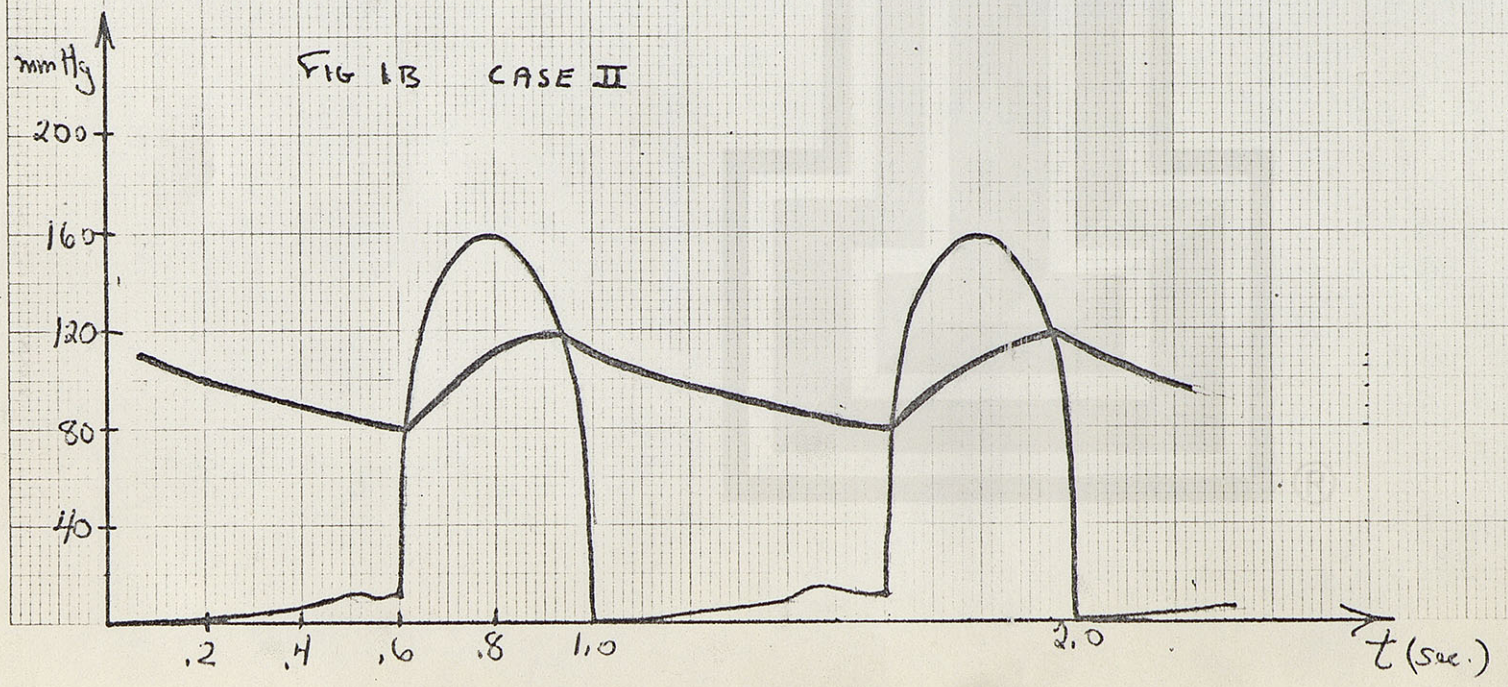


FIG 1B CASE II

Problem 2:

The following heart sounds and murmurs result from certain specific anatomic abnormalities. From your reading and knowledge of the physical determinants of heart sounds, present a brief and logical explanation of the following findings.

a) The heart rhythm is completely irregular--varying intervals between beats. The intensity of the first heart sound varies form beat to beat. Careful observation reveals that the intensity of the first heart sound is correlated with the previous inter-beat interval. Would you expect shorter intervals to be associated with louder or softer first heart sounds? Why?

b) What effects would elevated pulmonary artery blood pressure have on the components of the second heart sound? Why?

c) The first and second heart sounds are normal, with normal splitting of A_2 and P_2 with respiration. There is a systolic high-pitched blowing murmur approximately equally loud throughout systole. It is heard best over the lower sternum, and radiates upward toward the base of the heart. It gets louder during inspiration, and it is noted that the venous pressure in the neck increases noticeably during systole.

d) The first heart sound is normal. The second heart sound is single, with no splitting heard. It was loudest at the left second intercostal space. There was a short "diamond-shaped" systolic ejection murmur heard over the right 2nd intercostal space which radiated into the carotid arteries. There was also a diastolic murmur, high-pitched and decreasing in intensity during diastole. It was heard best over the right sternal border.

Problem 3:

I. A simple lumped parameter model of the circulation is the so-called "Windkessel" model shown in Figure 1. C represents the arterial capacitance, and R the peripheral resistance. The heart is represented by the current source I(t) which generates a train of impulses of area ΔQ (corresponding to stroke volume) at a frequency, f. In the model i(t) represents blood flow in the aorta, and V(t) represents arterial pressure.

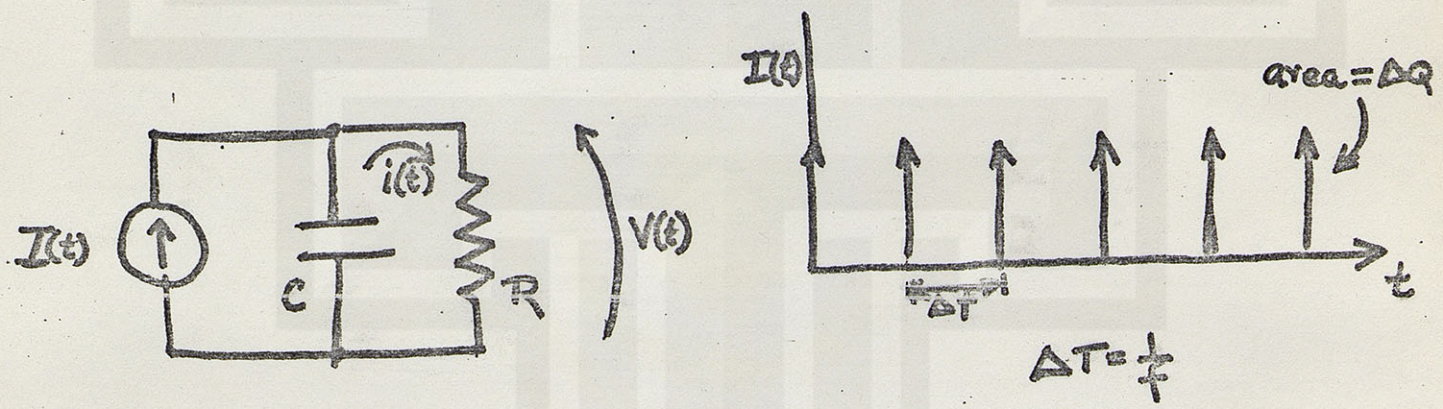


Figure 1

a) Using this model, express the steady-state arterial pressure v(t) in terms of R, C, ΔQ and f.

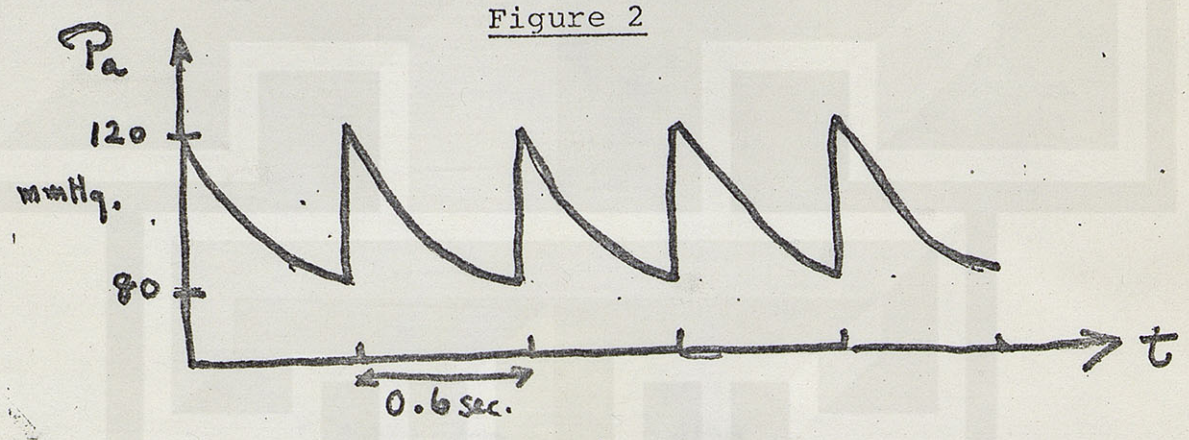
b) Derive an expression for the systolic pressure, diastolic pressure, and mean arterial pressure.

c) Sketch the general shape of the curves relating the above pressures to stroke volume, heart rate and peripheral resistance.

d) How does pulse pressure (systolic-diastolic) vary with arterial capacitance?

II. During a heart catheterization, aortic pressure was measured continuously. A measurement of cardiac output was made and was determined to be 5500 cc/min. The aortic pressure tracing taken at the same time is shown in Figure 2.

Figure 2



a. Calculate the lumped arterial capacitance, $\frac{\Delta Q}{\Delta P}$, for the equivalent Windkessel model.

b. Calculate the peripheral resistance, R_p.

c. Having "calibrated" the system once, and assuming the value of C to be essentially constant throughout the remainder of the catheterization beat-by-beat blood pressure may be obtained. Under what conditions might it <u>not</u> be reasonable to assume a constant C?

Problem 4:

a) From Table I enclosed, calculate typical Reynolds numbers for peak systolic flow and mean flow Reynolds numbers for the major elements of the systemic circulation.

b) Suppose one of the main arteries, e.g. the femoral artery, is reduced in diameter by the presence of severe atherosclerosis; if the constriction is localized so that the constricted diameter, d, is 1/3 of the undisturbed diameter, D, what is the characteristic Reynolds number at the peak of systole of the fluid flowing through the constricted region?

TABLE 1

Some quantities relevant to the circulation

Blood volume	6 litres
Number of red cells	5×10^6 mm^{-3}
Number of white cells	10^4 mm^{-3}
Specific gravity of blood	1·06
Viscosity of blood	Variable. 0·03 poise in large tubes
Cardiac output	6 litres min^{-1}
Heart rate	80 beats min^{-1}
Stroke volume	70 ml
Duration of systole	0·3 s
Duration of diastole	0·5 s

Dimensions of the circulatory bed

Vessels	Pressures (mmHg)	Diameter (cm)	Blood velocity (cm s^{-1})	Contained volume (ml)
Aorta and large arteries	120/80 mean 100	3–1	100/0 mean 25	300
Small arteries	120/70 mean 90	1–0·1	10–1	400
Capillaries	20	0·0008	0·05	300
Small veins	10	0·5	1	2300
Main veins	0–5	4	0/25 mean 10	900
Heart	—	—	—	360 (diastole)
Pulmonary artery	25/10 mean 15	4	50/0 mean 15	130
Pulmonary capillaries	5	0·001	0·02	110
Pulmonary veins	0	1	10	200

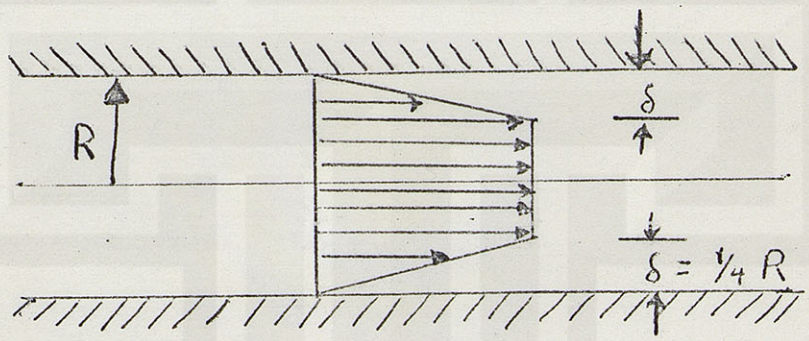
The figures are very approximate idealizations for an adult man.

Problem 5:

I. From the data of Table 1, estimate the mean and peak shear rates in the aorta, major arteries, and arterioles using:

a) A Poiseuille velocity profile

b) A top-hat profile as shown below.



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Supplementary Notes on Hemodynamics excerpted from HST 090 Notes.

February 8, 11, 1974
Lectures 3,4,

Cardiovascular Pathophysiology
HST 090 Dr. Mark

HEMODYNAMICS AND THE PERIPHERAL CIRCULATION

Part II: Hemodynamics

I. Introduction

A detailed examination of blood flow in the peripheral circulation reveals enormous variety and complexity. Such phenomena as pulsatile flow in viscous elastic vessels; propagation of waves in multiply-branched, tapered elastic conduits; capillary flow; anomalous viscosity of blood; turbulence; etc., are all observed in the cardiovascular system. A considerable effort has been expended at developing quantitative models for many of these phenomena by numerous investigators (references 1,2,4,6,7,8,10). Although the detailed description of cardiovascular fluid mechanics can become quite complex, it is quite possible to gain considerable insight using rather simple models. Our objectives will be to explore some of these simple models since they are quite helpful in understanding the normal and pathologic physiology of the cardiovascular system.

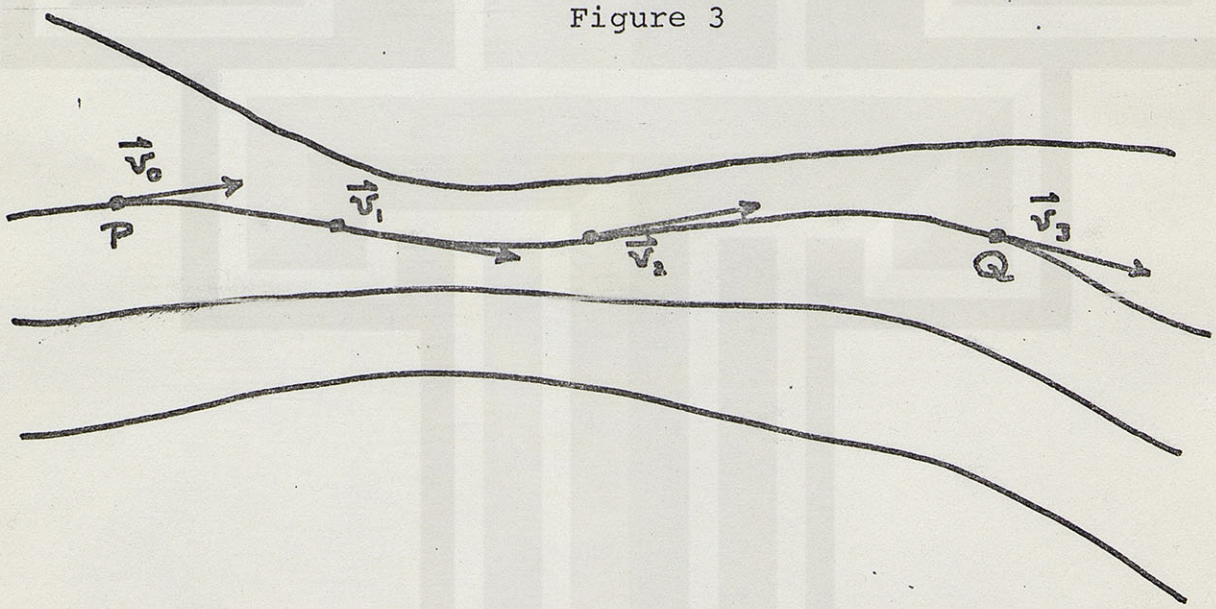
II. Stationary Flow.

At every point in a flowing liquid we can define the velocity, a vector which in general is a function of both position and time, $\vec{V}(x,y,z,t)$. In <u>stationary</u> or <u>steady</u> flow the velocity vectors do not change with time, and hence are functions only of location.

At any point P of space, then, the particle at that point possesses the same vector velocity, $\vec{V}_o$, no matter at what instant of time it is observed. If an individual particle were labeled (by dye for example) and its motion traced, it would move from point P to point Q (see Figure 3) along a definite path. Every succeeding particle coming to P would then follow

the same path to Q assuming the same velocity vector field. Lines of flow or stream lines may be drawn through the medium, tangent at every point to the velocity vector. By definition liquid can never flow from one line to another. Simularly, one may define tubes of flow, the elements of their surfaces being lines of flow. We may consider the liquid to flow through these tubes, as water flows through a pipe, never pausing outside since the velocity is always tangential to the surface of the tube.

Figure 3



III. Continuity Equation

If we consider steady flow we may consider a convenient control volume with longitudinal boundaries coincident with a tube of flow and transverse boundaries at right angles to the stream lines as shown in Figure 4. By definition no flow occurs across the longitudinal boundary. The mass of fluid which crosses surface A, in time dt is

$$dm_1 = \rho A_1 v_1 dt$$

where ρ is the fluid density, A_1 is the area of the transverse boundary at P_1, and v_1 is the velocity at P_1. Similarly at P_2 the mass of fluid crossing A_2 is

$$dm_2 = \rho A_2 v_2 dt$$

If there are no sources or sinks within the control volume, and if the fluid is incompressible such that ρ is constant,

$$dm_1 = dm_2$$

$$A_1 v_1 = A_2 v_2 \qquad (1)$$

Thus, the velocities in a given tube of flow must vary inversely with the cross-section of the tube. Equation (1) is a simple form of the continuity equation, and holds for incompressible fluids.

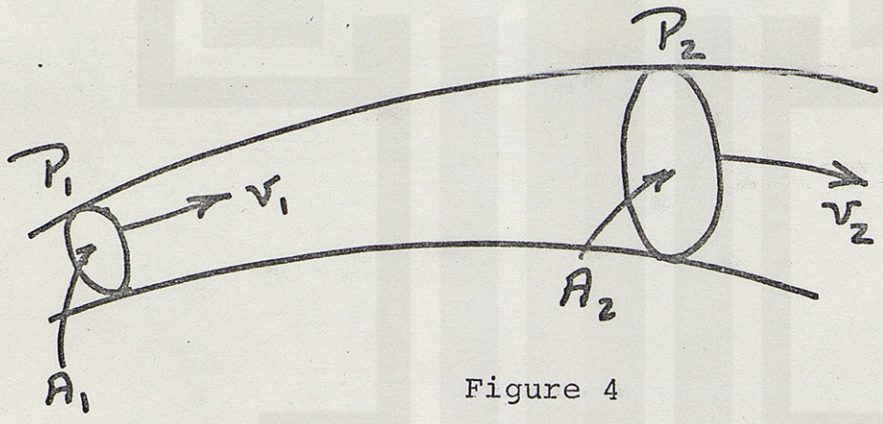


Figure 4

A more general formulation of the continuity equation begins by considering a differential control volume $\Delta x \Delta y \Delta z$ in a region where density and velocity are functions of position in space and time. (Figure 5). We compute the flux of mass per second through each face into the cube to get, for the three directions

$$-\left[\frac{\partial(\rho v_x)}{\partial x}\Delta x\right]\Delta y\Delta z, \quad -\left[\frac{\partial(\rho v_y)}{\partial y}\Delta y\right]\Delta z\Delta x,$$

$$-\left[\frac{\partial(\rho v_z)}{\partial z}\Delta z\right]\Delta x\Delta y$$

From the principle of conservation of matter, the sum of these (total flux of mass into the cube per second) must equal the time rate of change of mass within the control volume--

$$\frac{\partial}{\partial t}(\rho\Delta x\Delta y\Delta z)$$

Since $\Delta x\Delta y\Delta z$ is independent of time we may combine the above expressions and factor out $\Delta x\Delta y\Delta z$ to get

$$\frac{\partial\rho}{\partial t} + \frac{\partial(\rho v_x)}{\partial x} + \frac{\partial(\rho v_y)}{\partial y} + \frac{\partial(\rho v_z)}{\partial z} = 0 \qquad (2)$$

In vector notation this may be written:

$$\frac{\partial\rho}{\partial t} + \vec{\nabla}\cdot\rho\vec{v} = 0 \qquad (3)$$

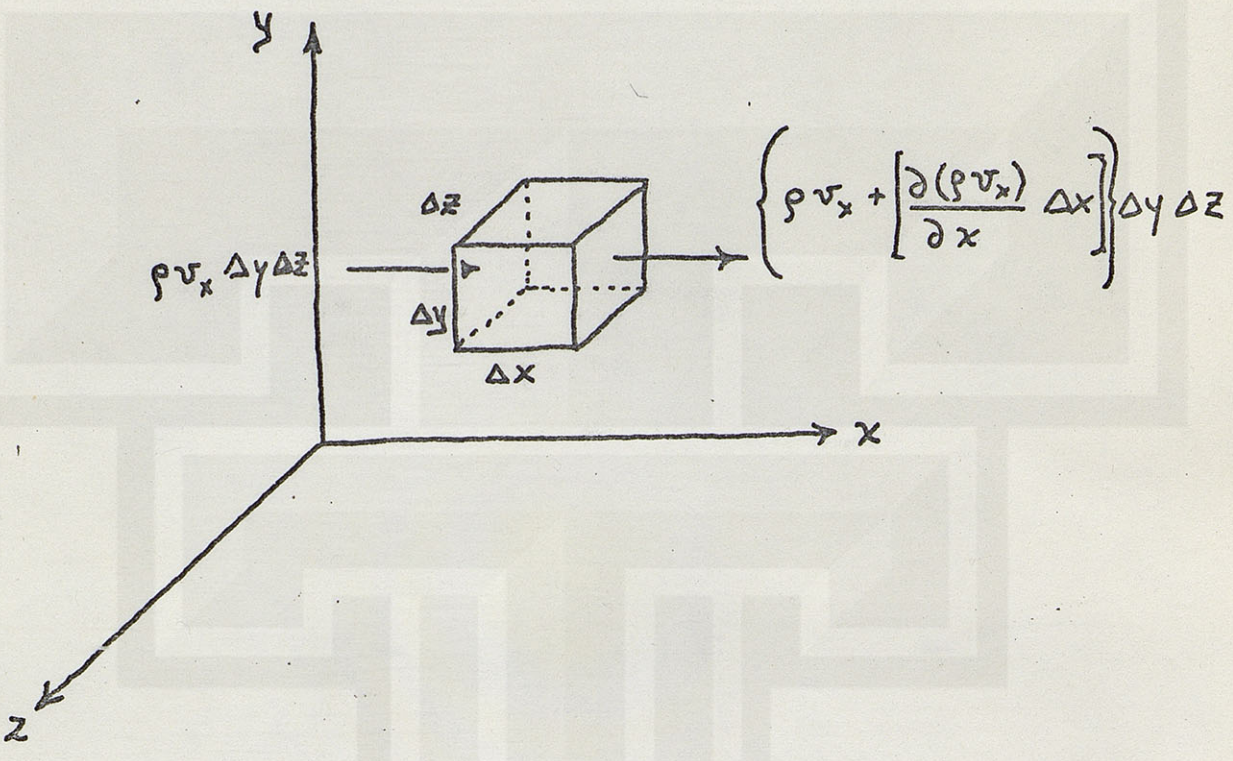
In the case of an incompressible fluid, ρ= constant, and equation (2) becomes

$$\frac{\partial v_x}{\partial x} + \frac{\partial v_y}{\partial y} + \frac{\partial v_z}{\partial z} = 0 \qquad (4)$$

or

$$\vec{\nabla}\cdot\vec{v} = 0 \qquad (5)$$

Figure 5



IV. Beronoulli's Equation

Bernoulli's theorem, which is derived in most standard texts (references 3,9) states that the total energy per unit volume along any stream line in steady incompressible flow is constant, though of course the value of the constant will in general change from one stream line to another. The equation is:

$$1/2\ \rho v^2 + P + \rho gh = \text{constant} \qquad (6)$$

where ρ is the density of the fluid in grams/cc, v the velocity in cm/sec, P the pressure in dynes/cm^2, g the acceleration of gravity (980 cm/sec^2), and h the height of the fluid above some arbitrary reference in cm. Instead of gram-centimeter-second units, we may use millimeters of mercury for all terms (1 mmHg = 1,330 dynes/cm^2). In the above expression 1/2 ρv^2 is the kinetic energy per unit volume along the stream line, while p and ρgh

appear as the potential energy per unit volume due to pressure and the earth's gravitational field respectively. It should be noted that the relation expressed in equation (6) assumes no energy losses due to friction--it applies only to ideal fluids, not to viscous flows. Nevertheless, its application in situations where dissipation of energy is negligible is extremely helpful. Several illustrative examples follow:

(a) Consider the simple example shown in Figure 6. A tube of varying cross-sectional area carries fluid in a horizontal direction. We wish to determine the relation between P_2 and P_1.

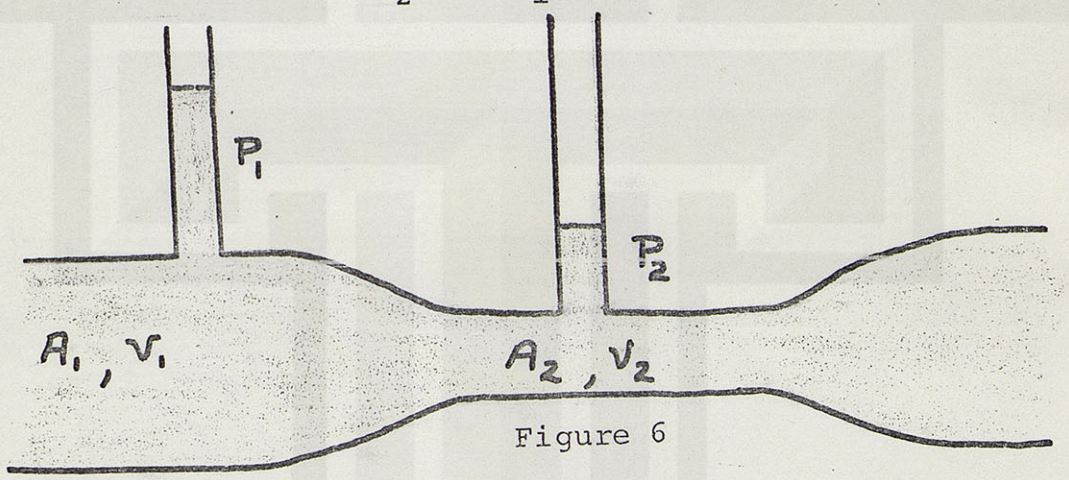


Figure 6

Since the tube is horizontal, we may neglect the gravitational term in equation 6. The equation of continuity permits us to relate the cross-sectional areas and velocities:

$$A_2V_2 = A_1v_1$$

$$v_2 = \frac{A_1}{A_2} v_1$$

Bernoulli's principle states:

$$1/2\ \rho v_1^2 + P_1 = 1/2\ \rho v_2^2 + P_2$$

$$P_2 = P_1 - 1/2\ \rho (v_2^2 - v_1^2)$$

$$P_2 = P_1 - 1/2\ \rho v_1^2 \left[\left(\frac{A_1}{A_2}\right)^2 - 1 \right]$$

Thus, in the narrowed portions of the tube, the pressure drops as the velocity increases. One interesting physiologic application of this finding is discussed by Burton (Chapter 10) in connection with the arterial narrowing due to atherosclerotic plaques. Consider the vessel shown in Figure 7.

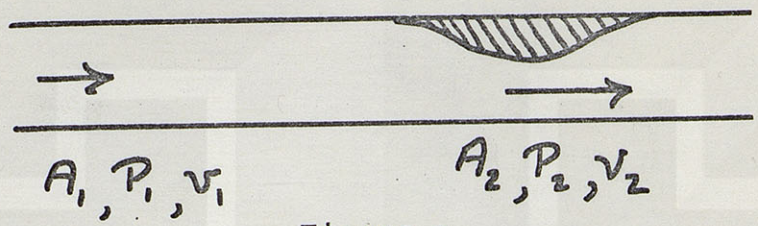


Figure 7

Assume the vessel's normal area to be A_1, with a mean pressure of 100 mmHg and a velocity of 30 cm/sec ($\rho \approx 1$). If the vessel is narrowed by the plaque to an effective area only one ninth as large as normal, what will the transmural pressure be at the point of narrowing? What are the possible consequences of this phenomenon? (Consider the Bronx cheer). Consider also the possible disastrous results of capillary ingrowth or cracks into plaques as illustrated in Figure 8. How might such geometry lead to rupture of the capillaries into the plaque?

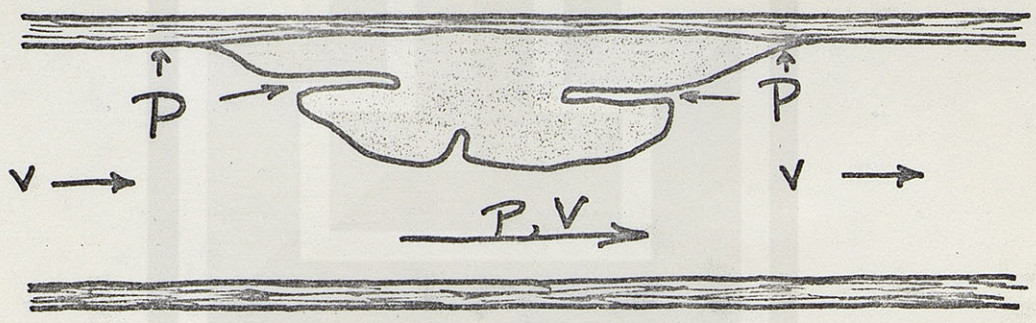


Figure 8

b. Pressure Measurement

If an obstacle is placed in a liquid flowing with velocity v_o, the liquid must come to rest just before the obstacle, and the streaming is divided into two branches one on each side of the obstacle. The original parallel streamlines are deformed as shown in Figure 9.

Figure 9

There is a stagnation point at the tip of the obstacle ($v_1 = 0$) and by the Bernoulli theorem,

$$P_1 = P_o + 1/2\ \rho v_o^2 \qquad (7)$$

where P_o and v_o are measured in that part of the liquid where the flow lines are straight.

Question: Extend this reasoning to the problem of measuring pressures in the arterial system with catheters. Compare the pressure readings which would be obtained from a catheter with the opening facing "upstream" (end pressure) versus one with laterally oriented openings (side pressure). Consider Figure 10 which shows the amount and relative importance of kinetic energy at different cardiac outputs. Note the rather significant effect in the pulmonary artery. (Here catheter opening usually points "downstream", and would underestimate pressure.

Figure 10

AMOUNT AND RELATIVE IMPORTANCE OF KINETIC ENERGY IN DIFFERENT PARTS OF THE CIRCULATION*

VESSEL	RESTING CARDIAC OUTPUT				CARDIAC OUTPUT INCREASED 3 TIMES		
	Velocity (Cm/Sec)	Kinetic Energy (Mm Hg)	Pressure (Mm Hg)	Kinetic Energy as % of Total	Kinetic Energy (Mm Hg)	Pressure (Mm Hg)	Kinetic Energy as % of Total
Aorta, systolic	100	4	120	3%	36	180	*17%*
Mean	30	0.4	100	0.4%	3.8	140	2.6%
Arteries, systolic	30	0.35	110	0.3%	3.8	120	3%
Mean	10	0.04	95	Neg.		100	Neg.
Capillaries	0.1	0.000004	25	Neg.	Neg.	25	Neg.
Venae cavae and atria	30	0.35	2	*12%*	3.2	3	*52%*
Pulmonary artery, systolic	90	3	20	*13%*	27	25	*52%*
Mean	25	0.23	12	2%	2.1	14	*13%*

*The cases where kinetic energy should not be neglected—that is, where it is more than 5% of the total fluid energy—are indicated by italic figures. When an artery is narrowed by disease processes, the kinetic energy becomes very important.
NOTE: Neg. = Negligible.

c. Calculation of Valve Areas

The Bernoulli principle provides an approach for estimating the size of valve areas using data obtained via cardiac catheterization. In Figure 11, the chamber represents the ventricle during ejection. Fluid is ejected through the orifice, and the cross-sectional

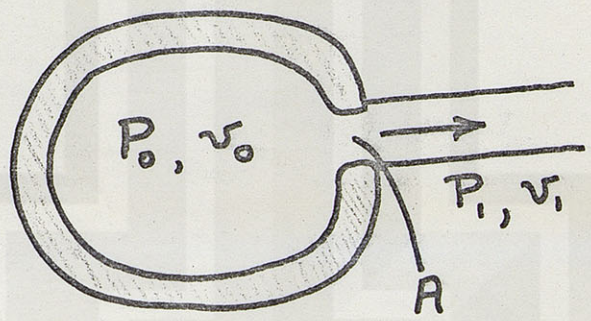


Figure 11

area of the jet is A. The pressure inside the chamber is P_o, and the velocity is v_o. The velocity and pressure of the fluid in the jet are v_1, P_1. Bernoulli's equation states:

$$P_o + 1/2\ \rho v_o^2 = P_1 + 1/2\ \rho v_1^2$$

$$1/2\ \rho (v_1^2 - v_o^2) = P_o - P_1$$

If we assume that $v_o << v_1$, this equation becomes:

$$1/2\ \rho v_1^2 = P_o - P_1$$

$$v_1 = \sqrt{\frac{2}{\rho}(P_o - P_1)} \qquad (8)$$

If the flow rate, Q, is known, we have

$$Q = Av_1$$

and

$$A = \frac{Q}{V_1}$$

Substituting from above, we have

$$A = \frac{Q}{k\sqrt{P_o - P_1}} \qquad (9)$$

In our derivation $k = \sqrt{2/\rho}$. The form of Equation (9) has been verified experimentally for diseased human heart valves, but the value of the constant, k, differs depending on the valve involved (reference 5). Q is measured in cc/sec; $P_o - P_1$ is the pressure gradient across the valve in mmHg; k is 44.5 for the aortic valve and 31 for the mitral valve; and A is the valve area in cm^2.

V. Viscosity

To this point, we have been considering ideal fluids in which no work is done in changing the shape of the fluid. All fluids found in nature depart from idealness to a greater or lesser extent. Some liquids such as glycerine or heavy oil depart widely from the ideal, and are known as viscous liquids.

The concept of viscosity was described by Sir Isaac Newton as a "lack of slipperiness" between adjacent layers of fluid. This lack of slipperiness produces an effective drag or force

between two layers moving past each other at different velocities. An understanding of this internal friction may result from consideration of a simple experiment. (See Figure 12) Suppose a viscous liquid, such as glycerine is placed on a glass plate of area A, and a similar plate is placed on top of the liquid. If the top plate is pulled horizontally with a constant force F, it is found that the top plate attains a constant velocity v. Since the liquid clings to the plates, a layer of fluid clinging to the top plate moves with the same velocity v, while the layer clinging to the bottom plate remains at rest.

The velocities of intermediate sheets of liquid are proportional to their vertical distance from the bottom plate. Assume the total thickness of the liquid is a. The impressed force is proportional to their vertical distance from the bottom plate. Assume the total thickness of the liquid is a. The impressed force is proportional to v and to A and is inversely proportional to a. Thus,

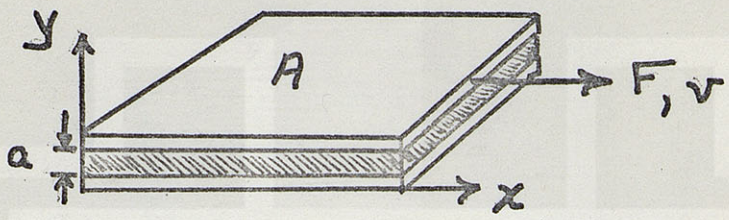
$$F = \mu A \frac{v}{a}$$

More generally, as a $\to$o we would have the following fundamental relation:

$$\frac{F}{A} = \mu \frac{dv_x}{dy} \qquad (10)$$

where y is measured perpendicularly to the direction of motion. The proportionality constant μ is called the coefficient of viscosity of the liquid and is expressed in dyne-sec/cm^2 or poise. Water has a viscosity of .01 poise or 1 centipoise. Whole blood viscosity is about 3-4 centipoise, and plasma is 1.5 centipoise. The viscosity of blood, however, is a function of hematocrit and of vessel size (see Burton Chapter 5). It may be considered as a Newtonian fluid if the radius of the vessel is greater than about 0.5 mm., and if the shear rate ($\frac{dv_x}{dy}$) exceeds 100 sec.$^{-1}$ (See Ref. 6).

Figure 12



VI. Laminar Flow in Rigid Tubes

The basic law governing the flow of a viscous liquid in a long cylindrical rigid tube was found by the French physician, Poiseuille, from careful measurements of laminar flow in tubes of various diameters. It is helpful to consider such laminar viscous flow as a first approximation to blood flow in small arteries. In laminar flow, we think of sheets of liquid as cylindrical tubes sliding over one another.

Consider a tube of radius r_o, length Δl, and a fluid of viscosity, μ. (See Figure 13.) The pressure at $x = 0$ is P_A, and at $x = \Delta l$ the pressure is P_B. Focus on a cylindrical shell of radius r, thickness dr, and length Δl. The net force acting on the shell due to the pressure gradient is:

$$F_p = (P_A - P_B)\, dA = (P_A - P_B)\, 2\pi r dr$$

the viscous forces will be a function of surface area and velocity gradient, and in general will be:

$$F_v(r) = \mu \cdot 2\pi r\, \Delta l \cdot \frac{dv}{dr}$$

The net viscous force acting on the shell would be

$$F_v(r) = F_v(r)\Big|_{r+dr} - F_v(r)\Big|_r = \frac{\partial}{\partial r}[F_v(r)]dr =$$

$$\frac{\partial}{\partial r}\left[\mu \cdot 2\pi r \ \Delta l \ \frac{dv}{dr}\right]dr$$

This force will act in a direction opposite to F_p. Since flow is stationary, the two forces must add to zero.

$$F_p(r) + F_v(r) = 0$$

$$(P_A - P_B)2\pi r\, dr + \frac{\partial}{\partial r}\left[\mu \cdot 2\pi r \Delta \frac{dv}{dr}\right] = 0$$

Integrating once, we have:

$$(P_A - P_B)\pi r^2 + \mu 2\pi r \ \Delta l \ \frac{dv}{dr} = k_1$$

Since $\frac{dv}{dr} = 0$ when $r = 0$, $k_1 = 0$.

Rearranging, we have

$$\frac{dv}{dr} = -\frac{(P_A - P_B)\ r}{2\mu\Delta l}$$

Integrating,

$$v = -\frac{(P_A - P_B)\ r^2}{4\mu\Delta l} + k_2$$

The second boundary condition is that $v = 0$ at $r = r_o$, and hence the equation becomes:

$$V = \frac{1}{4\mu}\frac{(P_A - P_B)}{\Delta l} r_o^2 \left[1 - \left(\frac{r}{r_o}\right)^2\right] \qquad (11)$$

Equation (11) is the equation of a parabola, and the velocity distribution is plotted in Figure 14.

Figure 13

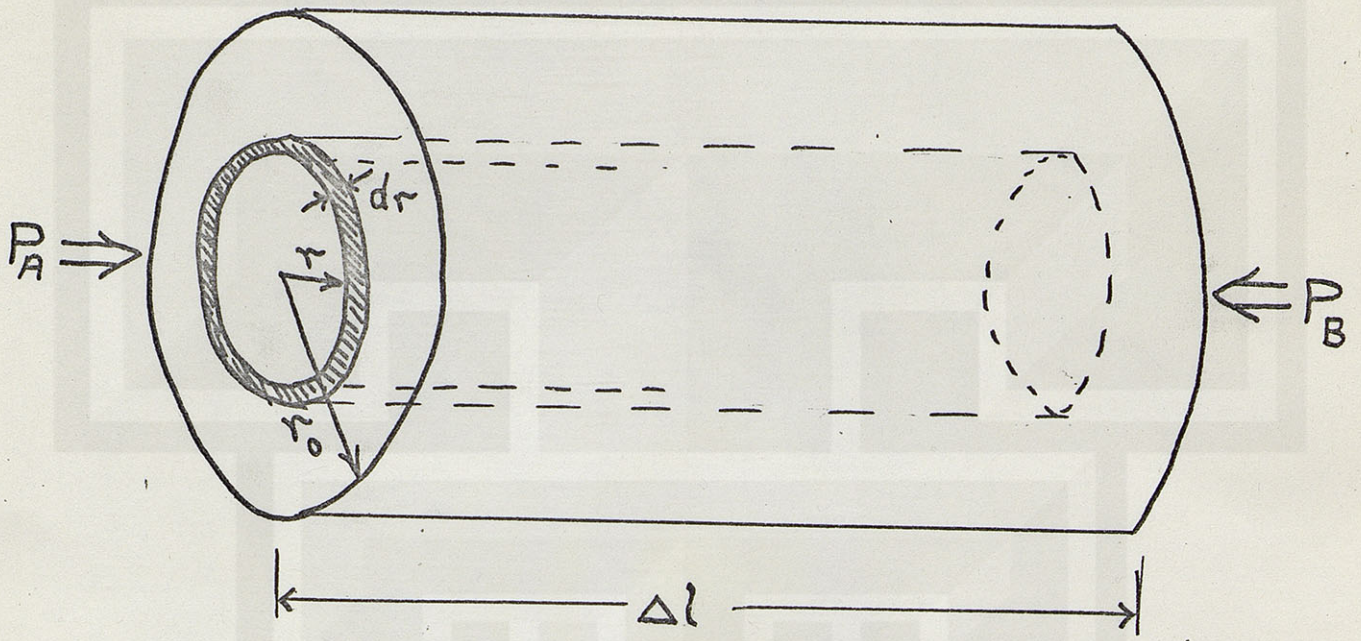
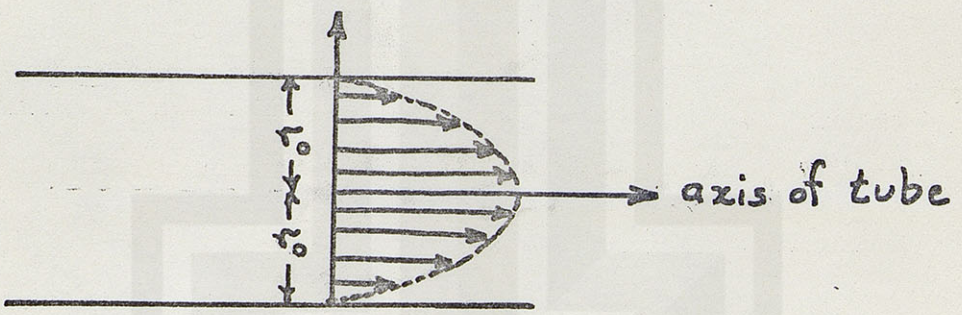


Figure 14



Knowing the velocities of each annulus, we may now calculate the relation between the total flow in the tube, $\dot{Q}$, and the pressure gradient across the tube $(P_A - P_B) / \Delta l$.

For each annulus the flow $\dot{dQ}$ would be

$$\dot{dQ} = v(r)\, dA$$

$$= \frac{(P_A - P_B)}{\Delta l} \cdot \frac{r_o^2}{4\mu} \left[1 - \left(\frac{r}{r_o}\right) \right] \cdot 2\pi r\, dr$$

Integrating from $r = 0$ to $r = r_o$, we obtain

$$\dot{Q} = \int_o^{r_o} \frac{(P_A - P_B)}{\Delta l} \cdot \frac{r_o^2}{4\mu} \cdot 2\pi r \left(1 - \frac{r^2}{r_o^2}\right) dr$$

$$\dot{Q} = \frac{\pi}{8} \cdot \frac{1}{\mu} \cdot \frac{r_o^4}{\Delta l} \cdot (P_A - P_B) \qquad (12)$$

Equation 12 is the well known Poiseuille's law which relates flow to pressure drop for rigid tubes. Note the division of the terms into a constant, a viscosity term, a geometric term and a pressure term. In particular, notice the strong dependence on tube radius r_o.

Equation 12 may be rewritten in the form

$$(P_A - P_B) = \dot{Q}R$$

where $$R = \frac{8}{\pi} \cdot \mu \cdot \frac{\Delta l}{r_o^4} \qquad (13)$$

Here R is termed the resistance of the tube and is directly proportional to length and viscosity and inversely proportional to the fourth power of the radius. Thus, one would expect that the major contribution to vascular resistance would be made by the small vessels. Thsi is borne out by consideration of the following table:

—Relative Resistance to Flow in the Vascular Bed: Calculated from Table 6 and Poiseuille's Law $\left(R \propto \frac{l}{r^4}\right)$

Aorta	4%	Venules	4 %
Large arteries	5%	Terminal veins	0.3%
Mean arterial branches	10%	Main venous branches	0.7%
Terminal branches	6%	Large veins	0.5%
Arterioles	41%	Vena cava	1.5%
Capillaries	27%		
Total: arterial + capillary = 93%		Total venous = 7%	

Note also that the strong dependence of the resistance upon vessel radius implies that sensitive regulation of flow is possible by changes in vessel diameter due to smooth muscle action.

Although Poiseuille's law has many engineering applications, and although it gives considerable insight into flow in the circulation; nevertheless, it cannot be rigorously applied to the circulation. It requires the following assumptions; as pointed out by Dewey and Jaffrin (reference 6).

(1) The fluid is homogeneous and newtonian. Blood may be considered as a newtonian fluid only if the radius of the vessel exceeds 0.5 mm and if the shear rate exceeds 100 sec $^{-1}$. This condition, therefore, excludes arterioles, venules and capillaries.

(2) The flow is steady and inertia-free. If the flow is pulsatile, the variable pressure gradient communicates kinetic energy to the fluid, and the flow is no longer inertia-free. This condition excludes the larger arteries.

(3) The tube is rigid so that its diameter does not change with pressure. This condition is never met in the circulatory system, particularly in the veins.

More precise models of blood flow in the circulation have been devised which take account of the properties of vessel walls, inertia, pulsatile flow, etc. which are beyond the scope of our consideration here. The interested student is

referred to references (4,6,7, and 8) for more sophisticated approaches.

In particular flow in the aorta is not Poiseuille flow. Blood enters the aorta as a bolus, with uniform velocity distribution over the cross-section. As the fluid flows down the aorta, the viscous drag from the walls has an increasing effect on the flow. In particular velocities near the wall tend to decrease, and the penetration of the drag into the fluid increases as the fluid continues down the conduit. Eventually the "boundary layer" penetrates to the center of the conduit and steady laminar flow is established. (See Figure 15.) The distance from the entrance to the point where fully developed flow occurs is termed the entrance length ℓ_E. The ratio $\ell_{E/D}$ where D is the diameter of the conduit is also related to the velocity and viscosity of the fluid. One might expect $\ell_{E/D}$ to be directly proportional to V and inversely proportional to μ. In fact, a theoretical entrance length which agrees reasonably well with experiment for non-turbulent flow is

$$\ell_{E/D} = 0.065R \text{ where} \qquad (14)$$

$$R = \frac{VD\rho}{\mu} \quad \text{(Reynold's number)}.$$

For the aorta $V \approx 25$ cm/sec., $D = 2.5$ cm. $\mu = 4 \times 10^{-2}$ $R \cong 1550$. Hence,

$$\ell_{E/D} = 100, \text{ or } \ell_E = 250 \text{ cm}.$$

This result implies that the velocity distribution in the aorta will tend to be uniform over the cross-section. Experimental results for dogs is shown in Figure 16.

Figure 15

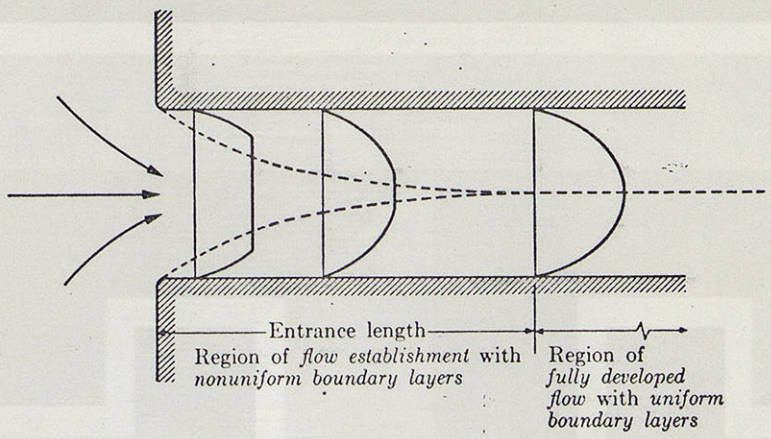
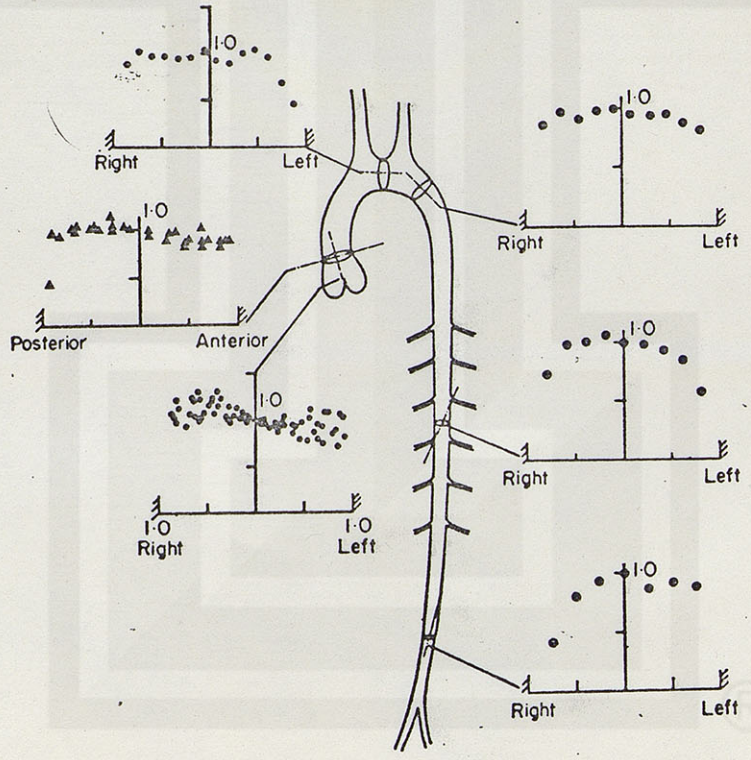


Figure 16



Velocity distribution in the aorta of dogs. For each graph the ordinate shows $\overline{U}/\overline{U}_{C.L.}$, mean velocity at each station normalized to the mean centre-line velocity, and the abscissa is R/R_W, the radial location of the measuring station normalized on the internal luminal radius.

Lectures 3,4 HST 090

VII. Definition of Vascular Resistance

Equation 13 states that the pressure drop between two points in the circulation is equal to the product of the flow and the resistance. Rewriting, it becomes:

$$R = \frac{\Delta P}{\dot{Q}} \qquad (15)$$

Equation 15 defines resistance as the ratio of pressure gradient to flow. If the flow is measured in cm^3/sec and P in $dynes/cm^2$, the units of R turn out to be dyne - sec/cm^5. If pressure is measured in mmHg and flow in cm^3/sec, resistance is expressed in "peripheral resistance units", or PRU. Note that if the mean pressure drop across the circulation were 80 mmHg and the cardiac output were 5 liters/min (which is about 80 cc/sec) the total peripheral resistance is close to 1 PRU.

Notice also that equations 13 and 15 are in the same form as the familiar Ohm's law in electric circuit theory,

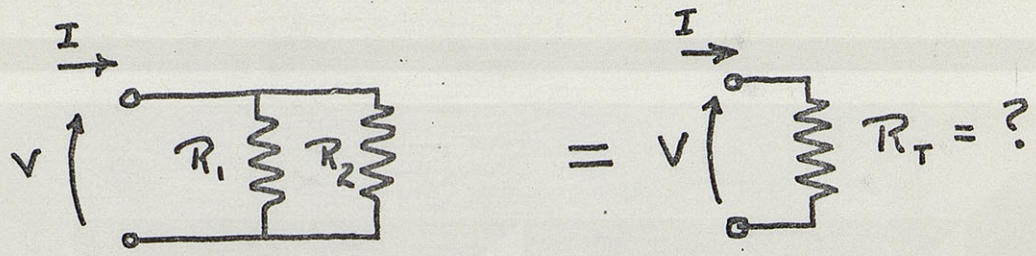
$$\mathcal{e} = \dot{\imath} R \qquad (16)$$

The analogies between fluid variables and electric variables are:

Fluid		Electric
Pressure, P	$\longleftrightarrow$	Voltage, $\mathcal{e}$
Flow, $\dot{Q}$	$\longleftrightarrow$	Current, $\dot{\imath}$
Resistance, $R = \frac{\Delta P}{\dot{Q}}$	$\longleftrightarrow$	Resistance, $R = \frac{\Delta \mathcal{e}}{\dot{\imath}}$

It is often helpful to represent vascular resistances in circuit terms.

Example: If two vascular beds of resistance R_1 and R_2 are connected in parallel, what combined resistance to flow would result?



Answer:

$$R_T = \frac{R_1 R_2}{R_1 + R_2}$$, remembering that

from circuit theory

$$\frac{1}{R_T} = \sum_{i=1}^{N} \frac{1}{R_i}$$

VIII. Turbulent Flow; Reynolds Number

At high enough velocities, the pattern of laminar flow described above breaks down into turbulence, in which the fluid particles move in irregular and constantly changing paths including vortex-like eddies. In turbulence, a substantial amount of the energy of flow is used to create the kinetic energy of these eddies. Sir Osborne Reynolds first treated the penomenon of turbulence quantitatively in 1883. Reynolds showed that the critical velocity of flow in tubes at which turbulence began depended on the viscosity, μ, and density, ρ, of the fluid, and the radius, r, of the tube. The relation was:

$$v_c = R\frac{\mu}{\rho d} \qquad (17)$$

where μ is the viscosity in poises, ρ is the density of fluid in grams/cc and d is the tube diameter. The dimensionless term R is called Reynold's number, and is

$$R = \frac{v_c \rho d}{\mu} \qquad (18)$$

For long straight tubes, Reynolds found that if R>2000, the flow became turbulent. This is reasonably true also for

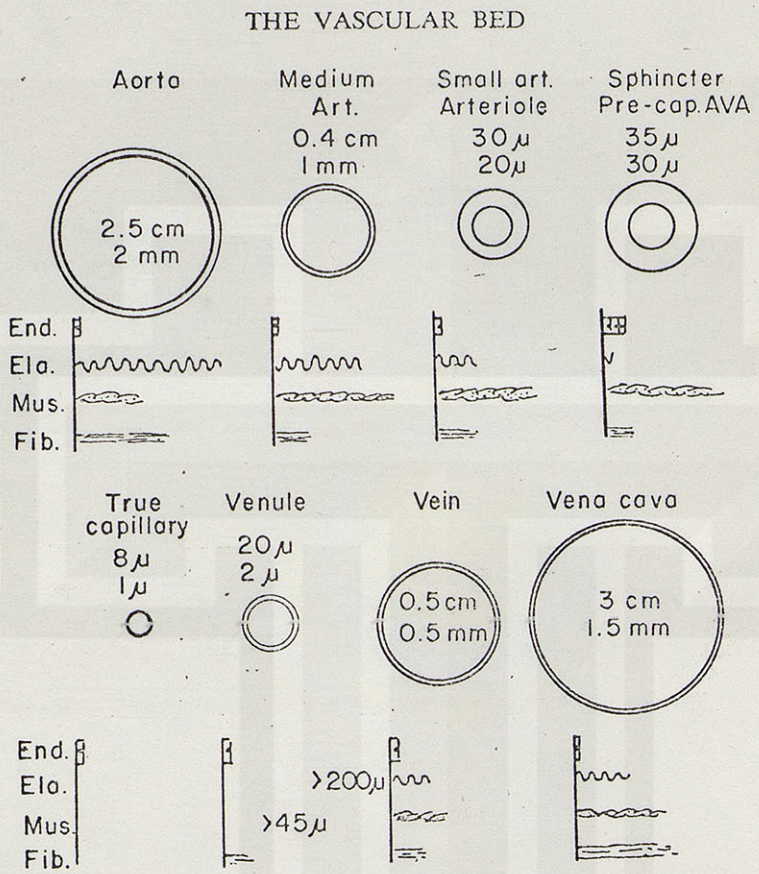
the circulatory system except in cases of stenosis or partial obstruction where turbulence may occur at Reynold's numbers much lower than 2000.

If then, conditions in the circulation provide sufficient velocity of flow in the cylindrical vessels, or a less, but critical, velocity through narrow orifices there will turbulence in the blood stream. This can create vibrations which will be shared by the elastic walls, and may be felt as a "thrill" and heard as a "bruit" or"murmur."

IX. Vascular Capacitance

The walls of blood vessels are not rigid, but rather they stretch in response to increased transmural pressure. The vessel walls contain four major elements: endothelial lining, elastin fibers, collagen fibers, and smooth muscle. The endothelium provides a smooth wall, and offers selective permeability to certain substances. The endothelial cells play very little part in the total elasticity of the walls. The elastin fibers are easily stretched (about six times more easily than rubber). The elastin fibers produce an elastic tension automatically as the vessel expands, and without bio-chemical energy expenditure. The collagen fibers resist stretch much more than do elastin fibers. However, these fibers are slack, and do not exert their tension until the vessel has been stretched. Thus, the more the vessel expands, the stiffer it becomes. The smooth muscle serves to produce an active tension by contracting under physiological control, and so changes the diameter of the lumen of the vessel. Figure 17 shows the relative mixtures of the four elements in walls of various vessels.

Figure 17



.—Variety of sizes, thickness of wall and admixture of the four basic tissues in the wall of different blood vessels. The figures directly under the name of the vessel represent the diameter of the lumen; below this, the thickness of the wall. *End.*, endothelial lining cells. *Ela.*, elastin fibers. *Mus.*, smooth muscle. *Fib.*, collagenous fibers. (From Burton, A. C.: Relation of structure to function of the tissues of the wall of blood vessels, Physiol. Rev. 34:619–642, 1944.)

Because of their ability to expand as transmural pressure increases, blood vessels may function to store blood volume under pressure. In this sense, they function as capacitance elements, and are similar in that sense to storage tanks. Such a tank is illustrated in Figure 18. The tank is of area A, and fluid of density ρ fills the tank to a height h. The pressure at the bottom of the tank is ρgh. Since the volume of the fluid is Ah, we may relate volume to pressure for

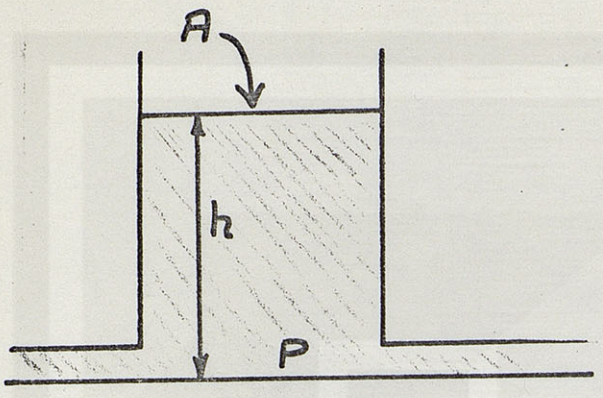


Figure 18A

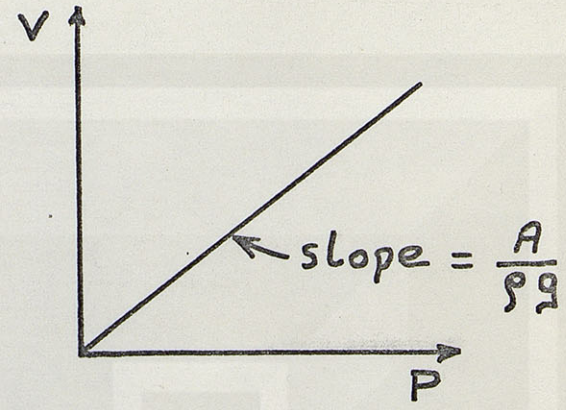


Figure 18B

this storage element:

$$V = \frac{A}{\rho g} P$$

A plot of volume vs. pressure would be a straight line as shown in Figure 18B. The slope of the V vs. P is the capacitance of the tank:

$$C = \frac{dV}{dP} = \frac{A}{\rho g} \qquad (19)$$

Volume-pressure curves for arteries and veins are shown (on different scales) in Figure 19. Note that the pressure is <u>transmural</u> pressure as opposed to driving pressure.

THE VASCULAR BED

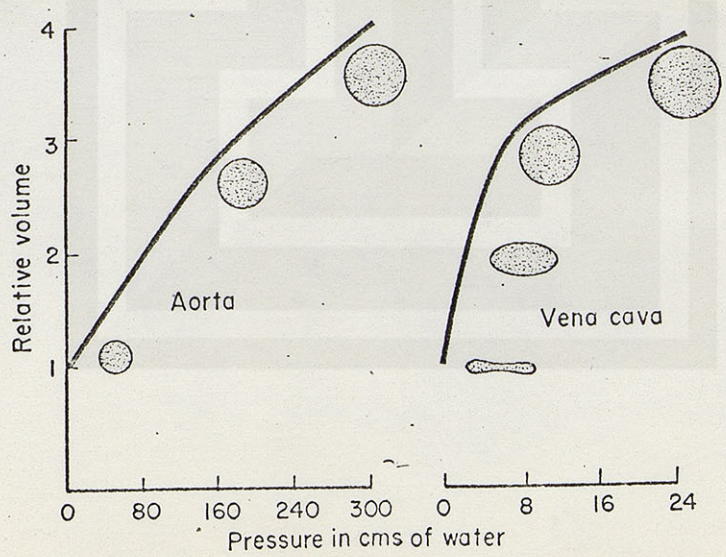


Figure 19

-Comparison of the distensibility of the aorta and of the vena cava. The way in which the cross-section of the vessels changes in the two cases is also indicated.

The slope of this line at any point is the incremental capacitance of the vessel at that particular pressure. Note that the capacitance (or compliance) decreases with increasing pressures. The capacitance also varies with age (how?). Note also that veins have a much larger capacitance than arteries and in fact, are often referred to as <u>capacitance</u> or <u>storage</u> vessels.

With the added concept of capacitance we may extend our table table of analogies between electrical and fluid variables:

Fluid Variable	Electrical Variable
Pressure, P	Voltage, $\mathcal{E}$
Flow, Q	Current, i
Volume, V	Charge, q
Resistance, $R = \frac{\Delta P}{Q}$	Resistance, $R = \frac{\Delta \mathcal{E}}{i}$
Capacitance, $C = \frac{\Delta V}{\Delta P}$	Capacitance = $\frac{\Delta q}{\Delta v}$

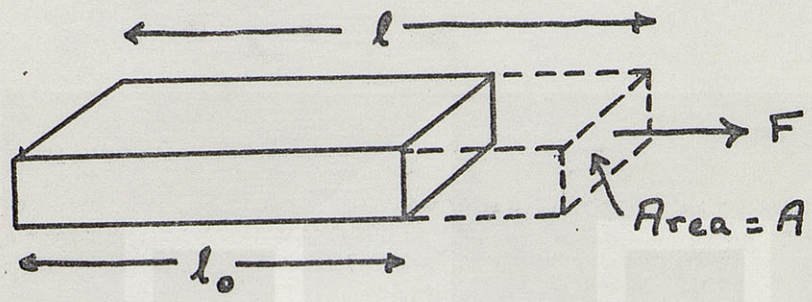
X. Hooke's Law, Laplace's Law

Two relationships which are helpful in understanding the elastic behavior of blood vessel walls are Hooke's Law and LaPlace's Law.

1. Hooke's Law

Consider a strip of material of initial length l_o, and cross-sectional area A (see Figure 20). A force F is applied to the strip, and in general

Figure 20



it will stretch to a new length, l. Hooke's law states that the force per unit area is proportional to the fractional increase in length. Thus,

$$\frac{F}{A} = E\left(\frac{l - l_o}{l_o}\right) \qquad (20)$$

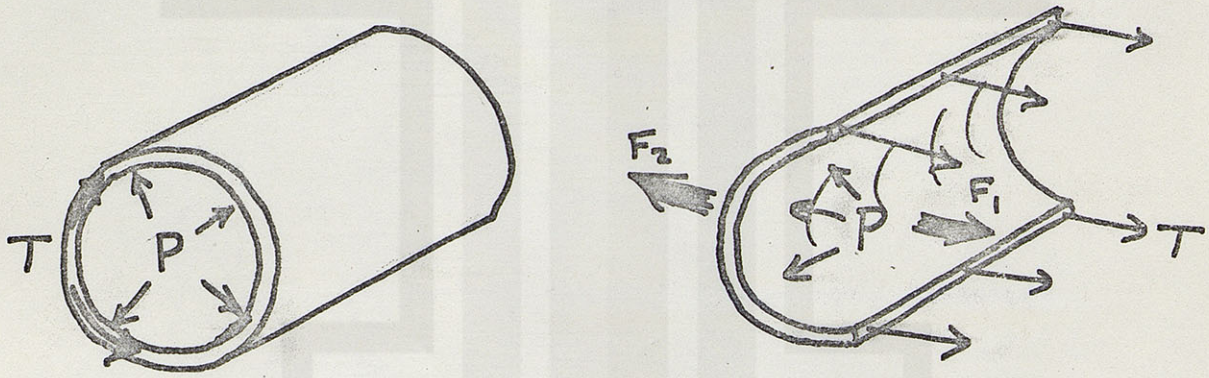
E is known as Young's modulus. For blood vessels, E is a function of pressure. The following table shows the variation of E with pressure for the thoracic aorta:

P, mmHg	E dynes/cm^2 x 10^{-6}
40	1.2
100	4.2
160	10
220	18

2. Laplace's Law

It is often of interest to relate the pressure inside a vessel or chamber to the tension in the walls. Consider a cylinder of length l and radius R with thin walls which is filled with a fluid under pressure P (Figure 21). There is a certain tension per unit length, T, in the walls which keeps the cylinder from exploding. We wish to

Figure 21



derive the relation between T and P. Imagine that the cylinder were split down the middle. The total force pulling the half-cylinder to the right would result from the tension T acting at each edge:

$$F_1 = 2Tl$$

In order for the vessel to be in equilibrium, an equal and opposite force must be provided by the pressure, P, acting against the area of the half-cylinder.

$$F_2 = P \cdot \text{Area} = P \cdot 2Rl$$

Since $F_1 = F_2$

$$2RlP = 2Tl$$

$$P = \frac{T}{R} \qquad (21)$$

Equation (21) is known as Laplace's Law for a thin-walled cylinder. Note that for a given transmural pressure, the tension required for equilibrium increases as radius increases and vice versa. What does this imply about stability of vessels under various conditions? Can you derive Laplace's Law for a spherical chamber?

XI. Pulse Wave Propogation in Arteries-Transmission Line Analogy

In this section, we wish to develop a simple model which illustrates how pulse propogation may occur in the arterial system. We will represent the artery as a thin-walled elastic tube characterized by an equation of state relating the area of the vessel, A, to the transmural pressure,p,

$$A = A(p)$$

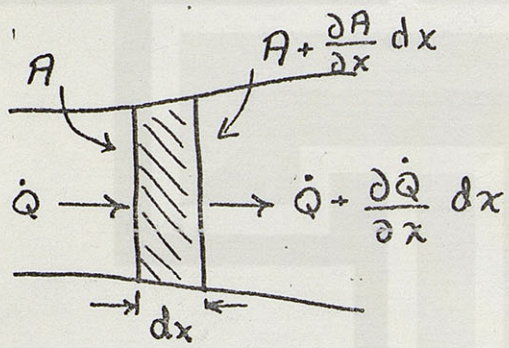
For simplicity we will assume a linear operating region and define a compliance per unit length C_u such that

$$C_u = \frac{\partial A}{\partial p} = \text{constant}$$

This implies $A = A_o + C_u P$. (22)

Consider a section of vessel as shown in Figure 22,

Figure 22



$\dot{Q}$ = volume flow rate

A = cross sectional area

The equation of continuity states that the net rate of increase in mass of the control volume, $\rho \frac{\partial A}{\partial t} \cdot dx$ must equal the net inflow into the control volume which is $-\rho \frac{\partial \dot{Q}}{\partial x} dx$

Hence,

$$\frac{\partial \dot{Q}}{\partial x} + \frac{\partial A}{\partial t} = 0 \qquad (23)$$

We will make an additional simplifying assumption in deriving the equation of motion--namely that to a first approximation the axial velocity is small compared to the wave velocity which permits us to ignore the non-linear terms in the acceleration. (The net flux of momentum into the control volume is assumed to be small compared to the time rate of change of momentum within the control volume.) Under those conditions, Newton's

law may be written:

$$\underbrace{\rho \cdot A \cdot dx}_{\text{mass}} \cdot \underbrace{\frac{\partial v_x}{\partial t}}_{\text{acceleration}} = \underbrace{-A\frac{\partial p}{\partial x} dx}_{\text{force= } \Delta\text{pressure} \cdot \text{area}}$$

Simplifying we have

$$\frac{\partial v_x}{\partial t} + \frac{1}{\rho}\frac{\partial p}{\partial x} = 0 \qquad (24)$$

Multiplying by A we obtain (noting that $\dot{Q} = Av_x$),

$$\frac{\partial \dot{Q}}{\partial t} + \frac{A}{\rho}\frac{\partial p}{\partial x} = 0 \qquad (25)$$

Using equation 22 differentiating with respect to t and substituting into eg. 23, we obtain

$$\frac{\partial \dot{Q}}{\partial x} + C_u \frac{\partial p}{\partial t} = 0 \qquad (26)$$

Equations 25 and 26 form the familiar equations governing losslers transmission lines, and ρ/A may be identified as L_u, the inertance per unit length.

If the compliance of the vessel is assume to be independent of pressure and location, and if the area of the vessel is assumed to be constant (no tapering and small perturbations with pressure), we may treat C_u and L_u as constants and solve equations 25 and 26.

Differentiating (25) with respect to x, and (26) with respect to t and eliminating the $\dot{Q}$ terms, we have:

$$C_u \frac{\partial^2 p}{\partial t^2} - \frac{A}{\rho}\frac{\partial^2 p}{\partial x^2} = 0$$

or

$$\frac{\partial^2 p}{\partial t^2} - \frac{A}{\rho C_u}\frac{\partial^2 p}{\partial x^2} = 0 \qquad (27)$$

Solutions are of the form

$$p = p(x-ct)$$

Thus pressure waves propogate at constant velocity, c, without distortion, where

$$c = \sqrt{\frac{A}{\rho C_u}} = \sqrt{\frac{1}{L_u C_u}} \qquad (28)$$

This velocity is known as the Moens-Korteweg wave speed.

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MASSACHUSETTS INSTITUTE OF TECHNOLOGY

Electrical and Mechanical Engineering Departments

6.022J/2.792J: Quantitative Physiology: Organ Transport Systems

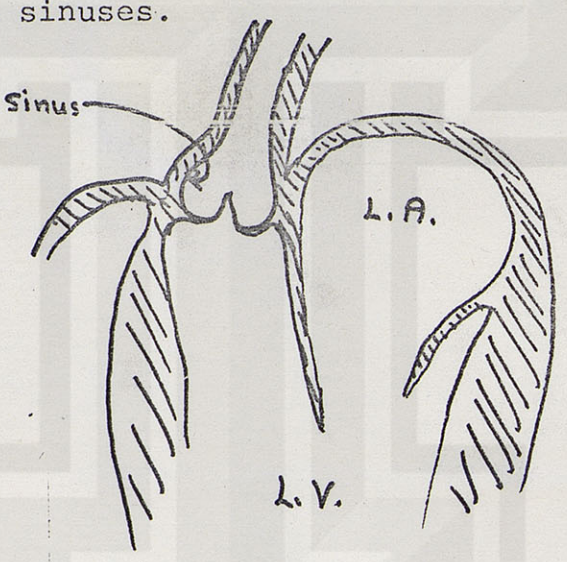
PART I: CARDIOVASCULAR PHYSIOLOGY (Cont.)

D. Fluid Mechanics of Heart Valve Action

(Ref. Bellhouse, B.J. The fluid mechanics of heart valves, in Cardiovasular Fluid Dynamics, Vol.I, ed. D. H. Bergel, pp. 261-285, Academic Press, 1972.)

1. Aortic Valve

The aortic valve consists of three thin (0.1mm) flexible, self-supporting cusps. Corresponding to each cusp, there is a bulge in the aortic wall called a sinus, (sinuses of Valsalva). The coronary arteries arise from two of these sinuses.



In the normal valve, blood flow is laminar, and furthermore, the reversed flow is less than 5% of the stroke volume. The fact that flow is observed to be laminar despite peak Reynolds numbers of near 10^4 suggests that the normal valve offers no obstruction to forward flow.

On the other hand the very low reverse flow at the end of systole suggests the valve may be almost closed before the end of systole. It also seems important that the ostia of the coronary arteries not be occluded during the heart cycle.

The movement of the valve cusps has been studied by Bellhouse by means of a model system, and they have demonstrated the importance of the sinuses. The observations are:

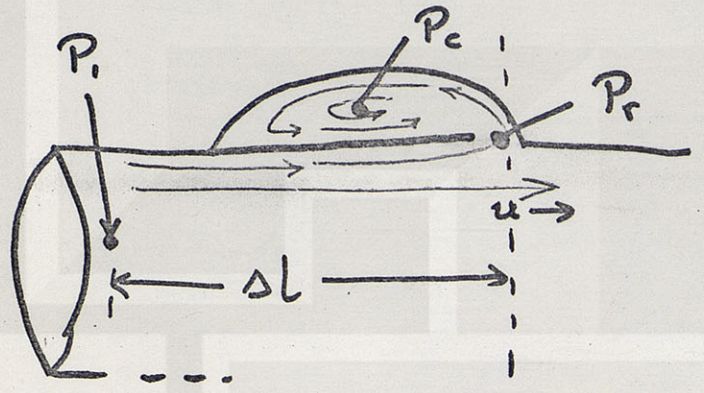
At the start of systole the valve cusps open rapidly and move out toward the sinuses. Vortices formed between the cusps and the sinus walls. The cusps did not flutter,

and flow entered each sinus at the ridge, curled back around the sinus wall, and then along the cusp to flow out into the main stream at the points of attachment of the cusp to the aorta. Thus, the valve leaflets are supported, during ejection, between the main stream and the trapped vortices.

After peak ejection velocity, as blood was being decelerated (but still moving out of the heart), the valve cusps move away from the sinuses, and are almost completely closed before the end of systole. Reverse flow was less than 5% of the forward S.V.

If the sinuses are occluded, the cusps open and touch the walls of the aorta during systole, and there are no trapped vortices. The valve closes by means of reversed flow above, and back flow increased to 25% of the forward flow.

The action of the valves may be explained on the basis of local pressure gradients in the region of the aortic root. During deceleration of the blood (later half of systole) there must be a reverse pressure gradient in the region of the aortic outlet. Specifically:



$$P_c - P_r = \frac{1}{2}\rho u^2$$
$$P_r - P_l = -\Delta l \rho \frac{\partial u}{\partial t}$$
$$\therefore P_c - P_l = \frac{1}{2}\rho u^2 - \Delta l \rho \frac{\partial u}{\partial t}$$

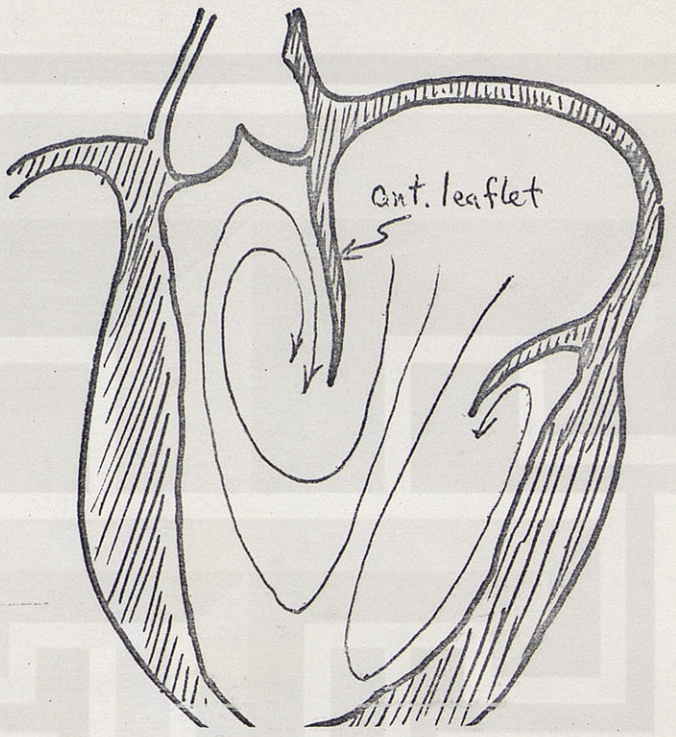
$P_c - P_l$ is the pressure across the valve leaflet, and positive gradients tend to close the valve.

When the valve is stenosed, a jet is formed which is not intercepted, in general, by the edge of the sinus. Vortices do not form, and the pressure measured in the region of the coronary ostia may be as much as 30 mmHg below the intraventricular pressure during maximum ejection rate. (What clinical implications follow?)

2. The Mitral Valve

As with the aortic valve, there is less than 5% regurgitation during systole across the mitral valve. This implies that the valve must be almost closed at the end of diastole. Two important mechanisms are involved.

a. Vortex formation in the ventricle during filling.



The vortex behind the anterior leaflet was stronger than the posterior vortex, tending to close the anterior leaflet first.

b. Deceleration of inflowing fluid.

As with the aortic valve closure mechanism, there is a reverse pressure gradient across the mitral valve during the deceleration of the inflowing blood. This reverse pressure gradient will tend to close the valve. A simple experiment illustrates the principle. At t = o the fluid column is released.