BSc School C Level 2a Module 6
Cardiovascular Mechanics

Notes for Cardiovascular Fluid Mechanics

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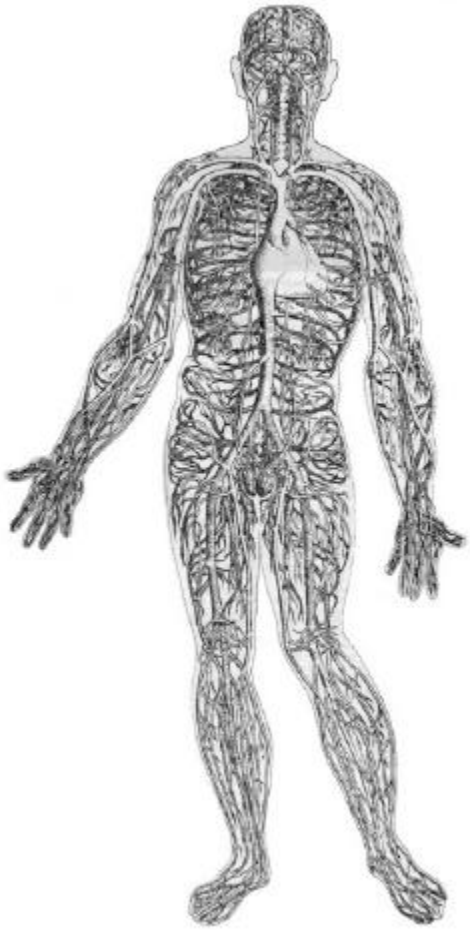


The Vein Man. Vesalius (1554)

Vesalius published the first illustrated atlas of anatomy. He is a very important figure in the history of science because he was one of the first to assert that it was important to 'look at nature' rather than to appeal solely to the classical writers such as Aristotle and Galen.

These notes are in two parts. First, there is a brief section entitled the Cardiovascular System describing the anatomy of the cardiovascular system which should be seen as a review.. There follows a more substantial section entitled Haemodynamics which should contain the primary content of the lectures for this module.

Books on Cardiovascular Dynamics:

Levick JR. (1991) *An Introduction to Cardiac Physiology*, 3rd Ed. Butterworth-Heinmann.

Caro CG, Pedley TJ, Schroter RS & Seed WA (1978) *The Mechanics of the Circulation*. Oxford Universtiy Press.

Nichols WW & O'Rourke MF (1998) *MacDonald's Blood flow in arteries: Theoretical, Experimental and Clinical Principles*. 4th Ed. Edward Arnold.

Milnor WR (1989) *Hemodynamics*, 2nd Ed. Williams & Wilkins.

Noble MIM (1979) *The Cardiac Cycle*. Blackwell.

Fung YC (1990) *Biomechanics: Motion, Flow, Stress and Growths*. Springer Verlag. 1990

Vogel S (1992) *Vital Circuits: On Pumps, Pipes and the Wondrous Workings of the Circulatory System.* Oxford University Press.

Various (1984) American Physiological Society *Handbook of Physiology*. Sections 2 & 3: Circulation & Respiration. American Physiological Society.

Bronzino JD (Ed.) *The Bioengineering Handbook*. CRC Press 1995

The Cardiovascular System

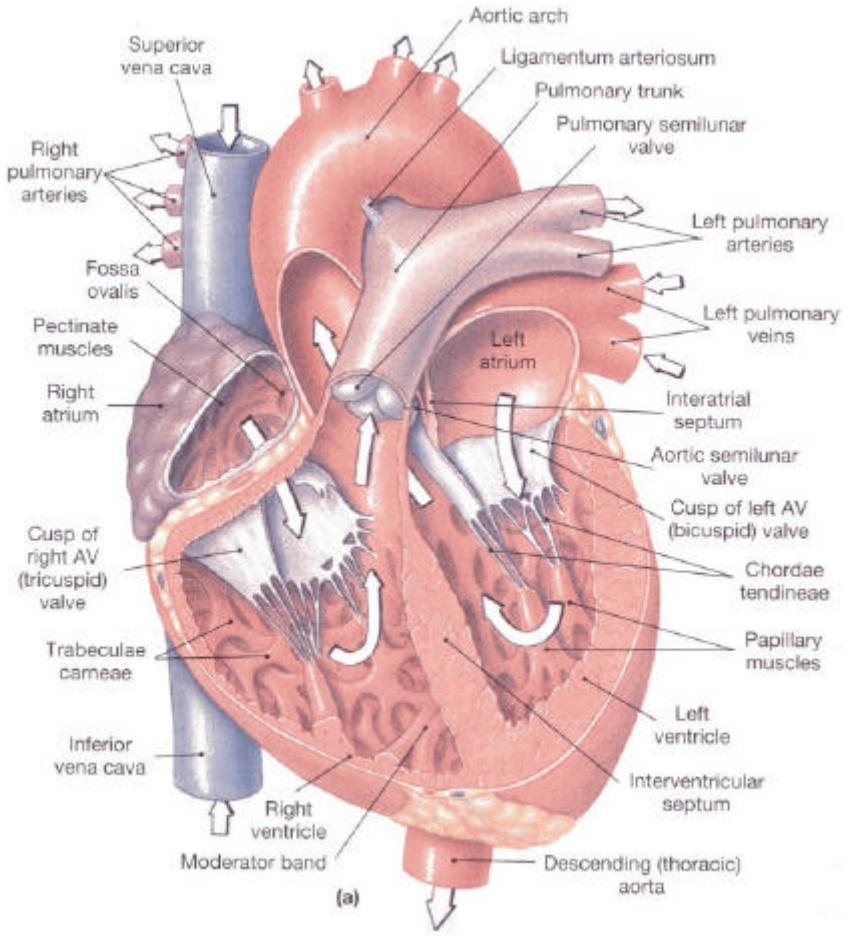
The Blood

Blood is composed of red blood cells suspended in the plasma which is primarily water containing ions, proteins and a relatively small number of cells. The volume fraction taken up by red blood cells, the haematocrit, is approximately 50%. This is very large given that the largest possible volume fraction without deformation of the red cells is about 60%.

The red blood cell, or erythrocyte, is biconcave disc shaped with a diameter of approximately 8 μm and a thickness of approximately 2 μm. It does not contain a nucleus, its contents being almost entirely haemoglobin, a protein which complexes with oxygen greatly increasing the ability of the blood to transport oxygen. The red blood cell is very deformable, which is important for its ability to traverse the microcirculation.

Red blood cells survive, on average, for about 120 days. The average trip around the circulatory system takes about 30 s, although the variation in circulation times is very large because of the very different path lengths that are possible. Compare, for example, the distance traveled around the coronary circulation to that around the circulation of the toe. The average red blood cell travels approximately 1000 km in its lifetime and makes approximately 1,000,000 circuits of the body.

The Left Side of the Heart



The Heart

A sketch of the heart showing its main structures and arterial and venous connections.
[adapted from FH Martini, Fundamentals of Anatomy & Physiology (3rd Edition)]

Left Atrium

Oxygenated blood comes from the lung to the left atrium via a number of pulmonary veins. The left atrium (LA) is a relatively thin walled compartment of the heart separated from the left ventricle (LV) by the mitral valve. When the pressure in the LV (P_{LV}) is greater than the pressure in the LA (P_{LA}) the mitral valve is shut and the LA acts like a fluid reservoir. When P_{LV} decreases because of LV relaxation, the pressure difference causes flow from the LA to the LV which opens the mitral valve.

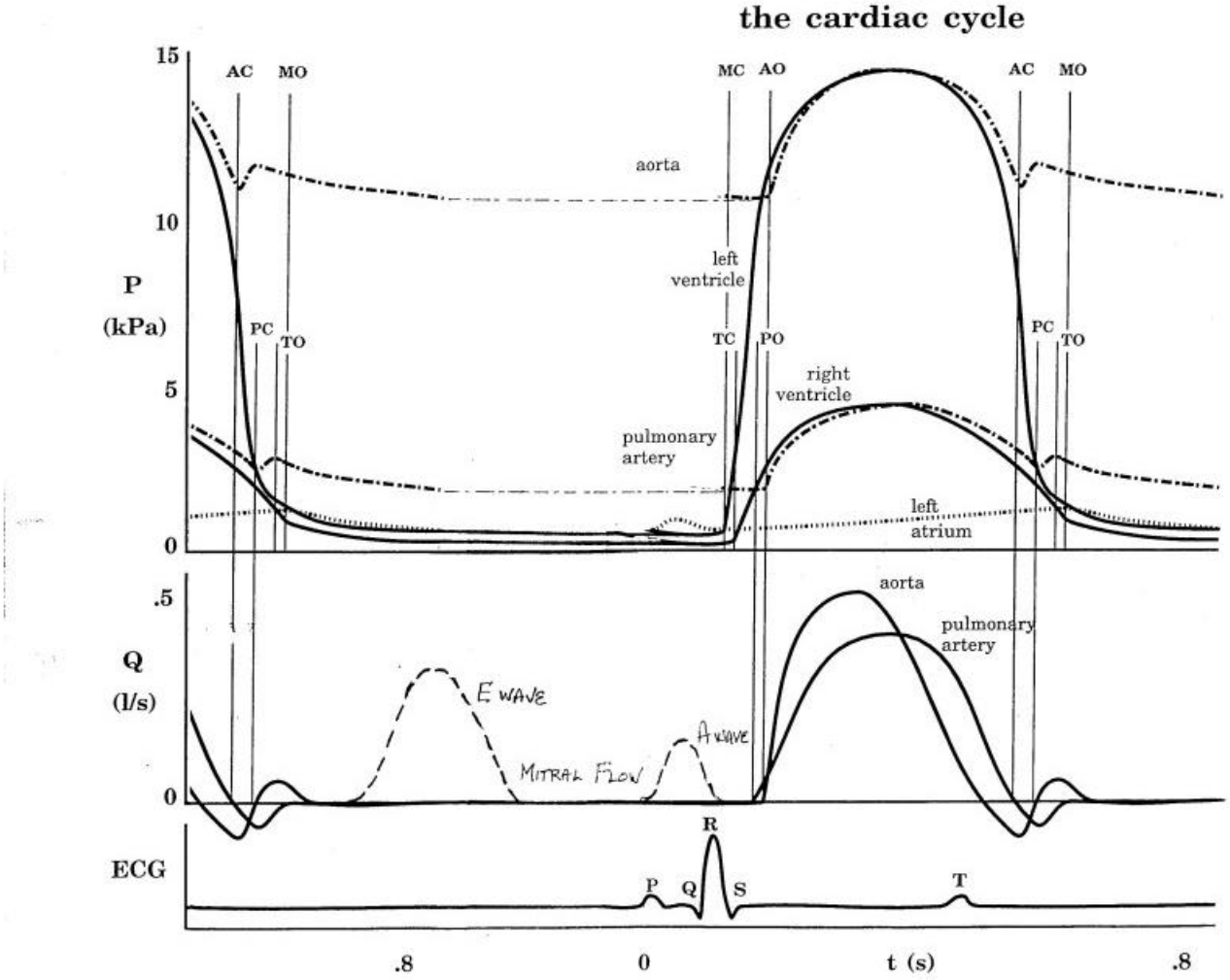
During diastole, there are normally two periods of flow from the LA to the LV. Early in diastole, there is a passive flow of blood, called the E wave, probably involving suction from the LV as it relaxes from its previous contracted state (this is still a somewhat contentious concept). The initiation wave of myocardial contraction starts in the atria and propagates to the ventricles via the AV node which delays the propagation. Thus, the contraction in the LA occurs before the contraction of the LV which raises P_{LA} causing further flow into the LV, called the A wave. In the healthy young person about 3/4 of the filling of the LV occurs during the A wave. In the normal elderly subject, the A wave is dominant. These transmitral velocities can be measured relatively easily by Doppler ultrasound and are important diagnostic measurements.

Left Ventricle

The left ventricle (LV) is the most muscular of the heart chambers. Its walls are approximately 1 cm thick which, when they contract, generate a pressure of approximately 15 kPa (although much higher pressures can be generated in exercise or in pathological conditions.

When the contraction wave reaches the LV, the pressure, P_{LV}, increases and when $P_{LV} > P_{LA}$ blood starts to flow back into the LA and the mitral valve closes, stopping further backflow. As long as P_{LV} is also less than pressure in the aorta PAo, the aortic valve remains closed. During this period when $P_{LA} < P_{LV} < P_{Ao}$, both the mitral and aortic valves are closed and so the volume of the ventricle must be constant. This period is called the isovolumic contraction period

When P_{LV} becomes greater than P_{Ao}, the aortic valve opens and blood flows from the LV into the aorta. As contraction continues, P_{LV} continues to rise and blood continues to accelerate into the aorta. Eventually the contraction ceases and P_{LV} starts to decrease. During the deceleration phase, P_{LV} is slightly less than PAo (see below for the relationship between pressure gradient and the acceleration of blood) but blood flow continues because of the inertia of the blood. Eventually, the velocity of the blood decreases to zero and then begins to reverse. The reverse flow sweeps the aortic valves closed. This marks the end of ventricular systole.



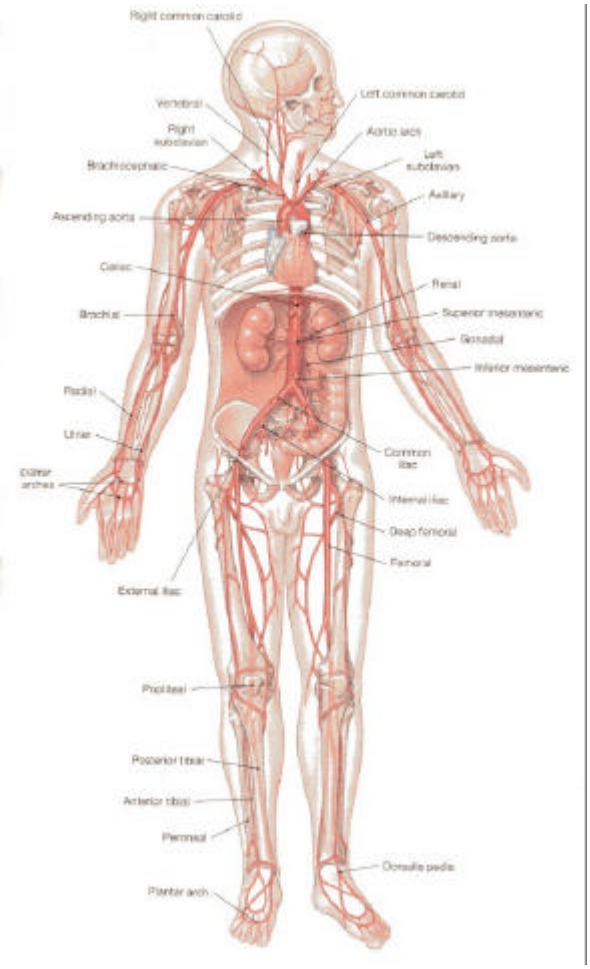
Pressure, Flow and ECG during the Cardiac Cycle

[adapted from Nichols & O'Rourke, McDonald's Blood Flow in Arteries (3rd Ed.)]

After aortic valve closure, relaxation of the myocardium continues. Again there is a period when $P_{LA} < P_{LV} < P_{Ao}$, and both the mitral and aortic valves are closed, so that LV volume must be constant. This is called the isovolumic relaxation phase. Myocardial relaxation, a metabolically active process, continues until $P_{LV} < P_{LA}$. When this happens, the mitral opens and the filling phase of the cycle starts.

The Systemic Arteries

The arterial system is a complex mainly bifurcating tree structure with a few more complex interconnections in, for example, the cranium, the hand, the foot and the liver. The arteries are lined by a single layer of endothelial cells which provide a continuous surface between the blood and the cells and connective tissue of the artery wall. The endothelial cells (taken together, possibly the largest organ of the body) are metabolically very active and are thought to transduce certain properties of the blood flow. The endothelial layer is relatively fragile and could not bear the mechanical loads on the artery wall.



The main mechanical support of the artery wall is provided by the media which is a lamellar structure composed of elastin, smooth muscle, collagen and other connective tissue components such as fibrogen, proteoglycans and glycoproteins. The elastin bears most of the load under normal physiological conditions. The smooth muscle can be activated either neuronally or humorally so that the elasticity, or tone, of the artery wall can be controlled. The collagen probably serves to prevent too much distension of the artery wall under abnormal conditions, acting like a 'string bag' around the more elastic elements, elastin and smooth muscle.

Flow enters the aorta at the aortic root, which is a relatively distensible portion of the aorta. Pressure and flow propagate along the artery in the form of an elatic tube wave first described by Thomas Young (1808). The elastic wave speed is dependent upon the local distensibility of the artery which varies from artery to artery. The wave speed is approximately 5 m/s in the aortic root and increases to 10-20 m/s in the less distensible distal arteries.

There are up to 20 bifurcations along any one path through the arterial system. Thus, there are up to 2^{20} (approximately 1,000,000) arteries and arterioles. I would guess that about 1000 have names, the rest are just known as the small arteries and arterioles and are defined by their size.

At each bifurcation the diameter of the daughter vessels is generally less than the diameter of the parent vessel, but the combined areas of the daughter vessels is generally greater than the cross sectional area of the parent vessel. Similarly the length of the daughter vessels is generally less than the length of the parent vessel.

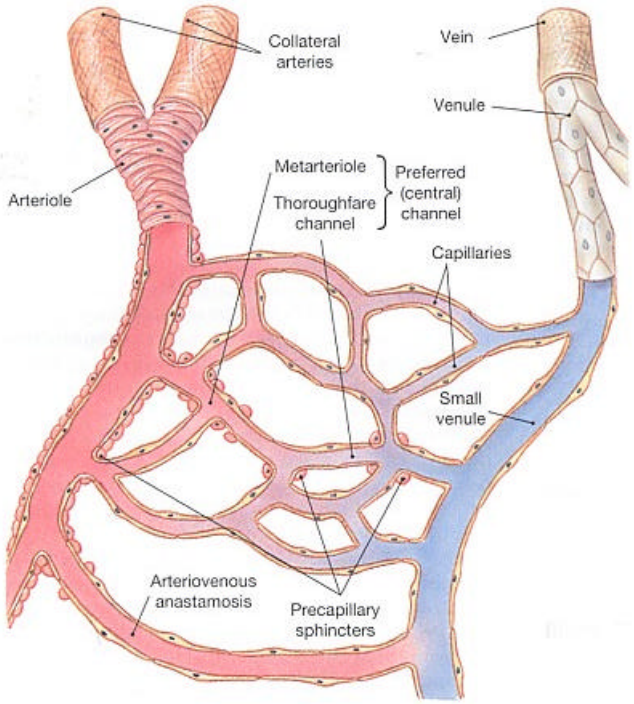
The fractal model of arteries

If the arterial system was fractal (i.e. if each generation scaled like every other generation) then the ratio of the sum of daughter areas to the area of the parent vessel would be about 1.2, the ratio of a daughter to parent diameter would be about 0.75 and the ratio of the length of a daughter to the length of the parent would be about 0.65. The number of arteries, their total cross section, and their diameter and length after 10 and 20 bifurcations are given in the table.

Symmetrical Fractal Arteries		
	after 10 bifurcations	after 20 bifurcations
number	$(2)^{10} = 1024$	$(2)^{20} \sim 10^6$
total area	$(1.2)^{10} = 6.2\ A_0$	$(1.2)^{20} \sim 40\ A_0$
diameter	$(.75)^{10} = .056\ d_0$	$(.75)^{20} \sim .003\ A_0$
length	$(.65)^{10} = .013\ L_0$	$(.65)^{20} \sim .0002\ L_0$

Note, however, that in cases where detailed measurements of arterial trees have been made, the fractal description of arteries has been shown to be a very poor approximation to reality.

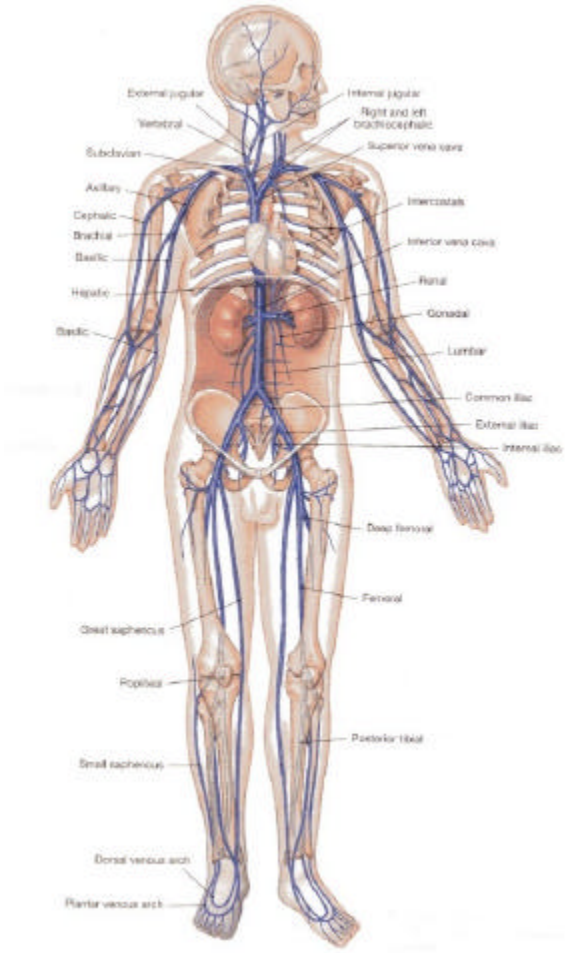
The Systemic Microcirculation



The microcirculation is made up of the smallest blood vessels; the arterioles, the capillaries and the venules. The structure of the microcirculation varies greatly between different tissues and so it is difficult to characterise it generally. One feature common to all capillary beds is that the network of vessels is much more complex than the relatively simple bifurcating, tree structure of the arteries and veins.

The main resistance to flow lie in the arterioles and capillaries. The arterioles at particular sites are surrounded by muscle, the prearteriole sphincters, which can change the local resistance to flow by contracting. This is the mechanism that controls the flow of blood to individual organs and regions. Because of the large pressure drop in the arterioles, the capillary pressure is relatively low compared to arterial pressure but larger than venous pressure.

The Systemic Veins



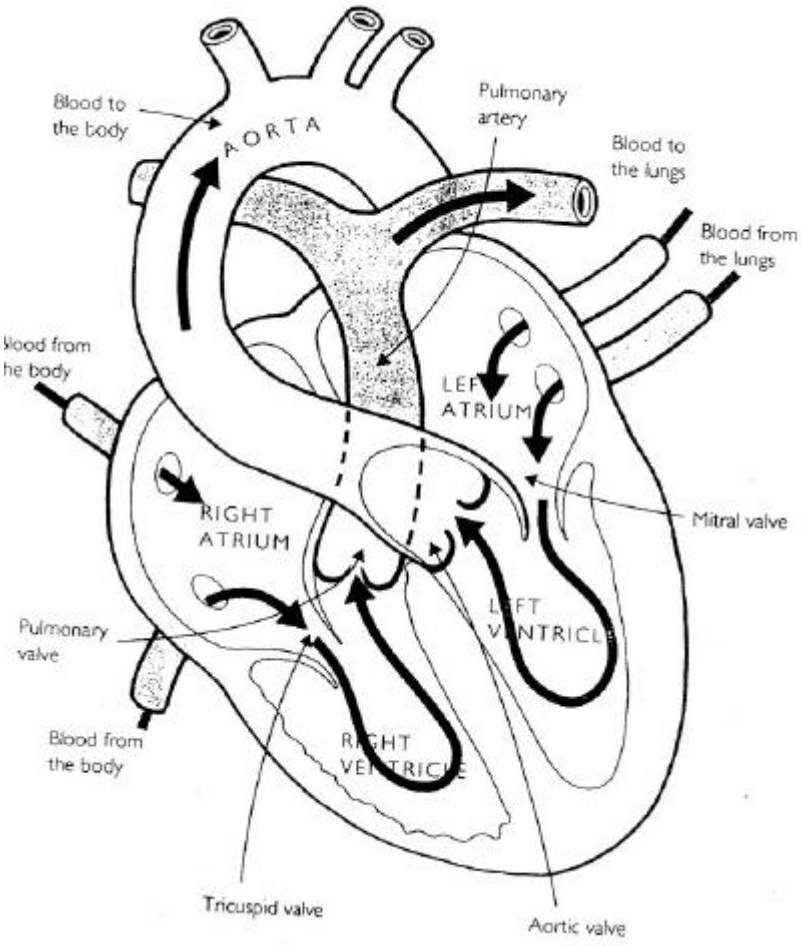
The venous system is roughly parallel to the arterial system. With a few exceptions, the corresponding artery and vein lie next to each other with the vein being slightly larger cross section than the artery. About 60-70% of the total blood volume is in the venous system.

The walls of the veins, like those of the arteries, are covered by a single layer of endothelial cells that separate the blood from the connective tissue of the vein wall. The vein walls are much thinner than the arterial walls containing little elastin or smooth muscle, reflecting the much lower pressure in the veins.

On unique feature of veins is the existence of valves that are oriented to allow flow toward the heart and prevent back flow toward the periphery. These valves are crucial to the operation of the muscle pump whereby veins which are embedded in skeletal muscle are compressed when the muscle contracts, pushing blood toward the heart. This mechanism is very important in the return of blood from the leg to the heart when standing.

It may be of interest that giraffes have valves similar to human venous valves in the arteries of their neck.

The Right Side of the Heart



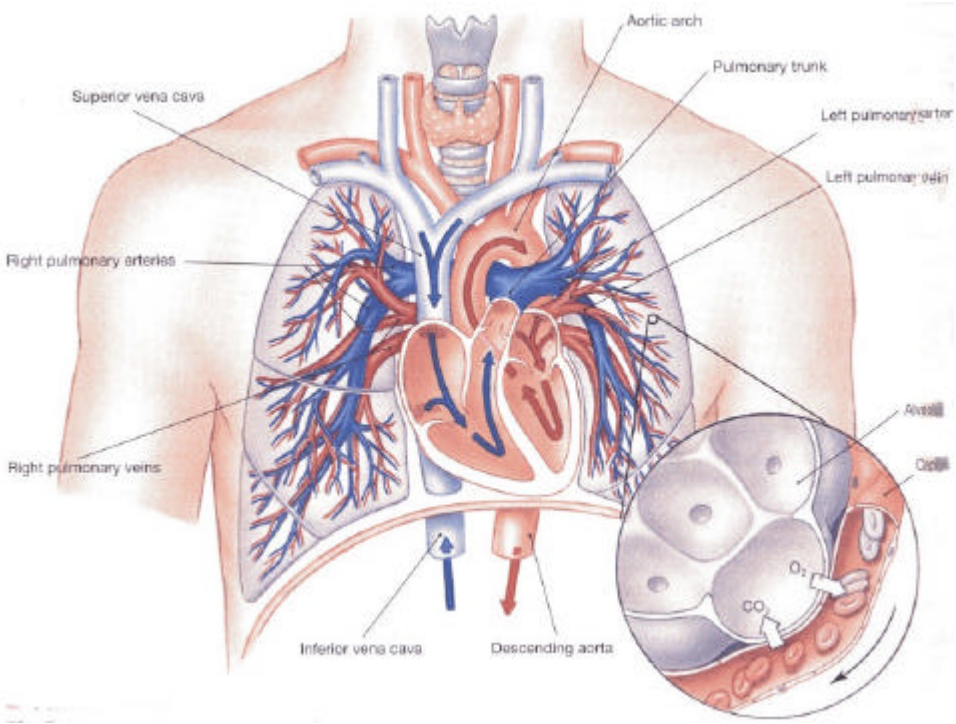
The myocardium of the right heart is much thinner than in the left heart. Apart from that, its structure is similar to the left heart: a right atrium (RA) separated from the right ventricle (RV) by the tricuspid valve and a right ventricle separated from the pulmonary artery by the pulmonary valve.

Blood flows from the RA to the RV during diastole when the pressure in the RV falls below the pressure in the RA, first passively (the E wave) and then actively as a result of RA contraction. The contraction of the RV causes the pressure to rise closing the tricuspid valve and eventually opening the pulmonary valve. The pressure generated by the RA (peak pressure less than 5 kPa) is much lower than that generated by the LV (systolic pressure approximately 15 kPa).

Since the average output of the RV must equal that of the LV, the lower pressure in the pulmonary circulation means that the resistance of the pulmonary microcirculation must also be lower than that of the systemic circulation.

The Pulmonary Circulation

The pulmonary arteries are similar in size and structure to the systemic arteries except that the walls are much thinner, reflecting the lower pulmonary pressure. Because they are more distensible than systemic arteries, the wave speed in the pulmonary arteries is much lower, about 2 m/s compared to approximately 10 m/s. The pulmonary arteries bifurcate very symmetrically and regularly.



The pulmonary capillaries surround the alveoli so densely that flow in them is often modelled as flow between two sheets rather than flow in individual tubes. The walls of the pulmonary capillaries and the alveoli are very thin and permeable to faciliate gas transport between inspired gas and the blood.

The pulmonary veins run parallel to their corresponding arteries. Like systemic veins, they are relatively thin walled and somewhat larger than their corresponding artery. The pulmonary veins are an important region for pooling of the blood. Oxygenated blood returns to the left atrium via 6-8 pulmonary veins, thus completing the circulation of blood.

The Coronary Circulation

The coronary circulation deserve special mention when considering the mechanics of the cardiovascular system because of its importance clinically and because of the special nature of flow through it.

In order to generate the high blood pressures during systole, the myocardium contracts increasing its interstitial pressure to levels greater than systolic pressure. Thus the pressure surrounding the intramyocardial blood vessels during systole is generally greater than the internal pressure causing the vessels to collapse. As a result, flow in the coronary arteries is stopped (or even reversed due to the backward displacement of the blood in the collapsing vessels) during systole. The intramyocardial pressure decreases as the myocardium relaxes during diastole and the collapsed vessels reopen allowing blood to flow. Thus, the perfusion of the myocardium occurs almost entirely during diastole.

The need to perfuse the myocardium during diastole is regarded by many as the reason for the high systemic arterial pressure in mammals.

Haemodynamics

Basic Concepts

1. Control Volume

Because it is very difficult to follow a 'piece' of fluid in motion, it is convenient to define a 'control volume' and consider what happens to the fluid in that volume.

2. Conservation of Mass

Since mass must be conserved, the rate of increase of mass in the control volume will be equal to the net flux of mass into the control volume. Written in equation form

d(Mass in control volume)/dt = (Mass flux in) - (Mass flux out)

Incompressible Fluid

If the density of the fluid is constant (incompressible) then the mass conservation equation can be written more simply in terms of volume. Blood is incompressible. Gases, such as air, are not incompressible.

d(Volume)/dt = (Volume flow rate in) - (Volume flow rate out)

***Examples**:*

A. During early systole there is a period when the pressure in the LV is greater than the pressure in the LA, so the mitral valve is closed, and less than the pressure in the aorta, so the aortic valve is also closed. If we consider the LV as the control volume, there is no flow in and no flow out during this period. Therefore the rate of change of the LV volume is zero and so the volume is constant. This is the isovolumic contraction phase of the cardiac cycle.

B. When LV pressure exceeds aortic pressure, the aortic valve opens and blood flows into the aorta. The mitral valve is closed and so, again taking the control volume to be the LV volume, the rate of change of LV volume is equal to the volume flow rate into the aorta.

3. Conservation of Momentum

Newton's second law applies to fluids. In its most fundamental form it says that the rate of change of momentum of a body is equal to the net force applied to the body. In its more familiar form it says that the net force is equal to the mass of the body times its acceleration, where acceleration is the rate of change of velocity. In the form of an equation

$$F = ma$$

where F is the net force, m is the mass of the body and a is the acceleration.

In fluids it is usually more convenient to think of stress, defined as force per unit area, instead of force. The principle stresses are pressure, the force per unit area acting normal to a surface, and shear stress, the force per unit area acting parallel to a surface.

The other principle force acting on a fluid is gravity. The force due to gravity is usually called the weight, W, which depends on the mass of the body, m, and the acceleration due to gravity, g.

$$W = mg$$

A. Hydrostatic pressure

If a stationary column of fluid is acted upon by gravity, there must be a force acting to counteract gravity. That is, the net force acting on the fluid must be zero or else, according to Newton's second law, there would be an acceleration and the fluid could not be stationary. This force is called the hydrostatic pressure. If the column of fluid has a height, h, the difference between the force acting on the column due to the pressure at the bottom of the column (acting upward) and the force acting on the column due to the pressure at the top (acting downward) must equal the weight of the water. In equation form

hydrostatic pressure = (density)(acceleration due to gravity)(height)

or

$$P_H = \rho g h$$

B. Inviscid (frictionless) flow

If viscosity is negligible, then the flow depends only on the pressure gradient, change in pressure per unit distance. Consider flow in a section of tube of length, L, and cross section area, A (our control volume). If the pressure at the inlet to the section is P_1 and at the outlet is P_2, then the net force acting along the tube is $(P_1 - P_2)A$. The volume is AL and so the mass in the control volume is ρAL. Thus according to Newton's second law

$$(P_1 - P_2) = \rho L a$$

where a is the acceleration of fluid in the control volume, r is the density and we have canceled the A which appeared on both sides of the equation.

$(P_1-P_2)/L$ is the pressure gradient, the change in pressure per unit area. Thus, we see that the acceleration of fluid is proportional to the pressure gradient

Note: Cardiologists commonly refer to the pressure difference measured at two different locations in the heart and vessels as the 'pressure gradient'. This is terminological inexactitude and should be avoided. In order to measure the real pressure gradient it would also be necessary to measure the distance between the two sites of measurement.

***Example**:*

Assume that a pressure difference of 1 mmHg (133.3 Pa) is applied to blood over a distance of 1 cm. The density of blood is approximately 1050 kg/m3. The resulting acceleration of the blood is

$$a = 133.3/(0.01x1050) = 13 \text{ m/s2}$$

The acceleration due to gravity is 9.8 m/s2, and so we see that this relatively small pressure difference will produce an acceration of more than '1g'.

Also note that the peak velocity in the aorta is of the order of 1 m/s and the period of systole during which the blood is accelerating is approximately 100 ms. For the blood to go from rest to 1 m/s in 0.1 s the acceleration is of the order of 1/0.1 = 10 m/s^2. In other words, the observed acceleration in aorta is produced by a pressure gradient of approximately 1 mmHg/cm. This is a very small drop in pressure relative to the mean pressure in the aorta of more than 100 mmHg.

C. Steady flow

If a flow is steady, there is no acceleration and Newton's second law tells us that the net force acting on the fluid in the control volume must be zero. If the flow is viscous, this means that the viscous forces (viscosity is like fluid friction) are balanced by pressure forces. In many cases this can be expressed in the form of a resistance, the fluid analogy of Ohm's law

$$\text{Pressure difference} = \text{Resistance} \times \text{Flow rate}$$

Usually this relationship is not exact, the relationship between pressure and flow is most often nonlinear, but it is a very convenient and useful approximation.

D. Reynolds number

It is important to remember that words like 'big' and 'small' are relative, to be meaningful scientifically they must be followed by 'compared to ...'. For example, a velocity of 1 m/s observed in an artery is big compared to the velocity at which fluid moves through a capillary, about 1 mm/s, but is small compared to the speed at which waves propagate along the arterial system, about 10 m/s.

Another way to express this point is that the number used to express a dimensional number depends upon the units used. A velocity of 1 m/s is also equal to 1000 mm/s and to 0.001 km/s.

A convenient way to compare quantitative parameters, very commonly used in fluid mechanics, is to represent them as nondimensional numbers. A nondimensional parameter is represented by a simple number. For example, given two lengths b and c, it is meaningless to ask if b is small but it is meaningful to ask if b is small compared to c. An easy way to represent this comparison is by the ratio b/c, which is nondimensional. If $b/c < 1$ then b is small compared to c.

The most important nondimensional parameter in fluid mechanics is the Reynolds number, which is the ratio of inertial and viscous effects in the flow. It is defined as

$$Re = \rho UD/\mu = (\text{inertial forces})/(\text{viscous forces})$$

where ρ is the density, U is the velocity, D is the diameter of the vessel and μ is the coefficient of viscosity, a material property of the fluid.

Examples:

a) Large artery.

$$r = 1050 \text{ kg/m}^3$$

$U = 1\ m/s$
$D = 2\ cm$
$m = 0.04\ Ns/m^2$

Therefore, the Reynolds number is

$$Re = (1050)(1)(0.05)/(0.04) = 5000 >> 1$$

This means that the flow in large arteries is dominated by inertia, not viscosity.

b) Arteriole.

The density and coefficient of viscosity are the same as for a large artery (Is this true a capillary where the diameter is smaller than the size of a red blood cell?)

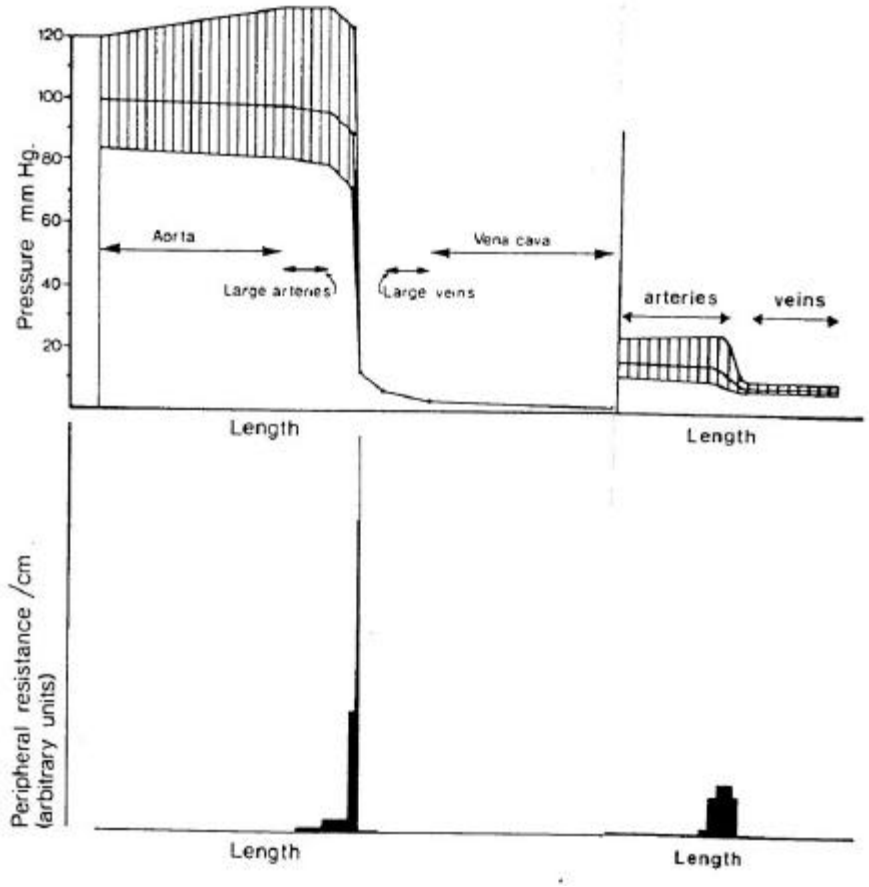
$U = 1\ mm/s$
$D = 0.1\ mm$

Therefore, the Reynolds number is

$$Re = (1050)(0.001)(0.0001)/(0.04) = 0.25 < 1$$

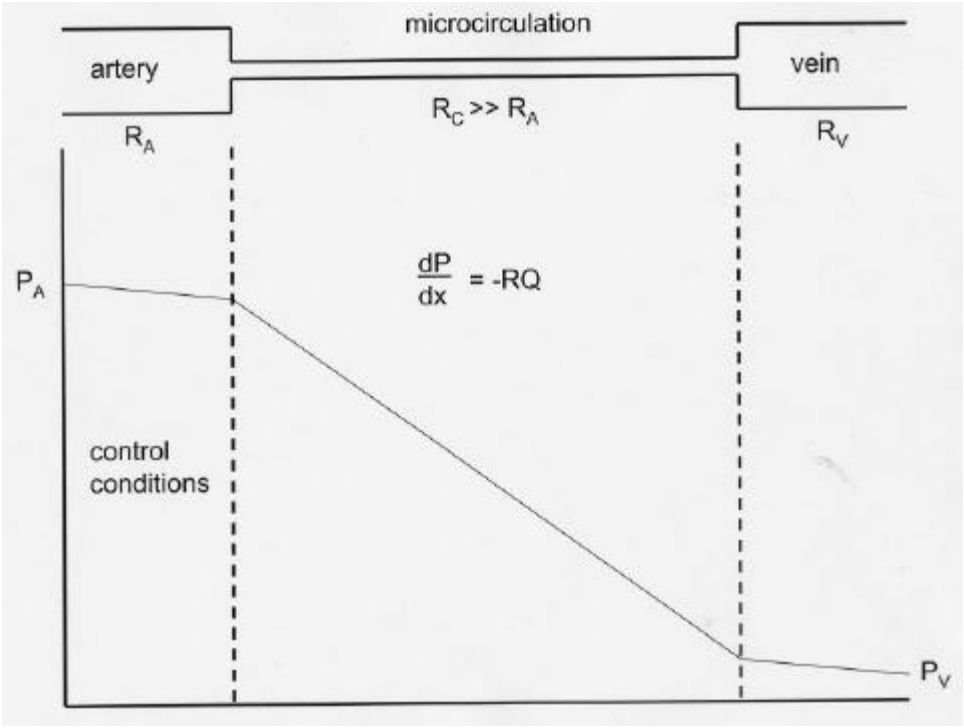
This means that viscous effects are dominant in arterioles.

Description of Flow in the Cardiovascular System

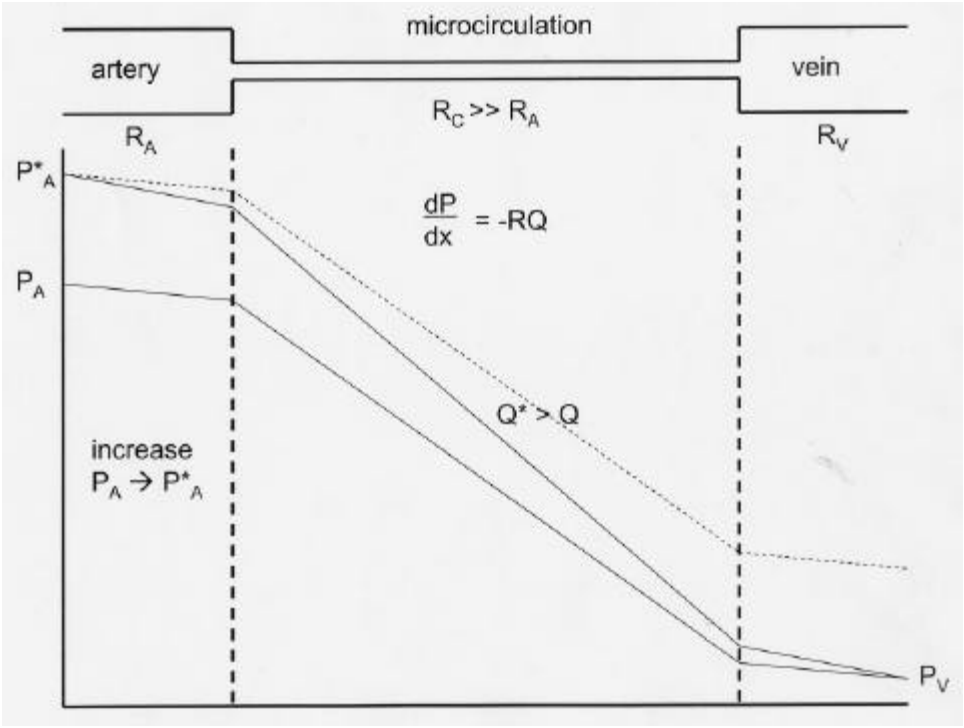


Adapted from: Nichols and O'Rourke, McDonald's Blood Flow in Arteries

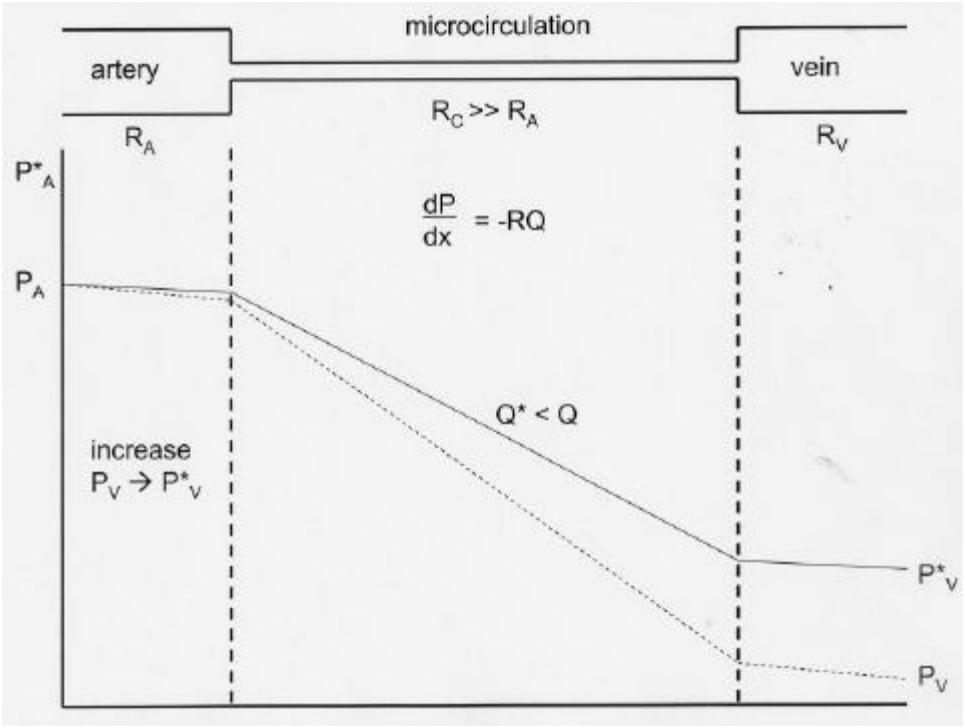
Determining the mean flow:



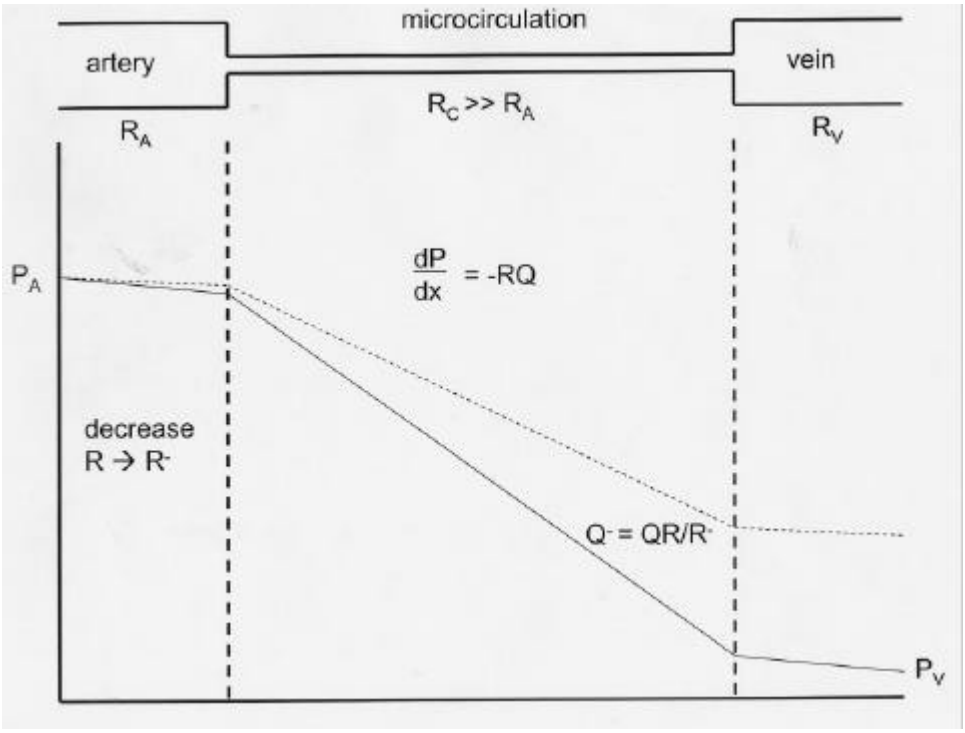
As a simple example of what determines the conditions of pressure and flow in the cardiovascular system, we consider the very simple case of a single artery with resistance R_A connected by a single capillary with resistance R_C to a single vein with resistance R_V. For a simple flow, the pressure gradient dP/dx = -RQ, where Q is the volume flow rate. This is analogous to Ohms law relating voltage, current and resistance. Like the real circulation we will assume that R_A and R_V are very small while R_C is large. Assume that we impose control conditions on this simple system where the pressure at the inlet of the artery is P_A and the pressure at the outlet of the vein as P_V. Since the pressure gradient in each vessel depends upon Q, the problem is to find the value of Q which leads to the specified pressure drop across the combined vessels. (Remember that pressure gradients and flow go together in the sense that you cannot have flow without a pressure gradient and if there is a pressure gradient there will be acceleration and hence flow of the fluid.)



If we increase the pressure at the inlet to the artery from P_A to P_A*. and keep the same P_V and Q then the pressure distribution will become the dotted line in the figure, where the gradients are the same as in the control condition but they start at the higher pressure. This does not lead to the pressure P_V and therefore cannot be the solution. The solution is found by increasing Q so that the pressure gradients are increased until the pressure at the outlet is P_V. Thus, increasing the pressure at the inlet leads to an increas in Q.



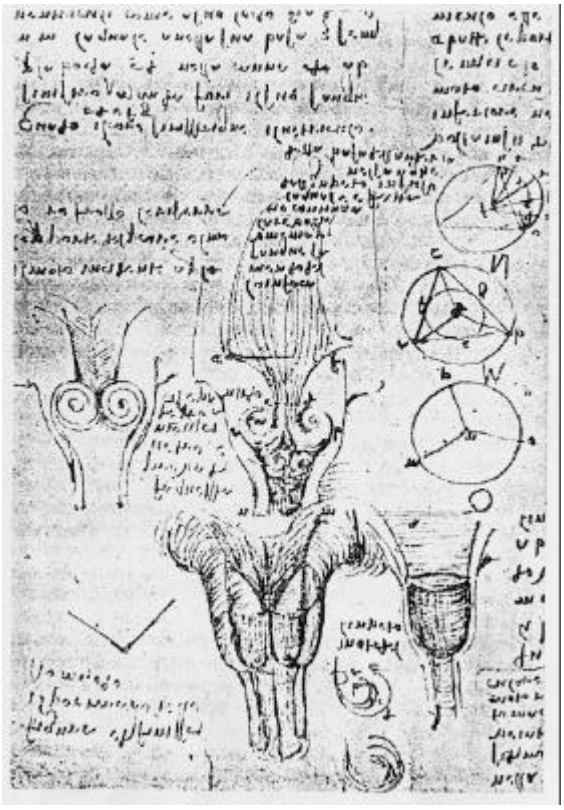
If, instead, we increase the pressure at the outlet of the vein from PV to PV* and keep the same PA and Q, then the pressure distribution will be the same as the control condition (the dotted line in the figure). This does not give the right outlet pressure. The solution in this case comes from decreasing Q which leads to smaller gradients in each of the vessels until the outlet pressure is equal to P_V*. Thus, increasing the outlet pressure leads to a decrease in Q.



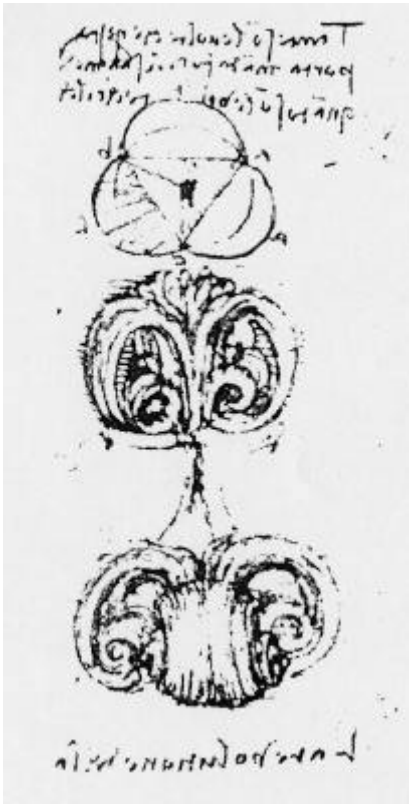
Finally, if we decrease the resistance in the capillary from R to R*, keeping the same inlet and outlet pressures as in the control condition, then we find that the smaller gradient leads to the dotted line in the figure which does not give the required P_V at the outlet. The solution is to increase Q until the pressure gradient is equal to that which was observed in the control conditions. The new flow rate, $Q^* = Q R_C/R_C^*$. Thus, reducing the resistance in the capillary will lead to a higher flow rate. This is the mechanism by which the body adjusts the distribution of flow in the circulation. By decreasing/increasing the resistance in the microcirculation vessels (by relaxing/contracting the muscles in the walls of the arterioles to increase/decrease their diameter) the flow to that region can be increased/decreased.

Flow in the systemic arteries:

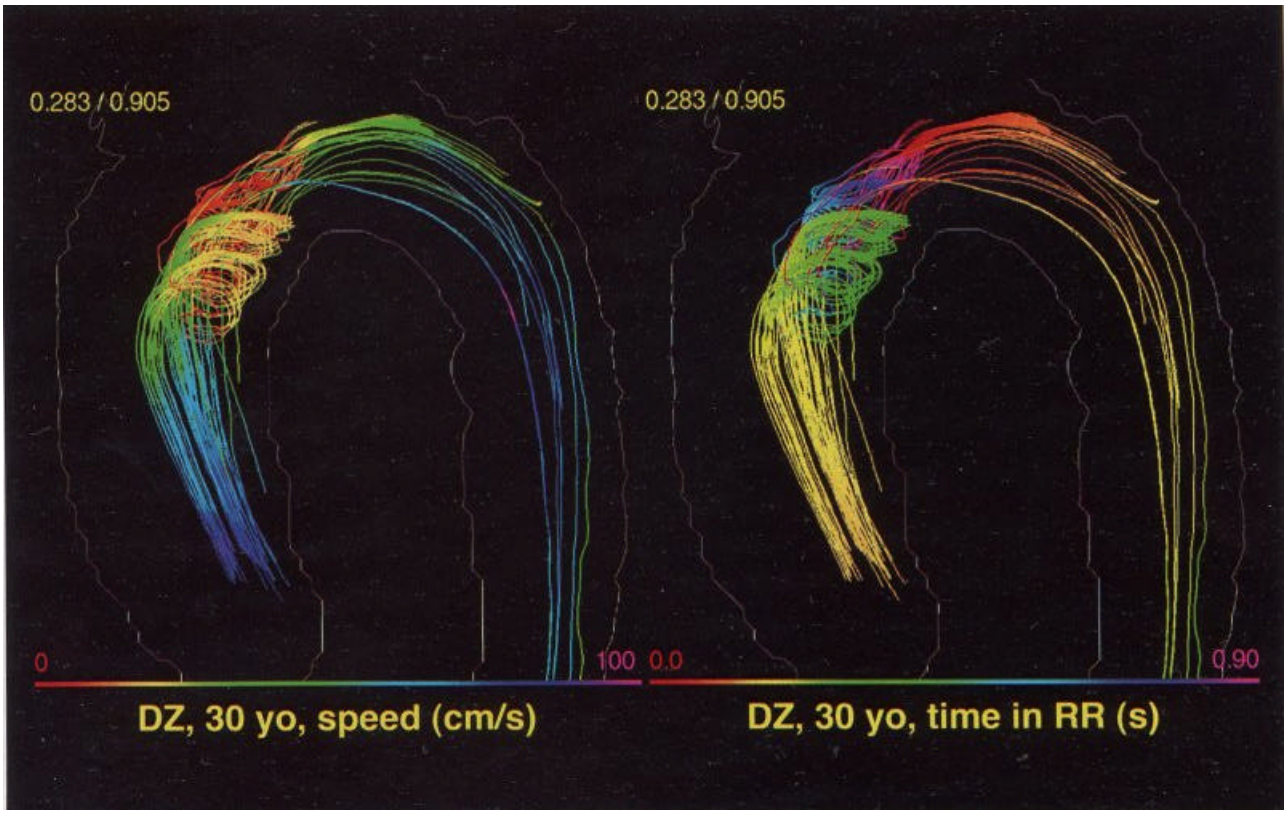
Pressure and velocity are highly pulsatile in the aorta due to the intermittant contraction/relaxation phases of the cardiac cycle. The peak pressure, systolic pressure, is approximately 15 kPa. The minimum pressure, diastolic pressure, is approximately 10 kPa. The mean pressure is approximately 12 kPa. The velocity goes from zero to approximately 1 m/s during the early part of systole. During late systole the velocity in the aortic root decreases and reverses slightly. It is this backward velocity that closes the aortic valve.



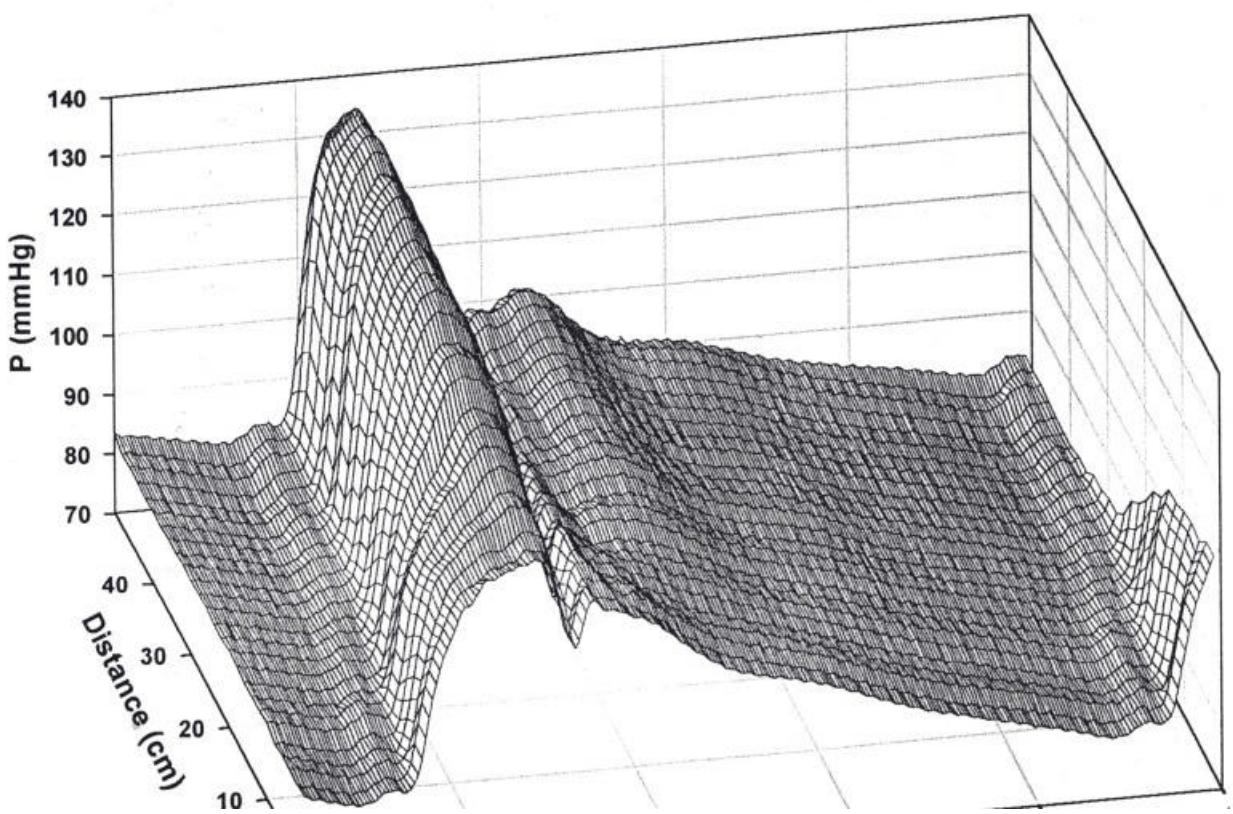
The flow in the ascending aorta is particularly complex. The intermittent flow through the aortic valve produces a vortex flow (very similar to a smoke ring) in the sinuses of Valsalva. These can be seen in the sketches of Leonardo da Vinci. These vortices help in the closing of the valve in late systole when the flow starts to reverse.

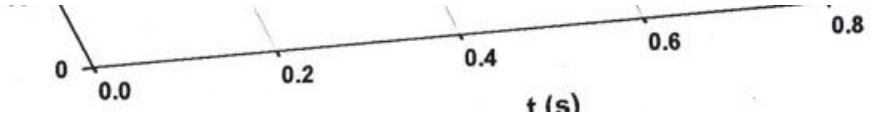


The complexity of the flow can be seen from the MR images (*used with the permission of H. Bogren, UC Davis*). These show 'fluid particle paths' for fluid originally in the left ventricle (LV). The position of the ventricle and the walls of the aorta are shown as faint lines bounding the flow. On the left the colour coding is for the instantaneous speed of the fluid, on the right the same particle paths are shown but colour coded for the time during the cardiac cycle (RR interval). We see that fluid originally in the LV is ejected into the ascending aorta during early systole at relatively high speed along relatively straight paths. Towards the end of systole, the flow slows down and the particle paths start to swirl around in a very complex way, some of the blood moving back towards the aorta. This swirling flow continues during most of diastole. At the start of the next systole, the blood is again accelerated and convects around the aortic arch into the descending aorta. This flow is very complex and very different from that described in most of the text books (and many of the specialist books on haemodynamics). The message here is not the exact nature of the flow, but simply that it is very complex and poorly understood.

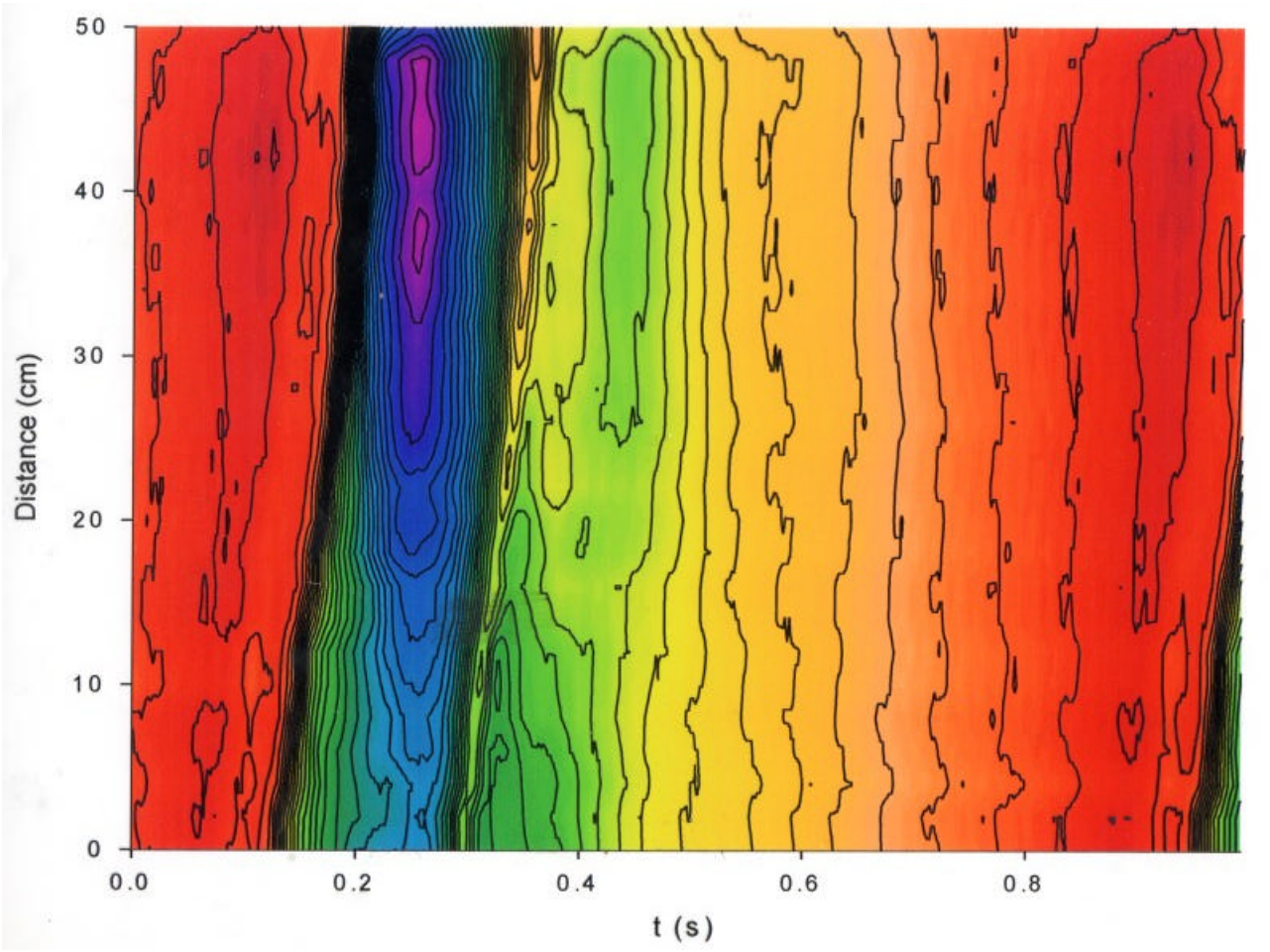


The pressure and flow generated by the injection of blood into the aortic root from the LV propagates toward the periphery by means of a wave. The wave is a pressure/velocity wave where the distension of the elastic arterial wall by the pressure represents the potential energy and the velocity of the blood represents the kinetic energy. The wave speed depends upon the local distensibility of the artery and varies from about 5 m/s in the relatively distensible aortic root to 10-15 m/s in the more muscular, less distensible peripheral arteries. Because these waves can be reflected from different parts of the arterial system, the resultant pressure and velocity waveforms alter in a very complex way as we move distally.





The pressure waveform in the canine central arteries measured by J.J. Wang using a catheter based pressure transducer. The pressure was measured every 2 cm from the aortic root to the aorto-iliac bifurcation. Note how the systolic pressure increases with distance away from the aortic root. There are also large changes in the dicrotic notch and during early diastole.

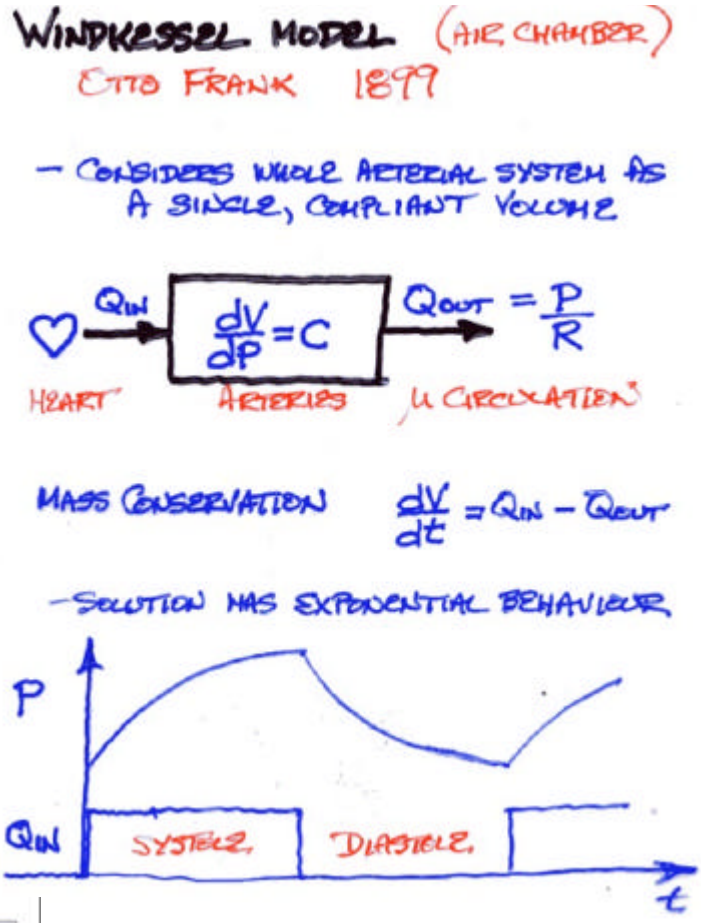


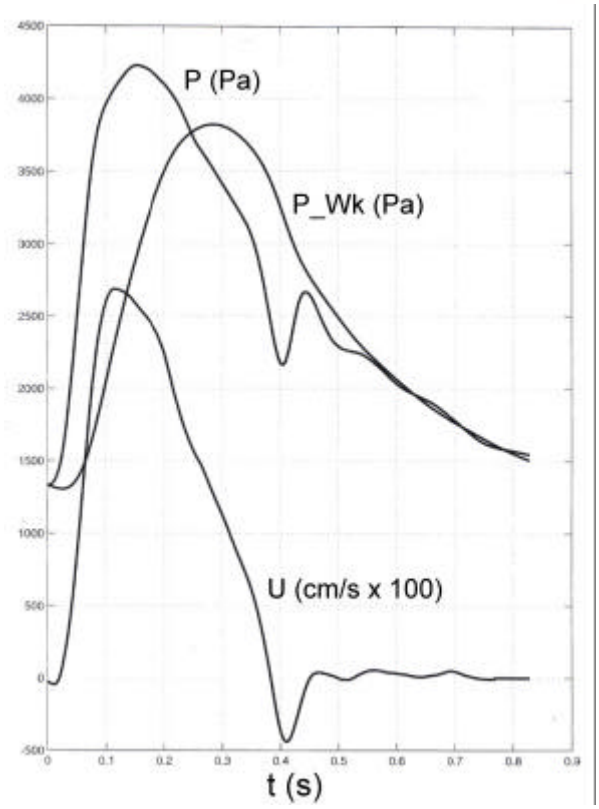
The same data as in the previous illustration represented as a contour map. Note the slope of the contours associated with the front of the pressure pulse. Since the two axes are distance and time, the slope is a velocity and indicates the local wave speed. The increase in the peak pressure can be seen from the colour coding where purple represents the highest pressures.

The Windkessel Model

The primary mechanical function of arteries is to serve as a conduit for blood to the peripheral microcirculation where exchange of gases, nutrients and wastes occurs. Because they are elastic, they also serve another function, as a hydraulic capacitor which serves to smooth out the highly pulsatile nature of flow from the heart.

Otto Frank (1898) compared this mechanism to the Windkessel (literally air chamber) used on early fire pumps to convert the pulsatile pumping action into relatively smooth flow. The theory considers the whole of the arterial system to be a single compliant compartment with blood flowing into it from the LV during systole, Q_{in}, and out of it through the microcirculation, Q_{out}. Because of the high resistance of the microcirculation, Q_{out} can be modelled very well by a hydraulic Ohm's law. With this assumption, it is possible to solve for the pressure of the arterial compartment, yielding an exponential function increasing during systole and falling during diastole.





The Windkessel model is an excellant description of the pressure waveform during diastole when there is no flow into the arterial system, but is a poor description of systolic flow.

The difference between the predicted Windkessel pressure and the measure pressure is related to the generation of arterial waves.

This example is from data measured in the main pulmonary artery of the dog.