Cardiovascular Fluid Dynamics
Draft

K.H. Parker and D.G. Gibson

Department of Bioengineering
National Heart and Lung Institute
Imperial College of Science, Technology and Medicine
London SW7 2AZ, U.K.

26 September 2005

The heart is a pump whose sole function is to perfuse the body with blood. Blood, like all matter, is subject to the laws of mechanics and a better understanding of the physical principles underlying the motion of blood can be useful to the cardiologist.

This article is divided into two parts; in the first we introduce some of the basic ideas of fluid mechanics and in the second we demonstrate how these can be applied to the heart and arteries. Although blood flow is extremely complex, too complex to be described in full detail, the basic principles that we present will allow us to capture the essential properties of blood flow.

Mechanics is a very old subject, nearly as old as medicine [Forbes & Dijksterhuis, 1963], and it is intimately entwined with the development of applied mathematics. This close connection with mathematics has meant that mechanics is generally perceived to be highly mathematical, enabling physicists and engineers to predict and design with precision. Unfortunately, this perception can discourage the less quantitative to learn the basic principles of mechanics. We feel that the concepts of mechanics can be learned and applied without the mathematics in a way that can benefit cardiology. That is the purpose of this essay. We will use a few equations and a number of symbols as shorthand, but no complex mathematics.

1 The Basic Principles of Fluid Mechanics

1.1 Dimensional integrity

Dimensions[1]

Probably the most fundamental physical principle is that of dimensional integrity. All physical quantities have dimensions which, in mechanics, can be expressed in terms of the basic dimensions mass $[M]$, time $[T]$ and distance $[L]$. It is very important to remember that all physical properties carry their dimensions with them; a parameter that

---

[1]The word '*dimension*' is used commonly to refer to spatial extent. However, we are using the broader physicist's definition: *"A physical property, such as mass, length, time, or a combination thereof, regarded as a fundamental measure of a physical quantity."* For example, velocity has the dimensions of distance/time.

does not have the dimensions of pressure cannot be a pressure. Similarly, values of pressure, however they are measured, will carry with them the unique physical dimensions of 'pressure'.

Furthermore, an equation involving physical quantities denotes not only equality in magnitude but equality in dimension. Thus it is just as nonsensical to say 'two seconds equals two metres' as it is to say 'two equals four'.

Units

The basic dimensions should not be confused with the units in which they are measured. For example, time can be measured in seconds, minutes, hours, days, weeks, years, *etc.* but all of these units have the basic dimension $[T]$. The SI convention for units has made things much simpler by reducing and regulating the proliferation of units. Thus in SI units mass is measured in kg, time in s and distance in m. Unfortunately, not all countries ascribe to the SI convention and even within the convention non-standard units such as $mmHg$ are permitted. We will use SI units throughout this essay and so the dimensions of the various properties should be evident.

	definition	dimensions	units	symbol
time	basic dimension	T	s	t
mass	basic dimension	M	kg	m
distance	basic dimension	L	m	x
velocity	distance/time	$\frac{L}{T}$	$\frac{m}{s}$	U
acceleration	distance/time^2	$\frac{L}{T^2}$	$\frac{m}{s^2}$	a
force	mass $\times$ acceleration	$\frac{ML}{T^2}$	$N\ (=\frac{kg\,m}{s^2})$	F
density	mass/volume	$\frac{M}{L^3}$	$\frac{kg}{m^3}$	ρ
pressure	force/area	$\frac{M}{LT^2}$	$Pa\ (=\frac{N}{m^2})$	P
viscosity	stress/velocity gradient	$\frac{M}{LT}$	$Pa\,s$	μ

Table 1: *The dimensions and units of some of the entities commonly used in cardiology.*

As an example of the reasoning behind the dimensions given in Table 1, consider *viscosity*. Experiments show that the shear stress (the force/unit area) that acts on a surface when a fluid flows over it is proportional to the gradient of velocity away from the surface, $\frac{dU}{dy}$, where U is the velocity parallel to the surface and y is the distance perpendicular to the surface. The constant of proportionality, defined as the *coefficient of viscosity*, depends upon the nature of the fluid (honey is more viscous than water) and other properties such as temperature (cold honey is more viscous than warm honey). Since the velocity gradient has the dimensions of velocity divided by distance, $\frac{M/T}{M} = \frac{1}{T}$, and stress has the dimensions, $\frac{M}{LT^2}$, then the coefficient of viscosity must have the dimensions $\frac{M}{LT^2}/\frac{1}{T} = \frac{M}{LT}$.

A corollary of the rule that all parameters have their own dimensions is that parameters with different dimensions cannot represent the same thing. For example, 'contractility' of a ventricle has been variously described by the ejection fraction, which is the ratio of

two volumes and is therefore dimensionless; V_{max}, the maximum normalised myocardial shortening rate which has the dimensions s^{-1} and end diastolic elastance which has the dimensions of pressure/volume kPa/m^3. Since these three entities have different dimensions they must be different. One needs to know no cardiology at all to say this.

1.2   Newton's second law

The starting point for all mechanics is Isaac Newton (1642-1727) whose second Law states that the rate of change of momentum of a body is proportional to the net force acting upon it [Newton, 1687]. Momentum is defined as mass times velocity, and if the mass of the body is constant then this law can be written in the familiar form

$$F = ma$$

where F is the net force, m is the mass and a is the acceleration. The acceleration is the rate of change of velocity and velocity is the rate of change of position of the body.[2]

Applying dimensional integrity to Newton's second law, we see that the dimension of force must be equal to the dimension of mass times acceleration $[ML/T^2]$. The basic SI unit of force is, appropriately, the Newton (N) which is defined as the force that will cause an acceleration of 1 m/s^2 when applied to a 1 kg mass (i.e., 1 N = 1 kgm/s^2).[3]

Newton's law is relatively straightforward when it is applied to a rigid body like a cannon ball, but problems arise when you try to apply it to a fluid like blood. Euler (1707-1783), whose importance to the development of mathematics and mechanics is comparable to that of Newton, addressed this problem by introducing the concept of a 'control volume'. Using the Eulerian approach to fluid mechanics, a volume of space is identified and the behaviour of the material within the control volume is analysed.

When we consider control volumes, it is convenient to think not in terms of mass and force, but in terms of density (the mass per unit volume) and stress (force per unit area). It is further useful to divide stress into pressure (perpendicular force per unit area) and shear stress (viscous forces acting tangentially per unit area). The SI unit for pressure and shear stress is the Pascal, Pa = 1 N/m^2. The conversion between $mmHg$ and Pa is 1 $mmHg$ = 133.32 Pa. For cardiologists, it is more convenient to think in terms of kPa since 10 $kPa \approx 75.0$ $mmHg$.

Two of the basic principles that can be applied to blood in a control volume are the conservation of mass and momentum.

---

[2]These quantities are formally defined using calculus as the derivatives with respect to time. The fact that Newton 'invented' calculus in order to describe his ideas about mechanics is an example of the intimate connection between mechanics and applied mathematics.

[3]As a rule of thumb, it may be convenient to remember that 1 N is approximately equal to the weight of an apple.

1.3 Blood velocity, acceleration and flow.

Measurements of blood velocity are readily available using Doppler ultrasound methods and peak values are frequently quoted and correlated with other clinical or physiological variables. However, blood velocity is not necessarily the most informative measurement to make if basic mechanisms are being studied. As pointed out in the previous section, it is acceleration rather than velocity that is directly proportional to the net imposed force.

Since it is the magnitude and direction of forces acting on blood that are likely to be of clinical significance, it may be more useful to consider accelerations rather than velocities. The two are simply related: during periods when the acceleration is constant

$$U = at$$

where U is the velocity, a is the constant acceleration and t is the time over which the acceleration has operated. A lower than normal value for peak velocity may be the result of a lower acceleration acting for a normal time, or a normal acceleration (or force) acting for a shorter time. Conversely, acceleration and deceleration times may be measured: again, these depend not only on corresponding acceleration and deceleration rates but also on the value of peak velocity.

Blood velocity is not identical with blood flow;[4] their corresponding physical dimensions are L/T and L^3/T. I.e.

$$\text{Blood flow} = \text{Blood velocity} \times \text{Cross-sectional area of the flow}$$

This is the basis of the Gorlin equations discussed below. It is also the basis of estimates of stroke volume by aortic Doppler, where the cross-sectional area is that of the LV outflow tract. In the PISA (Proximal Isovelocity Surface Area) method, the cross-sectional area is the surface area of the hemisphere whose radius is measured on the 2D display and the velocity is that at which aliasing is assumed to occur.

This relation can be used to estimate the cross-sectional area of jets or orifices

$$\text{Area} = \frac{\text{Blood flow}}{\text{Blood velocity}}$$

It is particularly useful when, for technical reasons, the area cannot be measured directly.

Jet area within the heart or great vessels may also be significant. For example, mean right and left ventricular stroke volumes are identical in the normal heart, whereas peak forward tricuspid velocities are consistently about half of those across the mitral valve. It is therefore clear that right ventricular filling is achieved with a jet area approximately double that of the left.

---

[4]'Flow' is often used in a generic sense, meaning different things. We use it to denote 'volume flow rate', probably its most common usage. However, it is sometimes used as a synonym for 'velocity' or for 'mass flow rate'. Cardiologists sometimes refer to 'blood flow velocity', which normally refers simply to blood velocity. It is a phrase that dates from the early days of ultrasound when they believed that Doppler measured blood flow. The moral is that if you want to be precise, it is important to define what you mean.

1.4   Conservation of mass

In the body, mass is conserved though it can be transmuted into different forms. For example, some of the plasma content of blood can be transformed into interstitial fluid when it is filtered through the endothelium. But the total mass is constant. Applying this to a control volume, we obtain the requirement that the rate of change of the mass contained within the volume is equal to the net mass flux into the volume (the mass flux in minus the mass flux out). Mass flux is defined as the rate of flow of mass through a surface which has dimension of $\frac{M}{T}$. Since blood is very nearly incompressible (i.e. its density is constant), the conservation of mass can be expressed simply

$$\frac{dV}{dt} = Q_{in} - Q_{out}$$

where V is the volume of the control volume (m^3), Q_{in} and Q_{out} are the volume flow rates (m^3/s) in and out of the volume and d/dt denotes the rate of change (the derivative with respect to time). An equation derived from the conservation of mass is frequently referred to as the *continuity equation.*.

A control volume can be defined in any way that we want. It can be defined by the walls of a ventricle or a blood vessel or it can be taken somewhere in the middle of a blood stream if we are interested in the variation of velocities across a blood vessel. A familiar form of a control volume is the placement of the sample volume in Doppler ultrasound as displayed on the screen where the gates define a volume in space in which the average velocity is measured.

A very simple example of a useful control volume might be the volume defined by the left ventricle. Most of the volume can be defined by the endocardium of the ventricle but this does not provide a complete description of the volume because of the valves. When the valves are closed they can be used to define the volume of the ventricle, but when they are open it is necessary to complete the definition of the control volume by defining some surface across the open valve, say a plane at the root of the valve leaflets.

Although the flow in the cardiovascular system is never truly steady during life, there are periods locally when flow behaves as if it was steady, so called *quasi-steady flow*. During these periods, the rate of change of the volume is zero and the conservation of mass simply says that the volume flux [m^3/s] into the control volume must equal the volume flux out. Examples of quasi-steady flows are the small jets that form during systole or diastole due to small anastomoses or valve defects, the mid-systolic period of diastole when the heart rate is slow or the flow in the small vessels.

More importantly, the conservation equation must also be satisfied for average flows. Thus we can say, for example, that the average flow in the pulmonary arteries must equal the average flow in the systemic arteries because, on average, the volume of blood in either ventricle is constant even though there can be transient imbalances, for example as the result of a Valsalva or Mueller manoeuvre. This global application of the conservation of mass can be very informative.

1.5   Conservation of momentum

Strictly speaking, momentum (defined as mass times velocity) is not conserved in the same way as mass. Newton's second law tells us that momentum changes when forces are applied to a body and so the conservation of momentum is really an expression of how momentum varies in response to forces. Thus, the rate of change of the momentum in a control volume is equal to the net rate at which momentum is convected into the volume plus the net force acting on the volume. Written as an equation in words, the momentum equation is

$$
\boxed{\begin{array}{c} \text{rate of change} \\ \text{of momentum} \\ \text{in the volume} \end{array}} = \boxed{\begin{array}{c} \text{net flux of} \\ \text{momentum} \\ \text{into volume} \end{array}} + \boxed{\begin{array}{c} \text{net force} \\ \text{acting on} \\ \text{the volume} \end{array}}
$$

This says that there are two ways for the momentum in the control volume to change: a difference between the amount of momentum flowing into and out of the volume as a result of forces acting on the control volume. Because momentum is the product of mass times velocity, a small mass with a large velocity can have the same momentum as a large mass with a small velocity.

There are different types of forces that can act on a fluid. The most important are: gravity, a 'body' force that acts directly upon every element of the fluid; pressure, a force per unit area that acts perpendicular to the surface of the control volume; and viscous forces, shear stresses arising from the viscous nature of fluids and the local gradients of velocity. Pressure can be thought of as the potential of a fluid to do work and viscosity is a dissipative force that can be thought of as the 'friction' of fluids. The conservation of momentum is the application of Newton's second law to the fluid within a control volume and it tells us that there must be a 'balance' between the rate of change of momentum and the net forces.

When the flow is in steady state, the conservation of momentum says that there is a simple relationship between net force acting on the fluid and the net momentum flowing into the volume.[5] Consider a mug of tea that has just been stirred so that the tea is flowing round in the mug. If we consider a triangular sector of the mug to be the control volume, flow is entering the control volume through one plane of the triangle and leaving through the other. The speed of an element of fluid will remain constant but its direction will have changed. Since velocity is a vector quantity having both magnitude and direction, there is change in velocity. This means that the fluid has accelerated even though its speed has not changed. This acceleration must have been due to a force directed toward the center of the mug. In fact, it is a pressure force which can actually be seen as a slight dip in the surface of the tea going from the edge of the mug to the centre.

This everyday example of flow is a good example of the coexistence of the pressure and velocity in flows. Like the chicken and the egg, the question of which came first depends upon the circumstances. If one applies a net force to a flow it will cause velocity but, equivalently, if one establishes a particular flow it will establish the necessary pressure

---

[5]'Steady state means simply that there is no change in the system with time, not that there is no motion.

distribution. The conservation of momentum means that you cannot have one without the other. In a ventricle during systole, for example, the pressure is primarily established by the force of contraction of the myocardium and the velocities can be thought of as the resultant of the pressure. In a syringe pump, on the other hand, the displacement of the syringe imposes the velocity of the fluid and the pressures that are generated are the resultant.

1.6   The steady Bernoulli equation

This co-existence of pressure and velocity can be made quantitative in steady flows where viscous effects are negligible. Daniel Bernoulli (1700-1782) showed that along a streamline in such a flow

$$P + \tfrac{1}{2}\rho V^2 = P_0 \quad \text{(a constant)}$$

where P is the pressure, ρ is the density of the fluid and V is the velocity. This is the steady Bernoulli equation and it can be thought of as an expression of the conservation of energy in the flow. Since mass times velocity squared is kinetic energy and ρ is mass per unit volume, the second term is easily seen to be kinetic energy per unit volume of the fluid. By the integrity of dimensions, this means that P must also have the dimensions of energy per unit volume. [6] It is therefore natural to think of P as the potential energy per unit volume. The constant P_0 is usually referred to as the total or stagnation pressure, being the pressure that the fluid has when it is brought to rest.

The 'Modified Bernoulli' equation
In echocardiography, regular use is made of the so-called 'Modified Bernoulli equation'

$$\Delta P = 4V^2$$

which is commonly used to estimate the pressure difference between two chambers of the heart or a ventricle and an artery from a measurement of the velocity of a regurgitant or stenotic jet. This equation, without attendant explanation, is a good example of needlessly sloppy mechanics in cardiology. Inspection of the dimensions of this equation suggest that it is not dimensionally correct since pressure has the units $Pa = kg/ms^2$ while the right hand side has the units of velocity squared, m^2/s^2. In fact, the equation is remarkably accurate *if* P is measured in $mmHg$ and velocity in m/s. This is because the density of blood $\rho \approx 1050 \ kg/m^3$ and $1 \ mmHg = 133.3 \ Pa$ and so the $\tfrac{1}{2}\rho$ appearing in the Bernoulli equation can be expressed as $1050/(2 \times 133.3) = 3.938 \approx 4 \ mmHg \ s^2/m^2$. Thus, the equation is valid if we think of '4' as a dimensional constant depending upon the density of blood. While this may be a very convenient equation for use in the clinic, it encourages sloppy thinking mechanically. The exact equation is simple enough and its use, particularly in reporting results, should be encouraged.

---

[6]An alternative way to see this is to multiply the definition of pressure, force/area, by distance in both the numerator and denominator. Since force time distance is work which has the dimensions of energy and area times distance is volume, we get the same result, pressure has the dimensions of energy/volume.

This example exposes another common mechanical mis-usage in cardiology. The difference in pressure between two points in the cardiovascular system is commonly referred to by cardiologists as the 'gradient'. Properly, the gradient is the difference per unit distance and therefore involves the distance between the measurement sites. This incorrect usage of 'gradient' may be too well established for us to suggest that it be replaced by the more proper 'difference'. We would, however, strongly recommend that the enlightened cardiologist should appreciate the proper mechanical meaning of 'gradient' while tolerating the the accepted inaccurate usage in order to communicate with less enlightened colleagues. The importance of being precise should be apparent at several points in the discussion below.

Pressure

Because of its importance in cardiology, we bring together some important but frequently overlooked properties of pressure.

1. Pressure is defined as the force/unit area and therefore has the units of force/area. The SI units of force of force are $Pa \equiv N/m^2$. The most commonly used units in cardiology are $mmHg$. $1\ mmHg = 133.32\ Pa$. For cardiologists, it is probably easiest to remember that $75\ mmHg = 10\ kPa$.

2. In a fluid the pressure acts equally in all directions. The force exerted by a pressure on a surface is directed perpendicular to the plane of the surface.

3. Although pressure can be measured absolutely, *i.e.* relative to an absolute vacuum, it is almost universally measured relative to some 'gauge' pressure. In mechanics, the most common reference pressure is 'atmospheric pressure', the pressure exerted by the atmosphere which, of course, changes with altitude and weather conditions. In cardiology, it is common to use the 'mid-thorax pressure' as a reference pressure. In comparing measured pressures, it is very important that the reference pressure be defined and considered.

4. Properties such as pressure and temperature do not vary with the sample size. They are termed 'intensive' properties. 'Extensive' properties such as mass and volume do vary with the sample size.

5. Pressure is defined mechanically as force/area but its dimensions can also be expressed as energy/volume. Considered in this light, it is natural to think of the pressure as a potential energy per unit volume.

6. Although pressure in a fluid is a scalar that acts uniformly in all directions, a pressure gradient (the change of pressure with distance) is a vector that has both a magnitude and a direction and represents a force in a fluid. The pressure gradient can be different in different directions. It is pressure gradients that are associated with accelerations in a fluid.

1.7 Reynolds number and turbulence

The Reynolds number

It should be remembered that the Bernoulli equation is only valid in flows where the effects of viscosity are negligible; but how do we know if viscous effects are negligible? The answer lies in the Reynolds number (O. Reynolds, 1842-1912)

$$Re = \frac{\rho U D}{\mu}$$

where U is a velocity that is characteristic velocity (such as the mean or peak velocity), D is a characteristic length (typically the diameter of the vessel), ρ is the density and μ is the coefficient of viscosity which as a property of the fluid (the coefficient of viscosity of normal blood is approximately 0.004 $Pa\,s$.) The Reynolds number is a non-dimensional number which represents the ratio of inertial effects to viscous effects. It combines physical properties of the fluid, ρ and μ, and properties of the flow, U and D. $Re \gg 1$ indicate that viscous effects are negligible while $Re \ll 1$ indicates that viscosity dominates inertia.

Examples of Reynolds numbers in different vessels in the cardiovascular system
In all vessels the density of blood is taken as 1050 kg/m^3 and its viscosity is taken as 0.004 Pas. Since velocity is not constant during the cardiac cycle, the Reynolds number depends upon the characteristic velocity chosen. For example, in the aorta two values of Re are calculated, one for the peak velocity and one for the mean velocity. In the other vessels the mean velocity is used.

vessel	diameter	velocity	Re
Aorta (peak velocity)	2 cm	1 m/s	$1050 \times 0.02 \times 1/0.004 = 5250$
Aorta (mean velocity)	2 cm	20 cm/s	$1050 \times 0.02 \times 0.2/0.004 = 1050$
Small artery	1 mm	40 cm/s	$1050 \times 0.001 \times 0.4/0.004 = 105$
Arteriole	0.1 mm	1 cm/s	$1050 \times 0.0001 \times 0.01/0.004 = 0.26$

In the aorta the Reynolds number based on the peak velocity $Re \approx 5250 \gg 1$. This implies that inertial effects are much more important than viscous effects in aortic flow during systole. If the Reynolds number is based on the average velocity in the aorta, $Re \approx 100$ which is still $\gg 1$. In a small artery, $Re \approx 100$ so that inertia still dominates although viscous effects are starting to be important. In an arteriole, on the other hand, $Re < 1$ and so we can conclude that flow in the microcirculation is dominated by viscous effects. The Reynolds number alone can tell us much about the nature of the flow and an estimate of the Re should be made whenever a question about fluid dynamics arises.

Some examples of Reynolds numbers in the heart

Since the density and bulk viscosity of blood are relatively constant ($\rho \approx 1050\ kg/m^3$ and $\mu \approx 0.004 Pa\,s$), the Reynolds number is primarily dependent upon the length scale and the velocity involved. In most cases in cardiology, the relevant length scale is the diameter of the vessel or the jet.

- The Reynolds number of normal flow through the aortic valve ($D \sim 2\ cm$) at peak velocity ($U \sim 1\ m/s$) is $Re = (1050)(1)(0.02)/(0.004) \approx 5200$.

- The Reynolds number in aortic stenosis where the effective diameter of the valve is reduced to 2 mm and the velocity is increased to 4 m/s is $Re = (1050)(4)(0.002)/(0.004) \approx 2100$. Note that even though the velocity through the stenosis is considerably higher than in the normal valve, the Re is less because of the reduced length scale of the valve.

- Flow through a normal mitral valve with diameter $1.5 cm$ during early diastolic filling (the E wave) results in peak velocity of 1 m/s in the young. The associated Reynolds number is $Re = (1050)(1)(0.015)/(0.004) \approx 3900$. In the elderly the mitral valve diameter typically remains the same but the peak velocity of the E wave often falls to 40 cm/s. The Reynolds number therefore becomes $Re = (1050)(0.4)(0.15)/(0.004) \approx 1600$.

  In a severely stenotic mitral valve, the mitral orifice can have an effective diameter of 3 mm producing a peak jet velocity of 5 m/s. The associated Reynolds number is $Re = (1050)(5)(0.003)/(0.004) \approx 3900$.

Notice that all of the Reynolds numbers calculated above are much greater than one. This indicates that peak flows through the valves of the heart are highly inertial and relatively unaffected by viscous effects.

Turbulence

Turbulence in flows is notoriously difficult to define precisely but is something that is part of our general experience.[7] *Laminar* flow is smooth with regular streamlines while *turbulent* flow has local, random fluctuations in the direction and magnitude of the velocity so that the streamlines are irregular and highly unsteady. Many colour Doppler machines will display a property that is called turbulence and it is important to point out that this property is usually derived from the variance in the calculated Doppler shift in the sample volume and is not necessarily the same thing as fluid turbulence. Aliasing of the signal in colour Doppler measurements can also be confused with turbulence. A better indicator of turbulence in echocardiography is the breadth of the Doppler signal. When flow is laminar within the sample volume the line is very narrow indicating near uniformity of blood velocity but when it is turbulent the line is broadened.

The Reynolds number is important in assessing whether flow will be laminar or turbulent. In his experiments on steady flows in tubes Reynolds found that turbulence appears spontaneously when $Re \approx 2000$ and many texts state that this is the transition Reynolds

---

[7]Turbulence is so difficult to define precisely that some fluid dynamicists have borrowed from medical terminology to describe turbulence as a *syndrome* that is defined by its symptoms.

number for all flows. In fact, turbulence can occur at much lower Reynolds numbers when the flow is unsteady or when there is a source of small disturbances in the flow such as an edge or corner. Turbulence has been observed in the aorta during late systole at peak Re (the Reynolds number based upon the peak flow velocity) as low as 1240 [Nerem and Seed, 1972]. On the other hand, it is very unusual to see laminar flow when $Re \gg 2000$. This difference in the stability of accelerating and decelerating flows leads to the familiar 'lawn sprinkler' shape of the Doppler velocity signal when measuring velocity in the aorta; the line is thin during the acceleration phase in early systole and thicker as the flow decelerates in late systole.
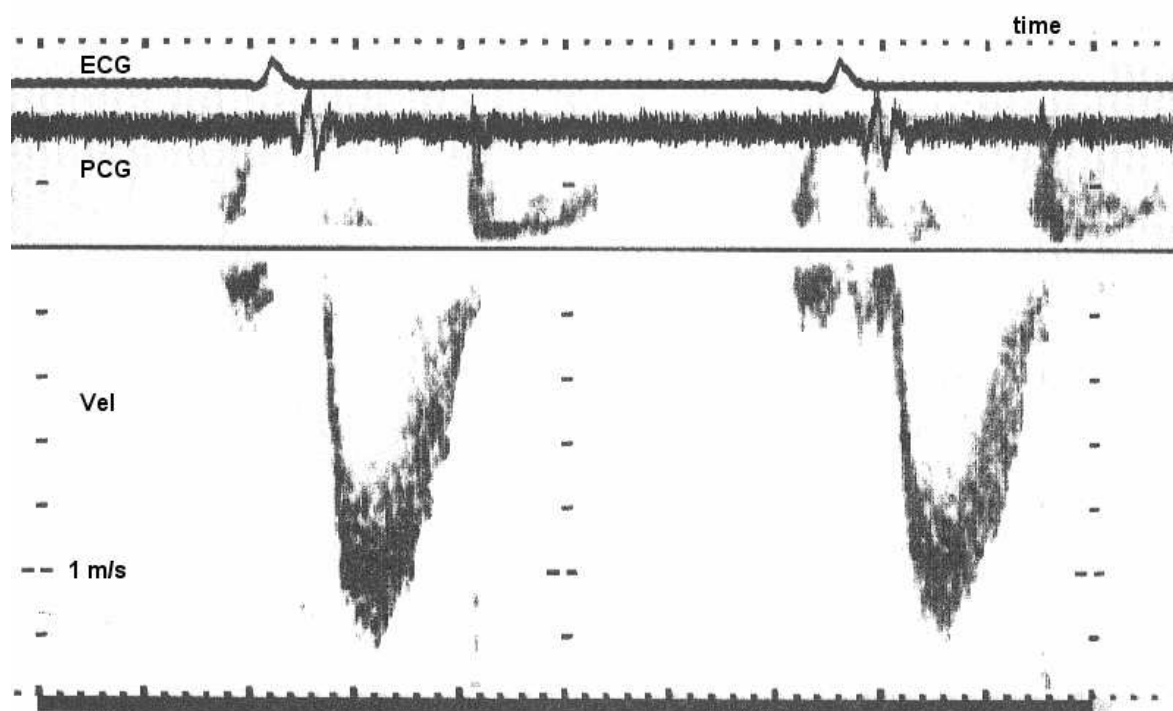

Figure 1: *Velocity in the aorta measured using range-gated Doppler directed into the ascending aorta from the apex of the heart. Time is increasing from left to right and the larger divisions on the scale indicate 200 ms. ECG - electrocardiogram, PCG - Phonocardiogram, Vel - velocity. The thin line during the acceleration phase at the start of systole indicates that the velocity in the sample volume is uniform. The thicker line during the deceleration phase during the last part of systole indicates that there are are elements of fluid in the sample volume moving with different velocities, probably due to turbulence.*

Another feature of turbulent flow is an increased loss of energy due to viscosity. As discussed below, this can be measured by a drop in pressure as blood flows through a stenosis, either a stenotic valve or a stenosed artery. Remembering that pressure has the same units as energy/volume, it is instructive to consider the magnitude of this energy loss as the kinetic energy of the blood is 'lost' through conversion to turbulence and eventually to heat through viscosity. The kinetic energy/volume of blood flowing at $1 \ m/s$ is the dynamic pressure $\frac{1}{2}\rho U^2 = 0.5 \ kPa$. While the velocity is very important to perfusion, its energy corresponds to approximately 1/20th of the normal diastolic pressure and, given the specific heat of blood, it will cause less than 1/1000th of a ºC change in the temperature of the blood.

1.8 The velocity profile

The no-slip condition

Although the Reynolds numbers for most flows in the large arteries and the chambers of the heart are large, viscosity cannot be ignored completely. This is because of the observation that the relative velocity of a fluid always goes to zero at a solid/fluid interface. This is known as the 'no-slip condition' and is an empirical observation of how fluids behave near solid walls rather than something that can be deduced from first principles.

The no-slip condition means that no matter how large the Reynolds number, there will be a region near any wall where viscosity dominates and the velocity goes to zero. If the wall is moving, like the wall of the ventricle, the no-slip condition says that the fluid next to the wall moves with the wall.

Boundary layers

The region near the wall is known as the *boundary layer* which is the region where the flow velocity goes from zero at the wall to the velocity far away from the wall. The thickness of the boundary layer is very difficult to predict in complicated flows like those in the cardiovascular system. Nevertheless, it is a useful concept that helps us to explain the qualitative behaviour of even these complex flows.

One of the consequences of the presence of boundary layers near the wall of the blood vessel is that there is always a velocity profile across the lumen of the vessel. Thus, using the peak velocity in the vessel $\hat{U}$ to calculate the flow, $Q = \hat{U}A$, will always result in an over-estimation.

1.9 The Coanda effect

The Coanda effect is the tendency of a moving column of fluid to follow a surface. [8] This is easily demonstrated in two phase flows such as a stream of water in air. For example, touching the edge of a thin column of water flowing out of a tap with a finger can produce a dramatic diversion of the flow.

The effect can also be observed in jets in a single fluid. For example, the jet formed during mitral prolapse is often directed along the mitral valve rather than axially relative to the plane of the mitral orifice. Once the jet makes contact with the wall of the atrium can often be seen to follow the wall of the atrium for some distance. The mechanical reason for this behaviour is poorly understood but, like separation, it can have a large effect on the bulk flow when it does occur.

[??Insert picture here??]

1.10 Accelerating and decelerating flow.

Most of the acceleration and deceleration in the cardiovascular system is caused by the pulsatile nature of the cardiac cycle. However, even if flow is steady the fluid can be

---

[8]The effect is named after the Romanian aerodynamicist Henri-Marie Coanda (1885-1972) who described it in the 1930's.

experiencing accelerations and it is useful to consider this simpler case first before going on to the much more complicated pulsatile flows.

The simplest example of accelerations in a steady flow arises when the cross-sectional area of the tube varies. If the flow is steady, then the conservation of mass tells us that the mass flow rate through any cross-section of the tube must be equal or else there would be accumulation of fluid somewhere, and the flow could not be steady. For an incompressible fluid such as blood, this means that the product of the average axial velocity and the cross-sectional area of the tube must be constant. Therefore, when the tube narrows the velocity must increase and when it broadens the velocity must decrease. This phenomenon is familiar to us in rivers where the velocity is high in regions where the river is narrow and slow where the river is broad.

By Newton's second law, these accelerations and decelerations must be associated with positive and negative pressure gradients. In fact, if there is no dissipation then we can predict how large a pressure difference there is between parts of the tube with different areas. If we denote the two positions as 1 and 2, then the conservation of mass requires the volume flow rate to be constant. From the conservation of mass

$$U_1 A_1 = U_2 A_2 = Q \quad \text{(constant)}$$

By the Bernoulli equation, the total pressure is also constant if viscous effects are negligible

$$P_1 + \tfrac{1}{2}\rho U_1^2 = P_2 + \tfrac{1}{2}\rho U_2^2 = P_0 \quad \text{(constant)}$$

Written in terms of the volume flow rate Q, the pressure difference is related to the areas

$$P_1 - P_2 = \tfrac{1}{2}\rho Q^2 \left(\frac{1}{A_2^2} - \frac{1}{A_1^2} \right)$$

When the tube is narrowing, the flow is accelerating and the local pressure gradient is positive. This positive pressure gradient acts uniformly on the fluid in the tube accelerating both the higher velocity fluid in the center and the slower moving fluid in the boundary layers near the walls of the tube. This has the effect of making the boundary layers thinner. This increases the wall shear stress but, in general, the flow remains laminar.

When the tube is broadening, the flow is decelerating and the local pressure gradient is negative. Again the deceleration is uniform across the tube but the deceleration of the slower moving fluid in the boundary layers can mean that the flow near the walls can actually change direction, flowing upstream. This phenomenon is called *separation* and can be very important, profoundly changing the qualitative nature of the flow. Separated flows are generally very unstable and can lead to large oscillations and eventually turbulence, even in steady flows.

This behaviour is familiar to the cardiologist both in stenotic valves and in stenoses in arteries. Flow upstream of a stenosis is generally laminar and regular. Downstream, however, the flow is much more complicated and frequently highly turbulent when the stenosis is severe. The ability of flows to separate from the walls in the presence of adverse pressure gradients is probably the largest single factor that makes flow difficult to
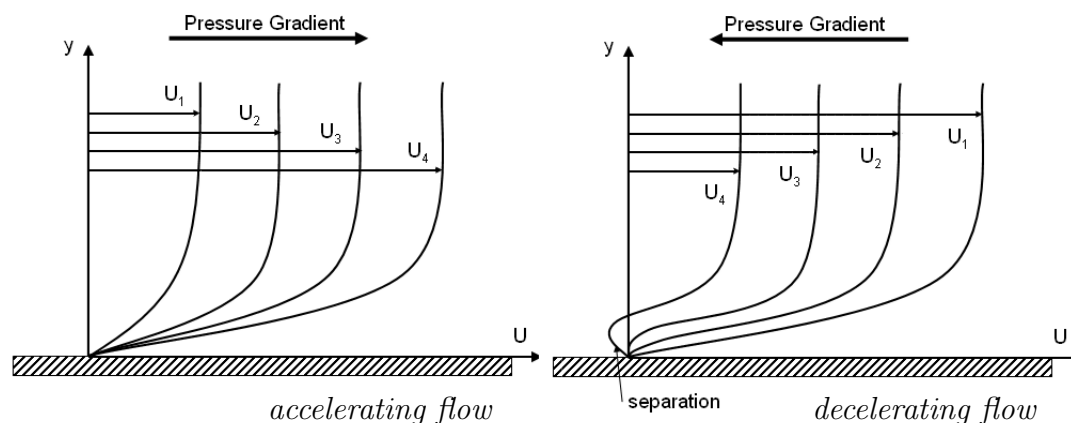
Figure 2: *Behaviour in the boundary layer in accelerating and decelerating flow. In accelerating flow, the boundary layer becomes thinner, while it gets thicker in decelerating flow. If the flow is decelerated sufficiently, the fluid near the wall can reverse direction while the flow far from the wall is still flowing forward. This phenomenon is called separation and it can have a profound effect on the overall flow.*

predict or control in complicated circumstances. Generally as we move downstream from a stenosis, the flow will eventually become laminar again. This process usually involves some recovery of the pressure that is 'lost' when the flow separates and forms a higher velocity jet in the middle of the vessel. This process is termed 'pressure restitution' and its effects can be significant although the magnitude of the pressures involved are relatively small.

Turbulent flow and sounds

Murmurs are generally said to be the result of turbulence arising from flow through stenoses or anastomoses. This is true but it is important to realise that not all turbulent flows in the cardiovascular system produce audible sounds. The sound energy produced by the fluctuating velocities in the fluid itself is very small. It is the interaction of these fluctuations with solid boundaries, such as the wall of a vessel or, more efficiently, a solid discontinuity such as the tip of a valve leaflet, that produce the loudest sounds. Thus, a jet directed against the wall of a ventricle will produce very much louder sounds than an identical jet directed into the centre of a ventricle.

1.11 Steady flow in curved tubes

When there is steady flow in a curved tube such as the aortic arch, there must be a pressure gradient from the outside toward the inside of the bend in order to provide the acceleration of the blood as it goes around the bend. This pressure gradient acts similarly on the high velocity fluid in the centre and the slower moving fluid near the walls of the tube. The acceleration that it produces causes the slower moving fluid in the boundary layers to move away from the outside of the bend towards the inside along the walls of the tube. When these two streams meet, they deflect into the centre of the tube producing a *secondary* flow of two counter-rotating helices superimposed upon the axially directed primary velocity in the tube. Thus, even if blood flow was steady, the curvature of the
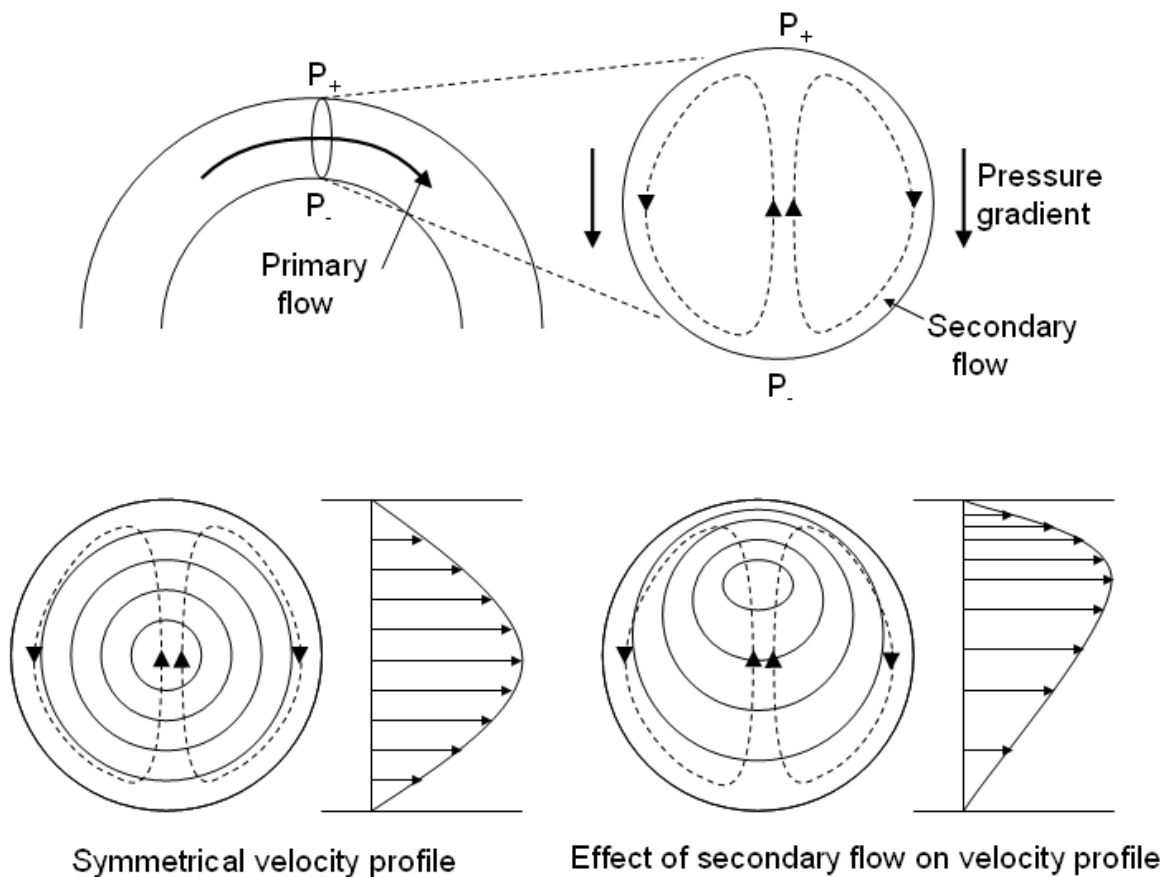
Figure 3: *Secondary flow can occur when the primary flow follows a curved path. The force needed to provide the acceleration due to the curvature induces a pressure gradient from the outside of the bend to the inside, as shown in the top panel. This pressure gradient accelerates the flow in the slower moving boundary layers near the wall of the tube, generating a pair of counter-rotating vortices in the cross-sectional plane of the tube. The secondary flow can perturb the primary flow, convecting the hight velocity fluid nearer to the outside bend of the tube, as shown in the bottom panels.*

arteries as they bend and bifurcate would give rise to very complicated flow patterns. This has been confirmed by CT during continuous flow in cardiopulmonary bypass [ref??].

1.12   Pulsatile flow

Flow in the cardiovascular system is highly unsteady, primarily because of the intermittent nature of the cardiac cycle of contraction although, as we have seen, other factors also conspire to make the flow unsteady. A pressure gradient applied to a fluid will cause the fluid to accelerate. The magnitudes of the pressures and accelerations involved can be most easily illustrated by a simple example. [9]

---

[9] Remember that only pressure gradients and differences and not absolute pressure can be calculated from velocities and accelerations. Although left atrial pressures have been estimated from transmitral flow velocities [ref.??], such estimates can never be rigorous.

Uniform Acceleration

As an example, we will calculate what happens to blood if a pressure gradient of 1 $mmHg/cm$ is applied to it. Consider a volume of fluid contained in a control volume of constant cross-section A and a length $L = 1$ cm. Assume that the pressure at one end of the control volume is 1 $mmHg$ higher than the pressure at the other end (note that it does not matter what the absolute pressure is, only the pressure difference). The net force acting on the control volume is $F = \Delta PA$. The mass of the fluid in the control volume is $M = \rho AL$ where ρ is the density of the fluid. By Newton's second law, the acceleration of the fluid in the control volume is, cancelling the area

$$a = \frac{F}{M} = \frac{\Delta P}{\rho L} = \frac{133.3}{1050 \times .01} = 12.7 \ m/s^2$$

The factor of 133.3 appears because 1 $mmHg = 133.3$ Pa and we have taken the density of blood to be 1050 kg/m^3. This acceleration is larger than the acceleration due to gravity (9.8 m/s^2) and is comparable to the acceleration of arterial blood during the early part of systole when blood accelerates from 0 to approximately 1 m/s in approximately 100 ms.

This result, a pressure gradient of 1 $mmHg/cm$ produces an acceleration slightly greater than the acceleration due to gravity, is a useful number to remember when thinking about cardiovascular fluid dynamics. On the one hand, because the acceleration of blood in the cardiovascular system is normally much less than 1 G, this means that the pressure gradients must be much less than 1 $mmHg/cm$ and so it is tempting to think of the pressures as rather uniform. This is done, usually without thought, when referring, for example, to *the* left ventricular pressure, implying that there is a single pressure within the left ventricle. On the other hand, it is a good reminder of the importance of seemingly small pressure gradients in determining the local blood flow. Returning, for example, to the left ventricle, it means that pressure differences too small to be measured clinically can produce large and clinically significant flows within the ventricle. In fact, the intra-ventricular pressure differences associated with intra-ventricular flows have been measured using very accurate multi-sensor pressure manometers [Spencer & Greiss, 1962].

In the highly unsteady flow typical of the cardiovascular system, the relationship between pressure and flow is generally very difficult. There are, however, a few conditions where the relationship becomes very simple and these provide a useful framework for understanding cardiovascular mechanics. The first simple case occurs at peak velocity. At this time the acceleration is zero and the momentum equation tells us that the pressure gradient must also be zero. This can be seen, for example, in the pressure difference ΔP between the left ventricle and the ascending aorta. During the acceleration phase at the start of systole $\Delta P > 0$, at the instant of peak velocity the pressures cross over so that $\Delta P = 0$, and during the deceleration phase $\Delta P < 0$. Note that there is still a positive flow out of the ventricle during the deceleration phase, but the negative pressure gradient is causing the flow to slow down until it eventually reverses, closing the aortic valve.

Note that acceleration must be accompanied by a decrease in pressure along the direction of flow. Thus, for example, the pressure in the outlet tract of the left ventricle must be greater than pressure in the aortic root during early systole when aortic flow is acceler-

ating. Similarly, when aortic flow is decelerating in late systole prior to the closure of the aortic valve, the left ventricular pressure will be lower than aortic pressure. Only at the time of peak aortic velocity, a time of zero acceleration, will the two pressures be equal. Just as acceleration requires a favourable pressure gradient, deceleration requires an adverse pressure gradient. Hence, for example, $P_{LV} > P_{Ao}$ during the acceleration phase of systole and $P_{LV} < P_{Ao}$ during the deceleration phase, where the subscripts 'LV' and 'Ao' refer to the left ventricle and the aorta.

It is sometimes useful to differentiate between *inertial* flows where the effects of viscosity are negligible and *resistive* flows that are dominated by viscous dissipation. As discussed above, inertial flows are associated with high Reynolds numbers and resistive flows with low Reynolds numbers. In an inertial flow, the acceleration is proportional to the pressure gradient and so there is a 'lag' in the velocity compared to the pressure while the acceleration is converted into velocity. In resistive flows, the velocity is proportional to the the the pressure and so the pulsatile pressure and flow are in phase with each other. Thus, a regurgitant jet through a valve stenosis with an effective diameter of 5 mm producing a velocity of 1 m/s would have a Reynolds number $Re = (1050)(1)(0.005)/(0.004) \approx 1300$ and would therefore be inertial. This would mean that the velocity of the jet would be expected to lag behind the pressure difference across the stenotic valve. On the other hand, a small jet produced by a 1 mm hole in a valve with a velocity of 50 cm/s would have the much smaller Reynolds number $Re = (1050)(0.5)(0.001)/(0.004) \approx 130$ and would be be resistive. The velocity in such a jet would be expected to follow the pressure difference across the valve with little or no lag.

The technological improvements in velocity measurements suggest that the relationship between acceleration and pressure gradients could be used to calculate pressure differences from measurements of blood acceleration. This approach has been applied to the estimation of pressure differences in left ventricle during filling in dilated cardiomyopathy, where mitral flow produces a distinctive jet in the ventricle [Fujimoto, *et al.*, 1995], and in the estimation of aortic pressure from velocity measurements by magnetic resonance imaging [Yang *et al.*, 1996].

1.13 The Windkessel

Stephan Hales (1677-1761) suggested in 1733 that the elasticity of the arteries could act like a cushion to smooth out the intermittent pressure pulses produced by the heart; serving the same function as the air chamber used in fire engine pumps of the time to produce a nearly steady outflow from the nozzle despite the periodic nature of the pumping. Otto Frank (1815-1944) put this idea into quantitative form in 1899 and the *Windkessel* (literally 'air chamber' in German) phenomenon has been the basis of much of our understanding of the pulsatile nature of the arterial system. Briefly, the entire arterial system is treated as a single compartment with a compliance C defined as the change of volume/change of pressure. Similarly, the flow through the entire microcirculation Q is described by a simple resistance R so that $P = RQ$ where P is the arterial pressure. This hydraulic capacitor/resistance system acts to smooth out the pulsatile inflow from the heart, delivering a relatively smooth flow through the capillaries.

During systole, this theory predicts pressure changes that are not very similar to measured pressures. During diastole, however, when there is no flow coming into the arteries, the theory predicts that the pressure should fall off exponentially with a time constant $\tau = RC$. This is very close to the observed pressure behaviour, particularly during late diastole, and so the theory has had a mixed reception over the years. Recent work has suggested that it might be useful to view the pressure in the aorta as the summation of a Windkessel pressure and an excess pressure which is responsible for generating waves in the elastic arteries [Wang *et al.*, 2003].

1.14 Waves in the arteries

Waves in the arteries is an old and venerable subject of scientific study, having been studied by Leonard Euler (1707-1783) in 1775 and Thomas Young (1773-1829) in his Croonian address to the Royal Society in 1809. It is well-known but not generally appreciated that the arterial waves involve simultaneous changes in pressure and velocity; it is impossible to have one without the other. Measured pressure and velocity waveforms are usually very different in shape, the velocity generally reaching its peak before the pressure. However, this is because the measured pressure and velocity at any point in the arteries are the summation of two separate wave, one travelling forward toward the periphery and another travelling backward toward the heart. Recognising this, it is possible to analyse simultaneously measured pressure and velocity data to separate the forward and backward travelling waves [Westerhof *et al.*, 1972; Parker & Jones, 1990].

Perhaps the most important thing to remember about arterial waves is the relationship between the pressure and the velocity expressed in the equation generally known as the *water hammer equation*

$$dP_\pm = \pm\rho c dU_\pm$$

where dP is the change in pressure induced by the wave, dU is the change in velocity, ρ is the density of blood, and c is the local wave speed which depends upon the distensibility (fractional change of area/change of pressure) of the artery. Note that if there were only forward travelling waves in the arteries, the water hammer equation tells us that the pressure and and velocity waveforms would be similar in shape throughout the cardiac cycle. Since they are not similar in shape, the equation tells us that there must be backward travelling waves in the aorta.

2 The Application of Fluid Dynamics to Clinical Cardiology

The basic principles of fluid dynamics discussed in the previous section can help us to understand a number of problems in clinical cardiology. Although the detailed description of cardiovascular flows is still beyond the capabilities and computer power of the fluid dynamicist, the general principles can be applied and can be informative. We have tried to illustrate this with some specific problems, discussing what is known and how a better understanding of fluid mechanics can be useful.

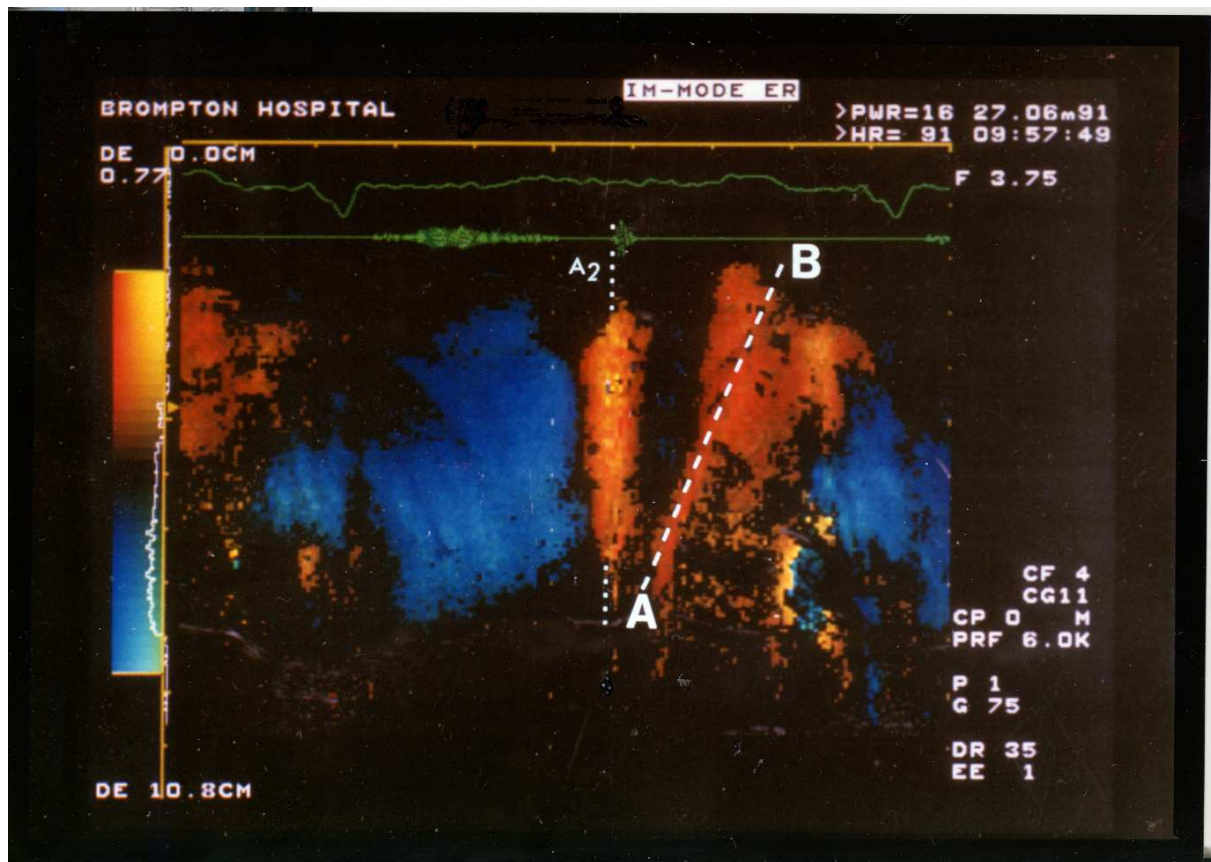## 2.1 The isovolumic contraction and relaxation phases

Figure 4: *Colour M-mode of the short axis of an incoordinate left ventricle. The start of the isovolumic relaxation phase coincides with the closure of the aortic valve indicated by the second heart sound (A2). Very high intraventricular velocities can be seen, approximately 1 m/s, during a time when both valves are closed. These velocities must be accompanied by intraventricular pressure gradients. The high velocities indicated by the line A-B are the flow from the mitral valve towards the apex of the ventricle. The slope of the line indicates the speed with which the jet formed by the mitral flow propagates into the ventricle during the E-wave of ventricular filling. The deviation of the velocity from the line A-B at the start of the P-wave on the ECG indicates the start of the A-wave of ventricular filling. In this patient, the E and A-waves overlapped.*

The instantaneous volume of the ventricular, particularly the right ventricle because of its irregular shape, is notoriously difficult to measure accurately. During the isovolumic phases of the cardiac cycle, there is no flow into or out of the ventricle and so the volume must be constant because blood is effectively incompressible.[10] Constant volume, however, does not mean that there is no flow within the ventricle during these periods. Any change

---

[10]There are a number of experimental papers that claim to have measured volume changes during the isovolumic phases [refs??], but close reading of their experimental methods reveal either that their definition of the ventricular control volume did not include the valve leaflets or that their measure of volume was approximate, e.g. based upon a limited number of intermural distances and an assumption about the shape of the ventricle.

of shape that occurs, such as a shortening of the long axis, must be accompanied by commensurate changes in some other dimension with an attendant flow of blood. Because this flow must be driven by a pressure gradient, there will also be pressure differences within the ventricle.

Although the velocities within the ventricles during the isovolumic phases are usually relatively small compared to the peak velocities seen in the large vessels and through the valve orifices, they are not necessarily negligible. It should be remembered that any distortion of the chambers of the heart must be accompanied by a movement of the fluid within them, as dictated by the conservation of mass.

2.2 Left Atrial Pressure

The instantaneous atrioventricular pressure difference can be estimated with considerable accuracy from blood flow acceleration or deceleration and its variation along the axis of flow.[11] However, knowing mean left atrial pressure is often more desirable clinically. A raised left atrial pressure is the direct cause of pulmonary congestion, and is an important diagnostic criterion of the clinical syndrome of heart failure. In spite of its apparent simplicity, estimation of left atrial pressure from the timing or velocities of blood flow is more complicated than that of the AV pressure gradient. A number of approaches have been used. Left ventricular isovolumic relaxation time shortens when LA pressure is raised, since the hight the atrial pressure the earlier the mitral valve opens. When measured from aortic valve closure to mitral cusp separation, this time is zero when mean LA pressure is approximately 30 mmHg. Such measurements are also likely to be affected by the level of aortic diastolic pressure and also the rate of fall of ventricular pressure. The first of these varies little in disease, but rate of LV pressure fall is commonly reduced in the presence of ventricular asynchorony and varies from patient to patient. The left atrial pressure measured by this approach is likely to be peak V wave which will be higher than the mean value.

The second method in common use depends on the characteristics of early diastolic LV filling. When LA pressure is high, peak E wave velocity is higher than would be predicted for age while deceleration time is short. Some cardiologists believe that further discrimination can be gained by using the ratio E/E', where E' is the peak velocity of early diastolic AV ring retraction. A high peak E wave velocity usually represents a high acceleration rate rather than a prolonged acceleration time. Similarly a short deceleration time is the result of a high deceleration rate. The conditions imply a high early diastolic AV pressure gradient which is rapidly reversed. This is not simply the result of the raised LA pressure, since there is usually a corresponding increase in LV diastolic pressure; indeed, in the absence of mitral valve disease mean LA and LV end-diastolic pressures are effectively identical. Stroke volume is usually low, and not likely to be increased in the absence of significant volume loading. Presumably, therefore, these large and rapid pressure gradient shifts represent low compliance of the artrioventricular chamber. In general compliance falls (i.e. stiffness increases) with distension in all cardiac chambers, and this may form the basis of the correlation with atrial pressure. However the extent to which it does so varies considerably from patient to patient. Furthermore, the LA pressure operative at

---

[11]This and the following section are discussed in more detail in [Gibson and Frances, 2003].

the time the measurements are made will be that during early diastole, i.e. one that is significantly less than the mean.

These estimates of mean LA pressure from early diastolic flow velocities can thus never be more than semiquantitative. They reflect atrial pressures at different times in the cardiac cycle, and depend on other unmeasured variables such as dynamic atrial or ventricular pressure-volume relations and flow propagation velocities. High correlations between mean LA pressure and flow velocities should not therefore be expected (and should be viewed with suspicion when they are reported).

2.3   The filling of the left ventricle

The filling of the ventricles generally takes place in two phases; 'early diastolic' filling during the early part of diastole (the 'E'-wave) and 'atrial systolic' filling at the end of diastole (the 'A'-wave). The origin of the A-wave is obviously the contraction of the atrial myocardium since it occurs immediately after the Q-wave in the ECG. The origin of the E-wave is less clear and there is currently some debate about the mechanics involved. The conservation of momentum requires that the trans-mitral acceleration of blood at the start of the E-wave must be driven by a positive pressure gradient from the atrium to the ventricle. Some researchers believe that blood is 'pushed' into the ventricle by the pressure in the atrium once the ventricular pressure falls below atrial pressure because of the relaxation of the myocardium [Wiggers, 1921; Katz, 1930]. Others believe that the A-wave is the result of diastolic suction generated by elastic strains in the ventricle wall caused by contraction of the myocardium to a volume smaller than its equilibrium volume during the ventricular ejection phase [Bloom, 1955; Brecher, 1956]. In fact, the two schools of thought are similar dynamically and differ only in their interpretation of the mechanics of the myocardium during relaxation; it is exceedingly difficult to ascertain the 'equilibrium' condition of the highly changeable myocardium.

Fluid dynamically, the positive pressure difference between the ventricle and atrium during systole and the isovolumic relaxation phase cannot generate mitral flow because the valve is shut. Once this pressure difference becomes negative at the end of the isovolumic relaxation phase, the valve cannot restrict the forward acceleration of the blood and the E-wave of trans-mitral flow begins. The flow will continue to accelerate as long as the pressure difference between the ventricle and atrium is negative. When the pressure difference is zero, acceleration is zero and the peak velocity is attained. The decreasing velocity during the latter portion of the E-wave is a deceleration and corresponds to a reversal of the ventricular-atrial pressure difference, which can occur because of a decrease in the atrial pressure as blood flow out of it into the ventricle, an increase in the ventricular pressure as it fills or, most likely, a combination of both effects. The fall in atrial pressure can dominate when there is some obstruction of pulmonary venous circulation and the ventricular pressure can rise very quickly with filling when there is restriction or constriction of the ventricle. In the normal heart at rest, there is usually a period of zero mitral flow, and therefore equal ventricular and atrial pressures, before the initiation of the next cardiac cycle causes the contraction of the atrium and the A-wave.

There is another mechanism for ventricular filling associated with atrial contraction that is 'silent' and frequently overlooked. As the myocardium of the atrium contracts it will

pull the mitral valve orifice towards the atrium. By this mechanism, blood originally in the atrium can suddenly find itself in the ventricle without having moved itself. This mode of filling will not be manifest in Doppler ultrasound measurements of mitral velocity and can only be assessed by techniques which can measure the relative movement of the myocardium and the blood.

We close this section with a comment about the use of *logarithmic pressure* in the assessment of the mechanical state of the ventricle. The stiffness of the myocardium, defined as the incremental change in pressure for an incremental change in volume, $\frac{dP}{dV}$, changes dramatically during the cardiac cycle as the myocardium contracts and relaxes, making it a difficult property to assess. However, at the end of diastole the normal heart should be in a state of maximal relaxation and considerable effort has been expended in analysing the end diastolic pressure volume relationship (EDPVR) as a means of characterising diastolic function of the LV [Burkhoff *et al.*, 2005]. It has been found in some studies that the stiffness of the LV at end diastole varies linearly with the distending pressure. If this is true, then the relationship between pressure and volume is exponential so that there is a linear relationship between $log(P)$ and V. This has led to the common use of logarithmic pressure in cardiology, in, for example, logarithmic pressure-volume loops. In doing this, it should be remembered that pressure is almost always measured as a gauge pressure relative to some reference pressure such as atmospheric or 'thoracic' pressure. The logarithm of pressure, particularly at the low pressures characteristic of diastole, is very sensitive to the choice of reference pressure and great care must be taken in the choice and maintenance of it during the measurements.

2.4 Why do pressures measured by echo and catheter sometimes disagree?

It can be extremely vexing for the cardiologist when, as sometimes happens, pressures measured in the heart by two different methods are different. An easy explanation is that one of the measurements is simply wrong. However, it is important to understand that the two methods are made in entirely different ways and that there are cases when both can be 'right' but different.

First, it should be remembered that measured pressures are usually pressure differences, either proper pressure differences ('pressure gradients') between two chambers or gauge pressures relative to some reference pressure. Pressures measured by echo techniques are limited to pressure differences between two sites giving rise to quasi-stationary jets whose velocity can be measured. Because Doppler ultrasound does not measure velocity, but only the component of velocity in the direction of the ultrasound beam, errors can arise through the incorrect measurement of the angle between the jet and the direction of insonation. Another source of error in echo measurements of pressure is the possibility that the jet is not quasi-stationary but is accelerating so that the application of the steady Bernoulli equation is incomplete.

Catheter measurements of pressure differences rely upon simultaneous measurements with two separate transducers, which can lead to problems when the response of the two transducers is not well matched[12], or with sequential measurements using a single transducer,

which can give rise to problems in synchronising the two waveforms. Because the pressure in both chambers is very dynamic, it is not guaranteed that the peak pressure difference will occur at any particular time in the cycle. A second problem with catheter mounted transducers is that they can modify the flow that they are measuring. A particular problem that can occur is to position the transducer so that it actually measures the stagnation pressure of the blood by bringing the blood to rest. Pitot tubes used to measure the velocity of airplanes intentionally make use of this possibility; one part of the tube is oriented into the flow so that air entering it is brought to rest and the pressure measured there is compared to the pressure measured at the side of the tube where the velocity is unimpeded. By Bernoulli's law, the difference between the two pressures is proportional to the square of the velocity of the airplane.

Finally, the most probably reason for two different 'correct' measurements of pressure is that they represent pressures made at different places in the chamber. As we have discussed previously, the intimate relationship between pressure and velocity means that it is dangerous to think that there is a single pressure within a cardiac chamber during most phases of the cardiac cycle. Just as the velocity varies at different places, the pressure will also vary.

2.5 Orifice flow (the Gorlin equation)

The Gorlin equation, which is used to calculate the area of stenosed valves [Gorlin & Gorlin, 1951), is another example of clinical echocardiography practice that could benefit from a more rigorous mechanical approach. The equation given by MedCalc [http://www.media.ch/medcalc/] is

$$\text{Valve Area} = \frac{\text{Cardiac Output}}{(\text{Flow Time} \times \text{Heart Rate}) \times \text{Valve Factor} \times \sqrt{\text{Valve Gradient}}}$$

The valve factor is taken to be 44.5 for aortic valve and 38.0 for the mitral valve when cardiac output is measured in ml/min, flow time in s, heart rate in $beats/min$ and the pressure in $mmHg$.

Fluid dynamicists treat orifice flow by assuming that the orifice introduces some losses into the flow through the generation of separation and turbulence. These losses are described by an orifice coefficient C that relates the flow for a given pressure drop across the orifice to that which would be observed if the flow was inviscid, $Q = C\hat{Q}$. Using the Bernoulli equation for steady flow, the pressure difference is related to the inviscid flow rate, $\hat{Q}$, through the equation

$$\Delta P = \frac{\rho \hat{Q}^2}{2A^2} \left(1 - \frac{A^2}{A_0^2}\right)$$

where P and A are the pressure and area at the orifice, P_0 and A_0 are the pressure and area upstream of the orifice and we have used to conservation of mass to write $U = \frac{Q}{A}$ and $U_0 = \frac{Q}{A_0}$. Experimentally, it is found that the orifice coefficient depends upon the geometry of the orifice and the Reynolds number of the flow. C varies from approximately

---

[12]A very simple test is to reverse the two transducers and repeat the measurement.

0.6 for high Re and sharp orifices to about 1 for smooth, rounded orifices at smaller values of Re. Rewriting this equation to give the area of the orifice and assuming that $A_0 \gg A$ so that the area ratio is negligible,

$$A = \frac{Q}{C\sqrt{\frac{2}{\rho}}\sqrt{\Delta P}}$$

Recognising that the volume flow rate through a heart valve is $\frac{\text{Cardiac Output}}{\text{Flow Time} \times \text{Heart Rate}}$, we see that the Gorlin valve factor is simply equal to $\sqrt{\frac{2}{\rho}}\,C$. Accounting for the units used in the Gorlin equation, the aortic valve factor 44.5 corresponds to $C = 0.88$ and the mitral valve factor 38.0 corresponds to $C = 0.75$. Both of these values are reasonable given the geometry of the valves and the Reynolds numbers involved.

Expressing the valve factors as orifice coefficients does not necessarily increase our ability to predict valve areas from measured flow parameters; it would be a brave engineer indeed who would predict the orifice coefficient for orifices as anatomically complex as stenosed heart valves. However, it does put the empirical relationship into a sounder mechanical context and allows us to draw upon the fluid dynamicists' experience with other flows to predict how the relationship might change with the changes in anatomy and haemodynamics observed in disease.

2.6   Pressure recovery

We have seen that pressure decreases as flow accelerates as it goes into a narrowing of the conduit and increases again as the flow slows down when the conduit broadens. If there are no viscous losses, the Bernoulli equation tells us that the pressure upstream and downstream of a stenosis will be the same, if the vessel area is the same. In real flows there are losses and we find that the pressure downstream of a stenosis is lower than the ideal pressure given by the Bernoulli equation. This process is called *pressure recovery*.

The degree of recovery of the pressure is describe by the pressure recovery coefficient

$$C_P = \frac{P - P_t}{P_{0t} - P_t}$$

where P is the pressure downstream of the stenosis, P_t is the pressure at the narrowest part of the stenosis (the throat) and P_{0t} is the total pressure in the throat ($P_t + \frac{1}{2}\rho U_t^2$). For the ideal flows with no losses, $C_P = 1$. For real flows, the value of C_P depends critically on the degree of separation of the flow due to the stenosis which depends upon the shape of the stenosis and the rate of increase of the cross-sectional area after it. For very irregular stenoses and very rapid area changes, C_P can be as low as 0.25. Because of the importance of this factor in the design of diffusers for pumps and engines, there is a great deal of experimental information about pressure recovery coefficients in steady flow. Much less is known about pressure recovery in unsteady flows such as those found in the cardiovascular system.

Separation of the flow is very closely associated with the pressure recovery. When flow separates it creates regions near the wall where the flow is recirculating, giving rise to

very long residence times in the regions of separation. In steady flow, these separation regions are usually described by the reattachment length, the distance downstream from the orifice where the flow near the tube wall is once again flowing downstream. This reattachment length describes the extent of separation and, to some degree, the effect of the separation on the flow. In unsteady flows, the separation region is dynamic and very difficult to describe. Because of its effect on the local shear stress, on the residence time of the blood behind the stenosis, and the degree of pressure recovery, it could be a very important important variable in separated flows.

[??insert picture here??]

2.7 Augmentation index

The *augmentation index* is defined from the aortic pressure waveform as

$$AIx = \pm\frac{P_s - P_i}{P_s - P_d}$$

where P_s is the systolic pressure, P_d is the diastolic pressure and P_i is the pressure at which an inflection point occurs [??do we need a figure??]. The positive sign is used when the inflection point occurs before the time of the systolic pressure and the negative sign is used when it occurs after.

Several different algorithms have been proposed to find the pressure P_i. If the P_i is defined as the pressure at the inflection point in the pressure waveform $P(t)$, then mathematically it is defined as the zero crossing of the second derivative of $P(t)$. However, the 'shoulder' of the wave is frequently determined manually or by the zero-crossing of the third or fourth derivative of $P(t)$.

The augmentation of the aortic pressure is generally assumed to be the result of the arrival of a backward wave arising from the reflection of the forward wave caused by the initial contraction of the left ventricle that enters the aorta when the aortic valve opens. From the water hammer equation, we know that any backward wave that increases the pressure (a *compression* wave) must simultaneously decrease the velocity (a *deceleration* wave). In studies where both the aortic pressure and velocity have been measured, this behaviour is seen at the time of the shoulder of the pressure waveform, reenforcing this interpretation.

Since most studies of arterial haemodynamics have been carried out using Fourier analysis where the waves are assumed to be superpositions of sinusoidal wavetrains with different frequencies and phases, it is difficult to apply their results to determine a time of arrival of a wave. Studies of arterial waves done in the time domain do allow one to determine the time of arrival of a wave, and they generally show that the shoulder of the pressure waveform does coincide with the arrival of backward waves. However, since the waves in these studies are determined by analysis of the measured pressure and velocity, there is a degree of circularity in these studies that should be taken into account.

3 Exercises

True/false questions:

1. A negative pressure difference found between the apex and the mitral valve in the left ventricle during early filling is evidence that the ventricle exerts a 'suction' force on the blood in the left atrium.

2. 'Pressure' has the same dimension as 'energy/volume'.

3. Left ventricular pressure is always greater than the aortic pressure throughout ejection.

Exercises

1. Calculate the Reynolds number based upon the peak velocity for the aorta assuming its diameter is 2 *cm*, the peak velocity is 1 *m/s* and the viscosity of blood is 0.004 *Pa s*. Calculate the Reynolds number in the same aorta based upon the mean velocity, 0.2 *m/s*. Calculate the Reynolds number based on the mean velocity in the main pulmonary artery of the same individual assuming that its diameter is 2.5 *cm*.

2. If a stroke volume of 80 *ml* is ejected from the left ventricle at a steady rate over a period of 100 *ms*, what is the Reynolds number in the flow through the aortic valve orifice if its diameter is 1.5 *cm*? If the aortic valve is stenosed so that the area is reduced to 1 *cm²*, what is the Reynolds number in the jet passing through the stenosis?

3. Assuming that the flow in the stenosed aortic valve in Exercise 2 is inviscid, what pressure difference do you expect between the left ventricle, cross sectional area 12 *cm²*, and the flow in the jet just downstream of the stenosis?

4. The coefficient of viscosity for a Newtonian fluid μ relates the shear stress τ to the velocity gradient, $\tau = \mu \frac{dU}{dy}$ where U is the velocity and y is distance. The dimensions of shear stress are $Pa(N/m^2)$.
a) What are the dimensions of μ?
b) In steady Hagen-Poiseuille flow the velocity gradient at the wall of the tube is $2U_0/a$ where U_0 is the velocity in the centre of the tube and a is the radius of the tube. If the aorta has a radius of 1 *cm* and the centre-line velocity is 1 *m/s*, calculate the steady wall shear stress.
c) Since aortic flow is pulsatile rather than steady, the assumption of Hagen-Poiseuille flow is not very good. A better assumption is that there is a thin boundary layer between the plug flow in the centre of the vessel and the wall where the velocity must be zero by the no-slip condition. If the centre-line velocity is 1 *m/s* and the boundary layer is 1 *mm* thick, what is the wall shear stress?
d) Compare the wall shear stress to the mean blood pressure.

5. a) What force would be required to accelerate one stroke volume of blood, 80 *ml*, from rest to 1 *m/s* in 100 *ms* if the blood behaves like a rigid body? If the diameter of the aorta is 2 *cm*, what length of aorta is occupied by one stroke volume (sometimes known as the stroke length? If the only force acting on the blood in aorta is the pressure force, PA, where A is the area, what is the pressure difference that corresponds to this force?

b) If flow from the left ventricle to the aorta is inviscid, what is the pressure difference between the left ventricle and the aorta if the velocity in the aorta is 1 m/s and the diameter of the left ventricle is 4 cm?

c) Compare these two estimates of the pressure drop between the left ventricle and the aorta during early systole.

6. Assuming that flow in a patient with aortic stenos is quasi-steady and inviscid during early systole, what pressure and velocity would you expect
a) in the left ventricle if it has a cross-sectional area of 12 cm^2,
b) in the left ventricular outflow tract with a cross-sectional area of 4 cm^2,
c) in the plane of the stenotic valve where the cross-sectional area is 0.5 cm^2 and
d) in the ascending aorta where the cross-sectional area is 4 cm^2.
If the flow is viscous, how would you expect these numbers to change?

7. Derive the relationship between the orifice coefficient used by hydraulic engineers and the valve factors that appear in the Gorlin equation discussed in Section 2.5.

4   References

Bellhouse BJ and Bellhouse FH (1968) Mechanism of closure of the aortic valve. *Nature, Lond.* **217**, 86-87.

Bernoulli D. (1738) *Hydrodynamica.* Paris. (Published as Hydrodynamics, translated from the Latin by Carmody T and Kobus H, Dover Publications, N.Y. 1968.)

Bloom WL (1955) Diastolic filling of the beating excised heart. *Am. J. Physiol.* **187**, 143-144.

Brecher GA (1956) Experimental evidence of ventricular diastolic suction. *Circ. Res.* **4**, 513-518.

Burkhoff D, Mirsky I and Suga H (2005) Assessment of systolic and diastolic ventricular properties via pressure-volume analysis: a guide for clinical, transitional and basic researchers. *Am. J. Physiol. - Heart* **289**, 501-512.

Euler L (1775) Principia pro motu sanguinis per arterias determinando. Opera postuma 2, 1862, p.814-823.

Forbes RJ and Dijksterhuis (1963) *History of Science and Technology, Vols. I & II.* Penguin Books, Harmondsworth, Middlesex.

Frank O (1899) Die Grundform des Arteriellen Pulses. Erste Abhandlung. Mathematische Analyse. *Z. Biol.* **37**: 483-526. (Reprinted in translation: K Sagawa, RK Lie and J. Schaefer (1990) *J. Mol. Cell Cardiol.* **22**: 253-277.)

Fujimoto S, Parker KH, Xiao HB, Inge KSK and Gibson DG (1995). "Early Diastolic Left-Ventricular Inflow Pressures in Normal Subjects and Patients With Dilated Cardiomyopathy - Reconstruction From, Pulsed Doppler-Echocardiography." Brit. Heart J. 74, 419-425.

---

[12]Answers to true/false questions: F, T, F.

Gibson DG and Francis DP (2003) Clinical assessment of left ventricular diastolic function. *Heart* textbf89, 231-238.

Gorlin R and Gorlin SG (1951) Hydraulic formula for calculation of area of the stenotic mitral valve, other cardiac valves, and central circulatory shunts, I. *Am Heart J.* **41**, 1-29.

Hales S (1733) *Statical Essays: containing Haemostaticks.* (Reprinted (1964) No. 22, *History of Medicine Series, Library of New York Academy of Medicine.* New York, Hafner Publishing.

Katz LN (1930) The role played by the ventricular relaxation process in filling the ventricle. *Am. J. Physiol.* **95**, 542-553.

Nerem RM and Seed WA (1972) An in vivo study of aortic flow disturbances. *Cardiovasc. Res* **6**, 1-14.

Newton I (1687) *Philosophiae naturalis principia mathematica.* 1st Edition, London.

Norton JM (2001) Toward consistent definitions for preload and afterload. *Adv. Physiol. Ed.* **25** 53-61.

Spencer MP and Greiss FC (1962) Dynamics of ventricular ejection. *Circ. Res.* **10**, 274-279.

Wang J-J, O'Brien AB, Shrive NG, Parker KH, and Tyberg JH (2003) Time-domain representation of ventricular-arterial coupling as a windkessel and wave system *Am. J. Physiol. Heart Circ. Physiol.* **284**, 1358 - 1368.

Westerhof N, Sipkema P, van den Bos GC and Elzinga G (1972) Forward and backward waves in the arterial system. *Cardiovasc. Res.* **6**, 648-656.

Wiggers CJ (1921) Studies on the consecutive phases of the cardiac cycle: I. The duration of the consecutive phases of the cardiac cycle and the criteria for their precise determination; II. The laws governing the relative durations of ventricular systole and diastole. *Am J. Physiol.* **56**, 415-438; 439-459.

Yang GZ, Kilner PJ, Wood NB, Underwood SR and Firman DN (1996) Computation of flow pressure fields from magnetic resonance velocity mapping. *Magn. Reson. Med.* **36**, 520-526.

Young T (1808) Hydraulic investigations, subservient to an intended Croonian Lecture on the motion of blood. *Phil. Trans. Roy. Soc. (London)* **98**, 164-186.