Physiological Fluid Mechanics

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In this course, I will describe some of the many phenomena in physiology that can be analysed using fluid mechanical techniques.

Most research in this area has focussed on blood flow, and in this course I will focus on this.

However, many of the techniques are quite general, and may be applied to many different systems (physiological or non-physiological).

Due to the short amount of time, I will only be able to give you a brief flavour of the research. If you are interested, I would recommend you read further, as there are several excellent books on the subject, some of which are listed on Page 130.
The cardiovascular system

- The main function of the cardiovascular system is to transport oxygen, carbon dioxide and nutrients between different parts of the body.

- It consists of a highly branched network of vessels and the heart, which acts as a pump.

**Figure:** ‘The Vein Man’ *De humani corporis fabrica* (On the Workings of the Human Body) (1543) by Andreas Vesalius (1514-1564). Working before Harvey’s discovery of the circulation of blood, Vesalius believed that the veins were the most important blood vessels responsible for taking blood from the liver where it was made to the tissues where it was consumed. Most of the vessels in his illustration are actually arteries. Although inaccurate in many details it gives an excellent impression of the complexity of the arterial system.
For a blood particle that starts in the left side of the heart, its journey around the cardiovascular system is as follows:

- **Left side of heart** → **systemic arteries** → **capillaries** → **systemic veins** → **right side of heart** → **pulmonary system (lungs)** → **left side of heart** → . . . .

**Vessels:**
- systemic arteries, containing about 20% of the blood,
- systemic veins, containing about 54% of the blood,
- pulmonary circulation, containing about 14% of the blood,
- capillaries, containing a small fraction of the blood,

and the heart contains about 12% (varies during heart cycle) (Noordergraaf, 1978).

**Figure:** Sketch of the cardiovascular system (Ottesen, Olufsen & Larsen, SIAM Mon. Math. Mod. Comp., 2004).
Arteries carry blood away from the heart. There are three groups:

- **The systemic arteries carry oxygenated blood to the organs of the body.**
  - The aorta is the largest artery, coming directly out of the heart and running down the torso. It has a large arch (the aortic arch) just above the heart (turns through $\sim 180^\circ$) and many bifurcations (points where the parent artery splits to feed two daughter arteries).
  - Other systemic arteries are the coronary, carotid, renal, hepatic, subclavian, brachial, iliac, mesenteric and femoral arteries and the circle of Willis. **Exercise:** Do you know where all these arteries are located?

- **Exercise:** What is special about the pulmonary arteries? The same special thing is true of the umbilical artery, which carries blood from a developing foetus towards the placenta. Why do you think this happens?

**Figure:** Schematic diagram showing the major systemic arteries in the dog, by Caro, Pedley, Schroter & Seed (1978).
Anatomy of the heart

The heart is the pump of the circulatory system, i.e. it is the source of energy that makes the blood flow.

The heart may be thought of as two pumps in series. Blood passes

- ...from the venous system ...
- ... into the atrium\(^a\) (low-pressure chamber), ...
- ... through a non-return valve ...
- ... into the ventricle (high-pressure chamber), ...
- ... and through another non-return valve ...
- ... into the arterial system.

\(^a\)In these notes, I have tried to highlight in colour important technical terms that you should be familiar with. Green highlighting is used to emphasise terms that are defined elsewhere in these notes, while red highlighting emphasises terms as they are being defined.

Figure: Diagram of heart, showing the major structures, by Ottesen et al., 2004).
Anatomy of the heart

The cardiac muscle structure

Figure: Arrangement of the muscle fibres in wall of the left ventricle.

Figure: Muscle fibre orientation in wall of the left ventricle (from Caro et al., 1978).

- The walls of the heart are composed of **myocardial tissue**.
- Myocardial tissue is made up of fibres that can withstand tension in the axial direction (along their length).
- The fibres are arranged in layers. The orientation rotates gradually as the layers are traversed.
- This arrangement makes the wall very strong in every direction.
Other possible arrangements of the cardiovascular system

Figure: Sketch illustrating different types of heart. The top row shows a linear heart (e.g. a snail heart), and the bottom row shows a looped heart, which is the type mammals have, by Kilner et al., Nature (2000).

Question: Do you think humans have a better arrangement?
The wall has three layers:

- **Tunica intima.** Innermost layer, a few microns thick. Composed of endothelial cells and their basal lamina. The endothelial cells act as a barrier between the blood and the wall.

- **Tunica media.** Middle layer, separated from the intima by the internal elastic lamina. Composed of smooth muscle cells, elastin, collagen and proteoglycans and determines the elastic properties of the wall.

- **Tunica adventitia.** Outer layer, separated from the media by the outer elastic lamina. A loose connective tissue containing collagen, nerves, fibroblasts and elastic fibres. In large arteries this also contains the *vasa vasorum* – a network of vessels providing nutrition to the outer regions of the artery wall.

**Figure:** Histological section of an arterial wall from Ethier & Simmons (2007).
The capillaries

- Capillary walls are also made of endothelial cells.
- The endothelial cells have gaps between them (unlike in the arteries), so that plasma can leak through, while the red blood cells remain in the arteries.
- Oxygen and nutrients are **convected** and **diffused** from the capillaries into the target tissues.
- In the tissues, oxygen is converted to carbon dioxide.
- This releases energy, which is used by the cells to perform their functions.
The capillaries

**Figure:** Pictures of the capillaries, by Gaudio et al., J. Anat. (1993).
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We study the bifurcation (splitting) of an artery shown in the figure. The cross-sectional areas of the vessels \( A_\star \), blood velocities \( u_\star \) and blood pressures \( p_\star \) are given and the density of the blood (mass per unit volume) is given by \( \rho = 1000 \text{ kg/m}^3 \). We will find:

- The velocities \( u_1 \) and \( u_2 \) of blood in the daughter vessels.
- The tethering force that holds the section of artery in place (provided by the surrounding tissue and neighbouring parts of the arterial wall).
The bifurcation is **symmetric**, so the flows in the two daughter vessels are identical ($u_1 = u_2$).

The vessels are **rigid**, that is the walls do not deform.

The blood is:
- **incompressible**, that is the density $\rho$ of the blood (mass per unit volume) is constant, and
- **inviscid**, that is there are no **viscous** forces.
- **steady**, that is, it does not change with time.

The flow is:
- **uniform**, that is the velocities $u_0$, $u_1$ and $u_2$ at the inlets and outlets are constant (rather than functions of the position), and
- **axial**, that is the direction of the velocity is along the tube and perpendicular to the surfaces.

These assumptions simplify the problem enormously, but they are only valid in some cases. Even so, there are cases in which the following analysis yields an answer close to reality.
To analyse the flow we will use two important general principles:

- **Mass conservation:** The total amount of mass in the system remains fixed (mass cannot be created or destroyed).

- **Momentum conservation:** The total amount of momentum in the system changes as a result of forces acting upon it. Newton’s second law tells us that the rate of change of momentum equals the force. If no force is acting then the momentum stays constant.

These principles are used in some form or another for most problems in fluid mechanics.

We can apply these conservation laws to a **control volume** – a particular region of a fluid. The forces acting on the fluid can be classified into:

- **Surface forces:** Pressure and stress forces that act at the surface of the fluid. (Stress forces arise in viscous fluids due to interaction of the fluid with the boundary.)

- **Body forces:** Forces that act over the interior of the fluid, for example we often consider gravity and viscous forces.
**Method to apply mass conservation**

- Choose a **control volume**. In this case, we choose the control volume to be all the blood in the region shown in the diagram.

- Find the **mass flux** into the control volume (this has dimensions of mass per unit time). In this case it is the flux $\dot{m}_0$ into the **parent** artery.

- Find the **mass flux** out of the control volume. In this case it is the sum of the two (identical) fluxes $\dot{m}_1$ and $\dot{m}_1$ out of the daughter arteries.

- The flux in must equal the flux out (this is mass conservation). In this case $\dot{m}_0 = 2\dot{m}_1$, which we can use to find the velocity $u_1$ in the daughter arteries.

**Note:** in this case the mass inside the control volume is constant because:

- The volume of the control volume is constant, because the arteries are **rigid**.

- The blood is **incompressible**, meaning its density (mass per unit volume) is constant (at any point and at any time).

In some problems the mass in the control volume changes in time. We account for this using the rule:

\[
(Mass \text{ flux in}) = (Mass \text{ flux out}) + (Net \text{ rate of increase of mass in control volume}). \quad (1)
\]

Equation (1) is true in all situations.
Application of mass conservation

To find the mass flux into the parent artery:

- Every unit of time, a length $u_0$ of blood flows into the artery.
- Therefore, every unit of time, a volume $A_0u_0$ of blood flows into the artery (this is the volume flux $Q_0$ – dimensions volume per unit time).
- Therefore, every unit of time, a mass $\rho A_0u_0$ of blood flows into the artery. This is the mass flux $\dot{m}_0$ through the parent artery (dimensions mass divided by time).

Similarly the mass flux out of each of the daughter arteries is $\dot{m}_1 = \rho A_1u_1$. Mass conservation implies:

$$\dot{m}_0 = 2\dot{m}_1 \Rightarrow \rho A_0u_0 = 2\rho A_1u_1 \Rightarrow u_1 = \frac{A_0}{2A_1}u_0 = 0.75 \text{ m/s.} \quad (2)$$

**Note:** the formula $\dot{m} = \rho Au$ gives the mass flux of a fluid across any surface, provided that:

- The fluid is incompressible.
- The flow is uniform and perpendicular to the surface.
Method to apply momentum conservation

- Find the **momentum flux** into the control volume (this is a vector quantity and has dimensions $MLT^{-2}$, momentum per unit time, the same dimensions as force). In this case it is the flux $\dot{M}_0$ into the parent artery.

- Find the **momentum flux** out of the control volume. In this case it is the sum of the two fluxes $\dot{M}_1$ and $\dot{M}_2$ out of the daughter arteries (note that $\dot{M}_1 \neq \dot{M}_2$ since $\dot{M}_1$ and $\dot{M}_2$ point in different directions).

- Find the resultant force acting on the fluid in the control volume. In this case the force comes from **pressure** forces acting on the sides and ends of the vessels (we are neglecting gravitational, viscous and stress forces).
Conservation of momentum implies:

\[
(Momentum \, flux \, out) - (Momentum \, flux \, in) = (Forces \, acting \, on \, fluid \, in \, control \, volume),
\]

\( (3) \)

**Note:** The formula (3) is only true for **steady** flows, that is flows that do not depend on time. If the flow is time-dependent we must account for the rate of change of momentum in the control volume too:

\[
(Momentum \, flux \, out) - (Momentum \, flux \, in) + (Rate \, of \, increase \, of \, momentum \, within \, control \, volume) = (Forces \, acting \, on \, fluid \, in \, control \, volume).
\]

\( (4) \)
Application of momentum conservation

In the case of our bifurcation, the forces acting on the blood in the control volume are the pressure forces on the ends of the blood in the control volume (which we can calculate) and the pressure forces from the walls of the arteries on the blood in the control volume (which is not easy to find, so we eliminate it). Equation (3) gives

\[ \dot{M}_1 + \dot{M}_2 - \dot{M}_0 = (\text{Pressure force on ends of blood in CV}) \]
\[ + (\text{Pressures force from walls on blood}). \] \hspace{1cm} (5)

The force exerted by the blood on the walls is equal and opposite (Newton’s third law):

\[ (\text{Pressure force of blood on walls}) = -(\text{Force from walls on blood}) \] \hspace{1cm} (6)

And since the walls are in equilibrium the resultant force acting on the walls must zero (the rest of the force is supplied by the tethering force:

\[ (\text{Resultant force on walls}) = (\text{Pressure force of blood on walls}) + (\text{Tethering force}) = 0. \] \hspace{1cm} (7)

Therefore

\[ (\text{Tethering force}) = -(\text{Pressure force of blood on walls}) \]
\[ = (\text{Pressure force from walls on blood}) \]
\[ = \dot{M}_1 + \dot{M}_2 - \dot{M}_0 - (\text{Pressure force on ends of blood in CV}). \] \hspace{1cm} (8)
Application of momentum conservation (ctd)

- We now use Equation (8) to calculate the tethering force.
- We need to find the momentum fluxes $\dot{M}_0$, $\dot{M}_1$, $\dot{M}_2$, and the pressure forces on the ends of the parent and daughter vessels.
- First we find $\dot{M}_0$. As noted before, every unit of time a volume $A_0 u_0$ of blood enters the parent vessel. This blood has momentum $\rho u_0$ per unit volume. Therefore the momentum flux has magnitude $(A_0 u_0)(\rho u_0) = \rho A_0 u_0^2$, and it points in the same direction as the velocity vector $u_0$ (note that, by definition, $u_0$ is the magnitude of $u_0$). Hence
  \[ \dot{M}_0 = \rho A_0 u_0 u_0 = \rho A_0 u_0^2 i \]  
  (where $i$ is the unit vector in the axial direction).
- **Note:** the formula $\dot{M} = \rho A u u$ is generally true as long as the blood is incompressible and the flow is uniform and perpendicular to the surface.
- Similarly
  \[ \dot{M}_1 = \rho A_1 u_1^2 (i \cos \theta + j \sin \theta), \quad \dot{M}_2 = \rho A_1 u_1^2 (i \cos \theta - j \sin \theta) \]  
  (where $j$ is the unit vector perpendicular to $i$ in the plane of the bifurcation), and therefore
  \[ \dot{M}_1 + \dot{M}_2 - \dot{M}_0 = \rho (2A_1 u_1^2 \cos \theta - A_0 u_0^2) i \]  
  (11)
Application of momentum conservation (ctd)

- The pressure forces are given by the formula \((\text{Force}) = (\text{Pressure}) \times (\text{Area})\) and act in the direction normal to the surface. Therefore the forces are

\[
p_0 A_0 \mathbf{i}, \quad -p_1 A_1 (\mathbf{i} \cos \theta + \mathbf{j} \sin \theta), \quad -p_1 A_1 (\mathbf{i} \cos \theta - \mathbf{j} \sin \theta),
\]

on the ends of the parent and two daughter vessels respectively. The resultant pressure force is the sum of these three:

\[
(\text{Pressure force on ends of blood in CV}) = (p_0 A_0 - 2p_1 A_1 \cos \theta) \mathbf{i}
\]  

(13)

- Substituting in Equation (8) we obtain

\[
(\text{Tethering force}) = \left( -(p_0 A_0 - 2p_1 A_1 \cos \theta) + \rho \left(2A_1 u_1^2 \cos \theta - A_0 u_0^2 \right) \right) \mathbf{i} = -0.89375 \mathbf{i} \text{ N.}
\]  

(14)
Discussion of the model of a bifurcation

We have shown that the velocity in the daughter vessels is 0.75 m/s and the tethering force is about 0.89 N in the direction opposing the flow. The tethering force arises because of the change in total momentum at the bifurcation.

- There is no dependence upon the lengths of the parent and daughter vessels. This is because, away from the bifurcation, the vessels are symmetrical, so the pressure forces cancel our around the cross section.

- If viscosity were included, the tethering force would depend on the lengths because the walls would exert stress forces due to the interaction with the fluid all along their length. The stress forces all act in the same direction (in the direction opposing the flow) and do not cancel out (unlike the pressure forces). Using a viscous fluid model of the blood would also mean that the flow develops a profile (the velocity is no longer uniform over the cross section). We will investigate this further in the next section.

- If gravity is included it will also change the tethering force. In this case the weight of the fluid and the weight of the walls should be added on to the force.
Exercise

Consider the curved segment of an artery shown in the figure above. You are given:

- The segment of the vessel turns through an angle $\theta$, that is the angle between the normal vectors at the inlet surface and the outlet surface.
- **At the inlet:** pressure $p_1$, areas of the vessel $A_1$, blood velocities $u_1$ (assumed uniform), blood density $\rho_1$
- **At the outlet:** pressure $p_2$, area $A_2$, density $\rho_2$

Find:

- The outlet velocity $u_2$.
- The **tethering force** that holds the section of artery in place.

You may make simplifying assumptions similar to those made in the bifurcation problem.
The Reynolds Transport Theorem is a generalisation of the principles of mass and momentum conservation.

A **conserved quantity** in the system is one whose rate of change we can quantify in terms of other physical effects. For example:

<table>
<thead>
<tr>
<th>Quantity</th>
<th>Reason for changes in the total amount of the quantity in the system</th>
<th>Why is it conserved?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mass</td>
<td>Does not change</td>
<td>We can quantify zero(!)</td>
</tr>
<tr>
<td><em>Linear</em></td>
<td><strong>Forces</strong> acting on the system</td>
<td>We can analyse the forces and therefore quantify them</td>
</tr>
<tr>
<td><em>Angular</em></td>
<td><strong>Torques</strong> acting on the system</td>
<td>We can analyse the torques</td>
</tr>
<tr>
<td>Energy</td>
<td><strong>Heat</strong> being supplied</td>
<td>We can quantify the sources of heat</td>
</tr>
<tr>
<td>Charge</td>
<td><strong>Electrons</strong> being added or removed</td>
<td>We can quantify the sources of electrons</td>
</tr>
</tbody>
</table>

The theorem is a conservation law that can, in principle, be applied to any conserved quantity.
The Reynolds Transport Theorem states that, for a conserved quantity $B$ in a control volume of the fluid,

$$
(Rate \ of \ change \ of \ B \ in \ system) = (Rate \ of \ change \ of \ B \ in \ control \ volume) + (Flux \ of \ B \ through \ control \ surface). \quad (15)
$$

Since, by definition of a conserved quantity, its rate of change can be quantified, we can find the left-hand side of the above equation in terms of other effects.

If the problem is **steady**, then the rate of change of $B$ in the control volume is zero.\(^1\)

\(^1\text{You might be forgiven for thinking that, since all rates of change ‘are zero’ in steady systems, the rate of change of B in the system should be zero in a steady system too. However, this is not true and it is to do with a difference between the Eulerian and Lagrangian viewpoints. The system is steady if all rates of change are zero at fixed points in space (the Eulerian viewpoint). On the other hand, the ‘system’ in Equation (15) is attached to the particles (the Lagrangian viewpoint). Therefore in a steady system, an individual particle may change its properties as it passes through different spatial locations (for example it may change its speed). For example, the bifurcation problem is steady, but particles change speed as they go from the parent to the daughter vessels.}
The flux through the control surface (the boundary of the control volume) is found in general by considering an infinitesimal area of the surface and finding the amount of $B$ that flows through it. Summing over the surface gives the total flux through the surface.

In the cases we considered so far, the calculation was simpler because the control volume had fixed inlets and outlets and the fluid flowed uniformly and perpendicularly across them (for example on Page 19 we obtained the formula $\dot{m} = \rho Au$).

To derive a general formula, we define $\beta$, the amount of $B$ per unit mass or intensive amount of $B$:

<table>
<thead>
<tr>
<th>Conserved quantity, $B$</th>
<th>Usual formula</th>
<th>$\beta$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mass</td>
<td>Mass</td>
<td>( \frac{\text{(Mass)}}{m} = 1 )</td>
</tr>
<tr>
<td>Linear momentum</td>
<td>Mass times velocity</td>
<td>( \frac{\text{(Momentum)}}{m} = u )</td>
</tr>
<tr>
<td>Angular momentum</td>
<td>Perp dist times momentum</td>
<td>( \frac{\text{(Ang momentum)}}{m} = r \times u )</td>
</tr>
</tbody>
</table>

The amount of $B$ per unit volume is $\rho \beta$. 
To find the infinitesimal volume, consider a small rectangle of area $dA$ on the surface. During a short time $dt$, a parallelepiped of fluid flows through the surface. The volume is the area of the base multiplied by the perpendicular height. In this case the base has area $dA$ and the other edges are given by the vector $u \, dt$. The volume is therefore $(u \cdot n) dA \, dt$, where $n$ is the unit normal vector to the surface.

Hence the amount of $B$ crossing the infinitesimal surface in the infinitesimal time is $\rho \beta (u \cdot n) dA \, dt$ and the rate of crossing is $\rho \beta (u \cdot n) dA$.

Therefore

$$(\text{Flux of } B \text{ through control surface}) = \int_{\text{Control surface}} \rho \beta (u \cdot n) \, dA$$  \hspace{1cm} (16)
In the previous example we assumed uniform flow. This neglects the effects of the viscosity of the fluid. Viscosity is a kind of internal friction in the fluid. It also enforces a no-slip boundary condition on rigid surface, meaning that the velocity of the fluid at the wall equals the velocity of the wall. For the pipe, this enforces the flow to be zero at the walls. Hence the velocity is slow around the edges and rises to a maximum in the centre of the pipe. Therefore the flow has a profile in space (the velocity $u$ is a function of position).

The resulting flow looks approximately like that shown in the diagram below.

In this section we will calculate an equation for the velocity of the blood as a function of the position in the artery.
In order to investigate the flow profile, we develop a simplified model. Finding the profile enables us to estimate useful quantities and make predictions. We make the following simplifying assumptions:

- **Geometry:** The artery is a circular cylindrical tube and the walls are rigid
- **Fluid:** The blood is an *incompressible* Newtonian fluid
- **Flow:**
  - The flow is *steady* (does not depend on time)
  - The flow is in the *axial* direction (parallel to the tube) and is *axisymmetric* (looks the same after rotating the tube)
  - The flow is *fully-developed* (it looks the same on every cross-section of the tube)

To find an equation for the velocity profile we need some governing equations.
The differential equations of fluid mechanics

We analyse the flow using the differential equations of fluid mechanics, which express respectively mass and momentum conservation in a fluid.

**The Continuity Equation**

- This equation expresses mass conservation in a fluid.
- It may be derived by applying the Reynolds Transport Theorem for mass conservation (1) to a small cube with side length $\delta x$ and letting $\delta x \rightarrow 0$.
- The continuity equation is

  $$\frac{\partial \rho}{\partial t} + \nabla \cdot (\rho \mathbf{u}) = 0,$$

  \hspace{1cm} (17)

  where $\rho(x, t)$ is the density and $\mathbf{u}(x, t)$ is velocity.
- In the case of incompressible fluids, $\rho$ is constant, meaning that the continuity equation (17) becomes

  $$\nabla \cdot \mathbf{u} = 0.$$  \hspace{1cm} (18)

- Hereinafter we assume that the fluid is incompressible.
Conservation of momentum

- By applying the Reynolds Transport Theorem for momentum conservation (4) to a cubic box of side length $\delta x$ and letting $\delta x \to 0$, we obtain

$$\rho \left( \frac{\partial u}{\partial t} + (u \cdot \nabla) u \right) = F,$$

(19)

where $F(x, t)$ is the force per unit volume acting on the fluid.

- The expression $\partial u / \partial t + (u \cdot \nabla)u$ is the acceleration of a particle moving with the fluid. ‘Moving with the fluid’ expresses the fact that we are using the Lagrangian frame rather than the Eulerian frame of reference.

- Therefore the left-hand side of (19) represents the rate of change of fluid momentum per unit volume in the Lagrangian frame of reference while the right-hand side represents the force per unit volume acting on the fluid. Thus (19) is just Newton’s second law expressed per unit volume.
Stress–strain relationships

To find an expression for the force per unit volume, $F$, appearing in (19), we need to find the relationship between the viscous forces acting on the fluid and the velocity field of the fluid. This is usually done by relating the stress and strain tensors of the fluid to give a constitutive equation for the fluid behaviour.

- The **stress tensor** $\sigma$ is a $3 \times 3$ matrix in 3 dimensions. If an imaginary cut were to be made in the fluid on a plane with unit normal $n$, the force per unit area on this plane would be $\sigma n$. The body force per unit volume in the fluid is

$$F = \nabla \cdot \sigma = \left( \frac{\partial \sigma_{ji}}{\partial x_j} \right)$$

which is short for

$$\sum_{i=1}^{3} \sum_{j=1}^{3} \frac{\partial \sigma_{ji}}{\partial x_j} e_i.$$  \hspace{1cm} (20)

- The **strain tensor** $\epsilon$ is also a $3 \times 3$ matrix in 3 dimensions, defined by

$$\epsilon_{ij} = \frac{1}{2} \left( \frac{\partial u_i}{\partial x_j} + \frac{\partial u_j}{\partial x_i} \right)$$

in Cartesian coordinates $x_i$, where $u = (u_{ij})$ is the fluid velocity.
Types of fluids

We categorise fluids according to the nature of the relationship between the stress tensor and the strain tensor:

- Newtonian fluids obey a stress–strain relationship of the form
  \[ \sigma = 2\mu \varepsilon - pI, \]
  \[ \text{(22)} \]
  where \( p \) is the pressure and \( \mu \) is the viscosity (which is constant),

- Inviscid fluids obey
  \[ \sigma = -pI, \]
  \[ \text{(23)} \]

- while other fluids have a more complicated relationship between the stress and strain tensors, which may depend on the history of the fluid motion, as well as the current state.
Navier–Stokes equation

We substitute (20) and (22) into (19). For incompressible fluids, we have $\nabla \cdot \mathbf{u} = 0$ (18), so the viscous force term simplifies, and we obtain the Navier–Stokes equation

$$
\rho \left( \frac{\partial \mathbf{u}}{\partial t} + (\mathbf{u} \cdot \nabla) \mathbf{u} \right) = -\nabla p + \mu \nabla^2 \mathbf{u}.
$$

(24)

The term $-\nabla p$ is the pressure force per unit volume and $\mu \nabla^2 \mathbf{u}$ is the force per unit volume due to the viscous stresses.

**Exercises: derive the continuity and Navier–Stokes equation**

- Derive Equation (24) from (20), (22) and (19).
- Derive the left-hand sides of the continuity and Navier–Stokes equations from first principles by applying mass and momentum conservation to a cubic box of side $\delta x$. This might be quite time-consuming, but it will give you insight and practice in manipulating tensors.
Model to find flow profile in an artery (ctd)

We return to the model of a artery. We have equations (18) and (24) expressing the mass and momentum balance for an incompressible Newtonian fluid, so we return to the model of an artery that we formulated earlier. We want to find the velocity field \( \mathbf{u}(x, t) \) and the pressure \( p(x, t) \).

- We work in cylindrical polar coordinates \((r, \theta, z)\), as shown in the figure.
- We write the components of \( \mathbf{u} \) as \( u_r, u_\theta, u_z \), and we may write each of \( u_r, u_\theta, u_z \) and \( p \) as functions of \( r, \theta, z \) and \( t \).
- We use the assumptions we made earlier to simplify the equations:
  - Steady flow implies that the components of \( \mathbf{u} \) and \( p \) do not depend on \( t \).
  - Flow in the axial direction implies that the \( u_r \) and \( u_\theta \) are both zero (we are left with \( u_z \) and \( p \)).
  - Axisymmetric flow implies that \( u_z \) and \( p \) do not depend on \( \theta \).
  - Fully-developed flow implies that \( u_z \) does not depend on \( z \).

We are left to find the functions \( u_z(r) \) and \( p(r, z) \).
Model to find flow profile in an artery (ctd)

- The continuity equation and azimuthal component of the Navier–Stokes equation are automatically satisfied. The radial and axial components of the Navier–Stokes equation respectively become

\[
0 = -\frac{\partial p}{\partial r}, \quad 0 = -\frac{\partial p}{\partial z} + \frac{\mu}{r} \frac{\partial}{\partial r} \left( r \frac{\partial u_z}{\partial r} \right). 
\]  

(25)

- Equation (25a) implies that \( p \) is a function of \( z \) only, while differentiating (25b) with respect to \( z \) gives

\[
\frac{\partial^2 p}{\partial z^2} = 0
\]

(26)

and solving this subject to \( p = p_1 \) at \( z = 0 \) and \( p = p_2 \) at \( z = L \) gives

\[
p = p_1 - \frac{(p_1 - p_2)}{L} z.
\]

(27)

Hence the pressure field drops linearly along the pipe, and it is constant across the cross-section. Substituting this expression for \( p \) into Equation (25b), we obtain the general solution

\[
u_z = C_1 + C_2 \ln r - \frac{(p_1 - p_2)}{4 \mu L} r^2
\]

(28)

Since \( \ln r \) is unboundedly large near \( r = 0 \), we must have \( C_2 = 0 \). The no-slip boundary condition implies that \( u = 0 \) at the boundary \( r = \sqrt{A/\pi} \), and hence we obtain the Poiseuille profile:

\[
u_z = \frac{(p_1 - p_2)}{4 \pi \mu L} (A - \pi r^2).
\]

(29)
Poiseuille flow

Generalisation of formula to calculate mass flux

On Page 19 we showed the flux of mass through one of the arteries is given by $\dot{m} = \rho Au$. However, in order to derive this we assumed the flow is incompressible, uniform and perpendicular to the cross-section.

The current arterial model does not have uniform flow, so we must adapt the formula. In this case the length of fluid per unit time crossing the surface is $u_z$, which varies over the surface. The volume crossing an infinitesimal area $dA$ per unit time is $u_z dA$ and the mass per unit time is $\rho u_z dA$. Hence

$$\dot{m} = \int \rho u_z dA = \int \int \rho u_z r \, dr \, d\theta. \tag{30}$$

We could have also derived this result from the formula (16) for flux of a quantity across a surface (using $\beta = 1$ for mass):

$$\dot{m} = \int_S \rho (\mathbf{u} \cdot \mathbf{n}) \, dS, \tag{31}$$

where

- $S$ is the surface and $dS$ is a small element of the surface area,
- $\rho(x, t)$ is the fluid density (now a function of space and time),
- $\mathbf{u}(x, t)$ is the fluid velocity (a vector function of space and time),
- $\mathbf{n}$ is the unit normal vector (constant if the surface is fixed and flat).

The formula above is true for any flow (in the present case, $\mathbf{u} \cdot \mathbf{n} = u_z$).
Poiseuille flow

• The flow is\(^2\)
  \[ p = p_1 - \frac{\Delta p}{L} z, \quad u_z = \frac{\Delta p}{4\pi \mu L} \left( A - \pi r^2 \right) . \]  

\((32)\)

• The volume flux along the pipe is

\[ \int_{\text{Cross-section}} u_z r \, dr \, d\theta = \frac{A^2 \Delta p}{8\pi \mu L} \]  

\((33)\)

which, for a given pressure gradient, is proportional to the fourth power of the diameter. Therefore if a pipe is replaced by one with double the diameter (keeping other parameters constant) the flux will increase by a factor of 16.

---

\(^2\)Note that we have only shown that this solution does satisfy the Navier–Stokes equations. We have not shown that it is the only solution, or even that it is stable. In fact, for large pressure gradients along the tube, Poiseuille flow becomes unstable and the flow can become unsteady or even turbulent.
Stress induced by the flow

- Endothelial cells (lining the arterial walls) respond to the shear stress they experience from the blood flow. The initiation process of atherosclerosis is thought to be linked to the shear stress profile and time-dependence.

- Therefore it is of some interest to find the shear stress in an artery, which is the force per unit area that the fluid exerts on the wall. This is given by \( \sigma n \), where \( n \) is the outward pointing vector from the wall, which, in this case, equals \( -\hat{r} \).

- The analogy of (21) in polar coordinates is quite complicated, and it is probably best to look it up rather than trying to work it out. We obtain

\[
\sigma (-\hat{r}) = (-p\mathbf{l} + 2\mu\varepsilon) (-\hat{r}) = p\hat{r} - \mu \frac{\partial u_\theta}{\partial r} \hat{\theta} - \mu \frac{\partial u_z}{\partial r} \hat{z}.
\]  
(34)

The normal stress is therefore simply the pressure, while the shear stress (the tangential components) decomposes into the axial shear stress, proportional to the radial derivative of the axial velocity, and the azimuthal shear stress, proportional to the radial derivative of the azimuthal velocity.
In the case of Poiseuille flow, the axial shear stress is

$$-\mu \frac{\partial u_z}{\partial r} \bigg|_{r=1} = \frac{\Delta p \sqrt{A}}{2 \sqrt{\pi} L}. \quad (35)$$

while the azimuthal shear stress is zero and the normal stress is \( p_1 - \Delta p \frac{z}{L} \).

The total drag force is the integral of the shear stress over the surface, which equals

$$\frac{(\Delta p \sqrt{A})}{(2 \sqrt{\pi} L)} \times (2\pi L \sqrt{A/\pi}) = A\Delta p.$$  

This result could also have been derived by observing that there is no acceleration of the fluid, and so the resultant force on the fluid must be zero. The forces acting on the fluid are the pressure force on the circular ends of the fluid and the stress forces coming from the walls. The pressure force on the ends of the fluid is \( A\Delta p \) in the direction of the flow. Therefore the total stress force from the walls is \( A\Delta p \) opposing the flow. Since the pressure forces cancel out due to the circular symmetry, this force is the drag force from the walls (the force of the walls on the fluid). Therefore the total shear stress force experience by the cells (the force of the fluid on the walls) equals \( A\Delta p \).

Exercise: Find an alternative derivation by using the Reynolds Transport Theorem to show that the drag force equals \( A\Delta p \).
Beyond Poiseuille flow

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Assumptions of Poiseuille flow

- Flow is axisymmetric
- Flow is fully developed and the vessel is rigid with a uniform cross-section
- Vessel is straight
- Flow is steady
- Artery is large enough that a Newtonian fluid model is appropriate

We will now discuss each of these five assumptions in turn. In particular, we will discuss when they should be relaxed and what effect this has on the flow. In each of the cases, a detailed description of what happens could take up a few lectures, so you are recommended to read further if you are interested.
To derive Poiseuille flow, we assumed the flow was axisymmetric. However, this need not be the case, as there are other solutions of the equations with broken symmetry. However, these are only observed for high Reynolds numbers, when the flow becomes turbulent. Another reason for the flow to become non-axisymmetric is if the pipe is not perfectly circular.

**Reynolds number:** This is a dimensionless quantity used to describe a fluid flow. It is probably the most widely used dimensionless number in fluid mechanics. It is defined by

\[
Re = \frac{\rho U L}{\mu},
\]

where
- \(\rho\) is the fluid density,
- \(U\) is a characteristic velocity scale,
- \(L\) is a characteristic length scale, and
- \(\mu\) is the viscosity of the fluid.

The Reynolds number gives a qualitative description of the flow, and is an order-of-magnitude estimate. The precise value is usually not important. For example, when describing the flow, the difference between a flow with \(Re = 60\) and one with \(Re = 80\) is often unimportant, but the difference between one with \(Re = 100\) and \(Re = 1000\) is important.

---

3 *Turbulence:* The study of turbulence constitutes an entire branch of fluid mechanics in its own right! Briefly, turbulent flows are characterised by behaviour on many different length scales from the scale of the container in which the experiment is being run down to lengths of a few intermolecular distances. Energy flows between difference lengthscales – the so-called cascade of energy.

4 An example of an exception to this might be if we set up an experiment and run it repeatedly in the same configuration with slightly different flow rates. We could define a Reynolds number based on some lengthscale in the experiment and use the precise value of \(Re\) to describe the flow.
1. Non-axisymmetric flow: Exercise

- What is the Reynolds number of Poiseuille flow?
- As a characteristic velocity you could use the maximum velocity and as a characteristic lengthscale you could use the pipe diameter.
- What difference does it make if you use the average velocity to describe the flow, or the pipe radius or pipe length instead of the pipe diameter?
2. Non-fully-developed flow: Introduction

Arteries are usually long compared with their diameter and have a regular cross-section, so the assumption of fully developed flow is often reasonable. However:

- The cross-section may not be constant along the pipe, for example due to:
  - **Arterial taper**, which tends to occur in most arteries (see figure).
  - **Elasticity** in the arterial walls cause the cross-sectional area to change in response to a change in pressure, resulting in non-uniformities in the cross-section along the pipe. See also S. Payvandi’s talk on Saturday.

- There may be **entrance effects** due to a change in the vessel, for example:
  - Flow in the ascending aorta is unlikely to be fully developed, since it is affected by the profile as it leaves the ventricle.
  - Flow in a daughter artery just after a bifurcation is affected by the splitting of the flow in the parent vessel.

Entrance effects persist for a distance proportional to $a \cdot Re$, where $a$ is the radius of the artery and $Re$ is the Reynolds number.

**Figure:** Change in radius and cross-section area of the canine aorta (by Caro et al., 1978).
3. Effect of arterial curvature on the flow

- The derivation of Poiseuille flow assumes the artery is straight.
- In reality, most arteries are curved, which has a significant effect on the flow structure.
- Happily, it is still possible to gain some insight using analytical techniques in the case when the centreline of the artery (the locus of centres of cross-sections of the pipe) is circular.

The other assumptions are the same as those used in the derivation of Poiseuille flow.

We assume the blood may be modelled as a Newtonian fluid and use the Navier–Stokes equations with no-slip boundary conditions.

We work in terms of a natural coordinate system of the pipe. Instead of $x$, $y$ and $z$, we use the distance along the pipe, $s$ and polar coordinates $r$ and $\theta$ in the cross-section, see figure.

After nondimensionalisation, the system has just two nondimensional parameters:

- The nondimensional curvature $\delta = a/R$, and
- The nondimensional flux $\rho Q/\mu a$. 

Figure: Sketch of coordinate system
Simplification:

- If we take the limit as $\delta \to 0$, then the system tends to a straight pipe (and the solution would be Poiseuille flow).
- However, we first make a rescaling, in which we consider long length scales and high velocities in the axial direction.
- Now when we take the limit as $\delta \to 0$, the centrifugal terms do not vanish (although the Coriolis terms do so).\(^5\)
- The equations are significantly simpler, notably in that the coordinate system effectively becomes like cylindrical polar coordinates.

\(^5\)The centrifugal and Coriolis terms can be understood if the problem is considered in the Lagrangian frame of reference, moving with the particles. As the particle moves along the pipe, it has to follow the bend in order to stay in the tube. Therefore if the particle tries to stay at the same point in the cross-section, it will experience effectively a force pulling it to the outside of the tube, which we may interpret as a centrifugal force in the frame of reference of the particle. Similarly there is a Coriolis force.
3. Effect of arterial curvature on the flow (ctd)

If the nondimensional flow is assumed small then a **series solution** may be found using analytical techniques. The series solution takes the form

\[
    u_r = Q^2 u_{r,0} + Q^4 u_{r,1} + \ldots, \quad u_\theta = Q^2 u_{\theta,0} + Q^4 u_{\theta,1} + \ldots, \quad u_s = Q u_{s,0} + Q^3 u_{s,1} + Q^5 u_{s,2} + \ldots. \tag{37}
\]

- The leading-order flow, \(Q u_{s,0}\), is Poiseuille flow, which is in the axial direction.
- The largest correction to this flow the components \(Q^2 u_{r,0}\) and \(Q^2 u_{\theta,0}\), which describe **colorrDean flow** across the cross section of the pipe. This flow has two vortices one in each half of the pipe cross section, and is illustrated in the figure on Page 52.
- The next correction, \(Q^3 u_{s,1}\) causes the location of the maximal axial velocity to be moved towards the outside of the bend of the pipe.

Poiseuille flow has a linear relationship between flow and pressure gradient. Interestingly, in the relationship between flow and pressure gradient in the curved pipe, the first few terms in the flow profile do not contribute to the axial flow rate. The departure from a linear relationship between flow and pressure gradient occurs at the fifth power:

\[
    Q = C_0 D + C_1 D^5 + \ldots, \tag{38}
\]

where \(C_0\) is the coefficient in Poiseuille flow.

**Torsion**: The analysis may also be extended to helical pipes (see Zabieliski & Mestel, 1998).

See also S. Payvandi’s talk at the end of the course.
3. Effect of arterial curvature on the flow

(a) Steady pressure gradient drives flow down the pipe. Flow predominantly in the axial direction (almost Poiseuille profile).

(b) Secondary flow. Centrifugal force acts towards the outside of the pipe bend.

Inside of pipe bend

Outside of pipe bend

Dean vortices

Figure: Dean flow. (a) Longitudinal cross-section, showing Poiseuille-like profile, (b) transverse cross-section, showing secondary flow, which forms Dean vortices.
4. Effect of unsteadiness in the flow

Flow in the cardiovascular system is not steady, but is rather highly pulsatile. This is illustrated in the graph below, which shows pressure and flow in the aorta.

Figure: Measured pressure and flow over the cardiac cycle

Slow oscillations of the pressure gradient When the oscillations in the pressure gradient are sufficiently slow, there is enough time for Poiseuille flow to develop, meaning the profile at any fixed time looks like the Poiseuille flow solution with the same pressure gradient. We call this a quasi-steady solution, because the time-derivative terms in the Navier–Stokes equations may be neglected.
In this case, we cannot neglect the time-derivative term in the Navier–Stokes equations. We consider the flow in the same model as before, but now \( p_1 \) and \( p_2 \) are functions of time. We still assume:
- Flow is axisymmetric
- Flow is in the axial direction
- Flow is fully developed (independent of \( z \))

As before, the continuity equation and azimuthal component of the Navier–Stokes equation are automatically satisfied. The radial and axial components respectively become

\[
0 = -\frac{\partial p}{\partial r}, \quad \rho \frac{\partial u_z}{\partial t} = -\frac{\partial p}{\partial z} + \frac{\mu}{r} \frac{\partial}{\partial r} \left( r \frac{\partial u_z}{\partial r} \right). \tag{39}
\]
4. Unsteady flow (ctd.)

Equation (25a) implies that $p$ is a function of $z$ and $t$ only, and, by differentiating (39b) with respect to $z$, we obtain as before

$$p = p_1 - \frac{\Delta p}{L}z,$$

where $\Delta p = p_1 - p_2$.  \hfill (40)

To solve Equation (39b), we need to prescribe $\Delta p$ as a function of time. For simplicity, assume that the time-dependence oscillates sinusoidally, say $\Delta p = p_0 e^{i\omega t}$, and, assuming that $u_z = U_0 e^{i\omega t}$ and substituting into Equation (39b), we obtain,

$$\frac{a^2}{r} \frac{\partial}{\partial r} \left( r \frac{\partial U_0}{\partial r} \right) - i\alpha^2 U_0 = -\frac{a^2 p_0}{\rho L},$$

where $\alpha = \sqrt{\rho \omega a^2 / \mu}$ is the Womersley number. \hfill (41)

The homogeneous equation of (41) (found by replacing the right-hand side with zero) has solutions of the form $c_1 J_0(kr/a) + c_2 Y_0(kr/a)$, where $k = \sqrt{-i\alpha}$ and $J_0$ and $Y_0$ are Bessel functions ($J_0$ is the Bessel function of the first kind of zero order and $Y_0$ is the corresponding Bessel function of the second kind).
4. Unsteady flow: Bessel functions

**Figure:** Bessel functions of the first and second kinds together with their orders $n$ (by Professor K. H. Parker)

- $J_0(x)$ is regular at $x = 0$ and tends to 1 there, whereas $Y_0(x) \sim \frac{2}{\pi}(\ln(x/2) + \gamma)$ for $x \ll 1$ (where $\gamma$ is Euler’s constant).
- For large $x$, $J_0(x) \sim \frac{\sqrt{2}}{(\pi x)} \cos(x - \pi/4)$ and $Y_0(x) \sim \frac{\sqrt{2}}{\pi x} \sin(x - \pi/4)$
4. Unsteady flow: Bessel functions (ctd)

Figure: Modified Bessel functions of the first and second kinds together with their orders $n$ (from efunda.com)
4. Unsteady flow (ctd.)

- A particular solution of (41) is $U_0 = -ip_0/(L\omega)$. Thus the general solution is
  \[ U_0 = c_1 J_0(kr) + c_2 Y_0(kr) - \frac{ip_0}{L\omega}. \] (42)

- We apply the regularity condition $U_0$ finite at $r = 0$ and the no-slip condition $U_0 = 0$ at $r = \sqrt{\pi/A}$ to obtain the amplitude of the axial flow profile:
  \[ U_0 = -\frac{ia^2 p_0}{\alpha^2 L \mu} \left( 1 - \frac{J_0(kr)}{J_0(k\sqrt{A/\pi})} \right). \] (43)

- This is a Womersley flow profile:
  \[ u_z = -\frac{ia^2 p_0}{\alpha^2 L \mu} \left( 1 - \frac{J_0(kr)}{J_0(k\sqrt{A/\pi})} \right) e^{i\omega t}. \] (44)
4. Unsteady flow exercise

- For small values of $\alpha$ the flow is **quasi-steady** (the time-derivative terms in the Navier–Stokes equations are unimportant).

- What about the high-frequency limit? Use the asymptotic form of the Bessel functions for large arguments to investigate the flow structure. What do you notice? Try simulating the flow.
5. Non-Newtonian flow: Introduction

So far in this course, we have treated the blood as a Newtonian fluid:

- Newtonian fluids have a stress–strain relationship of the form
  \[ \sigma = 2\mu \varepsilon - pI, \]  
  \[ (45) \]
  
  where \( \sigma \) is the stress tensor, \( \varepsilon \) is the strain tensor, \( p \) is the pressure and \( \mu \) is the viscosity (assumed constant). See Page 36.

- This is a remarkably good approximation to the behaviour of many real fluids in widely differing flows. We now consider its validity as a model for blood.

\[ \text{Proteins [~4%]} \]
\[ \text{Ions [<1%]} \]
\[ \text{Other [<1%]} \]
\[ \text{Water [50%]} \]
\[ \text{Formed elements [46%]} \]
\[ \text{Red cells [45%]} \]
\[ \text{White cells [<1%]} \]
\[ \text{Platelets [<1%]} \]

**Figure:** Overall composition of human blood. Plasma is composed of water, ions, proteins and ‘other’, while the composition of the formed elements is shown on the right-hand diagram. The numbers quoted are for males (the red blood cell fraction is approximately 4% lower in females). From Ethier and Simmons (2007).
5. Non-Newtonian flow: Mechanical behaviour of the red blood cells

Blood:

- contains a multitude of constituents with different mechanical properties (see pie charts),
- therefore it is far from being an ideal fluid,

In fact, the complex mechanics of the red blood cells is usually the cause of the greatest inaccuracy associated with a Newtonian model. Developing an accurate model of these mechanics is challenging for the following reasons:

- **Cell–cell interactions**: Red blood cells occupy 45% of the blood volume, and therefore blood is a highly concentrated suspension. Cells are closely packed and cell–cell interactions are important.

- **Alignment of cells**: Red cells are not spherical; they have a biconcave shape (see figure). If the fluid shear is low, the cells are orientated randomly, but in high shear they align with the direction of the shear. The effective viscosity of the blood is much lower when the cells are aligned than when they have random orientations. Thus the effective viscosity decreases as shear increases (shear-thinning property of blood) justified.

(List continues on next page)
5. Non-Newtonian flow: Mechanical behaviour of the red blood cells (ctd)

- **Deformation of cells:** As they flow along the arteries, the red blood cells deform significantly, meaning that a rigid-particle model to describe them is probably inaccurate. In order to pass through the narrow capillaries the cells adopt a completely different shape, see figure.

- **Particulate nature of cells:** The cells have diameter of around 8 µm, and in small vessels this is a substantial proportion of the vessel diameter. In the capillaries it may be larger than the vessel diameter. With such relatively large particles in a small vessel, a continuum model may not be justified.

**Figure:** Observed shapes adopted by red cells when travelling through a glass capillary of diameter 7 µm. The differences between the shapes of the three cells are thought to be due to the position of the cell within the capillary. The pictures are reproduced well by a mathematical model: to obtain a similar configuration to that in the top picture the cell is placed symmetrically. To obtain the middle picture the cell is offset by 0.5 µm towards the top. Finally to obtain the bottom picture the cell in the model is offset by 1 µm. From Secomb, Styp-Rekowska & Pries (2007).
5. Non-Newtonian flow: Implications of red cell behaviour

- Despite these complexities, it is actually reasonable to treat blood as a Newtonian fluid under many circumstances, for example if the vessel is relatively large compared with the red cell size, and the shear stays in a fairly narrow range of values.

- In small vessels in particular, the Newtonian model may not be the most appropriate. In this case we need to account for the different properties of blood under different shear rates.

- In a model of the capillaries we should take account of the red cells individually.
5. Non-Newtonian flow: Relationship between stress and shear rate

- In smaller vessels the **effective viscosity** of the blood is highly dependent on the local fluid flow, in particular the **shear rate**.

- To discuss realistic blood models we need to formulate a working definition of the effective viscosity.

- We do this by analogy with the case of a Newtonian fluid.
  - Assume the flow is a simple shear \( u = u(y) \hat{x} \), where \( \hat{x} \) is the unit vector in the \( x \)-direction and consider the force it exerts on a surface with unit normal \( \hat{y} \).
  - The **shear stress** \( \tau \) (component of stress that is tangential to the surface) is given by

\[
\tau = \mu \frac{\partial u}{\partial y} = \mu \dot{\gamma}, \quad (46)
\]

where \( \dot{\gamma} = \frac{\partial u}{\partial y} \) is the shear rate.

  - **Exercise:** Prove the relationship (46) starting from the relationship (45) between the stress and strain tensors.

- Newtonian fluids have the important property that the viscosity, \( \mu \), is constant.

- To find the effective viscosity of a non-Newtonian fluid, we need to measure the **shear stress** and the **shear rate** in an experiment. By analogy we define the effective viscosity

\[
\mu_{\text{eff}} = \frac{\tau}{\dot{\gamma}}. \quad (47)
\]
5. Non-Newtonian flow: Blood models

Shear-thinning fluid model:
- Shear-thinning fluids have an effective viscosity $\mu_{\text{eff}}$ that is a decreasing function of the shear rate $\dot{\gamma}$.
- Their properties under shear may be defined by specifying the functional form of $\mu_{\text{eff}}(\dot{\gamma})$, that is the dependence of $\mu_{\text{eff}}$ on $\dot{\gamma}$.
- We therefore have
  \[ \tau = \mu_{\text{eff}}(\dot{\gamma}) \dot{\gamma}. \]  
  (48)
  The bulk properties of the fluid can be taken as being Newtonian.

Viscoelastic model:
- In common with many biological materials, the effective viscosity of blood depends on the history of the shear rate as well as the present value. Such fluids are called viscoelastic, and their properties are a combination of fluid and solid properties.
5. Non-Newtonian flow: Blood models (ctd)

**Particulate model 1:**
Models that incorporate the red blood cells individually. *Eg* Tsubota, Wada & Yamaguchi (2006):

- 2D model
- Cell membranes (outer surfaces) treated as a spring network
- Plasma treated as a Newtonian fluid.

Advantages of such models:
- Provide detailed insight into events during cell–cell interactions.

Problems with these models:
- Difficult to formulate the model so that it reproduces realistic behaviour
- Very computationally expensive to run simulations
- Therefore can only be performed for a small number of cells or a short time.
5. Non-Newtonian flow: Blood models (ctd)

Particulate model 2:
On the capillary scale an even more detailed treatment is required. Secomb (in *Modeling and simulation of capsules and biological cells*, Pozrikidis (ed.), 2003) discusses analytical models of red blood cell motion in narrow tubes. Under the following assumptions
- Tube is sufficiently narrow that cells pass through in single file
- Cell and plasma flow are axisymmetric
- Membrane of cell is deformable but area-preserving
- Gap between cell and vessel wall is small compared to the vessel radius and axial length of gap. This allows *lubrication theory* to be be used (see Page 9).
- The cell membrane is assumed to be instantaneously in equilibrium

The fluid flow exerts a stress on the cell membrane, which causes it to deform, and in turn the deformation changes the shape of the gap and hence the fluid flow. The governing equations are given by:
- lubrication theory for the fluid flow, and
- mechanical equilibrium of the cell membrane is enforced by balancing the normal stress, the tangential stress and the bending moments acting on the membrane.

Solving the coupled problem determines the configuration of the cell and the plasma velocity. The cells move faster than the surrounding plasma. This leads to the prediction that there is a different circulation time for the plasma and the cells!
Lubrication theory for fluids: example of its application

The model by Secomb (2003) relied on a technique in fluid mechanics called **lubrication theory**. As a brief introduction to the subject I will illustrate it with an example.

**Cell travelling through capillary**

![Cell travelling through capillary](image)

**Enlarged view of green box**

![Enlarged view of green box](image)

**Figure:** Example of a scenario where lubrication theory may be applied. A cell moves steadily with speed $U$ along a vessel with a narrow gap at the walls.

Lubrication theory applies if one dimension of the space occupied by the fluid is much smaller than the other(s). For example in the figure above, and assuming derivatives with respect to the third coordinate may be neglected (that is the system is planar 2D):

- The fluid velocity at the vessel walls is zero (no-slip condition) but the fluid velocity at the surface of the cell equals the cell velocity. Therefore changes in the $x$-velocity $u$ are on the order of $U$, that is $|\Delta u| \sim U$, and $|\partial u/\partial y| \sim |\Delta u/\Delta y| \sim U/h$.

- The change in fluid velocity as we move through a distance $L$ in the $x$-direction is likely to be at most $U$, and therefore $|\partial u/\partial x| \sim U/L$. 
Lubrication theory for fluids: example of its application (ctd)

- The continuity equation,
  \[ \frac{\partial u}{\partial x} + \frac{\partial v}{\partial y} = 0, \tag{49} \]
  implies that \( |\partial v/\partial y| \sim U/L \); hence \( |\Delta v| \sim hU/L \).

- Nondimensionalise
  \[
  x = L x^*, \quad y = h_0 y^*, \quad h(x) = h_0 h^*(x^*), \quad u = U u^*, \quad v = h_0 U v^*/L, \quad p = \rho U^2 p^*. \tag{50}
  \]
  where \( h_0 \) is a typical value of \( h(x) \). Note that \( x^*, y^*, u^*, v^* \), are all order 1, but we have not yet determined the order of \( p^* \).

- The nondimensional governing equations are
  \[
  u^* u_{x^*}^* + v^* u_{y^*}^* = -p_{x^*}^* + \frac{u_{x^* x^*}^*}{Re} + \frac{u_{y^* y^*}^*}{\epsilon^2 Re}, \quad u^* v_{x^*}^* + v^* v_{y^*}^* = -\frac{p_{y^*}^*}{\epsilon^2} + \frac{v_{x^* x^*}^*}{Re} + \frac{v_{y^* y^*}^*}{\epsilon^2 Re}, \tag{51}
  \]
  \[
  u_{x^*}^* + v_{y^*}^* = 0, \tag{52}
  \]
  where \( \epsilon = h_0/L \ll 2 \), \( Re = UL/\nu \) and subscripts indicate differentiation. We may cancel the viscous terms that have a repeated \( x^* \)-derivative since they are much smaller than the viscous terms with a repeated \( y^* \)-derivative.

- The quantity \( \epsilon^2 Re \) is called the **reduced Reynolds number**. We assume it is small, which places an upper bound on the possible flux.
Assuming a pressure gradient drives flow in the $x^*$-direction, the pressure gradient term $p_{x^*}$ must appear in the leading-order balance. **Exercise:** What is the correct scale for the pressure?

The $y^*$-momentum equation is dominated by the pressure gradient term and becomes

$$p_{y^*} = 0 \Rightarrow p^* = p^*(x^*).$$

**Exercise:** Find the dominant balance of the $x$-momentum equation. Solve the equation to find $u$. Can you find $v$? What is the explanation?
Contents of this section

1. Anatomy of the cardiovascular system
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   - Introduction to the problem
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   - The multitude of vessels
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   - Foundations
Some more facts about the cardiovascular system

- So far we have studied models of a single artery.
- Next we consider ways to model the cardiovascular system as a whole.
- First we must review a few more facts about the arterial system.
- In this section we will mainly look at the arterial pressure distribution.

Properties of arterial flow that make modelling difficult

- Flow is pulsatile (periodic with a strong ‘pulse’ at the start of the cycle) and highly unsteady (both pressure and velocity vary widely over the cycle).
- Variation over longer timescales: During exertion, both the rate of the heartbeat and the stroke volume (volume per beat) increase. Cardiac output varies from about 5 l/min when at rest to about 25 l/min during strenuous exercise.
- Biomechanics of vessel walls: These have a complicated structure. They spend most of their lives in a deformed state. They are usually deformed. Arteries tend to be inflated and veins can be strongly collapsed.
Properties of arterial flow that make modelling it difficult (ctd)

- **Wide ranges of scales:**
  - **Diameters** range from around 30 mm (in the vena cava) to around 8 µm (in the capillaries).
  - **Average velocity** of blood during the cardiac cycle ranges from 0.5 m/s in large arteries to less than 1 cm/s in the capillaries.
  - **Reynolds numbers** range from $Re \approx 4500$ to $Re \approx 0.001$. Recall that
    \[ Re = \frac{Ud}{\nu}, \tag{54} \]
    where
    - $U$ characteristic velocity (eg average velocity),
    - $d$ characteristic lengthscale (eg vessel diameter),
    - $\mu$ viscosity of blood,
    - $\rho$ density of blood
  - Typically in the large arteries, $Re \gg 1$, while in small vessels and capillaries, $Re \ll 1$. 
More about the cardiovascular system

The multitude of vessels

Total cross-sectional area of the arterial bed

- The number of arteries with a particular cross-sectional area increases faster than the area decreases along the arteries. This means the total cross-sectional area of the arterial bed increases with distance from the heart (see figure).\(^a\)

- For a given bifurcation, we define the **branching ratio** as the fractional change in cross-sectional area, that is, the sum of the areas of the two daughter arteries divided by the area of the parent artery. In the human cardiovascular system this can vary between 0.79 and 1.29. We will see that this ratio influences the reflection properties of waves reaching a bifurcation point (see Wave Intensity Analysis, Page 94).

\(^a\)Strictly speaking, we should show that the rate of change of the logarithm of the number of branches increases at a faster rate than the rate at which the logarithm of the area decreases. **Question:** Can you see why this is?

**Exercise:** What implications does this area change have for the blood velocity? **[Hint:** Consider mass conservation.]

---

**Figure:** diagram showing that the cross-section area of the vascular bed grows peripherally (by Caro et al., 1978).
Clinical measurement of blood pressure

- The **transmural pressure** is usually the most relevant pressure both clinically, i.e. in terms of its implications for patient health, and also in terms of its relevance for the biomechanics. It is defined by

\[
P_{\text{(transmural)}} = P_{\text{(inside vessel)}} - P_{\text{(outside vessel)}}. \quad (55)
\]

**Question:** Why do you think **transmural** as opposed to **absolute** pressure is more useful?

- **Gravity** has a significant influence on the blood pressure, see picture. To eliminate its effects, we separate the pressure into

  - the **hydrostatic pressure** due to gravity, and
  - the **dynamic pressure** due to the pumping of the heart (always positive):

\[
P_{\text{(transmural)}} = P_{\text{(hydrostatic)}} + P_{\text{(dynamic)}}. \quad (56)
\]

**Questions:**

- What assumption is being made when the pressure relative to atmospheric pressure is quoted, rather than the transmural pressure?
- Can you think of a reason why pressure readings are usually taken on the upper arm with the patient seated?
Clinical measurement of blood pressure (ctd)

Figure: Simultaneous records of pressure and diameter in the ascending aorta during a cardiac cycle (from Caro et al., 1978).

<table>
<thead>
<tr>
<th>Period of cardiac cycle</th>
<th>Systole</th>
<th>Diastole</th>
</tr>
</thead>
<tbody>
<tr>
<td>Related adjective</td>
<td>Systolic</td>
<td>Diastolic</td>
</tr>
<tr>
<td>Definition</td>
<td>When aortic valve open</td>
<td>When aortic valve closed</td>
</tr>
<tr>
<td>What happens</td>
<td>Pressure rises, peaks and falls. Ends at the <strong>dichrotic notch</strong></td>
<td>Pressure falls – good fit with exponential graph</td>
</tr>
<tr>
<td>Blood pressure</td>
<td><strong>Systolic b.p. = max b.p.</strong></td>
<td><strong>Diastolic b.p. = min b.p.</strong></td>
</tr>
</tbody>
</table>
Clinical measurement of blood pressure (ctd)

Devices such as a sphygmomanometer measure the systolic and diastolic blood pressure relative to atmospheric pressure:

**Sphygmomanometer operation:** A cuff is placed around the patient’s upper arm, and inflated to $p_{(cuff)} > p_{(systolic)}$. The pressure is gradually reduced to $p_{(cuff)} < p_{(diastolic)}$ while the operator listens through a stethoscope:

<table>
<thead>
<tr>
<th>Cuff pressure</th>
<th>What the operator hears</th>
</tr>
</thead>
<tbody>
<tr>
<td>$p_{(cuff)} &gt; p_{(systolic)}$</td>
<td>No sound</td>
</tr>
<tr>
<td>$p_{(cuff)} \approx p_{(systolic)}$</td>
<td>Ticking noise begins</td>
</tr>
<tr>
<td>$p_{(diastolic)} &lt; p_{(cuff)} &lt; p_{(systolic)}$</td>
<td>Ticking noise becomes a whooshing noise</td>
</tr>
<tr>
<td>$p_{(cuff)} \approx p_{(diastolic)}$</td>
<td>Noise stops</td>
</tr>
<tr>
<td>$p_{(cuff)} &lt; p_{(diastolic)}$</td>
<td>No sound</td>
</tr>
</tbody>
</table>

This allows the operator to find $p_{(systolic)}$ and $p_{(diastolic)}$.

- Pressure is usually quoted in **mm Hg** (millimetres of mercury).
- This is the blood pressure relative to atmospheric pressure. To find the absolute blood pressure, we must add atmospheric pressure.

**Questions**

- How are mm Hg related to Pa?
- Why does the sphygmomanometer work? **Hints:**
  - Think about what happens to the artery when the pressure outside is greater than the pressure inside (which is the case when the cuff pressure is above diastolic pressure).
  - When the transmural pressure is close to zero, the artery is very sensitive to pressure change, i.e. a small change in pressure causes a lot of difference in the cross-sectional area. So what happens if the transmural pressure is unsteady and very close to zero?
Hydrostatic pressure and its elimination

- Pressure increases as we go deeper under water. In fact, for every 10 m deeper under water, the equivalent of one atmosphere of pressure is added! Thus 10 m under water, the pressure is 2 atm.
- This is an effect of gravity. The pressure increases due to the weight of water on top.
- We may analyse this using a control volume approach.

Exercise:

- Consider a small cube of stationary fluid of side. Can you derive what is the pressure gradient in the vertical direction \(\frac{\partial p}{\partial z}\)? What about \(\frac{\partial p}{\partial x}\) and \(\frac{\partial p}{\partial y}\)?
- Solve the partial differential equations to find the pressure field \(p(x, y, z)\). This is the hydrostatic pressure.
- What happens if the density is not constant? This might be relevant for example when studying air pressure in the Earth's atmosphere, because the air becomes increasingly thin at high altitude.
When you did the exercise on Page 78, you will have noticed that you can only find the hydrostatic pressure up to an arbitrary constant (of integration).

Therefore hydrostatic pressure must always be specified relative to a reference point.

Questions:

- Note that the hydrostatic pressure can be positive or negative. Where is it negative?
- How would you find the dynamic pressure if you took a sphygmomanometer reading on the leg of a standing patient for example?

Hereinafter, we will refer to the dynamic pressure as the arterial pressure (implicitly assuming the hydrostatic pressure has been removed).
Estimation of cardiac power: The pressure–volume loop

Figure: Schematic plot of a typical pressure–volume loop of the left ventricle. (a) Passive filling phase (work done by blood on left ventricle), (b) isovolumetric contraction (no work done, elastic energy stored in heart muscle), (c) ejection phase (work done on blood by ventricle), (d) isovolumetric relaxation (no work done, but stored elastic energy is dissipated). The dashed lines indicate a simplified shape used for calculation. From Ethier and Simmons (2007)

We estimate the useful energy content of the fluid using Bernouilli’s theorem. This tells us that the useful energy content of fluid is represented by its head:

$$p_T = p + \frac{\rho U^2}{2} + \rho gz.$$  \hspace{1cm} (57)

---

Bernouilli’s theorem states that the head of a fluid is constant on streamlines of the fluid, as long as the flow is steady, incompressible, inviscid, no work done on or by fluid, no heat transfer. It may be derived by applying momentum conservation to an infinitesimal streamtube (a tube-like region of fluid whose curved sides contain streamlines). The conditions for validity are approximately satisfied in regions (a) and (c) of the figure. In (b) and (d), the rate of change of the head equals the rate of work done on the fluid per unit fluid volume.
In the case of the heart, $p_T \approx p$ is a good approximation to the energy stored per unit volume.

Therefore the net work done over a cycle is

$$W = \int_{\text{cycle}} p \, dV,$$

which is the area within the pressure–volume loop.

We can simplify by integrating around the dashed rectangle in the figure to get

$$W = \Delta p \Delta V$$

This gives an estimate of 1.44 J per cycle from the left and right sides of the heart combined, which corresponds to a power of 1.68 W. During a 75-year life, the heart beats approximately $2.76 \times 10^9$ times. The total energy could be used to lift a 50-ton weight to the top of Mount Everest!
Maximum and minimum pressures

**Figure:** Pressure distribution in the human cardiovascular system (from Ottesen et al., 2004).
Maximum and minimum pressures (ctd)

From the graph on Page 82, we see that:

- The highest pressures are achieved in the left ventricle during ejection. Pressure in the left ventricle is significantly higher than in the right ventricle.

- The pressure in the aorta and large arteries fluctuates significantly in time due to the pumping action of the heart. Maximum and minimum pressures are approximately **120 mmHg and 80 mmHg** (\(\sim 16000–10665 \text{ Pa}\)). These are the values measured by a sphygmomanometer.

- Note the growth of the amplitude of fluctuations along the aorta. This is due to wave reflections from the peripheral arteries (see section on wave intensity analysis).

- The pressure in the capillaries fluctuates very little.

- The pressure in the veins does not reflect the oscillations due to the heartbeat. Pressures are quite low and the veins can collapse (this does not usually happen in arteries). Instead there are small changes due to squeezing by the surrounding muscles. These are used to drive the blood. This mechanism is particularly important in the legs.\(^7\)

---

\(^7\)Sometimes older people or pregnant ladies experience swelling around their ankles. This is due to insufficient pumping in the veins to bring the fluid back up to the heart. When we rest we like to put our feet up; due to the smaller hydrostatic pressure difference, this will mean the heart does less work.
Pressure and flow waveforms

- The systolic pressure increases with distance from the heart.
- The wave becomes steeper and the amplitude (maximum minus minimum value) increases.
- The amplification continues (in the dog) up to the third generation of branches (which have a diameter of approximately 1-2 mm).
- Thereafter, both the oscillation and the mean pressure decrease.

The mean pressure steadily decreases with distance from the heart (this is hard to detect in the figure).

**Figure:** Pressure versus time at different sites in along the aorta (from Caro et al., 1978).
Pressure and flow waveforms (ctd)

Figure: Records of pressure and velocity at different sites of the arterial system (from Caro et al., 1978)
More about the cardiovascular system

Pressure and flow waveforms (ctd)

Figure: Simultaneous records of pressure and velocity at different sites in the human arterial system (from Ethier and Simmons, 2007).

How do we attempt to explain the complexities in these waveforms?
Calculation of wall tension

We relate the transmural pressure $p$ to the tension $T$ (force per unit length) in the wall. Assume:

- circular cross-section;
- infinitely long tube (we neglect side effects);
- constant diameter.
- homogeneous and isotropic wall,
- wall is in static equilibrium.

The law of Laplace states

$$T = pR,$$  \hspace{1cm} (60)

where $R$ is the radius of the vessel.

**Exercise:** Derive the law of Laplace either by considering the segment shown and resolving the total forces on it in the vertical direction, or by analysing an infinitesimal slice of the wall.
**Ex vivo pressure–radius relationship**

*Figure:* Pressure-radius relationship for the carotid artery of the rat. Each curve refers to a different condition of the vascular smooth muscle (VSM). (○) fully relaxed VSM, (•) normal VSM, (□) maximally contracted VSM (from Ethier and Simmons, 2007).

- The relationship between pressure and radius is highly nonlinear.
- The slope decreases at higher pressures, showing that the vessel is stiffer when the lumen is bigger.
- This behaviour reflects the strain-stiffening behaviour of the collagen and elastin in the arterial wall.
The similarity of the two graphs suggests that pressure and diameter are directly related, i.e. the diameter is a function of pressure.

**Question:**
Assuming the relationship is based on *mechanical principles* only. The relationship between pressure and diameter may be *direct*, meaning it is of the form

$$d = f(p), \quad \text{i.e.} \quad d(t) = f(p(t)).$$

(61)

Alternatively it may be a *functional relationship*, that is, of the form

$$d = F[p] \quad \text{i.e.} \quad d(t) = F\{p(s) : s \in \mathbb{R}\}.$$  

(62)

What does the fact that the relationship is direct rather than functional imply about the biomechanics of the arterial wall?
Mathematically derived pressure–area relationship for a thin-walled elastic tube

**Figure:** Relationship between transmural pressure and cross-sectional area of an elastic tube. Shapes of the cross-section are sketched at three locations.
**Windkessel model**

Figure: Top: Schematic diagram of Windkessel model. Bottom: Equivalent electric circuit. From Ethier and Simmons (2007)

- Developed by Otto Frank in 1899 (see translation by Sagawa et al., 1990)

- **Advantages of the model:**
  - Gives qualitative understanding of the physical processes involved.
  - Accounts for the distensibility of the vessels.

- **Disadvantages of the model:**
  - Results not very accurate.
  - No accounting for wave-like behaviour.
Windkessel model (ctd)

The components are:

<table>
<thead>
<tr>
<th>Component</th>
<th>Properties of component</th>
<th>Electrical representation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart</td>
<td>Produces prescribed flux</td>
<td>Supplies current $Q_H(t)$</td>
</tr>
<tr>
<td>Distensible arteries</td>
<td>Compliant chamber with constant stiffness</td>
<td>Capacitor with capacitance $C$ and charge $V$</td>
</tr>
<tr>
<td>Peripheral vessels</td>
<td>Constant resistance to flow</td>
<td>Resistor with resistance $R$ and current $Q$</td>
</tr>
<tr>
<td>Blood pressure</td>
<td></td>
<td>Potential difference $p_{\text{art}}$ across both capacitor and resistor</td>
</tr>
<tr>
<td>Veins</td>
<td>Zero pressure</td>
<td>Earth</td>
</tr>
</tbody>
</table>

We solve the model to find the flux $Q$ entering the peripheral circulation.

The equations are

\[
\text{Capacitor: } C = \frac{dV}{dp_{\text{art}}}, \tag{63}
\]

\[
\text{Resistor: } p_{\text{art}} = QR, \tag{64}
\]

\[
\text{Charge conservation: } Q_H = Q + \frac{dV}{dt}. \tag{65}
\]
Eliminating \( p_{\text{art}} \) and \( V \), we obtain the governing equation for the flux through the capillaries:

\[
RC \frac{dQ}{dt} + Q = Q_H. \tag{66}
\]

We representing the flow output from the heart by

\[
Q_H(t) = Q_0 + \sum Q_i \sin(\omega_i t) \tag{67}
\]

and solving Equation 67 (ignoring transients)

\[
Q(t) = Q_0 + \sum \frac{Q_i}{\sqrt{1 + (RC\omega_i)^2}} \sin(\omega_i t - \tan^{-1}(RC\omega_i)). \tag{68}
\]

The steady component of the flow is transmitted directly into the capillaries, whereas the oscillating components are damped and out of phase from their original waveforms.

As mentioned, the results of the model are not accurate. This is partly due to the fact that vessels in different locations within the arterial tree distend at different times during the cardiac cycle as pressure waves travelling along the arteries reach them. The Windkessel model, however, cannot account for these waves, since the arteries are lumped together.

To improve the results we need to investigate wave propagation within the arteries. One model that has had some success in providing insight into this is wave intensity analysis, which we will study in the next section.
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8. Wave intensity analysis
   - Foundations
Wave intensity analysis is rooted in the development of gas dynamics during and after the Second World War. The advent of supersonic flight, jet engines and rockets required a new approach to aerodynamics that could explain the ‘new’ phenomena that were being observed; particularly shock waves.

The mathematical tools for solving these problems were provided nearly a century earlier by Riemann who introduced the method of characteristics for the solution of hyperbolic equations (B Riemann, *Gesammelte mathematische Werke un wissenschaftlicher Nachlass*, 1860).

Although arteries have complex geometries, for many purposes it is sufficient to consider them as long, thin tubes using the one-dimensional approximation (see Appendix, page 1).

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8 These notes are a summary of material by Professor Kim Parker. The full notes are available at http://www.bg.ic.ac.uk/research/intro_to_wia/welcome.html
What is a wave? Illustration of two approaches
What is a wave? Illustration of two approaches (ctd)

Figure: (See page 96) Representation of a wave in terms of Fourier components (left). The top trace is the pressure waveform measured in the human ascending aorta. The 16 traces below show the Fourier components for the fundamental and first 15 harmonics (the mean pressure is not shown). The summation of these 16 traces give the original signal to greater than 95% accuracy (if all of the Fourier components are included, the original signal is recovered exactly).

Representation by successive wavefronts (right). Again the top trace is the measured pressure waveform. The 16 traces below are the successive wavefronts derived from this waveform. They represent the change in pressure over a time period equalling 1/16th of the cardiac period. They sum to give a good representation of the original waveform. If wavefronts were defined using the difference measured at each sampling time of the original data, the summation would be exact.
What is a wave? Representation by Fourier decomposition

There are many possible ways to decompose a ‘wave’. Two important representations are provided by Fourier decomposition and successive wavefronts.

Fourier decomposition:

- Historically, this has been the most common way to represent cardiovascular waveforms.
- Treats the measured waveforms as the superposition of sinusoidal wavetrains at the fundamental frequency and all of its harmonics. See the figure on the left of page 96, which shows a human aortic pressure waveform decomposed into Fourier components. The measured waveform is shown at the top of each panel and the decomposition below.
- This approach has been very successful; however, it is not the only way to decompose arterial waveforms into unique and independent components (there is actually an infinite number of ways to do this).
- Since Fourier analysis is carried out in the frequency domain, it can be very difficult to relate features of the Fourier representation to specific times in the cardiac cycle.
What is a wave? Representation by successive wavefronts

**Successive wavefronts:**
This is a second approach to approximate the wave.

- Waves such as tsunamis and shock waves (the sonic boom) are best described as solitary waves. It is more convenient to consider these as a sequence of small ‘wavefronts’ that combine to produce the observed wave.

- Wavefronts are the elemental waves in wave intensity analysis. They can converge on each other as they propagate, causing waves to steepen and eventually form shock waves, or they can diverge causing the the wave to become less steep as it propagates. This process is seen on the beach where the fronts of approaching waves crest and eventually break as the wavefronts at the foot of the wave are overtaken by the faster wavefronts near the crest of the wave.

- It is convenient and accurate to describe these wavefronts as the change in properties during a sampling period $\Delta t$, e.g. $dP = P(t + \Delta t) - P(t)$ (note that $dP$ depends upon the sampling period, unlike the differential).

- The plot on the right of page 96 shows the same pressure waveform decomposed into 16 successive wavefronts (note that a higher resolution can be obtained simply by using more wavefronts occurring at smaller intervals during the cardiac period).
What is a wave? Comparison of the Fourier and successive wavefronts decompositions

The difference between the Fourier and successive wavefronts approaches is fundamental to the understanding of wave intensity analysis:

- Both approaches give unique, complete representations of the measured waveform and the choice of representation is determined solely by convenience. The decomposition is not in any way intrinsic to the waveform.
- Therefore a description in terms of wavefronts can be converted into one in terms of Fourier components, and *vice versa*.
- Wave intensity analysis implicitly uses the successive wavefront representation.
- Most, if not all, textbooks on haemodynamics do not describe arterial waves using the successive wavefronts approach. To understand wave intensity analysis, it is therefore helpful to start by thinking about waves in an abstract, general way.
Is the cardiovascular system in a steady state oscillation?

Figure: Response measured in the left main stem coronary artery during a missing beat. The top trace shows the pressure in kPa and the bottom trace shows the simultaneously measured ECG.
Is the cardiovascular system in a steady state oscillation? (ctd)

- The figure on page 101 shows the pressure and ECG during a missing beat.
- A premature QRS complex occurs in the ECG just before 26 s. This results in a contraction of the left ventricle that was barely able to create enough pressure to open the aortic valve. The response to this missing beat is a smooth continuation of the exponential fall-off of pressure that is normally observed during diastole.
- Following the missing beat, the ECG is normal and the pressure is close to normal. The pulse pressure of the beat immediately following the missing beat has a slightly increased pulse pressure consistent with the potentiation of the ventricular contraction produced by the increased filling due to the preceding missing beat (the Frank-Starling mechanism). There is also a decrease in mean pressure that persists for about 4 to 5 beats before the oscillation returns to the state prior to the missing beat.
- During the missing beat we see a smooth continuation of the exponential fall-off of pressure that is normally observed during diastole. This is typical of an over-damped system. There is no hint of a slightly damped oscillation at the normal heart frequency that would be characteristic of an under-damped system. This behaviour indicates that the cardiovascular system is over-damped and, by definition, over-damped systems cannot exhibit steady-state oscillation.

Therefore the cardiovascular system is not in steady-state oscillation. It is probably better to think of each heartbeat as an isolated event that just happens to occur periodically because of the regularity of the normal heart beat under constant conditions.
Nomenclature

There is disparity in the nomenclature used in the literature, and here we discuss possible definitions.

Waves

One of the biggest problems in the communication of wave intensity analysis arises from the different meanings of the word ‘wave’:

- It is appropriate to use ‘wave’ to describe any disturbance that propagates in space and time (e.g. sound waves, light waves, radio wave, water waves).
- Most, but not all, waves are periodic.
- On Pages 96–100 we discussed two possible approaches, both of which could be described as ‘decomposing the waveform into waves’:
  - In this course we treat the waveforms of pressure and velocity as the superposition of succeeding wavefronts.
  - Another commonly used approach is to treat the waveforms as the superposition of sinusoidal wavetrains.

For clarity we adopt the following conventions:

- ‘waveform’: measured pressure or velocity waveforms
- ‘wavetrain’: a sinusoidal wave
- ‘wavefront’: a small, incremental wave
Wavefronts
Wavefronts can travel either forward or backward, and can increase or decrease the pressure and either increase or decrease the velocity. The incremental pressure and velocity are related by the water hammer equation (which depends on the direction of travel of the wavefront):

\[ dP \pm = \pm \rho c dU \pm. \]  

Wavefronts have three properties: direction of travel, type of pressure change and type of velocity change. The water hammer equation implies that any one property depends on the other two and so it is necessary to specify only two of them to describe the wavefront type fully.

- **Direction of travel:** We define the **forward direction** as the direction of mean blood flow\(^9\) and the **backward direction** as the opposite.
  - In **forward travelling wavefronts** the sign of the change in pressure equals the sign of the change in velocity.
  - In **backward waves** these two signs are opposite.

---

\(^9\)In the arteries this is an obvious choice, but it is contentious in the veins.
Effect on pressure: There are five descriptions of the effect on pressure that are in use:

- ‘compression–decompression’
- ‘compression–expansion’ (this can be confusing since, in elastic vessels, a decrease in pressure results in a decrease in the diameter of the vessel)
- ‘compression–rarefaction’ (less appropriate for descriptions of liquids than gases)
- ‘pushing–pulling’
- ‘blowing–sucking’ (refers to the sign of the difference in pressure between the two ends of the tube, could be interpreted as the changes relative to ambient pressure – with this interpretation ‘sucking’ waves would cause collapse in elastic vessels)

Here we use ‘compression–decompression’.

Effect on velocity: Acceleration waves increase the velocity in the direction of travel and deceleration waves decrease the velocity in the direction of travel.
Method of characteristics: Governing equations

Conservation of mass

\[ A_t + (UA)_x = 0, \quad (70) \]

where\(^\text{10}\)
- \(A\) is the cross-sectional area of the tube
- \(U\) is the average axial velocity
- \(x\) is distance in the axial direction
- \(t\) is time

Conservation of momentum

\[ U_t + UU_x = -\frac{P_x}{\rho}, \quad (71) \]

where
- \(P\) is the hydrostatic pressure averaged over the cross-section
- \(\rho\) is the density (assumed constant)

---

\(^{10}\)Note that we are using subscripts to denote partial derivatives. Thus \(A_t = (\partial A/\partial t)|_x,\ etc.\)
Method of characteristics: Governing equations (ctd)

**Tube law**

The constitutive equation for the elastic behaviour of the walls:

\[ A(x, t) = A(P(x, t)) \]  \hspace{1cm} (72)

This says that \( A \) and \( P \) are related by a functional relationship (which we have not yet specified)\(^{11}\).

\(^{11}\)Note: here we are assuming the relationship is the same all the way along the tube. It is possible to generalise the tube law to a function of \( x \), that is \( A(x, t) = A(P(x, t), x) \), or even \( x \) and \( t \) (but this is more difficult). Using \( A(x, t) = A(P(x, t), x) \) leads to non-zero growth of the Riemann variables in Equation (82).
Method of characteristics: Rearrangement of governing equations

Equations (70)–(72) can be rearranged:

\[ P_t + UP_x + \frac{A}{A_P} U_x = 0, \quad U_t + \frac{1}{\rho} P_x + UU_x = 0. \] (73)

which can be rewritten as

\[ \frac{\partial}{\partial t} \begin{pmatrix} P \\ U \end{pmatrix} + \begin{pmatrix} U \\ 1/\rho \\ A/A_P \\ U \end{pmatrix} \begin{pmatrix} P \\ U \end{pmatrix} = 0. \] (74)

The eigenvalues of the 2×2 matrix are \( \lambda_{\pm} = U \pm c \), where

- \( c = 1/\sqrt{\rho D} \) is the wavespeed
- \( D = A_P/A \) is the distensibility (a property of the vessel walls)

The arterial walls usually become stiffer when the pressure rises, meaning that the distensibility reduces and wavespeed increases.
Method of characteristics: Characteristics trajectories

We define characteristic trajectories $\xi_\pm(t)$ defined by

$$\frac{d\xi_\pm}{dt} = \lambda_\pm = U \pm c. \quad (75)$$

We rewrite the system in terms of each of the two sets of coordinates

$$(t_+ = t, x_+ = x - \xi_+), \quad \text{and} \quad (t_- = t, x_- = x - \xi_-), \quad (76)$$

In the forward-travelling frame $(t_+, \xi_+)$, we may show that the relationships to convert partial derivatives are

$$\left. \frac{\partial}{\partial t} \right|_x = \left. \frac{\partial}{\partial t_+} \right|_{x_+} + (U + c) \left. \frac{\partial}{\partial x_+} \right|_{t_+}, \quad \left. \frac{\partial}{\partial x} \right|_t = \left. \frac{\partial}{\partial x_+} \right|_{t_+}, \quad (77)$$

(and similar relationships hold in the backward-travelling frame).
Method of characteristics: Solution using Riemann invariants

In the forward-travelling frame Equations (73) become

\[ P_{t_+} - cP_{x_+} + \rho c^2 U_{x_+} = 0, \quad U_{t_+} - cU_{x_+} + \frac{1}{\rho} P_{x_+} = 0, \]  
(78)

and in the backward-travelling frame they become

\[ P_{t_-} + cP_{x_-} + \rho c^2 U_{x_-} = 0, \quad U_{t_-} + cU_{x_-} + \frac{1}{\rho} P_{x_-} = 0, \]  
(79)

From these equations we obtain the simpler forms:

\[ \frac{1}{\rho c} (78a) + (78b) : \quad U_{t_+} + \frac{1}{\rho} P_{t_+} = 0, \]  
(80)

\[ -\frac{1}{\rho c} (79a) + (79b) : \quad U_{t_-} - \frac{1}{\rho} P_{t_-} = 0, \]  
(81)

Equations (80)–(81) can be written in the very simple form

\[ \frac{\partial R_\pm}{\partial t_\pm} = 0, \quad \text{where} \quad R_\pm = U_\pm \pm \int \frac{dP_\pm}{\rho c} \quad \text{are the Riemann invariants.} \]  
(82)

We may integrate the equation to show \( R_+ \) is constant in time in the forward-travelling frame of reference and \( R_- \) is constant in time in the backward-travelling frame.
Since the two frames move forward and backward along the tube with speed $c$, this justifies our definition of $c$ as the wavespeed on page 108.

If the blood velocity were zero, waves would propagate forwards and backwards along the tube with speed $c$. When the blood flows, the waves are convected by the blood, which adds a forward velocity $U$ onto both the forwards and the backwards waves, making their velocities $U \pm c$.

If $U < c$ (subsonic conditions) then the forward waves propagate forwards ($U + c > 0$) and the backward waves propagate backwards ($U - c < 0$). If $U > c$ (supersonic conditions) then the backward waves would also propagate forwards. Supersonic conditions lead to many interesting phenomena in gas dynamics (e.g. you can’t hear a supersonic plane approaching you).

In humans the wave travels from the heart to the radial artery in approximately 0.1 s giving a wavespeed around 10 m/s. The blood typically moves 20 cm during 1 heartbeat (approximately 1 s), giving an average velocity of around 0.2 m/s. In general, the wavespeed in the arteries is an order of magnitude larger than the fluid velocities.

In this course we assume $U < c$.

The wavespeed $c$ depends on the distensibility $D = A^{-1}dA/dP$, which depends on the compliance or the stiffness of the artery. The stiffness is of clinical importance, and, since $\rho$ is approximately constant, the local wave speed determines arterial stiffness (stiffer arteries have a lower distensibility and hence a higher wave speed).
Method of characteristics: Solution using Riemann invariants (ctd)

To find the solution at \((x, t)\):

- Find the forward and backward waves that intersect at the point \((x, t)\)
- Determine the values of \(R_\pm\)
- Use the equations

\[
P = \frac{\rho c}{2} (R_+ - R_-), \quad U = \frac{1}{2} (R_+ + R_-)
\]

(83)

to solve for \(P\) and \(U\).

Conceptually this is very easy, but it is complicated due to the following:

- The path of the wave depends upon the local velocity and the local velocity depends upon the waves arriving there from upstream and downstream.
- The expression for the wave speed depends on the pressure, meaning we must solve integral equations to find \(P\) and \(U\) give the values of \(R_\pm\).

Generally, the Riemann variables are determined by the boundary conditions that are applied at the inlet and outlet of the vessel. In more complicated circumstances, changes can be imposed upon the vessel, for example, by applying external compression to it at some particular point. In these cases, the Riemann variables are also determined by the conditions imposed everywhere along the vessel, not just at its boundaries.
Wave intensity

Wave intensity is defined as

\[ dl = dP \, dU, \]

where

- \( dP \) is the change of pressure across a wavefront,
- \( dU \) is the change in velocity across a wavefront.

It has the following properties:

- It has the units of power/area, \( W/m^2 \).
- It is positive for forward travelling wavefronts (both compression and decompression) and negative for backward wavefronts.
- This means that if there are simultaneous forward and backward waves (which is often the case in arteries), the total wave intensity is the sum of the wave intensities of the two wavefronts that intersect at the measurement site at the time of measurement.
- The sign of the net wave intensity therefore reveals immediately if the forward or backward waves are dominant.
Wave intensity (ctd)

Figure: The first measurement of wave intensity in a human: ascending aorta. There are large negative peaks indicating a dominant backward wave in mid-systole that varies with the respiratory cycle. Large negative peaks such as these are in fact pathological.
A simple example of an isolated wave

We can illustrate the main features of wave intensity analysis using an example:

- Consider a syringe pump (heart) connected to a long, uniform elastic tube (aorta). The other end of the tube maintained at pressure $P = 0$.

- The piston of the syringe is moved according to the following:
  - $t < 0$: The piston and fluid are stationary and the fluid is at constant pressure.
  - $0 < t < 1$: The piston is accelerated.
  - $1 < t < 3$: The piston moves with constant speed.
  - $3 < t < 4$: The piston is decelerated.
  - $t > 4$: The piston is stationary.

The results are shown in the linked animation and sketched in the figure on Page 116.

Wave in tube
A simple example of an isolated wave (ctd)

The piston-end of the tube:
- expands during $0 < t < 1$ (acceleration),
- maintains a constant expansion during $1 < t < 3$ (constant velocity),
- contracts during $3 < t < 4$, and finally
- resumes its undeformed configuration ($t > 4$).

However, a wave continues to propagate along the tube even after the piston has stopped moving.

Figure: Some stills from the movie on Page 115. The $n$th picture is at the time $t = n$. The graph at the bottom shows the wave intensity at the final time ($t = 12$).
A simple example of an isolated wave (ctd): Observations

- The wave intensity of the wave in the figure on Page 116 is only non-zero over front and back of the wave (the expanding and contracting parts of the tube).
- In the middle of the wave (the part of the tube with constant expansion) the velocity is zero and pressure is constant (at a higher value), so $dU = 0$ and $dP = 0$ and hence $dI = 0$.
- Both the compression wavefront at the front of the wave and the decompression wavefront at the back have positive wave intensities, indicating that they are forward waves.
- The velocity is positive in the compression wavefront and negative in the decompression wavefront.
- Therefore, from the point of view of a fluid particle that starts at $x = x_0$ in the tube (the Lagrangian viewpoint):
  - $t < x_0 / c$: the particle is stationary as the wave has not reached it yet.
  - $x_0 / c < t < (x_0 / c) + 1$: the particle is in the compression wave and moves forwards.
  - $(x_0 / c) + 1 < t < (x_0 / c) + 3$: the particle is in the middle of the wave and is stationary.
  - $(x_0 / c) + 3 < t < (x_0 / c) + 4$: the particle is in the decompression wave and moves back to its original position.
  - $t > (x_0 / c) + 4$: the wave has passed and the particle remains stationary back in its original position.

Note that the movement of the wave and the movement of the fluid are very different.
The water hammer equations

A simple but important relationship between the change in pressure $dP$ and the change in velocity $dU$ in a wavefront can be derived from the method of characteristics solution:

- The Riemann invariants $R_\pm$ are constant as we move along the forward and backward moving characteristics respectively.
- Therefore $dR_\pm = dU_\pm \pm dP_\pm/(\rho c) = 0$ on the respective characteristics.
- Hence

$$dP_+ = \rho c \, dU_+ \text{ for forward waves, } \quad dP_- = -\rho c \, dU_- \text{ for backward waves.} \quad (86)$$

These are the water hammer equations.

The water hammer equations show that the pressure and velocity waveforms in the arteries are not independent of each other as is often thought. In unidirectional waves there is a simple linear relationship between $P$ and $U$. 
The water hammer equations: alternative derivation

Figure: Control volume used in the alternative derivation of the water hammer equations in stationary coordinates (left) and in coordinates moving with the wave, shown for the forward moving wave only.

Exercise: Derive the water hammer equations by applying mass and momentum conservation (see Page 17) to a control volume of the fluid that is moving with the wavespeed (see the diagram). In the moving coordinates, the wave is fixed and the fluid velocity is steady (independent of time).
Wave separation

- We assume that wavefronts are additive, that is \(dP = dP_+ + dP_-\) and \(dU = dU_+ + dU_-\).

- Using these equations and the water hammer equations (86), the forward and backward waves can be found in terms of the measured waves:

\[
dP_+ = \frac{1}{2} (dP + \rho c dU), \quad dP_- = \frac{1}{2} (dP - \rho c dU),
\]
\[
dU_+ = \frac{1}{2\rho c} (dP + \rho c dU), \quad dU_- = \frac{1}{2\rho c} (dP - \rho c dU).
\]

- Now if we are given starting values of the pressure, \(P_\pm|_{t=0} = P_{\pm,0}\), and velocity \(U_\pm|_{t=0} = U_{\pm,0}\), we may find their values at time \(t\) by summing the incremental differences in pressure and velocity, that is

\[
P_\pm = P_{\pm,0} + \sum dP_\pm, \quad U_\pm = U_{\pm,0} + \sum dU_\pm.
\]

- In the cardiovascular system:
  - We may take \(P_{\pm,0}\) to be the diastolic pressure.
  - During late diastole, the velocity in the arteries is usually near zero, so we take \(U_{\pm,0}^{12}\).

Using the pressure and velocity waveforms measured from an experiment, the relationships (87), (88) and (89) allow us to separate these into the forward and backward waves.

\footnote{This is not true in vessels such as the carotid arteries and the umbilical arteries during pregnancy, as there is usually a positive velocity throughout the cardiac cycle.}
Results of performing wave separation

**Figure**: Black curves show measured pressure (top) and velocity (bottom) in the human ascending aorta. The pressures and velocities of the separated forward and backward waves are also shown (blue – forward; red – backward).

- As required by the water hammer equations, the forward pressure and velocity differ only by a constant scale factor $\rho c$, and similarly for the backward pressure and velocity.
- For both pressure and the velocity, the forward and backward waveforms sum to give the measured waveform.
- Early in *systole* the backward waves are effectively zero, indicating there are no reflections in the ascending aorta, and the only waves are due to the contracting ventricle. After about 60 ms the backward waves start to become significant, although they are never larger than the forward wave.
Results of performing wave separation (ctd)

- During diastole (see figure):
  - The pressure and the separated forward and backward pressures all fall back to their starting values.
  - The velocity is almost zero, but the magnitude of the forward and backward velocities remain fairly large.
  - This shows we have relatively large, but self-cancelling forward and backward waves during this portion of the cycle.

However, the aortic valve is closed, so the aorta is cut off from the left ventricle and there is no forcing to drive new waves. It is difficult to find a reasonable explanation for the existence for the self-cancelling waves. A possible resolution is the reservoir–wave hypothesis.

- The difference between the waveform of the forward waves and that of the backward waves in the arteries occurs because their sources behave differently. Forward waves are mostly due to the heart, but backward waves are due to reflections of forward waves from different sites in the arteries and in the microcirculation.\(^{13}\)

\(^{13}\)There are also forward waves that are re-reflections of backward reflected waves and backward waves that are re-re-reflections of re-reflected forward waves, etc.
The incremental wave intensity for the forward and backward waves is

\[ dl_\pm = dP_\pm dU_\pm = \pm \frac{1}{4\rho c} (dP \pm \rho c dU). \]  

(90)

The measured wave intensity is given by the sum of the forward and backward wave intensities: \( dl = dl_+ + dl_- \)

Hence the wave intensity is

\[ l_\pm = l_{\pm,0} + \sum dl_\pm \]  

(91)
Reservoir–wave hypothesis

Figure: Black curves show measured pressure (top) and velocity (bottom) in the human ascending aorta. The pressures and velocities of the separated forward and backward waves are also shown (blue – forward; red – backward).

- As mentioned, the prediction of self-cancelling waves during diastole seems unphysical, and here we propose a resolution. A possible resolution is to set

\[ P(x, t) = P_r(t) + P_w(x, t), \]  

(92)

where

- \( P_r \) is the reservoir pressure (spatially uniform throughout the arterial system), which acts like the Windkessel pressure, and
- \( P_w \) is the pressure driving the forward and backward waves.

To find the reservoir pressure, we fit an exponential curve to the falling pressure during diastole. The curve should have the form

\[ P = P_0 + P_1 e^{(t-t_n)/\tau}, \]

where \( t_n \) is the time when the aortic valve closes and \( P_0, P_1 \) and \( \tau \) are constants. Then \( \tau = RC \), where \( R \) is the resistance to flow of the capillaries and \( C \) is the compliance of the arterial system.
Reflection and transmission of waves

In the derivations in this section, we assumed that the flow is one-dimensional. However, this is not the case at sites such as bifurcations or points where the cross-sectional area of the pipe changes suddenly (for example a sudden expansion of the pipe).

At sudden changes of cross-sectional area, part of the wave is reflected and part is transmitted. At bifurcations, part of the wave is reflected and part is transmitted along each of the daughter arteries.

For each site of discontinuity, we calculate reflection and transmission coefficients. These coefficients govern how an incident wave is split up into reflected and transmitted waves after it has passed through the bifurcation.

Most bifurcations are well matched for forward waves, meaning that the reflection coefficients, and hence the magnitudes of the reflected waves are small.

This necessarily means the bifurcations are not well matched for backward waves, which means that large-amplitude waves propagate all the way down the generations of blood vessels, but they do not propagate back up to the root. Thus the waves tend to remain in the small vessels with hardly any in the large vessels. This phenomenon is known as wave trapping.
Determination of the wavespeed: Foot-to-foot measurements

Three methods have been proposed to determine the wavespeed, and here we discuss each in turn. The first method uses foot-to-foot measurements:

The foot of the pressure waveform is given by the delay between the start of systole and the time when the pressure begins to rise.

Assuming there are no waves in the system at the end of diastole, the foot is the time when the first forward wave reaches the site of measurement.

The delay equals the distance along the aorta divided by the wavespeed.

Therefore the foot has a linear relationship to the distance along the aorta, and the constant of proportionality is the wavespeed.

In this way the wavespeed can be estimated from measurements such as those illustrated in the figure.

Figure: Blood pressure measurements over the cardiac cycle taken at six fixed sites that are spaced along the aorta with 10 cm between neighbouring sites.
Determination of the wavespeed: The $PU$-loop

The second method is graphical and is based on the graph of pressure against velocity taken at a fixed site.

- During early systole almost all the waves present in the system are forward waves, because the waves from the heart have not had time to reflect from the peripheral circulation.
- Therefore the pressure and velocity are given by the forward waves only: $P = P_+$ and $U = U_+$.
- Substituting into the water hammer equation, we obtain $dP = \rho c \, dU$, and hence $P = P_0 + \rho c U$.
- Hence the graph of $P$ against $U$ should be a straight line with slope $\rho c$.
- Therefore, by measuring $P$ and $U$ and finding the slope of the line in early systole gives the wavespeed (since $\rho$ is known).
- The time of first departure from linearity gives the first time at which the reflected waves return from the peripheral circulation.
- This method will fail if there are backward waves in the system, which may occur if not all the waves decayed fully during diastole, or if there is a reflection site close to the site of measurement.
Determination of the wavespeed: The *PU*-loop

**Figure:** Left: *PU*-loop for a ‘normal’ and an ‘occluded’ case. Right: Corresponding pressure traces. Note the linear portion of the *PU*-loop, marked by dashed lines, from where it is possible to make an estimate of the wavespeed.
Determination of the wavespeed: The sum-of-squares method

The third method is graphical and is based on the graph of pressure against velocity taken at a fixed site.

- The method is based on the hypothesis that the system minimises the wave intensity.
- Using this assumption we may show that

\[ c = \frac{1}{\rho} \sqrt{\frac{\sum dP^2}{\sum dU^2}}. \] (93)

This formula may be used to calculate \( c \) from experimental measurements.

- The method can be shown to be accurate if the forward and backward waves are uncorrelated, that is \( \sum dU_+ dU_- = 0 \).


