Flows through deformable airways*

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1 Introduction

Most of the vessels that carry fluids (liquids or gases) around the body are deformable. Obvious examples are veins and arteries, the ureter and urethra, and the gut.

The intra-thoracic airways of the lung are no exception. The airways are arranged as a bifurcating network, with each parent airway splitting roughly symmetrically into two daughter airways at each bifurcation. In humans, there are roughly 23 generations of bifurcations from the largest airway (the trachea) down to the terminal air units (the alveoli, of which there are around 300 million in an adult lung). The lungs fit tightly inside a space confined by the rib-cage and diaphragm. On inspiration, active (muscle-driven) expansion of this space leads to expansion of the airways, which draws air into the lungs. On expiration, muscles relax and the lung typically contracts passively via its natural recoil, expelling air. Air can also be actively pushed out of the lungs (for example during the respiratory manoeuvre called *forced expiration*).

The flexibility of airways has a number of physiologically significant effects. We will discuss two of these in detail.

- *Flow limitation.* During forced expiration, active contraction of the ribs and diaphragm has two effects. First, it elevates alveolar pressure, providing a pressure gradient from alveoli to mouth that drives air out of the lungs. Second, it also elevates pressure in the tissues outside the airways. This causes the airways to partially collapse, which can inhibit expiratory flow. The net effect is that increasing the effort of expiration can, at a given lung volume, limit the maximum possible flow rate and possibly lead to a reduction in expiratory flow rate (this is known as *negative effort dependence*). Flow limitation can be a serious problem for someone having an asthmatic attack: they can have more difficulty getting CO_2 out of their lungs than getting O_2 in.

- *Wheezing.* One of the few non-invasive techniques for assessing lung function is to listen to the sounds generated during breathing. A forced expiration, for example, is a noisy event. Forcing air through a deformable tube can generate spontaneous oscillations of the airway walls (rather like a flag flutters in the wind), leading to audible wheezing. Other familiar sounds include those made in the upper airways during obstructive sleep apnoea (snoring) and crackles associated with the popping of liquid plugs in obstructed airways.

In these lectures we will examine some simple mathematical models of the physical phenomena underlying these effects, and some of the general properties of flows through deformable tubes.

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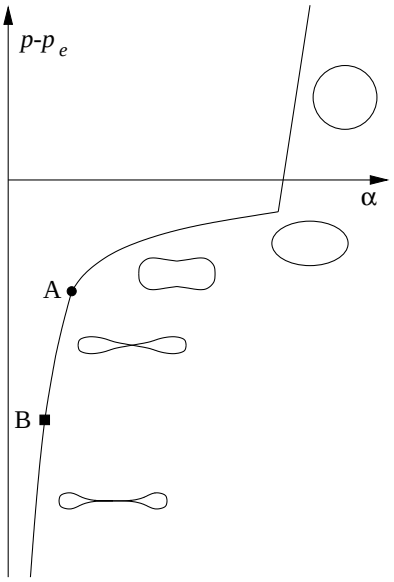

Figure 1: The relation between transmural pressure $p_{tm} = p - p_e$ and cross-sectional area α, showing typical tube cross-sections.

2 Deformable tubes

A typical lung airway has a complex internal structure, including an almost inextensible basement membrane and epithelium, a ring of smooth muscle, and tethering to surrounding tissue (Kamm 1999). It is helpful therefore to understand the behaviour of a simple physical analogue, namely a thin-walled elastic tube.

2.1 The Tube Law

Consider a long elastic tube subject to variable transmural (internal minus external) pressure $p_{tm} = p - p_e$. The relationship between p_{tm} and the cross-sectional area α is shown in Fig. 1. When $p_{tm} > 0$, the tube has circular cross-section, and is under an extensional hoop stress. A tube is typically very stiff in this state (the cross-sectional area changes very little as p_{tm} increases). If p_{tm} is reduced below 0, the tube initially remains circular, but is now under compression. At a critical pressure, the tube buckles, initially to an elliptical cross-section. The compression is now balanced by bending stresses in the most highly curved parts of the tube wall. In this state the tube becomes highly compliant: small reductions in p_{tm} lead to large reductions in cross-sectional area. As p_{tm} is reduced further, the opposite walls of the tube come into contact first at a point, and then along a line (points A and B in Fig. 1 respectively). Thereafter, the tube forms two distinct lobes in which bending stresses are large, and further area reductions are difficult.

We have here neglected tethering, or finite length effects, which can cause the tube to buckle into more than two lobes. Lung airways, for example, which have thick, inhomogeneous walls surrounding an inextensible epithelium, can buckle into a large number of lobes.

The calculation of the tube law for an axially-uniform thin-walled elastic tube (effectively an inextensible ring that resists bending) is given using Euler–Bernoulli beam theory in Flaherty, Keller & Rubinow (1972).

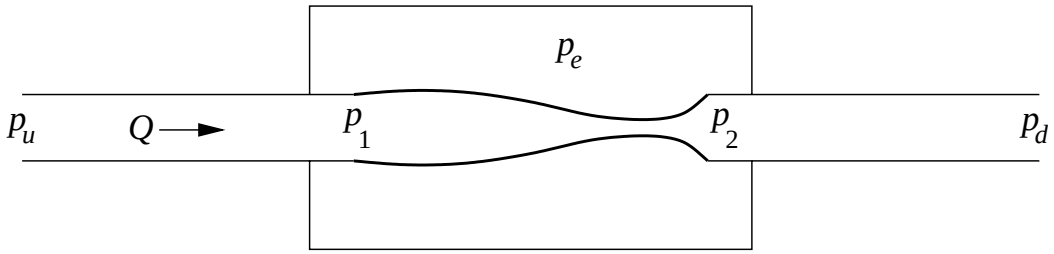

Figure 2: The Starling Resistor

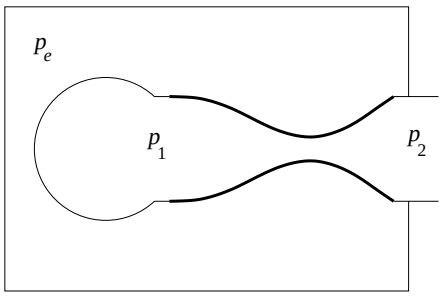

Figure 3: A collapsible-tube model of the lung

2.2 The Starling Resistor

Physiologists commonly use the device shown in Fig. 2, known as the Starling Resistor, as a simple bench-top model of a deformable airway. An elastic tube is mounted between two rigid tubes, and a flow of air or water with volume flux Q is driven through the system. The experimentalist can control p_e, the pressure outside the elastic tube, and one or other of p_1 and p_2, the pressures at the upstream and downstream ends of the elastic tube, by altering the pressures at either end of the apparatus p_u and p_d.

An analogous (and crude) model for the lung is shown in Fig. 3. The intra-thoracic airways are lumped into a single compliant tube, possibly having axially varying material properties. The alveoli, which are effectively elastic-walled sacs, are represented by a balloon attached to this tube at its upstream end. p_1 corresponds to alveolar pressure, and p_e to the pleural pressure in the tissues surrounding the airways. Fixing $p_1 - p_e$ corresponds to fixing the elastic recoil of the lungs, i.e. fixing the lung volume. p_2 corresponds to atmospheric pressure at the mouth.

Simulating a forced expiration at fixed lung volume using a Starling Resistor is therefore achieved by increasing $p_1 - p_2$, keeping the upstream transmural pressure $p_1 - p_e$ fixed. We find that the relation between the driving pressure $p_1 - p_2$ and the steady flow Q is as shown in Fig. 4(a). Increasing the pressure drop initially causes Q to increase: here, typically, $p_1 > p_2 > p_e$ and the tube is everywhere inflated. However, increasing $p_1 - p_2$ (for example by reducing p_2) leads to a reduction in $p_e - p_2$, which causes the tube to collapse at its downstream end (with $p_1 > p_e > p_2$). Collapse can then lead (surprisingly) to a reduction in flow-rate as $p_1 - p_2$ rises. This mimics *negative effort dependence*. Since Q cannot increase pass some threshold, this is an example of *flow-limitation*.

Other protocols with the Starling Resistor also yield nonlinear pressure-drop/flow-rate relations. Increasing $p_1 - p_2$ while holding $p_2 - p_e$ constant leads to *pressure-drop limitation* (i.e. a limit on the maximum possible pressure drop, Fig. 4(b)). And in a famous experiment (Fig. 4(c)), Conrad (1969) held p_e and the downstream outlet pressure p_d fixed, as well as the resistance of a valve in the downstream rigid segment, so that p_2 increased with the flux Q according to $p_2 = kQ^2 + p_d$. In this case,

- initially $p_e > p_1 > p_2$: the tube is collapsed along its entire length, offers a high resistance to

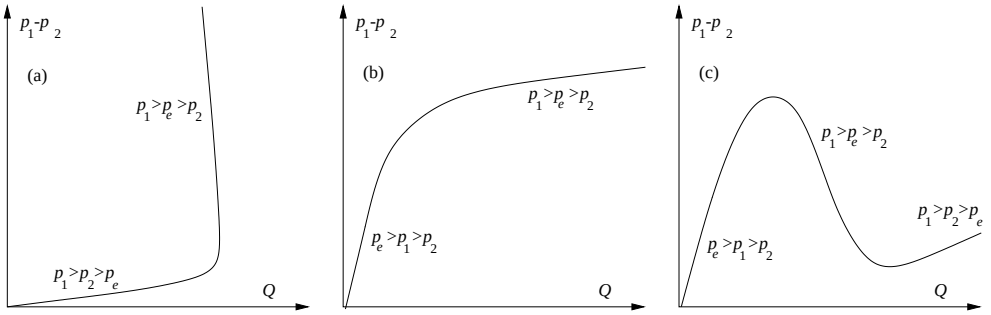

Figure 4: $p_1 - p_2$ versus Q with (a) $p_1 - p_e$ fixed, (b) $p_2 - p_e$ fixed, (c) Conrad's protocol. Time-averaged quantities are shown in cases when the flow becomes unstable.

flow, and $p_1 - p_2$ rises steeply to produce a small increase in Q; as Q increases, so do p_1 and p_2 because of the pressure drop across the valve;

- then $p_1 > p_e > p_2$: the tube inflates at its upstream end remaining collapsed downstream, the resistance to flow falls dramatically and $p_1 - p_2$ falls as Q increases;
- finally $p_1 > p_2 > p_e$: the tube is uniformly inflated, offers low resistance to flow and large increases in Q are provided by small increases in $p_1 - p_2$.

In practice, obtaining a steady pressure-drop/flow-rate curve is difficult when the flow is rapid (at large *Reynolds numbers*), because the Starling Resistor is prone to instabilities. Because the tube is highly compliant when it is partially collapsed (particularly when $p_1 > p_e > p_2$), small changes in the flow can lead to large changes in tube shape. The flow is strongly coupled to the shape of the wall, so shape changes lead to further flow changes. This flow-structure interaction can generate instabilities through a variety of mechanisms, that we shall explore below. Experimental measurements of the instabilities reveal a remarkably rich range of oscillations, characteristic of a complex nonlinear dynamical system. For reviews see Kamm & Pedley (1989), Pedley & Luo (1998) and Heil & Jensen (2002).

We will now examine some simple mathematical models for steady and unsteady flows in deformable tubes. While airways provide the primary motivation, these models can also be usefully applied to other systems such as blood vessels.

3 Governing equations

While early models of flow in the Starling Resistor were based upon ODEs (describing the behaviour of the system in terms of variables such as $p_1(t)$, $p_2(t)$, $Q(t)$ and $A(t)$ (the minimum area)), a more useful framework for our purposes is in terms of spatially one-dimensional models. We therefore introduce $\alpha(x,t)$, $u(x,t)$ and $p(x,t)$ as our primary dependent variables, where x measures distance along the tube and t is time. α is the tube's cross-sectional area, u the cross-sectionally averaged axial velocity and p the cross-sectionally averaged internal pressure. We need three governing equations for these three variables.

- Mass conservation: this is given by

$$\alpha_t + (u\alpha)_x = 0, \tag{3.1}$$

where subscripts x and t denote space and time derivatives. We can understand (3.1) by considering a short section of tube, between x and $x + \delta x$, where $\delta x \ll 1$. Any difference between the

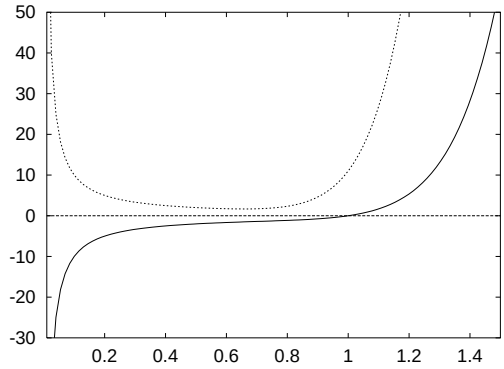

Figure 5: $\mathcal{P}(\alpha)$ (solid) and $\alpha P'(\alpha)$ (dashed) plotted versus α, with $\mathcal{P}$ given by (3.5).

volume fluxes $q = u\alpha$ entering and leaving this slice of tube must be accommodated by a change in the slice's volume, i.e.

$$(\alpha \delta x)_t = q(x,t) - q(x+\delta x, t). \tag{3.2}$$

In the limit $\delta x \to 0$, (3.2) reduces to (3.1). For steady flows, the flux $q(x)$ must be uniform along the length of the tube.

- Momentum conservation: this is given by

$$\rho(u_t + uu_x) = -p_x - R(u,\alpha), \tag{3.3}$$

where ρ is the fluid density (assumed constant) and $R > 0$ is a term representing frictional (viscous) effects. A formal derivation of (3.3) from the governing Navier–Stokes equations of fluid mechanics is not possible in general. However, (3.3) serves as a good model equation, provided R is chosen appropriately. It is basically an expression of $F = ma$ for a material slice of fluid. The term $u_t + uu_x$ is the fluid's acceleration (accounting for the fact that the slice can accelerate if it is swept into a region of faster moving flow, i.e. where $u_x > 0$). The force on the slice arises from the difference in pressures acting on either face ($p(x,t) - p(x+\delta x, t) \approx -\delta x p_x$), together with viscous resistance per unit length R.

- Pressure-area relation: this is given by

$$p - p_e = \mathcal{P}(\alpha). \tag{3.4}$$

Here, $\mathcal{P}(\alpha)$ is a nonlinear function representing the static pressure-area relation for a uniform tube, such as that shown in Fig. 1. While $\mathcal{P}$ can be computed using thin-shell theory, it is simpler to use an approximate function such as

$$\mathcal{P}(\alpha) = \left(\frac{\alpha}{\alpha_0}\right)^n - \left(\frac{\alpha}{\alpha_0}\right)^{-m} \tag{3.5}$$

where (for example) $n = 10$ and $m = \frac{3}{2}$. This satisfies $\mathcal{P}(\alpha_0) = 0$, where α_0 is the cross-sectional area when the tube is unstressed. $\mathcal{P}(\alpha)$ and $\alpha\mathcal{P}'(\alpha)$ in this case are shown in Fig. 5: the tube is very stiff when $\alpha > \alpha_0$ or as $\alpha \to 0$, but is highly compliant (low $\mathcal{P}'(\alpha)$) in between. Additional terms may be added to (3.5) to represent additional physical effects: for example

$$p - p_e = \mathcal{P}(\alpha) - T\alpha_{xx} + D\alpha_t + M\alpha_{tt}. \tag{3.6}$$

Here $T \geq 0$ represents in an *ad hoc* manner the effects of longitudinal tension; α_{xx} approximates the axial component of curvature of the tube. $D \geq 0$ corresponds to viscous damping in the wall, and $M \geq 0$ accounts for wall inertia (important for air flows, since the wall-mass of a length of thin-walled tube can be comparable to the mass of the air within it).

There are important analogies between (3.1, 3.3, 3.4) and the equations of compressible gas flow in a nozzle and the equations of shallow-water flow (Shapiro 1977). Features found in these systems (such as shocks and hydraulic jumps) are manifested also in collapsible tubes.

4 Models for steady flow

4.1 Choking

We can now explore steady flows governed by (3.1, 3.3, 3.4). For the present we take R to be a constant and set $T = M = D = 0$. Thus $q = u\alpha$ is uniform along the tube, and

$$\rho u u_x = -\mathcal{P}'(\alpha)\alpha_x - R. \tag{4.1}$$

It is convenient to define $c(\alpha)$ by

$$c = \sqrt{\frac{\alpha \mathcal{P}'(\alpha)}{\rho}}; \tag{4.2}$$

ρc^2 is plotted in Fig. 5. We shall see below that c is the wave-speed of small-amplitude pressure waves that can propagate along a uniform tube. We can then re-express (4.1) as

$$\rho(u^2 - c^2)u_x = -Ru. \tag{4.3}$$

We can distinguish two types of behaviour. If $0 < u < c$, so-called *subcritical* flow, $u_x > 0$, implying the flow accelerates and (since $q = u\alpha$ is uniform) the tube constricts as x increases. For $u > c$, so-called *supercritical* flow, $u_x < 0$, the flow decelerates and the tube expands as x increases. The precise behaviour of the tube depends on the form of the tube law. If we suppose for simplicity that c is constant (implying $\mathcal{P}(\alpha) = \rho c^2 \log(\alpha/\alpha_0)$), then we can integrate (4.3) to give

$$\tfrac{1}{2}\rho u^2 - \rho c^2 \log u = -R(x - x_0), \tag{4.4}$$

which is sketched in Fig. 6. For any initial condition, we see that the steady solution terminates at a finite value of x provided the tube is sufficiently long. This is called *choking*.

4.2 Non-uniform tubes

How, then, is it possible to have a steady flow in a long tube?

First, to obtain a transition from subcritical to supercritical flow we need to introduce an additional effect. In our simple lung model (Fig. 3), for example, this is provided by non-uniform material properties. Both the stiffness of the airway wall and the unstressed area α_0 may vary with position. To illustrate, assume $\mathcal{P} = K(\alpha - \alpha_0)$ where $\alpha_0 = \alpha_0(x)$ and K is constant. (We see from (3.4) that having variable $\alpha_0(x)$ is equivalent to having variable p_e, so that we can imagine changes in α_0 being provided by a cuff placed around an otherwise uniform tube). We then have

$$\rho u u_x - \frac{Kq}{u^2}u_x = K\alpha_{0x} - R. \tag{4.5}$$

To simplify the problem, suppose $R = 0$. Now (4.5) becomes

$$\rho\frac{u_x}{u} = \frac{K\alpha_{0x}}{(u^2 - c^2)}. \tag{4.6}$$

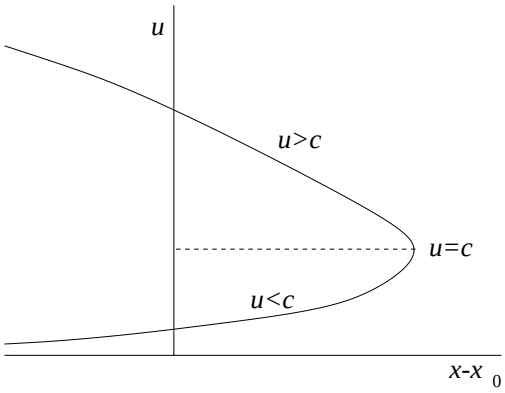

Figure 6: The solution of (4.4).

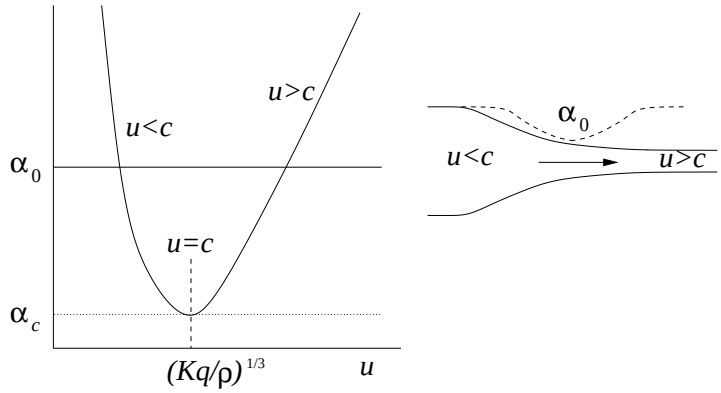

Figure 7: The graph shows how the solution of (4.7) is determined.

To have a smooth transition between sub- and supercritical flow, we must have $\alpha_{0x} = 0$ where $u = c$. We can for example imagine flow through a tube with a localised constriction (so that α_0 has a local minimum, as illustrated in Fig. 7) with $0 < u < c$ upstream of the constriction and $u > c$ downstream. We can then integrate (4.5) to get

$$\tfrac{1}{2}\rho u^2 + \frac{Kq}{u} = K\alpha_0(x). \tag{4.7}$$

The solution of (4.7) is illustrated graphically in Fig. 7. Where the flow is subcritical, u increases as α_0 decreases; a smooth transition to supercritical flow can occur provided $u = c$ where $\alpha_{0x} = 0$, so $\alpha_{0\,\min}$ must equal

$$\alpha_c \equiv \tfrac{3}{2}(K\rho^2 q)^{1/3}, \tag{4.8}$$

the minimum value of the function on the left-hand-side of (4.7); the flow is then supercritical as α_0 rises again. If $\alpha_{0\,\min} > \alpha_c$ then the flow is everywhere subcritical; if $\alpha_{0\,\min} < \alpha_c$ there is no steady solution for these parameter values and the flow *chokes*.

Shapiro (1977) gives an exhaustive survey of such transitions.

4.3 Elastic Jumps

If we re-introduce friction, our supercritical solution may still terminate at finite x (as in Fig. 6), unless we can somehow return to a subcritical state. This is accommodated through an *elastic jump*. This is an abrupt transition from super- to subcritical flow, represented by a shock-like solution of the governing equations. The conditions for the transition across the jump are statements of mass and momentum

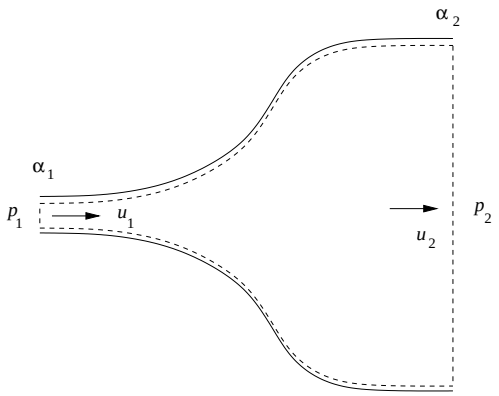

Figure 8: An elastic jump.

conservation; energy is dissipated within the jump, for example by flow separation and turbulence. Assuming the tube law can be applied throughout the jump, we have (see Fig. 8)

$$u_1\alpha_1 = u_2\alpha_2 \tag{4.9}$$

$$\alpha_1(p_1 + \rho u_1^2) + \int_{\alpha_1}^{\alpha_2} \mathcal{P}\,\mathrm{d}\alpha = \alpha_2(p_2 + \rho u_2^2). \tag{4.10}$$

Here terms of the form αp represent the horizontal force exerted by pressure on the dotted control volume. The terms $\rho\alpha u^2$ represent the momentum flux (the rate at which momentum enters and leaves the control volume).

Writing $q = u_1\alpha_1$, the momentum condition can be re-expressed using integration by parts to give the condition that

$$\Phi = \frac{\rho q^2}{\alpha} + \int^{\alpha} a\mathcal{P}'(a)\,\mathrm{d}a \tag{4.11}$$

has equal values either side of the jump.

Using this framework, sophisticated models for flow limitation during forced expiratory flow in the lung have been developed (e.g. Elad, Kamm & Shapiro 1987). Spatial non-uniformities allow a transition from sub- to supercritical flow, and further downstream an elastic jump allows a transition from super- to subcritical flow; its position is chosen to allow the correct pressure at the mouth to be accommodated.

5 Models for unsteady flow

We have seen that it is possible for steady flows to break down via choking, which reflects the non-existence of a steady flow for certain parameter values. Steady flows, if they exists, can also lose stability to oscillatory instabilities.

5.1 Wave propagation

Equations (3.1, 3.3, 3.6) form a closed system from which we can determine how pressure, speed and area are related during the propagation of a wave along a collapsible tube. To do so, we set $R = 0$ (ignoring viscous effects) and then linearise the governing equations, restricting attention to small-amplitude disturbances. We set

$$\alpha = \alpha_0 + \alpha_1 + \ldots, \quad u = u_0 + u_1 + \ldots, \quad p = p_e + p_1 + \ldots,$$

where α_0, u_0 and p_e are uniform and $|\alpha_1| \ll \alpha_0$, etc. Dropping terms that are quadratic in small quantities, we have

$$\alpha_{1t} + u_0\alpha_{1x} + \alpha_0 u_{1x} = 0 \tag{5.1a}$$

$$\rho(u_{1t} + u_0 u_{1x}) = -p_{1x} \tag{5.1b}$$

$$p_1 = \mathcal{P}'(\alpha_0)\alpha_1 - T\alpha_{1xx} + D\alpha_{1t} + M\alpha_{1tt}. \tag{5.1c}$$

Writing $\mathcal{P}'(\alpha_0) = \rho c_0^2/\alpha_0$, we can eliminate p_1, and then set

$$u_1 = \mathrm{Re}\left(\hat{u}_1 e^{i(kx-\omega t)}\right), \quad \alpha_1 = \mathrm{Re}\left(\hat{\alpha}_1 e^{i(kx-\omega t)}\right)$$

for some complex amplitudes $\hat{u}_1$, $\hat{\alpha}_1$, where Re denotes 'real part.' This representation describes wave-like disturbances with wavenumber k (assumed real), wavelength $2\pi/k$, and wave crests that propagate with speed ω/k. At a fixed location, disturbances are oscillatory functions of time with angular frequency ω (if it is real) and period $2\pi/\omega$. We seek ω as a function of k. Since

$$\exp\left[i(kx - \omega t)\right] = \exp\left[i(kx - \mathrm{Re}(\omega)t)\right]\exp\left[\mathrm{Im}(\omega)t\right],$$

waves will grow or decay in time if $\mathrm{Im}(\omega) > 0$ or < 0 respectively.

Noting that time derivatives of u_1 and α_1 equate to multiplication by $-i\omega$ and space derivatives to multiplication by ik, (5.1) reduces to

$$\begin{pmatrix} u_0 k - \omega & k\alpha_0 \\ (kc_0^2/\alpha_0) + (Tk^3/\rho) - (i\omega Dk/\rho) - (\omega^2 Mk/\rho) & u_0 k - \omega \end{pmatrix} \begin{pmatrix} \hat{\alpha}_1 \\ \hat{u}_1 \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \end{pmatrix},$$

so that a necessary condition for a solution to exist is that the matrix has zero determinant. This yields the following complex quadratic equation for ω as a function of k:

$$(u_o k - \omega)^2 - c^2k^2 - k^4\frac{\alpha_0 T}{\rho} + i\omega\frac{Dk^2\alpha_0}{\rho} + \omega^2\frac{Mk^2\alpha_0}{\rho} = 0. \tag{5.2}$$

We can examine a number of special cases of this *dispersion relation*. When $T = D = M = 0$, we have

$$\omega = (u_0 \pm c_0)k. \tag{5.3}$$

Neutrally stable waves therefore propagate with phase speed $\omega/k = u_0 \pm c_0$. As expected, c_0 is the speed at which small-amplitude pressure waves propagate down a uniform elastic tube in the absence of a mean flow. In subcritical flow ($0 < u_0 < c_0$), waves travel upstream (with speed $c_0 - u_0$) and downstream (with speed $c_0 + u_0$). In supercritical flow ($u_0 > c_0$), all waves travel downstream. This helps us understand how flow limitation in the lung arises: if there is a region of supercritical flow, then reducing the downstream pressure need not be felt upstream since disturbances cannot propagate upstream through the region of supercritical flow. Changes may instead be accommodated by changes in the location of an elastic jump, for example.

Setting $T > 0$ (with $D = M = 0$), we find that

$$\frac{\omega}{k} = u_0 \pm \sqrt{c_0^2 + \frac{T\alpha_0 k^2}{\rho}}. \tag{5.4}$$

We see that waves of different wavelengths travel at different speeds, a phenomenon known as *dispersion*. In particular, short waves (with large k) can in principle propagate upstream through a region of supercritical flow. Tension alone is not adequate to prevent choking from occurring in some situations, however. While it must be included in distributed 1D models of flow in the Starling Resistor (Fig. 2), in order that the system is of sufficiently high order to impose boundary conditions on both pressure and

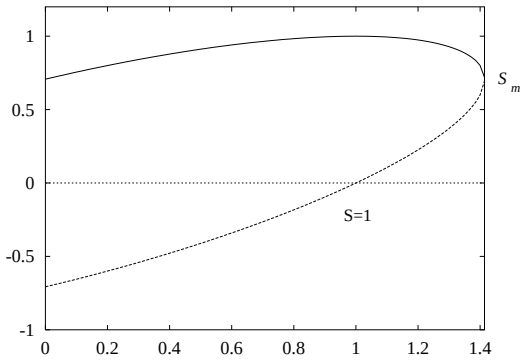

Figure 9: The real part of (5.7) for $\kappa = 1$.

height at either end of the apparatus, steady inviscid flow through this system can still choke (the tube area at some point goes to zero in finite time) if critical conditions are exceeded (Pedley 2000; Heil & Jensen 2002). This is an example of a so-called *static-divergence instability*: it has a direct explanation in terms of the Bernoulli effect: at a constriction in the tube, the speed rises (by mass conservation), the pressure falls (by Bernoulli) and so the constriction narrows further, via a direct instability.

When $D > 0$ (with $T = M = 0$), we expect that $\mathrm{Im}(\omega) < 0$, so that waves decay in time.

When $M > 0$ (with $T = D = 0$), then we find

$$\frac{\omega}{k} = \frac{u_0 \pm \sqrt{u_0^2 - (1 + (Mk^2\alpha_0/\rho))(u_0^2 - c_0^2)}}{1 + (Mk^2\alpha_0/\rho)}. \tag{5.5}$$

We see in this case the possibility that $\mathrm{Im}(\omega > 0)$, so that waves of fixed wavelength grow in time. For $0 < u_0 < c_0$ and $Mk^2\alpha_0/\rho \gg 1$ we see that there exists a root with

$$\mathrm{Im}(\omega) \approx \left[\frac{\rho(u_0^2 - c_0^2)}{M\alpha_0}\right]^{1/2} > 0, \tag{5.6}$$

for example. More generally, set $u_0 = Sc_0$ and $\kappa^2 = Mk^2\alpha_0/\rho$. Then

$$\frac{\omega}{c_0 k} = \frac{S \pm \sqrt{S^2 - (1+\kappa^2)(S^2 - 1)}}{1 + \kappa^2}; \tag{5.7}$$

the real part of this quantity is plotted in Fig. 9 for $\kappa = 1$. For $0 < S < 1$, neutrally stable waves propagate both upstream and downstream, at speeds no greater than c_0 because the wall increases the overall inertia in the system. For $1 < S^2 < S_m^2 \equiv (1 + \kappa^2)/\kappa^2$, waves propagate downstream only (which may lead to flow limitation). For $S > S_m$, there exists a wave with $\mathrm{Im}(\omega) > 0$ which grows in time, leading to instability.

The mean flow u_0 can therefore induce *travelling wave flutter* (TWF) in a flexible tube or channel, much like a flag flaps in the wind. Surprisingly, introducing wall damping D changes this instability to one of static divergence, in which the tube collapses through a non-oscillatory instability (Grotberg & Davis 1980). Introducing the effects of viscous dissipation in the fluid (via a term $-R = -\hat{R}u$ in (5.1b), for example) restores the oscillatory nature of the TWF instability, however (Grotberg & Reiss 1984).

TWF is a likely candidate for the generation of wheezing noises in forced expiration. It has been suggested that flutter is a signature of flow limitation, but the evidence supporting this is inconclusive (Grotberg 1994).

5.2 Other modes of instability

While TWF is a likely candidate for the generation of wheezing in lung airways, other modes of instability are observed in the Starling Resistor which could be important in both wheezing and the generation of snoring noises. For example, the Starling Resistor exhibits lower-frequency self-excited oscillations when the working fluid is water rather than air, in which case wall inertia is dominated by fluid inertia. One particular type of instability has recently been interpreted as a global mode of the whole apparatus, rather than a local mode such as we found for TWF (Heil & Jensen 2002). Much remains to be done to understand these oscillations in bench-top systems, yet more to relate them to realistic physiological conditions.

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