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SMR.378/17

WORKSHOP ON THEORETICAL FLUID MECHANICS AND APPLICATIONS

(9 - 27 January 1989)

PHYSIOLOGICAL FLOW AS A BRANCH OF FLUID MECHANICS

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PHYSIOLOGICAL FLOW as a branch of (1)

FLUID MECHANICS

i.e. seek an understanding of the forces which drive the motion of blood and other bodily fluids, and the motion that results from the forces. WHY?

(a) **DIAGNOSIS.** Interpret measurements of blood pressure, flow rate, heart sounds, breath sounds, etc.

(b) **CURE (BIOENGINEERING).**
Artificial heart valves, lungs, kidneys; coronary artery by-pass; cardiopulmonary resuscitation, etc.

(c) **PHYSIOLOGY (PURE SCIENCE).**
How animals work.

(d) **PATHO-PHYSIOLOGY.** Understand the origin and effects of disease.
E.g. ATHEROSCLEROSIS is associated with the force exerted by the blood on artery walls; ASTHMA and BRONCHITIS - excess work of breathing.

Flow systems to be discussed

CARDIOVASCULAR SYSTEM.
Blood flow in arteries, veins, arterioles, capillaries

RESPIRATORY SYSTEM
Airflow and gas mixing in the airways of the lung.

EXCRETORY SYSTEM
Mass transport between blood and urine in the kidney; peristaltic pumping along ureters to bladder; passive flow along urethra from bladder.

EPITHELIAL TRANSPORT
Proximal tubule in kidney
Gall bladder
Gut
Cornea,
Etc.

Newton's Law of Particle Motion

Mass x acceleration = Force
(inertia)

Fluid mechanics consists of the application of Newton's Law to every particle of a flowing fluid (gas or liquid).

The acceleration can be of two types:
- local (velocity of fluid at measuring point changes with time)
- convective (particle moves to a point where the velocity is different).

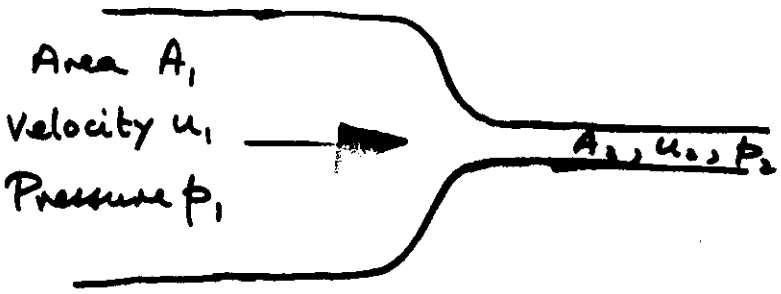
The forces are also usually of two types:
- pressure gradients (that drive the flow)
- viscous forces (that resist it)

(body forces such as gravity can be treated like pressure gradients).

Note that a fluid flows as if it were incompressible as long as $\frac{\text{fluid speed}}{\text{sound speed}} \ll 1$

Example

Steady flow in a constriction.
(Horizontal)



Conservation of mass: $u_1 A_1 = u_2 A_2$
(so $u_2 > u_1$)

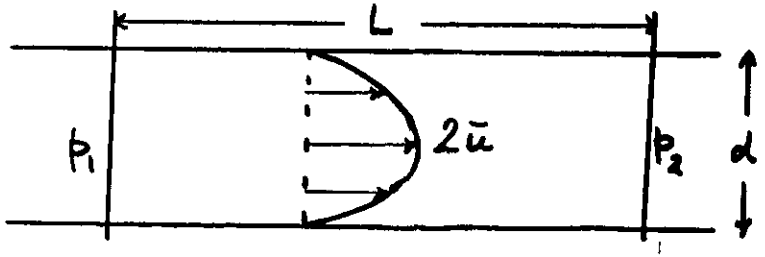
Consequences of Newton's Law
(Bernoulli's equation)

$$p_1 + \frac{1}{2}\rho u_1^2 = p_2 + \frac{1}{2}\rho u_2^2$$
(+ small viscous term)

(so $p_1 > p_2$)

ρ = fluid density

Steady flow in a long, straight, rigid tube, far from entrance.



No acceleration.

Parabolic velocity profile.

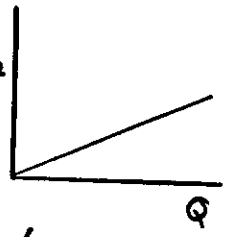
Volume flow rate $Q = \frac{\pi d^2}{4}\bar{u}$

Wall shear: $\tau = 8\bar{u}/d$

Pressure drop:

$$p_1 - p_2 = \frac{128\,\mu L Q}{\pi d^4}$$ Poiseuille

(μ = fluid viscosity)



Doesn't occur in any airways or blood vessels!

Arteries and airways are curved, branched, elastic and short, and the flow in them is unsteady. Thus steady, fully-developed flow in a long straight tube (Poiseuille flow) is irrelevant!

Moreover blood is a concentrated ($\approx 45\%$) suspension of deformable cells, mainly red blood cells. Fortunately, however, it behaves approximately as a Newtonian fluid ($\nu \approx 0.04\ cm^2\,s^{-1}$) in the arteries and veins. Only in the microcirculation must the non-Newtonian and inhomogeneous character of blood be taken into account.

Outline of lectures

A. Pulse wave propagation in arteries

B. Flow and wall shear stress in arteries

C. Pressure - flow relations in the airways

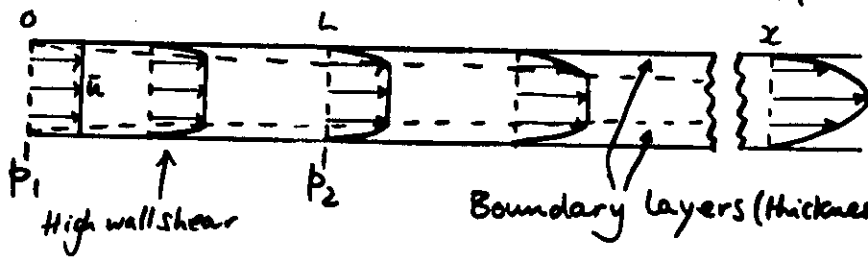
D. Flow in collapsible tubes.

Reference.

The Fluid Mechanics of Large Blood Vessels by T.J. Pedley (Cambridge University Press 1980)

Entry flow (steady, rigid)

$\delta \propto \left(\frac{\mu x}{\rho \bar{u}}\right)^{1/2}$



No slip at the walls, so fluid increasingly retarded in growing boundary layers.
Core flow accelerated (conservation of mass)

Entrance length: $x = 0.03\, d\, Re$

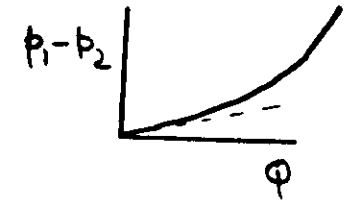
Reynolds number $\boxed{Re = \frac{\rho \bar{u} d}{\mu}}$ (ρ = fluid density)

Represents relative importance of inertia (convective accel.) and viscosity.
Only if $Re \ll 1$ does viscosity dominate.

Pressure drop: $p_1 - p_2 =$ [redacted]

(non-linear) $+ k_1 (\mu\rho)^{1/2} \frac{Q^{3/2} L^{1/2}}{d^4}$

$+ \frac{1}{Q}\int \frac{1}{2}\rho (u_2^3 - u_1^3)\, dA$



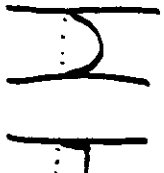
Oscillatory flow far from entrance (laminar)

Frequency f

Flow-rate $Q = Q_0 \sin(2\pi f t)$

Pressure drop $p_1 - p_2 = \Delta p_0 \sin(2\pi f t + \phi)$ (Δp_0: amplitude; ϕ: phase lead)

Small f: $\Delta p_0 = \frac{128 \mu L Q_0}{\pi d^4} = \Delta p_s$ (steady value)



$\phi = 0$

Quasi-steady flow.

Large f: $\Delta p_0 = \frac{\alpha^2}{8} \Delta p_s$, $\phi = 90°$

where α is the Womersley parameter:

$$\alpha = \frac{d}{2}\sqrt{\frac{2\pi f \rho}{\mu}}$$

"Small f": $\alpha \lesssim 4$. "Large f": $\alpha \gtrsim 4$

α is the ratio of the tube radius to the thickness of the oscillatory boundary layer. (see predicted and measured velocity profiles) Measures local accel. × mass / viscosity

R.M.S. wall shear ↑ as α ↑ for given Q.

Values of Re and α in blood vessels and airways DOG.

Vessel		Re	f(Hz)	α
Ascending aorta (canine)	mean	750	2	13
	max	4500		
Capillary		0.001	2	0.00!
Trachea (human) ($Q = 1\ \ell s^{-1}$)		4600	0.25	2.9
Terminal bronchiole		0.18	0.25	0.07

Thus:

Viscosity dominates inertia in the periphe of each system.

In large vessels inertia dominates, but only in arteries is the unsteadiness dynamically important. In airways the flow is quasi-steady.

Arteries are elastic.

Propagation of the pulse wave.

A balance between wall elasticity and blood inertia.

Simple theory predicts:

Wave speed c: $c^2 = \frac{A}{\rho}\frac{dp_{tm}}{dA}$

where A = cross-sectional area

ρ = blood density

p_{tm} = transmural pressure.

Viscosity is ignored: $\alpha \gg 1$.

If artery is thin-walled, wall thickness h, diameter d, Young's modulus E, then

$c^2 = Eh/\rho d$ (Moens-Korteweg; Young)

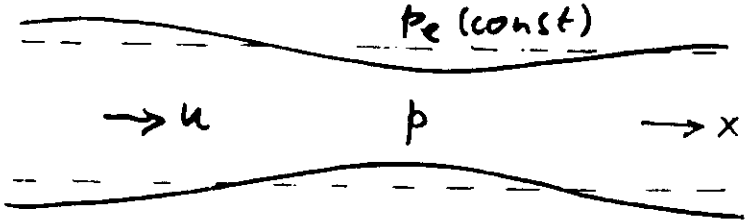
Prediction and measurement give

$c \approx 5\ m\,s^{-1}$ in proximal aorta

$\approx 8\ m\,s^{-1}$ in more peripheral large arteries,

compared with $u_{max} \approx 1\ m\,s^{-1}$.

Wave propagation in elastic tubes



Long wavelength approx., so area $A(x,t)$, pressure $p(x,t)$, velocity $u(x,t)$.

Cons. of mass: $A_t + (uA)_x = 0$ (1)

Cons. of momentum: $u_t + uu_x = -\frac{1}{\rho}p_x$ (2)

(neglecting friction)

Tube law: $p - p_e = \tilde{P}(A)$ (3)

Assume small amplitude (linear) waves:

$A = A_0 + A'$, $p = p_0 + p'$, u small,

and obtain the wave equation:

$$p'_{tt} = c^2 p'_{xx}$$

where $c^2 = \frac{A}{\rho}\frac{d\tilde{P}}{dA}$ (evaluated at $A = A_0$)

c = wave propagation speed

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The simple theory also predicts:

- no change of shape of wave-form as it propagates;
- velocity wave-form the same shape as pressure wave-form (but out of phase)

NEITHER IS TRUE.

The velocity wave-form shape can be explained if account is taken of blood viscosity (blood modelled as a homogeneous, Newtonian fluid!)

The peaking of the pressure wave can be explained in terms of wave reflection from arterial junctions.

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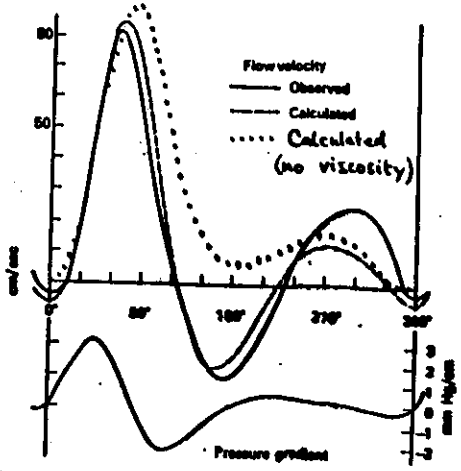

The agreement between the observed velocity wave-form and that predicted from the measured pressure gradient using rigid tube theory indicates that elasticity is unimportant in the determination of flow from pressure gradient (although it is crucial in determining the pressure gradient). Viscosity is important in determining flow.

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Velocity profiles

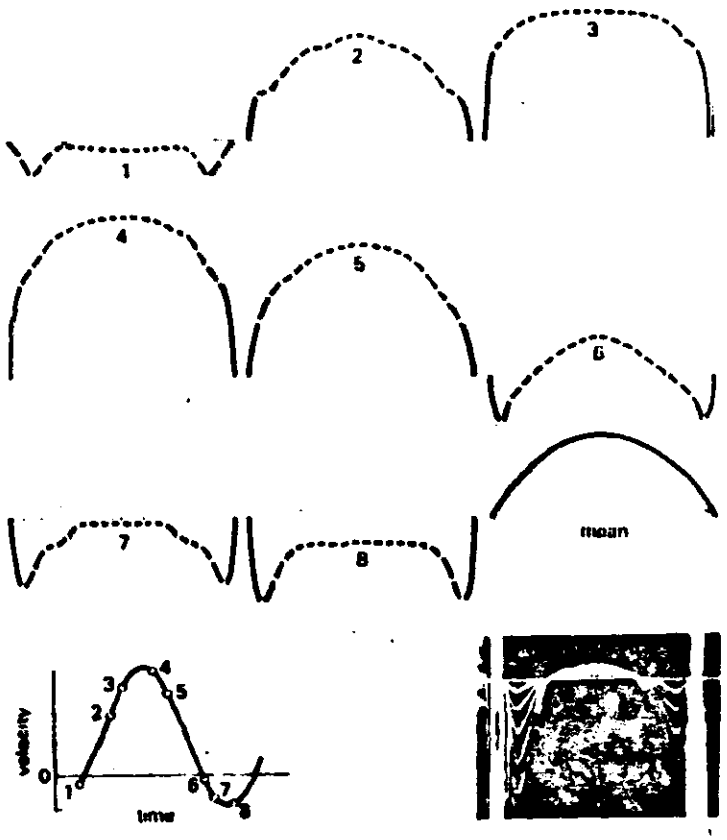


Fig. 12.42. Instantaneous velocity profiles at eight different times during a cycle of oscillating flow in a long, straight rigid pipe. The times are marked on the flow-rate wave-form which is shown at bottom left and which consists of a sinusoidal oscillation super-imposed on a steady flow, so that there is only a short period of backflow. The measurements were made far from the entrance of the pipe, so that the mean flow had a fully developed, Poiseuille profile (also shown). The flow conditions were chosen to be appropriate to those in the aorta:

mean Reynolds number $Re = 1785$
Womersley parameter $\alpha = 20{\cdot}5$
peak velocity 2·74 times mean velocity.

The measurements were made by 'marking' the flow with lines of tiny hydrogen bubbles, generated by pulsing electrical current *through a fine* wire stretched across the flow. These bubbles are carried with the flow, and are photographed after a known time interval (unset), the velocity at each point on the diameter during this interval can then be calculated. (Data reproduced by courtesy of Mr. P. Maston and Dr. M. Clamen.)

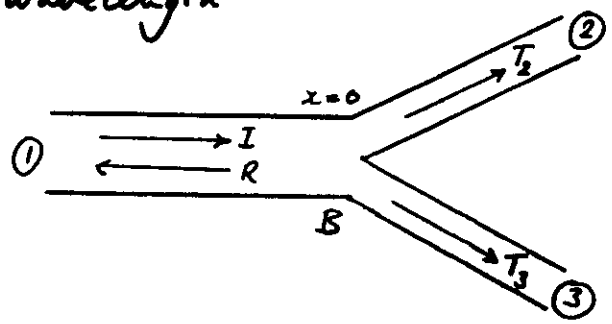
EXPERIMENT. $\alpha = 20.5$

Straight tube.

TR7

Wave Reflection (no viscosity)

Long wavelength



$(f_{max} = 1)$

Incident wave: pressure $p = P_I f(t - x/c_1)$

flow rate $Q = A_1 u = Y_1 P_I f(t - x/c_1)$, $Y_1 = \frac{A_1}{\rho c_1}$

Reflected wave: $p = P_R g(t + x/c_1)$, $Q = -Y_1 P_R g(t + x/c_1)$

Transmitted waves: $p = P_{Tj} h_j(t - x/c_j)$, $Q = Y_j P_{Tj} h_j(t - x/c_j)$ $(j = 2, 3)$

Continuity of pressure and flow rate at $x = 0$

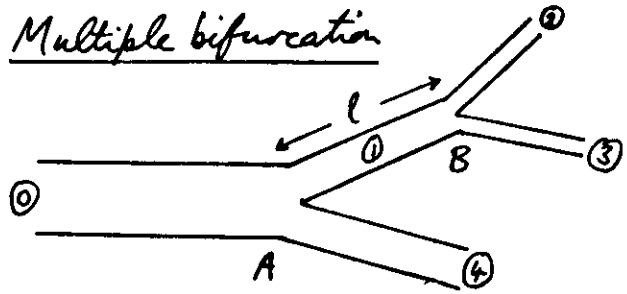
$\Rightarrow$ $h_2(t) = h_3(t) = g(t) = f(t)$ and

$$P_I + P_R = P_{Tj} \qquad (j = 2, 3)$$

$$Y_1 (P_I - P_R) = \sum_{j=2}^{3} Y_j P_{Tj}$$

Hence $$\frac{P_R}{P_I} = \frac{Y_1 - \Sigma Y_j}{Y_1 + \Sigma Y_j}, \quad \frac{P_{Ti}}{P_I} = \frac{2Y_1}{Y_1 + \Sigma Y_j}$$

Multiple bifurcation



At A in tube ① we have

$$\frac{Q}{p} = Y_{1\,eff} = \frac{Y_1 [f(t + \ell/c_1) - P_R/P_I\, f(t - \ell/c_1)}{f(t + \ell/c_1) + P_R/P_I\, f(t - \ell/c_1)}$$

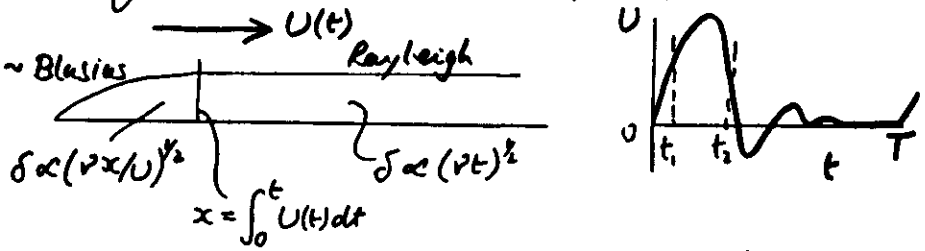
which is independent of t only for a single Fourier mode, for which $f(t) = e^{i\omega t}$.

Then $$\frac{Y_{1\,eff}}{Y_1} = \frac{\sum_{j=2}^{3} Y_j + i Y_1 \tan(\omega \ell/c_1)}{Y_1 + i \sum_{j=2}^{3} Y_j \tan(\omega \ell/c_1)}$$

Now the junction at A can be analysed in the same way as that at B, and hence the whole cardiovascular tree can be put together, as long as we know the wave-speed c and characteristic admittance Y for each branch.

Unsteady entry flow in a straight tube

(neglect curvature – as for a flat plate)



Assume motion starts from rest at $t=0$. At given x, the boundary layer will be purely diffusive (a Rayleigh layer) for $0<t<t_1$ and again for $t_2<t<?$; it will be advective (approx. a Blasius layer) for $t_1<t<t_2$, for some t_1, t_2.

Dimensionless boundary layer equation:

$$\underset{①}{(u_t - U_t)} + \underset{②}{(uu_x - u_y\int_0^y u_x\,dy)} = \underset{③}{u_{yy}}$$

with $u(t,0)=0$, $u(t,\infty)=U(t)$, $u(0,y)=0$.

Advective solution, for $t_1<t<t_2$: ② balances ③, ① is negligible.

Blasius →

$$u = U(t)\left[f_0'(\eta) + \frac{x\dot{U}(t)}{U^2(t)} f_{11}'(\eta) + \cdots\right], \quad \eta = y\left[U(t)/2x\right]^{1/2}$$

This breaks down at t_1, t_2 where

$$\epsilon = \frac{x\dot{U}}{U^2} = (\text{say})\ 0.5$$

Diffusive solution, for $0<t<t_1$, $t_2<t<?$

① balances ③, ② is negligible.

$$u = U(t) - \frac{2}{\sqrt{\pi}}\int_{\eta_0}^{\infty} U\left[t - \frac{\eta_0^2(t-t_0)}{\mu^2}\right] e^{-\mu^2}\,d\mu$$

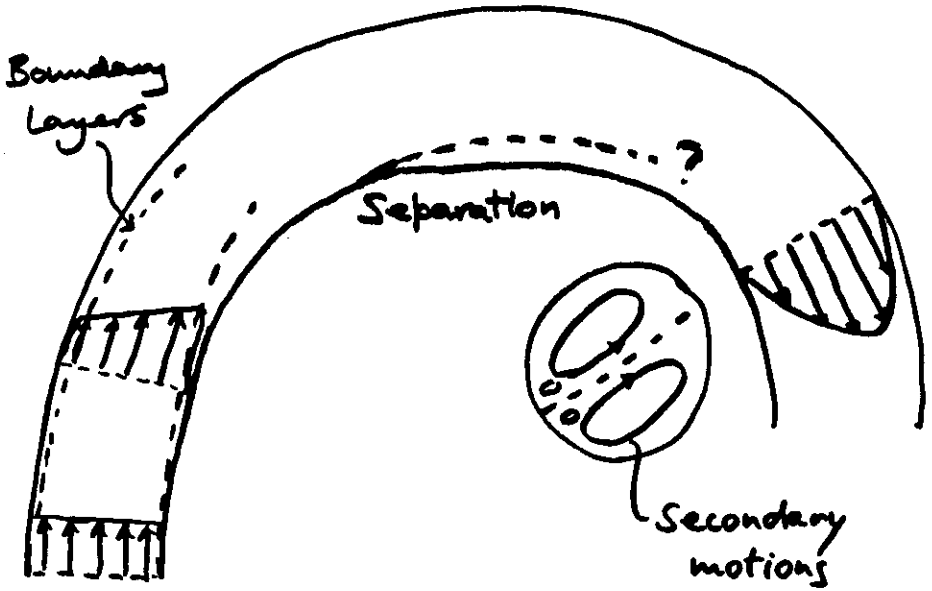
where $\eta_0 = \frac{1}{2}\,y(t-t_0)^{-1/2}$

and $t_0 = 0$ for $0<t<t_1$,

$t_0 \neq 0$ for $t>t_2$ – in this case t_0 is chosen to make the displacement thickness continuous at $t=t_2$.

Shear stress can then be calculated.

20 # Entry flow in a curved tube

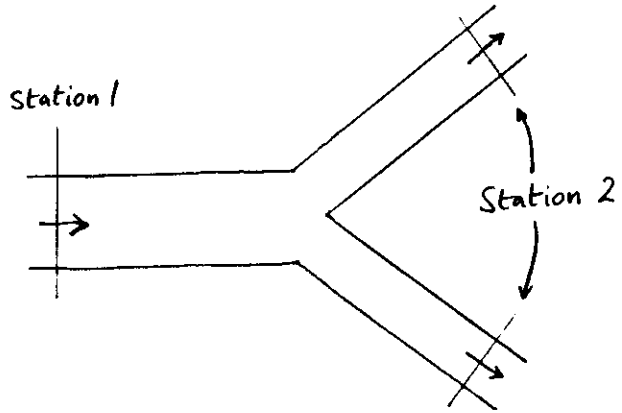


(Steady)

Similarly in branched tubes

T11

Pressure drop calculated from energy balance



Rate at which pressure force at 1 does work

- rate at which pressure force at 2 does work

= rate at which kinetic energy is gained at 2

- rate at which kinetic energy is lost at 1

+ rate at which mechanical energy is dissipated in the whole region through viscosity

Friction factor

$$C_F = \left[\text{total } \Delta P - \left[\begin{array}{c} \Delta P \text{ due to} \\ \text{changes in} \\ \text{kinetic energy} \end{array} \right] \right] \Big/ \tfrac{1}{2} \rho \bar{V}^2$$

Straight tube results

Poiseuille flow:	$C_F = 64(l/d)Re^{-1}$
Entry flow:	$C_F = k_3(l/d)^{1/2}Re^{-1/2}$
Turbulent flow:	$C_F = k_4\, l/d$

Inspiratory pressure drop in branched tube systems.

$$\Delta P = \Delta P_v + \Delta P_k = \tfrac{1}{2}\rho \bar{V}^2 (C_F - C_k)$$

$\bar{V}$ = average velocity in parent tube

C_k = kinetic energy constant (≈ 1.0 in trachea)

C_F = friction factor

$C_F \propto Re^{-1}$ (Poiseuille)

$C_F \propto Re^{-1/2}$ (Entry flow)

C_F = constant (Turbulent)

[$C_F = K_1/Re + K_2$ Rohrer]

C_F in general is computed from measured velocity profiles. In branched tubes most energy dissipation is in the boundary layer on the flow divider; hence use an entry flow model.

$$\left(\hat{p}_1 + \tfrac{1}{2}\rho \hat{q}_1^2\right) - \left(\hat{p}_2 + \tfrac{1}{2}\rho \hat{q}_2^2\right) = \mathcal{D}/Q$$

where $\hat{p}_1 = \frac{1}{Q}\int_{A_1} p u \, dA$, $\hat{q}_1^2 = \frac{1}{Q}\int_{A_1} |\underline{u}|^2 u \, dA$

$= \beta \bar{u}_1^2$ (say)

and dissipation rate is

$$\mathcal{D} = 2\mu \int_V e_{ij} e_{ij} \, dV \simeq \mu \int_V \left[\left(\frac{\partial u}{\partial r}\right)^2 + \frac{1}{r^2}\left(\frac{\partial u}{\partial \theta}\right)^2\right] dV.$$

Thus both $\mathcal{D}$ and the kinetic energy terms can be estimated from measured velocity profiles.

Let $y = \dfrac{\text{actual dissipation rate per unit length}}{\text{dissipation rate in Poiseuille flow}}$

$$z = \frac{\text{total dissipation rate } \mathcal{D}}{\mathcal{D} \text{ in Poiseuille flow}}$$

Entry flow model implies

$$y = \tfrac{1}{2}\gamma (Re\, d/x)^{1/2}, \quad z = \gamma (Re\, d/\ell)^{1/2}$$

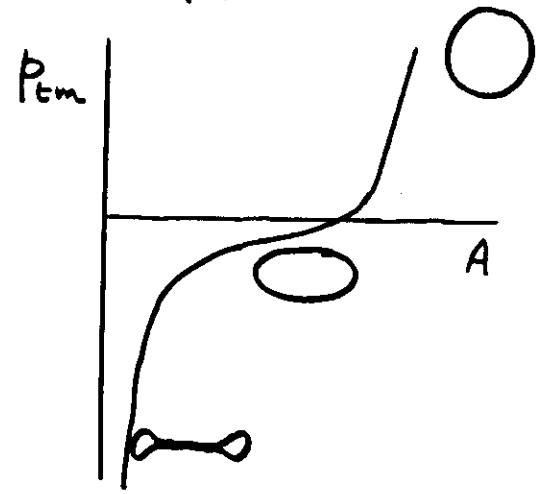
where γ should be a constant, and the experimental data indeed show γ and β to have no systematic dependence on Re:

$$\gamma \simeq 0.33, \quad \beta \simeq 1.7$$

We use these values to predict Δp in the lung.

FLOW IN COLLAPSIBLE TUBES

When the transmural pressure p_{tm} in an elastic tube is negative (i.e. compressive) there are large area changes for small changes in p_{tm}.



Even the small pressure changes associated with flow (viscosity, inertia) are enough to cause collapse.

PHYSIOLOGICAL EXAMPLES

VEINS above the heart (not skull)

VEINS entering the chest

(and ARTERIES leaving it during CPR?)

CORONARY BLOOD VESSELS in systole

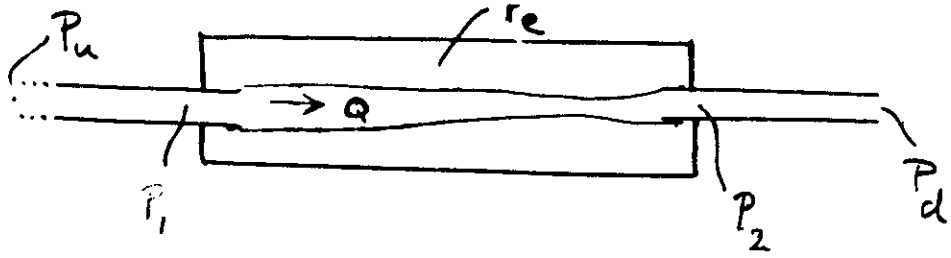
ARTERIES under a blood pressure cuff — Korotkoff sounds

LARGE AIRWAYS during forced expiration

(and smaller ones during chest compression?) — Wheezing.

URETHRA during micturition,

etc.



STEADY FLOW in a tube with uniform properties is governed by the differences between 3 pressures: P_u, P_e, P_d.

Measure $P_1 - P_2$ vs. $\dot{Q}$

(i) $P_u = P_1$, $P_d = 0$; $P_u - P_e$ = const.

Vary $\dot{Q}$ by varying P_u.

e.g. forced expiration, coronary circulation, chest compression.

Leads to FLOW LIMITATION

(ii) $P_e - P_d$ = const. Vary $\dot{Q}$ by varying P_u

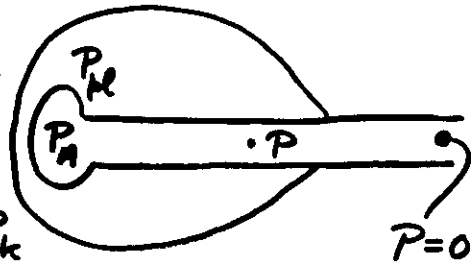
e.g. Cuff, Starling Resistor; Conrad

(iii) $P_e - P_2$ = const.; P_d has no importance when the const. exceeds a critical value

Brower et al., Bertram. — WATERFALL

Forced expiration

Pressure differences



(1) $P_A - P = \Delta P_V + \Delta P_k$

(2) $P_A - P_{pl} = \Delta P_{EA}$ (elastic recoil pressure of alveoli)

(3) $P - P_{pl} = \Delta P_{EB}$ (e_ r_ p_ of bronchi, reduced by tethering & interdependance)

(1) & (2) →

$$P - P_{pl} = P_{EA} - (\Delta P_V + \Delta P_k)$$

If this is less than ΔP_{EB} we have airway collapse, flow limitation. This only happens when $\Delta P_V + \Delta P_k$ is sufficiently large, i.e. during forced expiration.

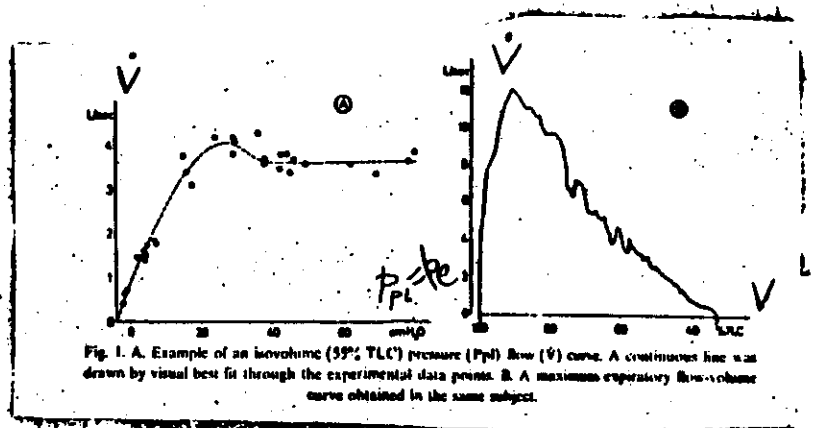


Fig. 1. A. Example of an isovolume (59% TLC') pressure (Ppl) flow ($\dot{V}$) curve. A continuous line was drawn by visual best fit through the experimental data points. B. A maximum expiratory flow-volume curve obtained in the same subject.

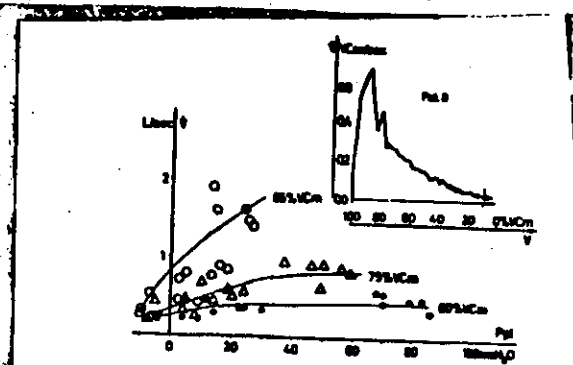


FIG. 3. IVPV curves constructed at three lung volumes in a patient with severe COPD. 85% VC_M is slightly past PEVR (see MEVV curve in insert): at this level, variability of flows is much larger than at lower lung volumes.

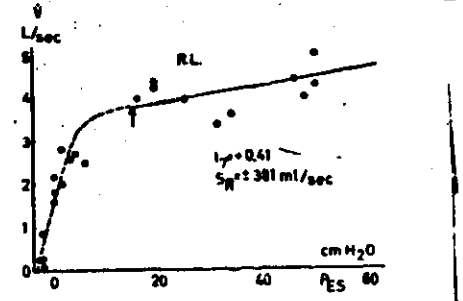


FIG. 6. IVPF curve at 55% TLC in *subject RL*. Even in plateau region (onset indicated by arrow), maximum flows increase further with transpulmonary pressure (P_{ES}). Slope of the fitted regression line is not statistically significant (*t* test, 0.41). Indeed residual standard deviation along regression line ($s_{y/x}$) is large.

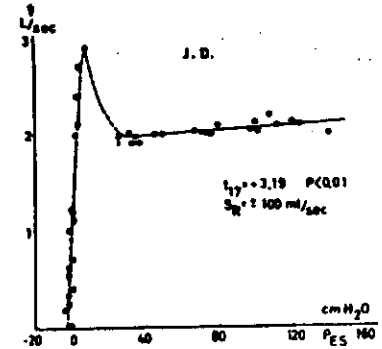


FIG. 7. In this example, the slope though small, is signific positive. On visual inspection, experimental points might as we located on a horizontal plateau.

UNSTEADY FLOW

(a) Deterministic, driven by imposed variations in P_u, P_e or P_d, or combinations

e.g. P_e : deep vein compression; CPR; venous return from lower limbs (muscle pump : valves).

P_d : venous flow during respiratory manoeuvres.

P_u and P_e : coronary vessels; respiratory manoeuvres.

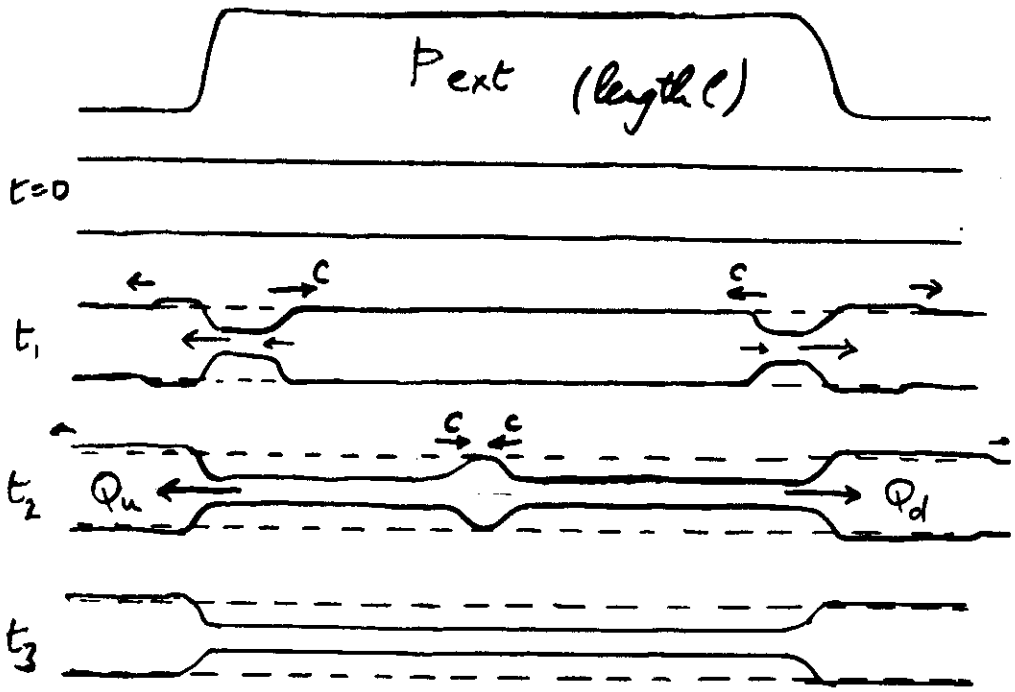
P_u : opening and closing of a vessel under a cuff (or a finger).

Can be analysed using the exhaustive one-dimensional theory of

Kamm & Shapiro (J. Fluid Mech. 95, 1-78 1979)

as long as the geometry and elastic properties are known. (NB: need to measure AREA of a vessel, not WIDTH)

The rate of emptying of a squeezed tube depends on the speed of propagation of the collapse waves, c.



The distribution of flow up- and downstream (Q_u and Q_d) depends on the relative impedances of those parts of the external system.

There will clearly be a time delay $\frac{\ell}{2c}$ before collapse is complete.

(b) Non-deterministic.

32

SELF-EXCITED OSCILLATIONS.

e.g. Korotkoff sounds; (Shimizu)
extracorporeal vessels – bypass, open heart or dialysis. (Matsuzaki)
Wheezing (flutter)

Note Large Reynolds number.

Many possible mechanisms
– theory at an early stage.

Conrad 1969 Lumped X

Cancelli + Pedley 1985 Distributed incomplete

(wave-speed limitation)

33

Tube law for small A

(dimensional analysis)

Assume no variation along tube;
line contact between opposite walls.

The only length-scale is radius of curvature at edges: R
(not tube diameter).

$p - p_e$ is sustained entirely by resistance to bending at edges.

Theory of beams: $M = \frac{EI}{R}$

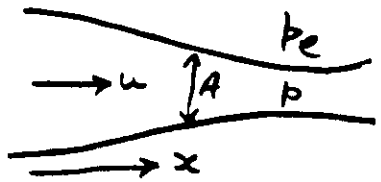
where M = bending moment (p.u. length)
E = Young's modulus of tube)
I involves wall thickness, not

Now M = length × force $\propto R^2 (p_e - p$

Hence $p - p_e \propto -R^{-3} \propto -A^{-3/2}$

Steady flow in a continuously-varying collapsible tube.

Area $A(x)$
Velocity $u(x)$
Pressure $p(x)$



Conservation of mass: $uA = \text{const}$

Momentum: $u\frac{du}{dx} = -\frac{1}{\rho}\frac{dp}{dx} - Ru$ (viscosity)

Elasticity: $p - p_e = \tilde{P}(A)$ (tube law)

Hence

$$\frac{dA}{dx} = \frac{-Ru}{\frac{1}{\rho}\tilde{P}'(A) - u^2/A}$$

$$= \frac{-RuA}{c^2 - u^2} \quad < 0 \text{ if } u < c$$

where $c^2 = \frac{A}{\rho}\frac{d\tilde{P}}{dA}$ (wave-speed)

No solution if u reaches the value c. (choking)

(Recovery possible after viscous collapse $u \ll c$, $Re \ll 1$)

5

For a finite tube held open at the ends the previous model fails anyway.

Need to include:

(a) longitudinal tension: $p - p_e = P(A) - T\frac{d^2A}{dx^2}$

(b) flow separation (and neglect other viscous losses):

$$\chi u\frac{du}{dx} = -\frac{1}{\rho}\frac{dp}{dx}$$

($\chi = 1$ upstream of separation at narrowest point, $= 0.2$ downstream)

Combine the equations to obtain

$$T\frac{d^2A}{dx^2} - \frac{1}{2}\chi\rho\frac{Q^2}{A^2} - P(A) = p_e - p_0$$

where Q = constant flow-rate

p_0 = a constant pressure

Model $P(A) = K_p\left[1 - (A/A_0)^{-3/2}\right] \quad A \le A_0$

$= K_p k\left(A/A_0 - 1\right) \quad A \ge A_0$

Boundary conditions (steady flow)

$A = A_0$ at $x = 0, L$

$p = k_2 Q^2$ at $x = L$, giving

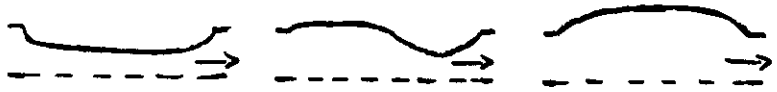
$$p_0 = (k_2 + \tfrac{1}{2}\chi\rho)Q^2$$

$P_u - p = k_1 Q^2$ at $x = 0$, giving P_u.

This non-linear system can be used to show (Jensen)

(i) When $\chi = 1$ (no separation losses)
no steady solution exists if Q exceeds a critical value, dependent on T.
Equivalent to choking ?

(ii) A solution exists for all Q.

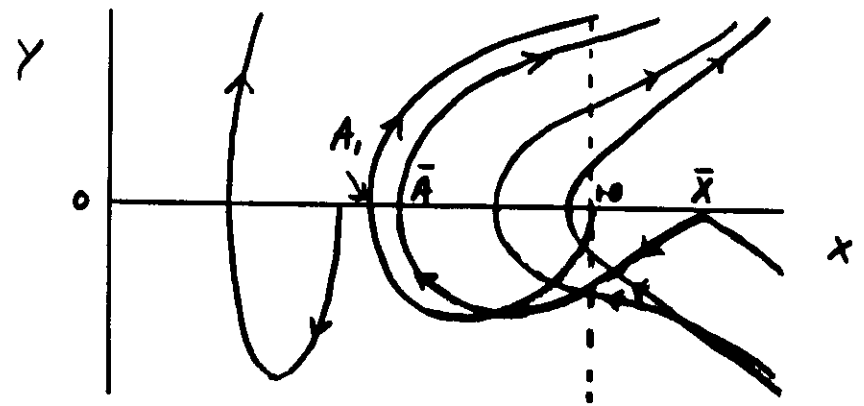


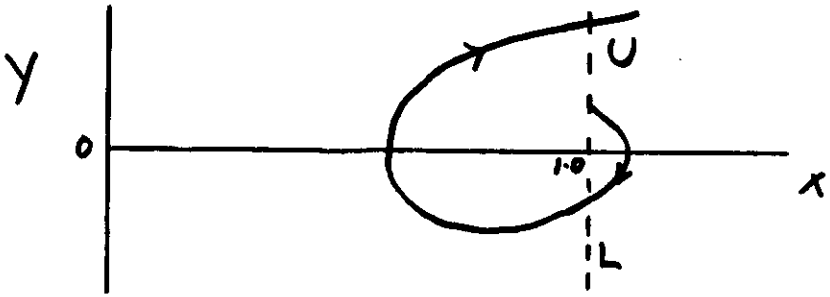
Questions.

(a) Are the steady solutions stable, when they exist?

(b) If not, or if they don't exist, what oscillations result?

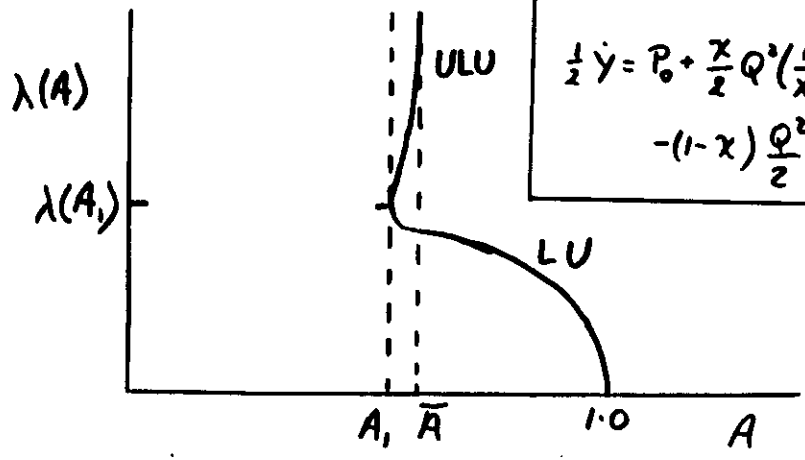
Phase plane



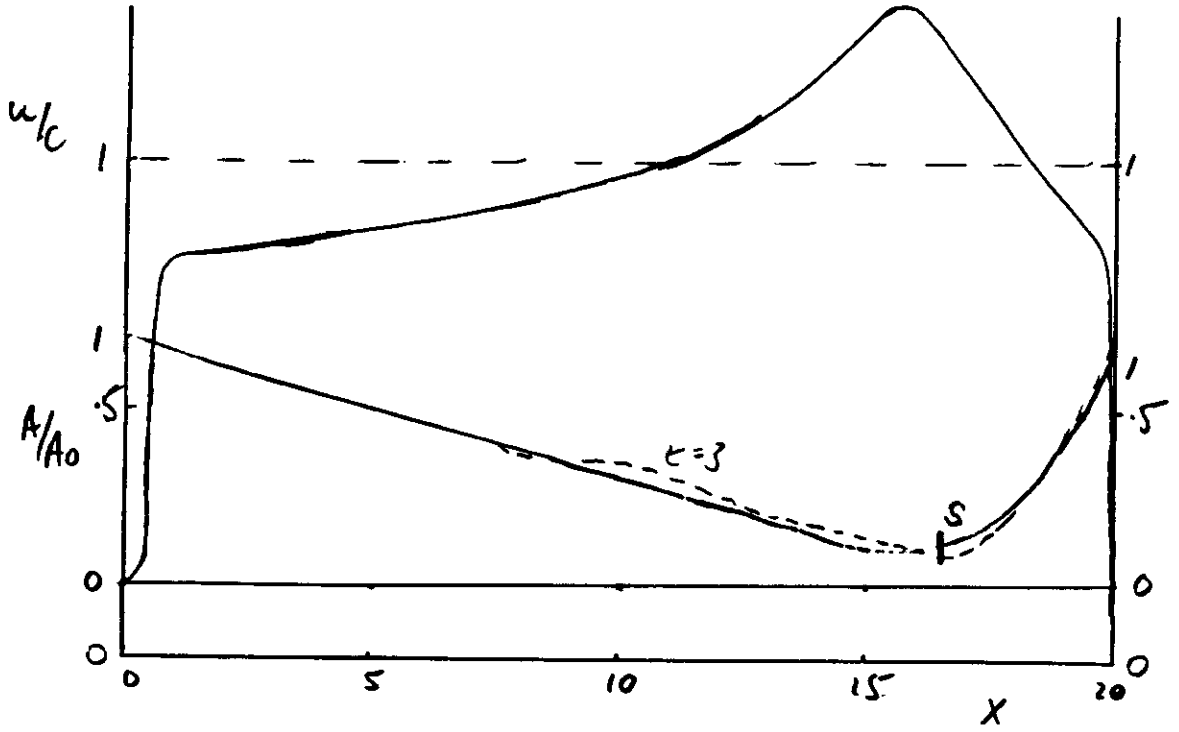


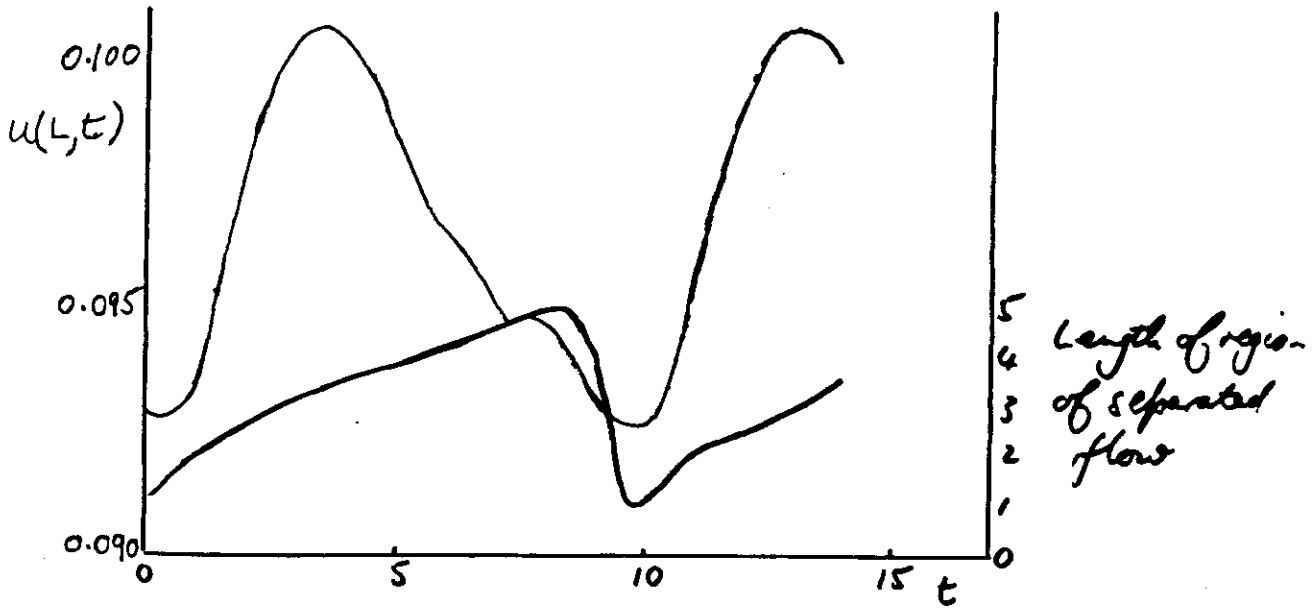
$X = \alpha \qquad \dot{X} = Y$

$$\tfrac{1}{2}\dot{Y} = P_0 + \frac{\chi}{2}Q^2\left(\frac{1}{X^2} - 1\right) + \mathcal{P}(x) - (1-\chi)\frac{Q^2}{2}\left(\frac{1}{A^2} - \frac{1}{X^2}\right)$$



$c^2 = \frac{A}{\rho}\frac{dP}{dA}$ H typical decay time (+ oscillations in A)





OSCILLATIONS

Results of new model. (C & P)

(i) The steady-state predicted from the time-independent equations is stable if the separation point is prevented from moving up and downstream as pressure waves pass.

(ii) When the separation point is allowed to move, the steady state is unstable, and oscillations occur, whenever $u > c$.

(iii) Oscillations sometimes (for small η_s) occur when $u < c$, as predicted by lumped-parameter models, but they are feeble and probably unimportant (compare experiment).

Cancelli & Pedley (JFM 157, 375, 1985) found in a numerical model that a requirement for large-amplitude oscillations was hysteresis in the flow separation process.

This, and other studies, imply that we need much more detailed fluid mechanical understanding of unsteady, internal, three-dimensional separated flows.

Precisely the same is required to assess arterial wall shear stress, so collapsible tubes are, after all, linked to atherogenesis.

Non-linear waves (Riemann)

$$A_t + (uA)_x = 0 \qquad (1)$$

$$u_t + uu_x = -\frac{c^2}{A}A_x \qquad (2)$$

Add $\pm \frac{c}{A} \times (2)$ to (1) and get

$$\frac{\partial}{\partial t} R_\pm + (u \pm c)\frac{\partial}{\partial x} R_\pm = 0$$

where $$R_\pm = u \pm \int_{A_0}^{A} \frac{c}{A}\, dA$$

(Riemann invariants)

Hence $R_\pm$ remain constant along paths in the $(x\text{-}t)$ plane given by

$$\frac{dx}{dt} = u \pm c \quad \text{respectively}$$

(characteristics)

Leads to the method of characteristics for integrating such equations

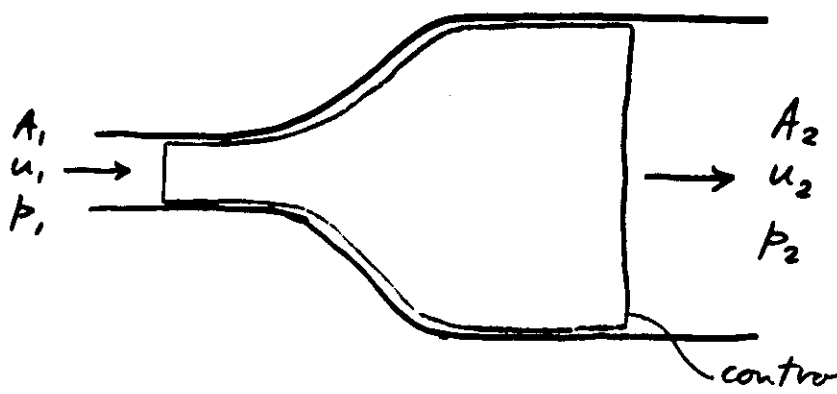
(Kamm & Shapiro, 1979, JFM 95, 1-78)

Or use finite-differences, iteratively

(Kimmel, K&S, J.B.E., to appear)

(Jan, K&S, J.B.E., 105 12-19, 1983)

Elastic jump conditions



Mass conservation:

$$u_1 A_1 = u_2 A_2 = Q$$

Momentum conservation:

$$A_1(p_1 + \rho u_1^2) + \int_1^2 p\, dA = A_2(p_2 + \rho u_2^2)$$

This term represents the longitudinal force exerted by the pressure on the non-parallel part of S.

If the tube law can be used for p in the jump (and even if not: Cowley, 1982, JFM 116, 459-473) NOT SHAPIRO this reduces to conservation of the quantity

$$\Phi(A) = \frac{Q^2}{A} + \int_A \frac{K_p}{\rho} A P'(A)\, dA$$

So, given Q & A_1 you can calculate A_2.

Notes (i) If upstream flow is supercritical,
44 downstream flow is subcritical.

$$\Phi(A_1) = \Phi(A_2)$$

$$\Rightarrow \int_{A_1}^{A_2} \left(c^2 - \frac{Q^2}{A^2}\right) dA = 0$$

So if the integrand is positive at one end of the range, and if it varies monotonically (which it does), then it must be negative at the other end. (and zero at A_Q in between)

(ii) There must be positive energy loss at the jump. (Separation → turbulence)

$$\left[\frac{p}{\rho} + \frac{1}{2}u^2\right]_2^1 = R > 0$$

Now $R = \frac{1}{Q}\int_1^2 \left(\frac{1}{A_Q} - \frac{1}{A}\right)\frac{d\Phi}{dA}\,dA$

and this is >0 if $A_1 < A_2$ and A_Q is a minimum of Φ, which it is in our case.

In fact this requires $M(A_Q) > 0$.

IIC (See Cowley; Oates (1975) Med. Biol. Eng. 13, 773-8)

Nature 329, 13-14, 3-9 Sept. 87

How giraffes prevent oedema

T.J. Pedley

An upright giraffe, by analogy with humans, ought to suffer massive oedema in its feet; moreover, when it lowers its head to drink, the blood should rush down into it and be unable to flow up again. New pressure measurements reported on page 59 of this issue[1] by Hargens *et al.* show why neither of these things happens, and also contain some surprising observations of highly variable venous pressure (P_v) in the leg and of a counter-gravitational gradient of P_v in the neck, which greatly improve our understanding of the circulation of blood in the giraffe. The latter result, in particular, demonstrates conclusively that the jugular vein is normally highly collapsed and therefore the circulation in the head and neck does not resemble a siphon, despite recent opinion to the contrary[2].

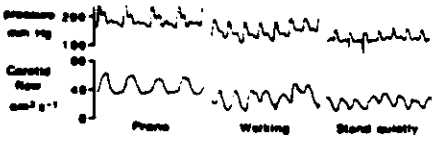
The gravitational (or hydrostatic) gradient of pressure with height means that, in an upright animal of height 3.5 m, the mean blood pressure in an artery in the head is as much as 110 mm Hg (about 1.5 m H_2O or 15 kPa) lower than at the level of the heart, which is about 200 mm Hg above atmospheric, double the human value, to stop the carotid artery from collapsing. In the foot, mean arterial pressure (P_a) will be 110 mm Hg greater, that is, 310 mm Hg, which, if the peripheral circulation were the same as in man, would lead to pooling of blood in the veins and to oedema.

Hargens *et al.*[1] made radiotelemetry measurements of P_a, P_v and tissue pressure (P_t) (all relative to atmosphere) in free-ranging animals, and have additionally recorded osmotic pressures and blood flow in sedated animals standing still. The means of the data in the feet of standing animals show $P_a \sim 260$ mm Hg; $P_v \sim 150$ mm Hg; and $P_t \sim 44$ mm Hg. In moving animals, however P_a varies between 70 and 380 mm Hg; P_t between −120 and +80 mm Hg; and P_v varies hugely, between −250 and +240 mm Hg.

Features of the peripheral circulation that inhibit oedema in a standing giraffe are first, a high resistance to flow in the thick-walled arterioles, which keeps P_v and hence capillary pressure P_c well below P_t; and second, a very tight skin and fascia in the lower legs (an 'anti-gravity suit'[3]), which allows P_t to be much higher than in man (where $P_t \sim 0$). Even so, there is a net filtration pressure of more than 80 mm Hg, and quietly standing giraffes will be susceptible to some oedema although one might expect P_t to continue to rise as oedema proceeds, assuming the fascia to be effectively inextensible.

In the ambulant giraffe, however, the well-known 'muscle pump' comes into play, as in man, squeezing blood up out of the lower veins as the skeletal muscles contract, and sucking it in again through the capillaries as they relax, backflow in the veins being prevented by the valves. A high arteriolar resistance would then explain the very low observed values of P_v (and hence P_c), because it prevents the compliant veins from filling rapidly; this explanation can be checked from the phase relations between P_a and P_t if the radiotelemetry equipment allows them to be recorded as functions of time.

The siphon model[2] of the circulation in the head and neck is based on the fact that if the blood vessels are rigid, or at least stiffly distended, gravitational pressure changes are irrelevant, $P_a - P_v$ at the heart being determined purely by a constant viscous resistance. But blood vessels are compliant and can collapse flat to a very small cross-sectional area when the internal pressure falls significantly below the external, and even in man the jugular vein is often in a collapsed state[4]. (Veins in the head do not collapse because the skull is a constant-volume box filled with incompressible tissue and fluid, so incipient collapse causes a corresponding fall in extra-vascular pressure.) The gravitational contribution to P_v at the top of a standing giraffe's neck is around −100 mm Hg (right atrial pressure being close to atmospheric), not the observed +13 mm Hg (ref. 1). The difference cannot be accounted for by compartmentalization of the vein, as although there are valves in the jugular vein of a giraffe (unlike man), they work the wrong way round. The required mean pressure gradient of about 80 mm Hg m^{-1} must come from viscous resistance to flow in the vein itself. However, if the jugular vein were fully open, with diameter 2.5 cm (ref. 2), it would take a flow rate of above 1,500 l min^{-1}, 500 times the flow in the carotid artery[5], to generate this gradient, according to Poiseuille's law. Realistic flow rates require a much larger resistance which can come only from severe collapse of the vein. The analogy is closer to a waterfall than to a siphon[6].



Phasic waveforms of carotid pressure and flow in the giraffe during spontaneous activity (redrawn Fig. 6 from ref. 5).

What about venous return with the head down? This is where the valves come in, but as in the legs it requires a muscle pump to help move the blood up, and this I believe comes from rhythmic movements of the jaws.

All the data I have discussed above refer to mean blood flow, averaged over many heart beats. But, nearly 20 years ago, Van Citters *et al.*[5] measured the shapes of the pressure and flow-rate pulses in the carotid arteries of free giraffes, and found dramatic differences between prone, quietly standing and walking animals (see figure). I have seen no published explanation of these differences, although it should not be too hard to find one as the giraffe carotid artery is long and straight, so that relatively simple fluid-mechanical theories[4] should be applicable. □

Giraffes in their 'anti-gravity suits'.

1. Hargens, A.R., Millard, R.W., Pettersson, K. & Johansen, K. *Nature* **329**, 59–60 (1987).
2. Badeer, H.S. *Comp. Biochem. Physiol.* **83A**, 207–211 (1986).
3. Burton, A.C. in *Physiology and Biophysics* (eds Ruch, T.C. & Patton, H.D.) 523–542 (Saunders, Philadelphia, 1965).
4. Caro, C.G., Pedley, T.J., Schroter, R.C. & Seed, W.A. *The Mechanics of the Circulation* (Cambridge University Press, 1978).
5. Van Citters, R.L., Kemper, W.S. & Franklin, D.L. *Comp. Biochem. Physiol.* **24**, 1035–1042 (1968).
6. Permutt, S., Bromberger-Barnea, B. & Bane, H.N. *J. appl. Physiol.* **18**, 924–932 (1963).

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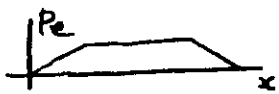
Mathematical Tripos Part III
Physiological Fluid Mechanics
Dr. T.J. Pedley
February 1988

Example Sheet II

1. **Low Reynolds number flow in a collapsible tube of slowly-varying elliptical cross-section.** Suppose that a pressure-area relation $p - p_e = P(A)$ exists for the tube, and that the shape of the cross-section varies with area in such a way that the major axis of the ellipse remains constant when $A < A_0$ (A_0 is the area at which the cross-section changes from circular to elliptical). Use lubrication theory to show that the differential equation for $A(x)$ when the tube is horizontal and the flow is steady is

$$\frac{dP}{dA}\frac{dA}{dx} = -\left\{\frac{dp_e}{dx} + 4\pi\mu Q(A^2+A_0^2)/A_0A^3\right\} \qquad (1)$$

where $p_e(x)$ is the distribution of external pressure with longitudinal distance, Q is the volume flow-rate and μ is the fluid viscosity. Investigate qualitatively the distribution of A with x in realistic conditions for a pressure cuff: e.g. Let $A = 1.1A_0$ at $x = 0$, let p_e vary trapezoidally, and take dP/dA to have a simple form, such as $dP/dA \propto A^{-5/2}$ for $A < A_0$, $dP/dA \propto A$ for $A > A_0$.



[To see the effect of a little fluid inertia, see my book section 6.2.]

2. A rigid tube of length 2L has a circular cross-section of non-uniform radius $a_0a(z)$ for $-1 \le z \le 1$, where a_0 is a typical radius and z is the longitudinal distance made dimensionless with L. Flowing in it is a viscous incompressible fluid of viscosity μ and density ρ. The pressure p at $z = 1$ is zero, while that at $z = -1$ is oscillatory with zero mean and period T:

$$p = \frac{L\mu W_0}{a_0^2}P(t) \qquad \text{at} \quad z = -1,$$

where t is time divided by T and W_0 is a scale for the velocity in the tube. The ratio $\epsilon = a_0/L$ is small, the Reynolds number $Re = a_0W_0\rho/\mu = O(1)$ as $\epsilon \to 0$, and the frequency parameter $\rho a_0^2/\mu T = \alpha^2 \epsilon\, Re$, where α^2 is also $O(1)$ as $\epsilon \to 0$. Analyse the flow to first order in ϵ, and calculate the flow-rate through the tube as a function of time. Deduce that it has a non-zero mean value if $P^2(t)$ has non-zero mean and if $a(1) \neq a(-1)$.

3. Incompressible fluid of density ρ flows steadily with flow rate Q in a collapsible tube of length L whose elastic properties are represented by the equation

$$p = \tfrac{1}{2}\rho C_0^2\frac{A^2}{A_0^2} - P_0 - T\frac{\partial^2 A}{\partial \hat{x}^2}$$

where $p(\hat{x})$ is the internal pressure, $A(\hat{x})$ is the cross-sectional area, A_0 is a reference area, $\hat{x}$ is the longitudinal coordinate, C_0 is a constant velocity, P_0 is a constant pressure and T is a constant proportional to the longitudinal tension in the wall. The external pressure is zero. The ends of the tube at $\hat{x} = 0, L$ are held open at area A_0, and $p(0) = \tfrac{1}{2}\rho C_0^2 - P_0$. The viscous retarding force at any $\hat{x}$ can be represented by a pressure gradient $-\rho R\, u\, A_0^2/A^2$, where R is a constant and $u(\hat{x})$ is the cross-sectionally averaged fluid velocity. Show that $\alpha = A/A_0$ is given by the following ordinary differential equation:

$$\sigma\frac{d^3\alpha}{dx^3} - \frac{d\alpha}{dx}(\alpha - q^2/\alpha^3) - q/\alpha^3 = 0$$

where $q = Q/C_0A_0 < 1$, $\sigma = TA_0R^2/\rho C_0^4$ and $x = R\hat{x}/C_0$. Write down the boundary conditions on α.

(a) Show that, when $\sigma \gg 1$,

$$\alpha \approx 1 - \frac{q}{6\sigma}\,x\,(\lambda^2 - x^2)$$

where $\lambda = RL/C_0$, and indicate how to derive a more accurate approximation.

(b) In the case $\sigma \ll 1$, show that a good approximation for α is given by

$$x = \frac{1}{5q}(1 - \alpha^5) - q(1-\alpha) \qquad (1)$$

as long as $\delta < x < x_1$, where δ is a small positive number and

$$x_1 = \text{Min}\left[\lambda, \frac{1}{5q} - q + \frac{4}{5}q^{3/2}\right]$$

is the position at which the solution breaks down. Find $\alpha_1 = \alpha(x_1-)$.

(c)* In case (b), show that if $x_1 = \lambda$, then in the neighbourhood of $x = \lambda$, α is given by that solution of

$$\frac{d\alpha}{d\xi} = (\alpha - \alpha_1)\left(\frac{\alpha}{3} + \frac{2\alpha_1}{3} - \frac{\alpha^2}{\alpha\alpha_1^2}\right)^{1/2}$$

which equals 1 at $\xi = 0$, where $\xi = \sigma^{-1/2}(x_1 - x)$.

4. * Write down the equations governing quasi-one-dimensional unsteady flow in a straight blood vessel inclined at an angle θ to the horizontal and possessing uniform elastic properties such that the difference between the internal and external pressures is given by

$$p(x,t) - p_e(x) = P(A) \equiv P_o + \frac{1}{2}\rho c_o^2 \frac{A^2(x,t)}{A_o^2}$$

where A is the cross-sectional area of the vessel and x,t are the longitudinal coordinate and time; ρ is the blood density and P_o, c_o, A_o are constants. The effect of viscosity can be represented by a longitudinal resistive force, per unit length of tube, equal to $\rho AR(A)u$, where $u(x,t)$ is the average blood velocity across a cross-section (assumed positive).

(i) When $\theta = 0$, $p_e = 0$, when there is no mean flow, and the undisturbed cross-sectional area is $\bar{A}$, find the speed of propagation and attenuation factor of small amplitude waves of angular frequency ω.

(ii) When $\theta \neq 0$, $p_e = 0$, show that steady flow with volume flow-rate qA_oc_o and uniform cross-sectional area A_1 is possible if

$$q = \frac{A_1 g \sin\theta}{R(A_1) A_o c_o} .$$

(iii) With upstream area and flow-rate held constant (at the values given in (ii)) a cuff is inflated round the vessel and applies to it an external pressure given by

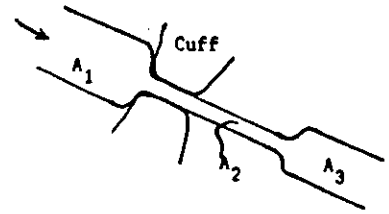
$$p_e = P_e(1 - x^2/\ell^2) , \quad |x|<\ell$$

$$= 0 , \quad |x|>\ell .$$

Assuming that ℓ is sufficiently small for gravity and friction to be locally negligible, and that $q < \alpha_1^2$ where $\alpha_1 = A_1/A_o$, show that steady flow remains possible, and the tube opens out to area A_1 downstream of the cuff, if $P_e < P_c$ where

$$P_c = \frac{1}{2}\rho c_o^2 (q - \alpha_1^2)^2/\alpha_1^2 .$$

(iv) If $P_e = P_c$ (with gravity and friction still negligible), show that a steady transition is possible in which the area decreases to $A_2 = \alpha_2 A_o$, where $\alpha_2 = q/\alpha_1$. Show that the flow downstream of the cuff is supercritical, i.e. its speed exceeds the local wave propagation speed.



(v) Show that a spontaneous elastic jump can occur from area A_2 to area $A_3 = \alpha_3 A_o$, where

$$\alpha_2\alpha_3(\alpha_2^2 + \alpha_2\alpha_3 + \alpha_3^2) = 3q^2 ,$$

and show that the flow downstream is subcritical. (Assume that $p - p_e = P(A)$ throughout the jump, and that gravity and friction are still negligible). Calculate the total head loss across the whole cuff/jump system.

(vi) What happens if $P_E > P_c$?

5. Extend the analysis of the lumped parameter model of collapsible tube oscillations to take account of one or more additional effects, and report on your findings.

E.g. Wall inertia;

Wall viscoelasticity;

Phase lag between reduction in constricted area and separated flow energy loss;

Finite upstream resistance and inertia (so u_1 is not constant);

Anything else you can think of.

[This is an open-ended opportunity to develop your own simple model of a complex physical phenomenon, taking account of what <u>you</u> think to be the important factors.]