5 In vivo dynamic testing of hydrocephalus shunts

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Abstract

The accumulation of excess cerebrospinal fluid in the ventricles of the brain results in hydrocephalus, a condition that is fatal if left untreated. The usual remedy is to insert a shunt into the ventricles of the brain, which drains excess fluid away, moderated by a pressure dependent valve. It is important that the system functions properly so that a reasonable intercranial pressure is maintained. Unfortunately, pressure measurements in the ventricles are highly invasive, while pressure measurements in the shunt outside the skull may not detect any blockage in the catheter inside. Here we develop a model primarily aimed at detecting in vivo a blockage or other shunt malfunction using noninvasive measurements, so that shunt valves can be adjusted accordingly. We then extend this to discuss the possibility of flow regulation.

5.1 Introduction

5.1.1 Cerebrospinal fluid

Cerebrospinal fluid (CSF) is a watery liquid produced in the parenchyma, with approximately 60% from the chronic pleas. Normal production is at a constant rate of approximately 20ml/hr, although this varies from 14 to 36ml/hr. In healthy individuals, the CSF flows from the lateral ventricles into the third ventricles, down the audacity towards the sanitary sinus and the spinal sad. It is reabsorbed in the top of the cranial vault, through granulations near the sanitary sinus. There is usually about 150ml of CSF in the intercranial space at any given time, with 25ml in the ventricles, 30ml in the spinal subarachnoid space and 75ml in the cerebral subarachnoid space.

5.1.2 Hydrocephalus

Hydrocephalus is a serious condition resulting from an excess of CSF in the ventricles of the brain. This may build up either over a long time or relatively quickly, and is the result of insufficient absorption (compared to production) of CSF. This leads to expansion of the ventricles, compression of the brain and, for non-infants, an increase in intercranial pressure (ICP), leading to the occlusion of circulation and cell death. To offset an increase in ICP, blood pressure (BP) will increase to maintain cerebral perfusion pressure (CPP),

$$CPP = BP - ICP,$$

so that sufficient oxygen and glucose are supplied to the brain. This rise in BP is, however, limited, after which cerebral viability quickly declines. In infants whose skull bones are not yet fused, increasing ICP will result in an expansion of the head, which offsets some of the compression, but is obviously still very damaging and painful.

We will not consider communicating hydrocephalus, which involves an excess of CSF in only the spinal column, since it is treated with lumbo-peritoneal shunts emanating from the lower spinal canal.

5.1.3 Hydrocephalus shunts

Hydrocephalus is treated by draining excess CSF from the ventricles through a shunt. A catheter is inserted through the brain into the ventricles. Tubing under the skin then drains the fluid into the heart via the jugular (a ventriculo-atrial shunt) or into the stomach (a ventriculo-peritoneal shunt). The latter is currently favoured because it avoids the difficulties of surgery near the heart, but can cause operational difficulties (see section 5.1.5). Outside the skull is a pressure operated valve designed to regulate the shunt. Just above this is a small reservoir, from which pressure measurements can be taken, as shown in Figure 32.
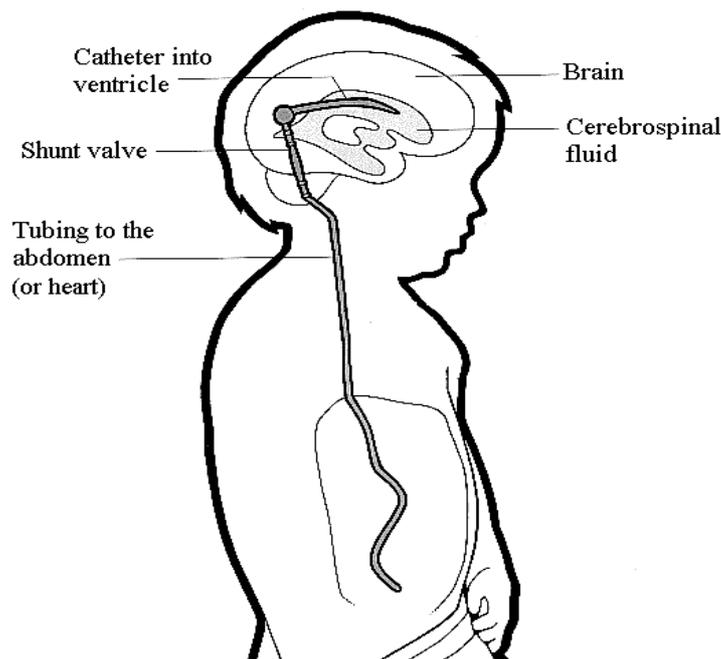

Figure 32: A hydrocephalus shunt implanted in an infant, in this case draining into the abdomen. Modified with permission from an original diagram at www.spinabifida.org.

Commercial shunts are designed to respond at set pressure differentials, and are categorized as low ($\approx$ 0 mmH$_2$O), medium ($\approx$ 60 mmH$_2$O) or high ($\approx$ 120 mmH$_2$O). More sophisticated models exist which are manually adjustable, so that settings may be altered without the need to replace the system. Most shunts are now manufactured as a single unit comprising a catheter, a valve and tubing. This has both improved surgery time and decreased the risk of disconnection (previously a problem). This does, however,

mean that other malfunctions (see section 5.1.5) require the complete replacement of the system.

The Orbis-Sigma valve is designed to maintain a constant flow rate across a range of transient pressure variations, such as when a patient sneezes. The resistance to flow increases with increasing pressure, within a given range, and is meant to avoid overdrainage, discussed in section 5.1.5. Outside this range, the flow to pressure response is approximately linear, the aim being that for high pressures the resistance is near zero to allow for rapid decompression.

5.1.4 Anti-siphoning devices

While shunts may operate perfectly while recumbent, when an individual stands up pressures within the head drop into the negative range so that the system becomes a siphon, which may result in overdrainage of the ventricles (see section 5.1.5).[1] The original solution was an anti-siphon device inserted into the tubing, which responded to atmospheric pressure through the skin. When the fluid pressure in the shunt dropped below this (as occurs with siphoning) the device shuts off flow (a diaphragm is pushed down through the pressure differential) until pressure within the ventricles returns to atmospheric. Such systems may malfunction if atmospheric pressure is not properly detected, as occurs if there is encasement by scar tissue.

This problem may be overcome by the use of the Pudenz-Schulte Delta valve shunt. Although this works in a similar manner by preventing drainage when there is negative pressure, it does not need to perceive atmospheric pressure to operate. In what follows we therefore focus only on the horizontal case, since the above mentioned devices mean that the transient effects of standing can be negated. The issue of hydrostatic forces is discussed in greater detail in section 5.2.

The horizontal-vertical valve, designed to open only when the patient is lying down, is primarily used in communicating hydrocephalus and is therefore not relevant here.

5.1.5 Shunt malfunction

A shunt is said to be malfunctioning if it is draining at an inappropriate rate. Underdrainage means that insufficient CSF is removed from the ventricles, so that symptoms are not fully alleviated. Even more damaging can be overdrainage, which may result in collapse of the ventricles. If this is very rapid, the brain may be torn away from the inner surface of the skull, causing bleeding (subdural haemorrhage), which may induce compression of the brain. If this leads to slit ventricles then the sides may stick together so that they do not fully reopen when CSF is reintroduced. Slower underdrainage can result in low ICP with symptoms including severe headaches.

Incorrect drainage is often the result of blockage, either of the catheter (in the ventricles) or of the tubing (more common in the stomach than the heart). The former is more common and is a result of a build up of the cells that produce CSF in the holes of the catheter. This results in a pressure difference across the valve, to which it responds, different to that between the ICP and outlet (atmospheric), which is what should be regulated.

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[1]We measure pressure relative to atmospheric, so that atmospheric pressure is zero.

Pentriculo-peritoneal shunts may also introduce a variable outlet pressure. While any drainage into the heart occurs at close to atmospheric pressure, pressure in the abdomen is less constant and can be strongly affected by digestion. Anti-siphoning devices mediate the problem of negative pressure, but not other variations, and the extended use of such shunts (see section 5.1.3) has seen an increase in such malfunctions. Since the consequences of incorrect drainage are very severe, it is important that any shunt malfunction is quickly diagnosed and corrected.

5.1.6   Malfunction diagnosis and patient monitoring

The symptoms of under or overdrainage include headaches, visual disturbance, vomiting, fits, behavioural changes and concentration deficits. These may present in any combination and make diagnosis difficult. Psychological differences in particular may not be noted as significant in growing children and teenagers, yet these are the individuals going through the most significant physical changes.

Ventricle size can be monitored using CT scans, but the number of these should be minimized due to the use of radiation and are therefore not desirable for regular use. MRI scans are also now an option, but are expensive and time consuming and not readily available. The main problem with this approach is that ventricle size is not critical, and may vary significantly between individuals. Thus regular measurements are required for comparative analysis, and need to be able to detect subtle changes, especially in developing individuals.

Of primary importance is the regulation of ICP, usually in the range of 100-180 mmH_2O in healthy individuals. This can be measured by the insertion of a catheter into the ventricles, a highly invasive procedure. These are currently available either as fluid filled tubes which measure pressure remotely, or the more advanced version with a tip mounted pressure sensor. These are often inserted into patients in critical care, and may be left in for about a week to provide a continuous measurement. For longer term monitoring, however, a new hole would have to be introduced in the skull and through the brain every time, making it an undesirable option. The use of timpanic membrane displacement as a noninvasive and harmless measure of ICP has recently been developed, but is currently not widely available (see [1]).

For individuals with a shunt inserted, pressure measurements may be taken in the reservoir just outside the skull. This may be useful in determining whether the valve is operating at its set level, but may not be an accurate reflection of the pressure inside the skull if there is any obstruction in the catheter. It is, however, the only option that casuses no intercranial damage. In the following section, we develop a model that focuses on optimizing the use of this measurement for the successful diagnosis of shunt malfunction.

It should also be noted that the validation of satisfactory operation is as important as the detection of a failure. The replacement of a shunt, which usually means the entire system as a single unit, involves major surgery which carries a not inconsiderable mortality risk.

5.2   Model

We will now develop a simplified model of the shunt drainage mechanism, noting that it may be reasonable to measure pressure in the shunt reservoir outside the skull but that, at present, routine measurements of true ICP are not possible. Of diagnostic importance is the detection of a malfunction, caused by a blockage or otherwise, which results in incorrect drainage and hence dangerously high or low pressure in the ventricles.

5.2.1   Fluid flow

The amount of excess CSF flowing through the shunt will be no more than the total amount of CSF produced, usually 14-36 (mean 20) ml/hr. This flow rate of 3.8-10$\times 10^{-9}$ (mean 5.6 $\times 10^{-9}$) m^{3}s^{-1} has, assuming Poiseuille flow down the pipe[2] which has a typical diameter of approximately 1mm, a maximum velocity of 1.0-2.5$\times 10^{-2}$, (mean 1.4$\times 10^{-2}$) ms^{-1} with a pressure gradient of 4.0-10.4 (mean 5.8) mmH$_2$O (4.0-10.1$\times 10^5$, mean 5.6$\times 10^5$, Pa). The pressure drop due to flow in the pipe, typically less than 1m in length, is therefore considerably less than the pressure difference between ICP and atmospheric pressure. This is primarily induced by the valve and any other blockage, and we therefore neglect pipe resistance at this stage.

   We define Q to be the net excess CSF produced (that produced less that which is absorbed) and q as the flow through the shunt. The latter may of course be zero if the valve is closed. Since CSF is virtually incompressible, the rate of change in total ventricle volume V_v is

$$\frac{dV_v}{dt} = Q - q. \tag{5.1}$$

5.2.2   Brain compliance

Because the skull has a fixed volume (except for infants, for whom visible extension may occur, see section 5.1.2), any increase in ventricle volume must be compensated for by a decrease in brain volume. The volume of the ventricles is therefore dependent on the pressure inside the skull; more precisely, the pressure inside the ventricles P_v and also any difference from ICP. Although pressure in the skull is relatively uniform, we should not neglect the effect of arterial pressure, P_a, which introduces a strongly time dependent component. Ventricle volume will therefore be a function of the difference between ventricle and arterial pressure, $V_v \equiv V_v (P_v - P_a)$, with the precise relationship determined by the compliance of the brain.

   The relationship between cerebral pressure and volume is complex, especially at extreme values (see, for example, [2]). For our model we assume a linear relationship of the form

$$V_v = V_0 + k (P_v - P_a) , \tag{5.2}$$

which is a reasonable approximation for values that do not deviate dangerously far from the optimal range. More complex forms capable of modelling the entire pressure spectrum could be introduced at a later date, although these may require the numerical solution of the resulting equations. We have obtained analytical results for the more accurate

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[2]The Reynolds number is small.

logarithmic relationship

$$V_v = V_0 + \ln\left(\frac{P_v - P_a + k}{k}\right),$$

but these are algebraically complex and we will not discuss them here.

5.2.3   Vessel obstructions

We consider a blockage with simple resistance r_b in the catheter, the most common place for occlusion to develop. This could also represent a blockage at the outlet (or anywhere else) since this also induces a reduced pressure drop across the valve compared to true ICP. Since a shunt is a single unit it is not important to determine exactly where a fault has occurred, since any rectification involves the replacement of the entire' system.

If P_r is the pressure in the reservoir, any flow q through the (partially blocked) catheter takes the form

$$q = \frac{P_v - P_r}{r_b}, \tag{5.3}$$

provided flow is possible (in other words, the valve is open; see next section).

5.2.4   Shunt valve dynamics

There are many different types of valve on the market, and these have a variety of flow rate responses to pressure differences. Some of these are highly nonlinear, such as the Orbis-Sigma valve discussed above (see section 5.1.3), and are dependent upon the valve mechanism involved. The majority of valves, however, have a quasi-linear (nonlinear for only very low flow rates) response, and it is these that we consider here. Thus there is a straightforward resistance r_v to flow when the valve is open, above some critical closing pressure P_c. Note that this may be less than the pressure P_o required to open the valve, due to, for example, stiffness in membrane valves. Flow through the valve is given by

$$q = \begin{cases} 0, & \text{valve closed,} \\ \dfrac{P_r - P_c}{r_v}, & \text{valve open.} \end{cases} \tag{5.4}$$

Note that we have assumed that the outlet pressure at the valve is atmospheric.

5.2.5   Pressure variations

As mentioned previously, our model will assume that the patient is lying down (standard when monitoring measurements are taken). The use of anti-siphoning devices justifies neglecting the transient dynamics of standing and lying, while hydrostatic differences in the vertical position may be simply incorporated by scaling the relevant pressures appropriately.

Arterial pressure P_a is of course highly oscillatory, which is a phenomenon we exploit below. A typical heart rate is 1-1.5Hz, with each pulse having an amplitude of at least 20%. It is reasonable to assume that the baseline (mean) value remains relatively constant over the short time interval used for any diagnostic test. Should the frequency of the heart cycle prove too rapid, or the amplitude too variable, it is possible to induce more controlled oscillations into the baseline BP using a Lower Body Negative Pressure

Chamber (LBNPC) (see [3]). This allows for smooth sinusoidal variations with an amplitude of 5-10% to be imposed on arterial pressure, at any frequency below about 0.15Hz. Whichever oscillations we focus on, arterial pressure is simply modelled as variations with amplitude A and frequency ω, through

$$P_a = A_0 + Ae^{i\omega t}. \tag{5.5}$$

The full model system is shown in Figure 5.2. The dynamics of any flow are determined by the system of equations (5.1)-(5.5).
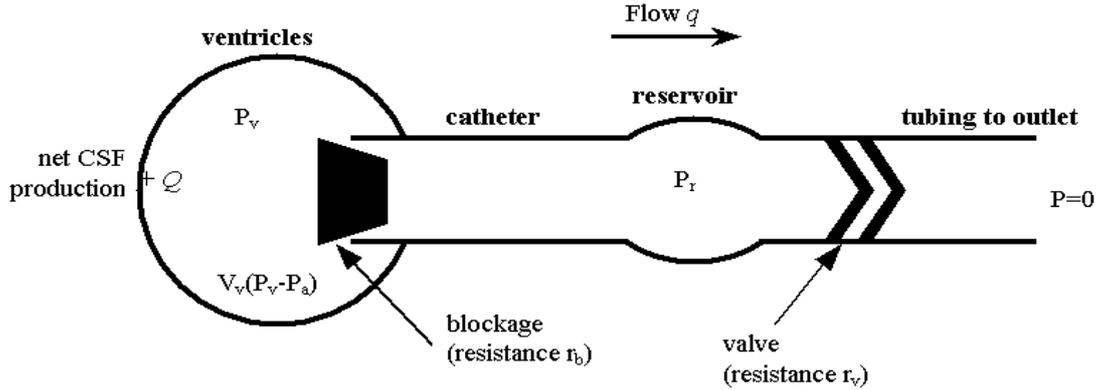

Figure 33: The model system neglects the relatively small pressure drop induced by the catheter and tubing. The only significant resistance to flow is given by the shunt valve and any possible blockage.

5.3 Solutions

Because of the modelling assumptions we have made, we can derive explicit solutions of the governing equations. We note, however, that the equations appropriate to the clinical applications that we describe below could easily be solved numerically, so that more complex expressions could be incorporated into the model. In particular, our expression for brain compliance (5.2) could be modified to make it valid for all physiological pressure variations.

5.3.1 Steady state dynamics

Since the valve, once opened, remains open for any flow rate $q > 0$, the system always converges to an equilibrium solution $q = Q$, provided there is a net excess of CSF production ($Q > 0$) and that the volume oscillations are not too large. By this we mean oscillations which induce a pressure drop in the reservoir below the valve closing pressure: a condition for this to hold may be derived from the solution (5.13) by considering $P_r \geq P_c$. For simplicity we show convergence to the equilibrium for the case where any volume oscillations are neglected ($A = 0$). As we shall see, these oscillations can simply be superimposed (provided $P_r \geq P_c$), so that the solution oscillates about the equilibrium point in the pressure-volume state space rather than simply asymptoting monotonically to equilibrium.

If we consider a valve which has just shut ($P_r = P_c$, $q = 0$) at $t = 0$, it will remain shut until $P_r = P_o$. Differentiating (5.2) when $q = 0$ and substituting into (5.1), we have

$$\frac{dP_v}{dt} = \frac{Q}{k}.$$

Since $P_r = P_v$ when there is no flow, it follows that

$$P_r = P_v = \frac{Q}{k}t + P_c$$

while the valve is closed. The valve reopens once sufficient fluid has built up in the ventricles to raise the pressure to P_o. This occurs when $t = t_o$ where

$$t_o = \frac{k}{Q}\left(P_o - P_c\right).$$

When the valve opens, since V_v, the volume of the ventricles, cannot change instantaneously, we have $P_v = P_o$. The flow is now governed by the valve and blockage resistances through (5.3) and (5.4), and hence P_r must change instantaneously to

$$P_r = \frac{P_c r_b + P_o r_v}{r_b + r_v},$$

as shown in Figure 34. The solution then converges exponentially fast to the equilibrium solution,

$$(q, P_r) \to (Q, Qr_v + P_c) \quad \text{as } t \to \infty, \tag{5.6}$$

along the line given by the valve resistance,

$$q = P_r - P_c r_v.$$

This behaviour is also shown in Figure 34. In addition, (5.3) implies that

$$\lim_{t \to \infty} P_v = Q(r_v + r_b) + P_c. \tag{5.7}$$

In fact, the equilibrium solution

$$(q, P_r, P_v) = (q^*, P_r^*, P_v^*) \equiv (Q, \ Qr_v + P_c, \ Q(r_v + r_b) + P_c) \tag{5.8}$$

given by (5.6) and (5.7) is a global attractor for all systems with positive net CSF production ($Q > 0$). We give a full derivation of the solution in the next section.

Note that whilst the equilibrium pressure in the ventricles, P_v^*, is dependent on any blockage (measured by the resistance r_b), that in the reservoir, P_r^*, is not. Thus any static clinical measurement taken in the reservoir, as opposed to taken invasively in the ventricles, will only help to evaluate the performance of the valve and not detect any malfunction due to occlusion.
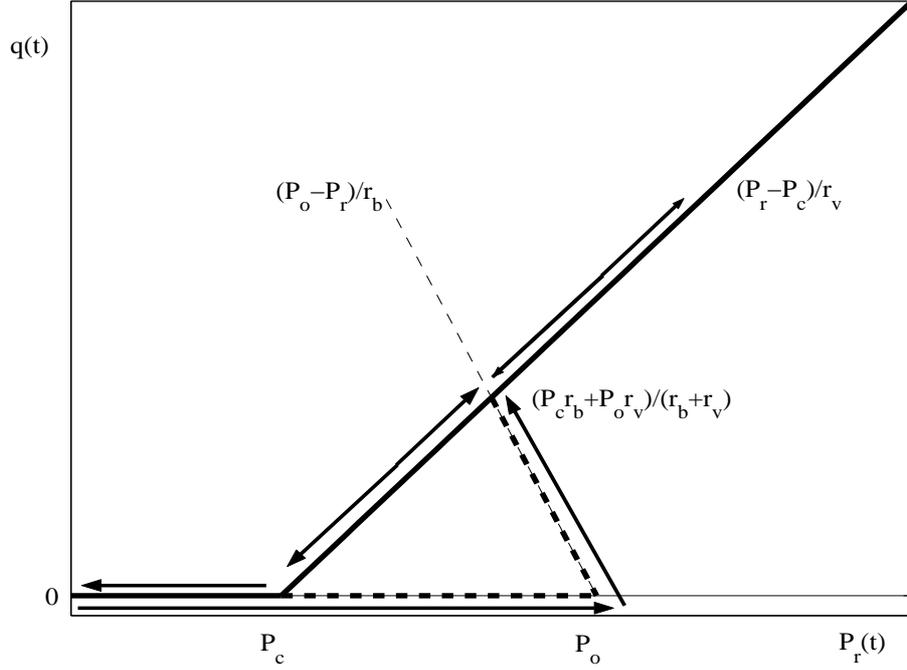
Figure 34: The dynamics of the system for positive CSF production (in the absence of volume oscillations) result in global convergence to the equilibrium solution, (5.8). If there is no blockage ($r_b = 0$) the jump from zero to positive flow as the valve opens is vertical. As $r_b \to \infty$, the starting value for flow tends to zero since, in the limit, no flow is possible. Once upon the open valve flow line $q = (P_r - P_c)/r_b$, the solution moves up and down dependent on the equilibrium flow rate $q = Q$.

5.3.2 Blockage diagnosis

In this section we consider methods of detecting the presence of any obstruction in the catheter by evaluating the resistance r_b. We will try to make use of the periodic variations in volume induced by arterial pressure, (5.5), and assume that there is some excess CSF flow so that the valve is open. As we showed above, the valve will always open eventually, provided that $Q > 0$.

We differentiate (5.2) and substitute into (5.1) to get

$$\frac{dP_v}{dt} = \frac{Q - q}{k} + i\omega A e^{i\omega t}. \tag{5.9}$$

By eliminating P_r from (5.3) and (5.4) we may substitute for q in (5.9) and derive a first order equation in the ventricle pressure,

$$\frac{dP_v}{dt} = \frac{Q}{k} - \frac{P_v - P_c}{\tau} + i\omega A e^{i\omega t}, \tag{5.10}$$

where

$$\tau = k\left(r_v + r_b\right) \tag{5.11}$$

is a constant with dimensions of time. Note that the more severe the blockage, the greater the value of r_b, and hence the longer the timescale τ. The solution of (5.10) with initial

condition $P_v(0) = P_0$ is

$$P_v(t) = P_c + (r_v + r_b)Q + \frac{\mathrm{i}\omega A}{\mathrm{i}\omega + \frac{1}{\tau}}e^{\mathrm{i}\omega t} - \left\{ P_c + (r_v + r_b)Q - P_0 + \frac{\mathrm{i}\omega A}{\mathrm{i}\omega + \frac{1}{\tau}} \right\}e^{-\frac{t}{\tau}}, \quad (5.12)$$

which, using (5.3) and (5.4), implies that

$$P_r(t) = \frac{r_v P_v + r_b P_c}{r_v + r_b}$$

$$= P_c + r_v Q + \frac{r_v}{r_v + r_b}\frac{\mathrm{i}\omega A}{\mathrm{i}\omega + \frac{1}{\tau}}e^{\mathrm{i}\omega t} - \frac{r_v}{r_v + r_b}\left\{ P_c + (r_v + r_b)Q - P_0 + \frac{\mathrm{i}\omega A}{\mathrm{i}\omega + \frac{1}{\tau}} \right\}e^{-\frac{t}{\tau}}. \quad (5.13)$$

and

$$q(t) = \frac{P_r - P_c}{r_v}$$

$$= Q + \frac{1}{r_v + r_b}\frac{\mathrm{i}\omega A}{\mathrm{i}\omega + \frac{1}{\tau}}e^{\mathrm{i}\omega t} - \frac{1}{r_v + r_b}\left\{ P_c + (r_v + r_b)Q - P_0 + \frac{\mathrm{i}\omega A}{\mathrm{i}\omega + \frac{1}{\tau}} \right\}e^{-\frac{t}{\tau}} \quad (5.14)$$

We can see that the system converges to a periodic solution with mean equal to the equilibrium (5.8) and amplitude proportional to that of the arterial variations A.

Whilst the value of the mean pressure in the reservoir is again independent of any blockage, both the amplitude of the oscillations and the rate at which the solution converges to equilibrium are not, and can, in principle be used to evaluate r_b, and hence diagnose a blockage.

Convergence rate

If we filter out the high frequency component of our solution (equivalent to neglecting oscillations and setting $A = 0$), the timescale upon which any perturbed solution converges to the equilibrium (5.8) is τ, given by (5.11). This could be observed by measurement of either the flow or pressure in the reservoir, and does not require invasive measurement of P_v. Although the timescale τ is directly proportional to the blockage resistance, it is also dependent upon the brain compliance parameter k which is difficult to estimate. The practicalities of dealing with this are considered in section 5.3.3.

Oscillatory solution

After transient effects have become negligible, the system is periodic with frequency ω and mean equivalent to the equilibrium (5.8). The amplitude of such variations in the reservoir pressure is given by

$$B = \frac{r_v}{r_v + r_b}\frac{\omega\tau}{\sqrt{1 + \omega^2\tau^2}}A. \quad (5.15)$$

Since, when $r_b = 0$, τ is about 5 minutes, and will be longer for nonzero r_b, we have $\omega\tau \gg 1$ so that

$$B \sim \frac{r_v}{r_v + r_b}A. \quad (5.16)$$

Thus the amplitude reduction between BP and observed reservoir pressure P_r is directly dependent upon the resistance of any obstruction, with $B \to A$ as $r_b \to 0$ (no blockage) and $B \to 0$ as $r_b \to \infty$ (complete occlusion).

5.3.3 Summary of unknown constants

Although this is a simple model, there are still a number of important unknowns. However, most of these can be estimated, if dynamic measurements are taken at the time the shunt is implanted.

The resistance of the valve, r_v, is often available from manufacturers, in addition to which independent measurements have been made for a range of shunts [4]. Failing this, if the functional form of the behaviour of the shunt is known, the resistance can be calculated by taken flow and reservoir pressure measurements. For the valve that we have considered, we can use (5.4) to give

$$r_v = \frac{P_r - P_c}{q}.$$

When the shunt is first inserted we expect the catheter to be clear, so that $r_b = 0$. The timescale at the time of insertion, τ_i, over which the solution asymptotes to its equilibrium, therefore gives an estimate of brain compliance using (5.11), with

$$k = \frac{\tau_i}{r_v}.$$

If k is assumed to remain relatively constant in an individual, this value can be used in subsequent measurements to calculate whether any blockage has developed. Measuring the new convergence timescale gives, again by (5.11),

$$r_b = \frac{\tau}{k} - r_v.$$

Continuous measurement of the periodic solution allows the blockage resistance to be evaluated without reference to the physiology of the brain, which may be changing, especially in growing individuals. For BP variations with amplitude A generating variations in reservoir pressure with amplitude B we have:

$$r_b = \frac{A - B}{B} r_v.$$

5.3.4 Flow regulation

Given constant excess CSF production, the use of an Orbis-Sigma valve (as described in section 5.1.3) should be satisfactory. If there is a significant change in Q, however, the patient will experience a large change in pressure, which could be detrimental. This is because the shunt is primarily designed to maintain a constant flow rate, irrespective of any transient pressure changes. It should not be forgotten, however, that what we are ultimately trying to control is the pressure in the ventricles, that is, maintaining $P_v(t)$ at some ideal value P_v^i.

Our model shows that, primarily because it is not possible to take regular pressure measurements inside the ventricles, any shunt capable of dealing with variations in CSF

production must be capable of measuring flow as well as pressure. The valve closing pressure can the be adjusted to the appropriate level (see section (5.1.3) according to one of the following:

$$\begin{aligned} P_c &= P_v^i - (r_v + r_b)\, Q, \\ &= P_v^i - \frac{\tau Q}{k}, \\ &= P_v^i - \frac{\tau}{\tau_i} r_v Q, \end{aligned}$$

depending on which parameters are available and most reliable.

The only alternative to this is to take both initial and subsequent convergence timescales, and balance the equation

$$(\tau_i - \tau)\, P_c = \left(\tau_i P_v^i - \tau P_r\right).$$

This has the advantage of requiring only experimental measurements (P_r, τ and τ_i) in addition to the desired ventricle pressure P_v^i and the valve closing pressure setting P_c. The reservoir pressure P_r will of course vary as P_c is adjusted (in a way dependent upon the flow rate which we assume is unknown), so that this type of adjustment can only be done on a trial and error basis. Since changes will be monotone, however, even a simple automated system should quickly evaluate the required setting.

5.4   Conclusions

Simple modelling has revealed that much diagnostic information on shunt performance is available through current measurement methods. The use of pressure measurements in the reservoir to detect catheter blockage provides an alternative to the invasive and damaging procedures necessary for intercranial pressure measurements. This method does, however, require dynamic measurements, since static pressure readings reveal only the pressure inside the reservoir, which may be significantly different to that inside the ventricles.

The model can easily be extended to include more complex physiological descriptions through numerical simulation. As discussed above, a more generic model of brain compliance can be introduced, as well as valves with different response functions. At this point it is appropriate to also include the relatively small resistance of vessels, and the hydrostatic effects of sitting rather than lying down. These are not of qualitative interest (and have hence not been included above) but will add to the quantitative accuracy of any numerical results.

Since reservoir pressure measurements are usually carried by inserting a needle through the skin, each procedure must be carried out by a trained individual and carries a small risk of infection. It it therefore highly desirable to develop a shunt with a built in pressure sensor. Current technology is advanced enough that a small pressure membrane would be a negligible addition to the current reservoirs used. We would assume that the main problem in developing such a tool, that of data transmission through the skin, can also be overcome by, for example, induction. Alternatively, these could be used directly by an automated shunt to adjust the valve response.

To truly control and monitor shunt performance we require a measurement of both flow and pressure, but this does not appear to currently be an option. If it is possible to measure pressure in the reservoir and record results externally, it may be more appropriate to focus efforts on developing a pressure sensor sufficiently small for it to become part of the catheter. This could then measure ventricle pressure directly - the variable

we are ultimately trying to control, and return data via the catheter to the reservoir. Mathematical analysis has revealed that shunt operation is relatively simple to predict if certain driving forces are known. It is perhaps more important that engineering advances be made to measure these successfully in a way which does not require additional invasive procedures.

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