Laboratory for Process and Product Design


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Abstract
A finite element model of an artificial arterial pulse waveform (APW) flowing through an elastic tube was constructed using linearized differential equations. Computational data were exhumed from the model using computer-aided simulations with the intention of facilitating the creation of a new mathematical model of cerebral ventricular space dynamics in acute hydrocephalus. Causal relationships between intracranial pressures (ICP) affecting cerebral spinal fluid behavior and the APW were established by appealing to data from the model. The arteriovenous blood velocity wave deformation can be attributed, nearly completely, to the damping effects of normally noted fluid dynamic phenomena of incompressible media traveling through an elastic tube. Further accounting for the biochemistry of cranial auto-regulatory mechanisms will yield insignificantly small physiological alterations in the traveling velocity wave. Verification of the model's credibility is demonstrated through its ability to show the blood velocity wave leading the arterial pressure wave, and muting of pressure and velocity flow peaks which are qualitatively consistent with clinical data.

1. Introduction
Due to the exigency of most diagnosed cases of hydrocephalus coupled with the lack of an analytically comprehensive model detailing its effects on the brain, the need for an accurately predictive model based on readily available pressure data is clear. There have been numerous attempts to establish such a model, but they generally sort themselves into a dichotomy of errors. The first category of models do well at mimicking the steady state response of the cerebral ventricular space, and perhaps even during some cases of hydrocephalus, but fail to describe the more pathological experimental results which have been seen in many K9 studies, such as the enlargement of a single ventricle and the non-responsive nature of the other ventricles when only one choroid plexus is removed. The second class provides a fairly reliable account of these causatively unexplained responses, but are usually built using parameters that are not feasible in a biological framework, such as a vessel wall which is too ridged, or ventricles that are far more elastic than is found in the body.

Building this mathematical model of cerebral blood flow velocity (CBFV) in will allow future finite element models to link changes in ICP to various deformations of the input APW, which has been widely theorized as a contributing cause of the development of hydrocephalus. Some theories speculate that the cardiac pulsations are primarily responsible for ventricular distension in many cases. If it is true that cerebrovascular pulsatile effects are directly responsible for most of the ventricular expansion that has been noted in hydrocephalus, then it is of paramount importance that any future proposed model contain an explanation of how ICP is effected by arterial distension, and how the APW shapes the pulsation of CSF in the ventricular space.

By using a tank and flow model we are able to mimic CBFV through the arterial system of the brain by modeling force, mass, and fluid flow equations for incompressible media flowing through an elastic tube. Each tank of the proposed model is representative of a single elastic tube element, and is connected to its successors by a ridged section, in which the velocity wave remains constant. As more tank elements are added to the model, the process becomes less discretized and behaves more like actual arteries. Within each of these cylindrical tanks there is a distensible lid, modeled by second order
differential equations, which responds to pressure and flow rates. These lids represent the elasticity and compliance of the arterial walls. By inputting the appropriate physiological values for artery length, elasticity, blood viscosity, cross-sectional area, and other essential numbers we can accurately predict how the CBFV wave will travel into the brain, and further use that data as an input to the ventricular portion of the future models which will better define outcomes of hydrocephalus. This cerebral blood supply model is also useful for observing pressure shifts due to controlled, isolated stenoses of various formans within the ventricular space. Elastic tank and flow models, used here to model cerebral blood supply to the choroid plexus, are easily tailored for use in ventricular spaces to show pressure gradations in CSF movement. As, illustrative examples, computational pressure and velocity data are presented.

2. The Literature Search

A great deal of effort needed to be put forth into area research of existing models, relevant cerebral anatomy, auto-regulation, physiological parameters, systolic and diastolic flow rates and pressure variances, and other similar concerns. This serves as the groundwork for the model, and will help ensure that it yields biologically meaningful data.

2.1. Basic Cerebrovasculature

The brain itself accounts roughly for only 2% of total body mass, but it consumes 25% of all available oxygenated blood. Nearly 80% of blood flowing to the brain is carried through the carotid arteries, while the remaining 20% is brought up the spine in the vertebral arteries which merge to form the basilar artery at the base of the brain. The arterial pressure is approximately 15 times the venous pressure, providing most of the incentive for blood flow. Once inside the cranium, this large volume of blood is feed into the Circle of Willis where it is then redirected to many other branching arteries that ascend into the cranial cavity. These larger arteries, which consist in the Middle Cerebral, Posterior, Superior and Inferior Cerebellar, and Cerebral Internal Carotid artery, become narrow as they approach the blood brain barrier (BBB), a lattice of tightly woven endothelial cells that form a casing around the brain impermeable to blood, water, and CSF. Once blood reaches these narrow interfaces, it perfuses through the BBB via the small pial arteries, which freely communicate with much of the cerebrovascular tree, then collects in the venules and empties out of the parenchyma through the jugular veins.

Circumventricular organs, several of which protrude from the brain's surface, offer an alternate route for blood to percolate into the brain parenchyma. Their functions are thought to be eclectic, though no conclusive research as been compiled describing any of them in their entirety. These organs participate, in some way, with CSF flow regulation and communication, and boarder the 3rd and 4th ventricle spaces.

The anterior choroidal artery supplies the choroid plexus with blood, along with numerous other small arterial vessels. The blood then passes through the plexus and out into the venous structure by the arachnoid villa. These villa are actual projections of the arachnoid membrane itself into the venous sinuses. Both blood and CSF exit from the choroid plexus here, and drain into the venous system.

Many biological values were compiled to facilitate the mapping of the model to actual human physiology. Some data obtained are displayed in figure 1, and more are presented with the supplementary CD (SCD).
<table>
<thead>
<tr>
<th>Ascending Aorta</th>
<th>4-8 L/min blood; 122/75.4mmHg S/D BP; Elasticity: 0.0036mm²/kPa/mm*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carotid Artery</td>
<td>180ml/min each; 7.3mm diameter</td>
</tr>
<tr>
<td>L. Jugular Vien</td>
<td>8.2mmHg; 6.9mm diameter</td>
</tr>
<tr>
<td>Basilar Artery</td>
<td>380ml/min; 2.5mm diameter</td>
</tr>
<tr>
<td>Arachnoid Villa</td>
<td>1.9-108mm/s blood</td>
</tr>
<tr>
<td>Blood Viscosity</td>
<td>0.001Kg/m²s</td>
</tr>
<tr>
<td>Heart to Plexus</td>
<td>3.53m of arteries</td>
</tr>
<tr>
<td>Total CBFV</td>
<td>750ml/min; 46ml/(100ml/min) blood volume</td>
</tr>
</tbody>
</table>

**Figure 1:** Physical properties table. +: Although the elasticity value is documented in the literature as 0.0036, the arterial compliance value used in the model is slightly different. (9)

2.2 CBF and Pulsatile Dissents

In general, cerebral blood flow in arteries towards the brain parenchyma is pulsatile, while the blood returning to the vena cava from the brain is steady. (26) The venous system houses 65-70% of blood in the body, and nearly 80% of blood volume in the brain. These stipulations add significance to our model, which produces smaller peaks in the pressure and velocity waveforms as it travels through the system.

Bergsneider’s, and others, hypothesis includes the possibility that as complications arise in the ventricular system, which prevent the proper movement of CSF through its usual channels, turbidity in these venous cavities increases. (3) This pulsatility in flow will impede CBFV due to a greater resistance that incoming arterial blood will experience and inertial effects. Standard auto-regulatory mechanisms dictate that compensatory measures for retarded flow velocities include capillary distension. (19) If there is no ventricular mediation, the result of the vasodilation is an increase in ICP. Thus, Bergsneider concludes that hyperventilation, which might be a clinically implemented procedure, would be a detriment, considering that blood flow is precisely what the brain lacks here.

Another issue not accounted for in modeling might be the assumption of a standard parabolic wave morphology during flow. Blood flow, most notably in the carotid artery, exhibits an ‘M’ shaped wavefront which will undoubtedly yield greater resistance from the tube dynamics. (33)

Further disagreements have arisen upon investigation of the impact on CBFV from diffusion of nutrients and other hemodynamically transported particles across ultramicroscopic pores in the cerebral capillary bed. Our contact at neurosurgery, Dr. Penn, has speculated that the losses incurred by mass flow rates due to this effect are wholly insignificant to CBFV. (25) However, there are some theoretical arguments which may disagree. The claim is that while particulate movement out of the blood stream is not noticeable, the Starling Hypothesis states that a “no-slip” boundary condition be imposed at the vessel walls. (11) Such non-linear representations of capillary blood flow are more apt to pick up lost friction and other friction-like forces.

One further dilemma is the possibility of electrochemical regulation of CBFV. It has been documented that sympathetic nerve activity induces small changes in cortical blood flow. (15) This type of interstitial interference may offer unforeseen contributions to turbulent flow or artificial pressure restrictions. (24)

Arising concerns seem boundless in the literature, from cilia induced fluid movement, to complicated hormonal chemistry. Not every aspect can be accounted for, and consequently, only the most pronounced forces have been modeled.

2.3. Data Collection Reliability

This is a brief look at Speck’s paper in the accuracy and potential improvements in the way we obtain cerebral fluid velocity data. (30) This is important only in that it is that very data which will provide assurance as to the accuracy of our model.
The claim made by this paper deals with the relative advantages of the modern static method of data acquisition, wherein the evaluation occurs once before and once after the contrast agent has been infused into the subject, over the dynamic method in which the contrast agent is more globular and helps measure a signal over some course and time. The dynamic method has been preferred, however, new advances in the contrast agent which prevent significant diffusion and create a “truly intravascular contrast agent” may now pave the way for more cost effective and precise reading using the static method.(37)

Reliability concerns regarding data acquisition from our model using MATLAB’s Simulink program are all but inconsequential. Data was gathered using a tolerance of at most 1E-4, and a step size of 1E-4. Integration bugs that caused small spikes in the data were noted, but did not affect the overall shape of the wave. All simulations were run in duplicate to isolate any integration error. Though, figures 4, 9, 10, and 11 were graphed with a sample rate of 1E-2 in order to make the volume of data manageable, the curve shape was maintained.

3. The Mathematical Model

We consider wave propagation in a system formed of elastic tubes of finite length, represented by tanks described with second order differential equations. The application of Newtonian equations used to describe the pumping action of the tank lids yielded response times on the order of a millisecond.(34) Because the action of the tank lids are nearly instantaneous in comparison to initial forcing displacement on the tube, which corresponds to cardiac pulsations, the lid acceleration has been neglected. Each tank has a fixed radial value, and varies only in the direction of the lid, like a piston, which affords us the advantage of a linear model.

More comprehensive models which included much of the cerebral arterial system were considered in figures 18 and 19. However, such complexities demanded work beyond the scope of my tenure with this project, and are likely unnecessary for an accurate model of hydrocephalus.

3.1. Overview of Mathematical Modeling

For the iteration of our balance equations and the Navier-Stokes equations we employed the modeling software, Simulink. This software is coded in MATLAB, and is a commercially available subdirectory in the MATLAB program. Simulink allows us to use an intuitive, pictorial representation of the equations we employed, and readily lends itself to computations of a highly dynamic system in both the time and Laplace domain. A mechanical delineation of the mathematics we use in Simulink is expressed in figure 2.

3.2. Governing Equations and Conditions

The network of equations which control the finite element model are presented here:
to provide a foundation for the results we achieve in computational simulations.

The basic pressure balance looks like,
\[ \Delta \rho_f - \Delta \rho_f(t) - \Delta \rho_2(t) = 0 \]  
Equation 1

This defines the pressure drop from one tube element to the next, over the ridged connectors.

The wall dynamics equation takes the form,
\[(\rho_f A_i \delta) y'_i(t) + k_{\delta} y'_i(t) + k_u y_i(t) - A_i \Delta \rho_i(t) - k_A A_i(t) = 0\]  
Equation 2

The effluent flow rate from each tank is given by,
\[ A_i y_i(t) - q_f(t) + q_i(t) = 0 \]  
Equation 3

The second pressure drop equation, which accounts for pipe segment length and radius is,
\[ \Delta p_{f1}(t) - \frac{8 \mu l}{r_1^2} u_1(t) = 0 \]  
Equation 4

And finally, the overall conservation of flow balance is given by the equation,
\[ \int_0^t q_f(t)dt - A \sum_{i=1}^2 y_i(t) - \int_0^t q_e(t)dt = 0 \]  
Equation 5

3.3. Formulation of the Model

All of the balance equations that were used to build our model are represented by Simulink, our computational modeling program, as a system of block diagrams. Each block performs some mathematical operation, and each relation between blocks performs some further representative function.

We will consider the interactions of the larger subsystems first. The first tank, labeled ‘Tank 1’ in figure 3, is the first element that the pressure and velocity waveforms see as they travel through the tube. The tank’s effluent is measured using the scopes that are connected to the exiting

![Diagram](image-url)

**Figure 3** The highest perspective of the finite element model. Biological parameters used are given in figure 13. Each arrow indicates the order of operations on each value. Closer perspectives of all tanks and source functions are given in following figures. (Filename ‘tubetrial_viii’ on SCD.)

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‘streams’ of the first tank. Volumetric flow, piston distension, fluid velocity, and rates of pressure change can be measured after each tank. The math of the first tank and all following intermediates are illustrated in figure 8.

The input to the tube is comprised of two constituents. The first is the force of any initial pressure on the ends of the tubes, and because there is a negligible amount of initial occlusion in arteries the source responsible for this work, labeled ‘a’ or ‘anull’, is set to zero in most trials. This force is placed on each tank independently.

The second, more interesting arterial force is the entering volumetric blood flow velocity waveform, labeled ‘CBFV Waveform’. This wave mimics the hemodynamics of the blood supply to the choroid plexus, and is applied only as an input to the first tube element. Figure 5 offers the precise mechanics of the production of this wave. By using sine waves and pulse generators we have developed a feed that closely resembles those waves that Verdonck has designed based on his research.(29) Although most other models of the cardiac velocity wave appeal to oscillating characteristic responses of ordinary differential equations, the variance due to this discrepancy in our model appears insignificant. (We opted not to use similar equations because of the difficulty that Simulink exhibits in interpreting complex numbers.) By starting with a base sine wave, we can add additional waves to it to produce the desired peaks and various peak amplitudes. By multiplying the supplementary sine waves with pulse generators, which take time dependant values of either zero or one, we can effectively turn these waves on or off for portions of the main sine wave’s period. This allows for a delicate manipulation of each peak and trough in the wave, thus offering a close fit to observable wave patterns, as shown in figure 4.

Subsequent additions to this volumetric source wave include variability of force of a contracting heart, and pulse rate. Greater flexibility and more exacting control over such variables allows for a more accurate response from our blood wave to the onerous conditions imposed on the body due to hydrocephalus. It is often the case that strenuous bodily states induce a quickening of heart rate, and it would be a valuable to track the effects of such a reaction on ICP if this model can be linked to Cristian Tsakiris’ model formulations. (34) Appendix 1 outlines the procedures for controlling the source.

Within the subsystem labeled ‘Tanks i1-i14’ in figure 3, and shown in figure 7, are 14 more tube elements which perform sequential operations on the progressing wave. Each one of these tanks behaves identically to the first tank with the exception of their inputs. Each tank, i1 through i14, takes the previous tank’s flow rate as its input, not the initial cardiac wave. The final tank is contained in the subsystem labeled ‘End Tank’, and is shown in figure 6. Scopes that measure the velocity and pressure of the blood are used to

![Choroid Artery Blood Velocity Vs. Flow Time](image)

**Figure 4:** Arterial volumetric velocity wave created by our pulse generator. Figure 4 shows the internal mathematics used to produce of the wave. This flow rate is the initial feed to the finite element model, from which all data are collected. (Filename ‘WaveDataPlot’ on SCD.)
Figure 5: The source waveform for volumetric blood flow into the choroid plexus. Each of the three sinusoidal sources converges into a single wave, producing only one output to the tube. A version of the source capable of accommodating physiological changes is offered in figure 14.

Figure 6: View within the subsystem labeled ‘End Tank’ in figure 2. This represents the final element in the tube which takes the cumulative stretching of the tube and other balance equations as its input.

Figure 7: View within the subsystem labeled ‘Tanks i1-i14’ in figure 2. In each subsystem shown here the mathematics are regulated by the identical set of equations that regulate tank 1, shown in figure 4. The lid displacement value, which comes from output 1 of each tank, are summed and used as the input to the final tank to satisfy the overall balance equations.

Figure 8: View within each individual tank, from tank 1 through tank 15. Note that though each tank has five outputs, only two are used to gather new data, and to interact with the bulk system. The remaining three outputs function as a check process and as a window into the minutia of the simulation results.
collect data on the final conformation of the wave.

3.4. Physiological Oversights

Due to extensive auto-regulatory mechanisms, the brain is capable of stabilizing blood flow over a wide range of arteriovenous pressure differences, which is CBF’s primary motive force. From pressures as low as 60mmHg, up to steady pressures approaching 150mmHg, cerebral blood volume remains at values near 50ml/100g/min.(10) The predominant method for CBFV regulation is through the distension and occlusion of pial capillaries and cortical blood vessels within the brain parenchyma.(19) Thus, CBFV will face a variable impedance as a function of pial artery diameter, seen in figure 18.

Other normative factors in blood flow include carbon dioxide concentrations, and oxygen levels in the ventricles, choird plexus, and general parenchyma. ICP increases, due to overproduction or under-absorption of CSF, also function to control CBFV by narrowing the arteries. Many of these precautions serve to ensure that despite low cerebral perfusion pressures, the brain retains a steady oxygen and nutrient supply. The resistance to cerebral venous outflow is greater with increasing ICP.(23)

It is due to these subtle additions to the basic math that has lead to a dissenting voice in the scientific community as to weather or not it is appropriate to model cerebral hemodynamics with linear differential equations. It has been noted that a linearization of the more precise mathematics has little effect on pressure and velocity measurements due to the small functional amplitudes of the waves in question.(8)

4. Mathematical Modeling Results

The principle motive for the presentation of our data is to show that the metamorphosis of the velocity wave in cerebral arteries dampens in the body, as it proceeds to the venous system, the same way it does in our model. Once we have shown that this is the case, the cumbersome auto-regulatory biochemistry that takes place in the brain can be partially discounted, and the bulk of the theory can rely on theoretical computational fluid dynamics.

**Comparison of Calculated Venous and Arterial CBFV Waveforms**

![Comparison of Calculated Venous and Arterial CBFV Waveforms](image)

**Figure 9:** Computational results of cerebral blood flow velocity waveforms through tanks 2 and 14. The two waves are shown to underscore the flattening of the primary and secondary peaks of the waveform as the fluid passes through the tube. Filename ‘MutedWaveOverlay’ on SCD)
4.1. Volumetric Flow Wave Deformation

We have data that conclusively show the deciding factor in CBFV wave deformation is the elastic nature of the cerebral arteries. By using biologically consistent parameters, we are able to see the arterial wave degrade into what we would expect to see in the arachnoid villa without appealing to any auto-regulatory mechanisms.

After passing through 14 elements of the model and traveling 3.54 meters in distance, the blood flow looks astounding like expected waves in the venous tree.(38) Figure 9 offers a perspective of the changing wave as it flows through the model.

After progressing through each tube element the wave becomes notably more muted, which would seem to indicate that these amplitude reductions are cumulative. The longer blood flows through the arteriovenous tubules, the less pronounced the wave’s peaks become. This is seen to be the case in clinical trials as well, indicating an accurate mapping of data gathered from this model to actual biological values. Figure 9 highlights the wave movement through the tube in such a manner, losing the well defined peaks as it flows through more elements in the tube.

4.2. Lagging Cardiac Pressure Wave

The heart acts as a pump by contracting its musculature to produce a large pressure wave that travels along the arterial network. The CBFV wave uses the APW as a vehicle to propelle itself through the vascular tree. The elasticity of the veins and arteries, however, is partially responsible for a delayed response of the blood’s movement through the tubes.(28) Unlike ridged pipes, pressure applied at a tube’s source is not felt instintaneously at its terminas. The walls of an elastic tube will resist the pressure by absorbing some of the pressure wave’s kinetic energy, which may be why in experimental measurements of hemodynamic pressure and velocity waves in subjects, the pressure wave is lagging behind the velocity wave.

Our mathematical model exhibits this lagging pressure wave characteristic. Figure 10 depicts the cardiac pressure wave coaxing the velocity wave through the body. Such data would seem to indicate a strong likeness between our model and actual physiological traits displayed by blood advancing from the heart to the parenchymal venules. This data, combined with other aforementioned demonstrated effects, endows the model with the credibility needed to foster a merging more complex models outlining that whole of the cerebral ventricular space.

While our model does reproduce this signature arterial particularity, it is presently unclear which aspect of our computational fluid mechanistic approach is responsible for this effect. While it is understood that this is noted in the body, the reason why such a feature must exist, and exactly what math describes it appropriately remains elusive.

4.3. Piston Distension

Simulink allows a wide breath of measurements to be gathered from the simulation. We are capable of measuring the stretch of the cylindrical tank’s lid as the
flow passes through it. Indeed, much of the past literature claims that at peak arterial systolic stress, major arteries, such as the aorta or internal carotid artery, dilate due to a rapid propagation of a large fluidic volume through these elastic tubes. (31)

As the peaks of the APW shrink, we expect to see smaller dilations accordingly. The natural radial extension in the internal carotid artery during peak systolic pressure is approximately 800\(\mu\)m in healthy young adults. (17) Our computational results are accurate at describing this phenomenon as indicated in figure 11. Because the area we model is deeper inside the brain parenchyma than the carotid artery, we expect an even smaller systolic variance, as the arteries will be less elastic and generally smaller in diameter. We achieve an extension of nearly 750\(\mu\)m after the media has traveled through the entire model. These data further establish the viability of using a finite element model to predict cerebral artery behavior.

5. The Physical Model

Efforts were made to construct a coarse physical reproduction of the pathophysiology involved with fluid movement and accumulation during hydrocephalus. This simple representation allows us to confirm that our ambitions of producing pressure drops between ventricular spaces by using fluid dynamic equations are worth while, in that it is at least possible to see such drops.

5.1. Physical Modeling Methods

By using standard items, 3-4mm diameter rubber tubing, water balloons, duct tape, electrical tape, a ten gallon aquarium, cooking oil, and various copper control valves we attempted to create the equivalent of the ventricular space as seen from a fluid dynamics perspective. Without taking care to achieve accurate biological values for elasticity and arterial dimensions, we used two balloons to simulate two ventricles, and a longer, larger tube for the sub-arachnoidal space. A visual aid of our physical model is provided in figure 12.

These balloons were connected to each other by two sets of tubing to allow fluid exchange between them. A ‘T’ junction led to the inflow which was closed upon filling the system to a steady state. To achieve a stable equilibrium, the balloons were filled to capacity just before they begin to stretch, thereby initially avoiding their elastic properties. Because the small difference in balloon elasticity manifests in large volumetric consequences, this method allowed each balloon to achieve the same approximate initial size.

![Lid Displacement Wave Propagation Through Elastic Tube Model](Filename 'LidDistensionGraph' on SCD.)
Inside the right balloon was another, smaller balloon which was separated from the water accumulating in the larger one. This smaller balloon was used to introduce pulsation into the system by filling it rapidly with air and subsequently deflating it through a third tube which did not enter the second ventricle, emulating the behavior of an active choroid plexus. The entire experiment was performed submerged in a water filled aquarium, representing ICP and elastic forces of the brain parenchyma.

5.2. Physical Modeling Results and Discussion

This experiment was executed with the intention of deciding whether a large pressure gradient across a small passage connecting two or more elastic chambers is a feasible mechanism to attempt to reproduce with our fluid dynamic equations. During the air-induced pulsations, the balloon with the smaller internal balloon expanded notably, but did not spill off its excess fluid into the second, vibrationless balloon as quickly or as completely as we had predicted. The pulsations induced by the first balloon on the second were markedly subdued. When distending the first balloon’s diameter by approximately 4-5cm, the vibrationless balloon saw a fluctuation of only 1-2cm. These results point to a deficiency in the coefficients of the

Figure 12: Schematic diagram of the physical model experiment. The diagram is numerically labeled to facilitate the description of the model in the text. 1-The elastic inlet tube which pulsed compressed air into the smaller orange balloon, and caused it to rapidly inflate and deflate. This system contains no water, and no air is ever leaked into the larger balloon, which is filled with water. 2-The smaller, orange balloon inside the larger right balloon, functioning as the choroid plexus. 3-The two larger, blue balloons meant to represent the left and right ventricles. These are filled with water and are submerged within an aquarium that functions as the sponge-like tissue of the brain parenchyma. 4-The large elastic tube that is exposed to atmospheric pressure outside the aquarium. Its meniscus is used as a pressure gauge for the system, and represents the sub-arachnoidal space. 5-A ‘T’ junction which connects both large balloons to each other, and to 4. 6-The oil-water udometer. This measures the pressure difference between the two blue balloons by the displacement of the oil.

Figure 13: Photograph of the double ventricle balloon experiment with induced asymmetric hydrocephalus in the right ventricle.
linearized equations currently being employed by Tsakiris. In his working model, obtaining substantial pressure variations across elastic pipe connections in 3mm tubes is difficult without extending the length of the pipe to increase its resistance. It may now be necessary to adjust his parameters so that the elasticity of the balloon takes a new value to account for its unwillingness to donate water to its neighbor balloon. Furthermore, the system dynamics observed serve as strong evidence that future models of cerebral ventricular space can be formed using fundamental fluid mechanics equations, because only elementary hydraulic principles were employed to manufacture these results. If this simple mechanical apparatus portrays the unstable ventricular volumetric equilibrium that Bering’s declared after his experiments, then surely these phenomena can explained without appealing to complicated auto-regulatory functions of cerebral biochemistry.

Some measurements were taken using the meniscus of the sub-arachnoidal space, which is exposed to the atmosphere, and an olive oil-water udometer that was constructed in this mechanical model to measure the pressure differences between the two balloons. A photograph of the udometer is shown in figure 15 and experimental data is shown in figure 14.

<table>
<thead>
<tr>
<th></th>
<th>Δl extended</th>
<th>Δp total</th>
<th>Δp oscillation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right Balloon</td>
<td>4-5cm</td>
<td>14cm</td>
<td>+/- 0.75cm</td>
</tr>
<tr>
<td>(3) (50cc vol)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left Balloon</td>
<td>1-2cm</td>
<td>14cm</td>
<td>+/- 0.75cm</td>
</tr>
<tr>
<td>(50cc vol)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Small Pulsing</td>
<td>3cm</td>
<td>14cm</td>
<td>+/- 0.75cm</td>
</tr>
<tr>
<td>Balloon (2)</td>
<td>(20cc vol)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Large External</td>
<td>n/a</td>
<td>12.5cm</td>
<td>+/- 1.50cm</td>
</tr>
<tr>
<td>Tube (4)</td>
<td></td>
<td></td>
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</tbody>
</table>

Figure 15: Water-oil udometer used to measure pressure variance between the two balloons in the mechanical model. The udometer is part of the closed ‘ventricular system’ of the model.

In a second case where the balloons were extended beyond their normal state into a moderately stretched position, there were other factors at work. It was shown that the balloons have a sort of memory of how they were filled, in that when hydrocephalus is induced via internal pulsation, either ventricle can be made to expand by having one begin with more water than the other. As the pulsations continue, and more pressure is placed on the balloon with...
greater liquid volume, this larger balloon tends to favor expanding itself rather than pushing excess water into its neighboring ventricle. When the sub-arachnoid space is then opened, allowing water run off to pass through it with one end open to atmospheric pressure, water will flow from the stretched balloon to this space, but will still be disinclined to travel into the unstretched balloon. One hypothesis is that the primary cause for this manner of transfer is that once the larger balloon expands, it becomes less ridged due to the nature of the rubber, while the other balloon remains un-stretched because that rubber offers more resistance to the initial stretch. Water flowing out through the sub-arachnoidal space indicates that junctions are not occluded, and water is free to move.

6. Conclusion

It appears promising to model tapering of the CBFV transfer function by fashioning a representative finite element elastic tube model. Our pulse rate and contraction strength dependant volumetric waveform may be combined with Cristian Tsakiris' working ventricular model in the future to produce a predictive composite, which may ultimately aid in the understanding and treatment of acute hydrocephalus. Although there are many biological oversights and contravening opinions sited from the literature, we believe that the data acquired from this model represents well the actual, experimentally measured cerebral hemodynamics. And while the mathematics was linearized for simplicity's sake, our computational simulations yield accurate results down to a very small order. Further research will be required to produce a truly comprehensive system that will offer a creative and insightful explication of the more elusive results obtained in laboratory experiments.

Acknowledgements

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References


*The design for this model was developed by working closely with the current version of Tsakiris’ own model, and we use identically similar equations within the specific tank elements, though the design as a whole is original.*
Appendix 1 (The cardiac pulse waveform generator)

There are 2 new variable components in this model.

1.) **To adjust the strength of the hearts pump**, increase the "Harder Cardiac Contraction Gain" value, which functions as a percent of the total amplitude. (i.e. a gain of 1.2 is 120% of the normal cardiac amplitude)

2.) **To increase heart rate**, the number of beats (periods) per unit time, the process is slightly more complicated. Take x to be your input value. If you wish the heart rate to be 150% of normal, take x to be 1.5. Input the x value into each of the 5 sources on the left. For each of the 3 sine waves, input the value in the field called “frequency”. The field should look like 4.713*x. In the 2 pulse generators change the field “period” to look like 2.665/x. Finally, in the generator labeled “Secondary Pulse Generator” you must input a value, y, into the field called “phase delay” to ensure the pulses are synchronous. The field should look like 0.68 – [y]. Obtain y from the rule of thumb equation: \( y = (0.1)x+2[(0.1)x-0.12] \). For example, in the case where \( x = 1.5 \) (a 50% increase in heart rate), \( y = 0.21 \). This adjustment is qualitative, and is performed to ensure a smooth wave. For normal operation or minimal increases, \( x \approx 1 \), disregard the rule and simply set \( y = 0 \).

![Figure 16: Adjustable source wave for the finite element model of an elastic artery. (Filename 'cardiacwavetrial2' on SCD.)](image-url)
Figure 17 represents numerous attempts to extend Simulink’s resources beyond its means. Most literature published in biomechanics journals appeals to a characteristic oscillatory response of a system of ordinary differential equations to act as the volumetric pulse wave that is seen by the arterial system, and created by the heart. However, such a response implies the use of transfer functions which are based on angular frequency and incorporate complex numbers into the exponentials. Simulink is ill equipped to handle complex equations, and the extent of the progress that was made using these models was done so by taking the complex resultant from the summing block, as a function of time, and then splitting it into its real and imaginary constituents. Once in this manageable, real form the hope was that it would then be subject to some function that would merge these two components to produce a real equivalent of the complex response. These efforts met with limited success, and in favor of continued toil in this area we opted to pursue a simpler, artificial approach, which diverges only slightly from these complex equations in computational results.

\[
Q(u) = Q_f e^{u} + Q_b e^{-u} 
\]

\[
g = 100 \times 10^9 \text{ N/m}^2 \text{ (Young's elasticity modulus)}
\]

\[
h = 0.001 \text{ m} \text{ (vesSEL thickness)}
\]

\[
p = 1050 \text{ kg/m}^3 \text{ (blood density)}
\]

\[
D = 0.00125 \text{ m} \text{ (local diameter)}
\]

\[
v = 0.3 \text{ (position ratio)}
\]
Appendix 2: (Original whole brain/body qualitative CBF model)

This model illustrates the primary path of cortical blood flow, which includes the Circle of Willis, the capillary bed shown as a variable resistor, and various other major cerebral arteries and communicating arteries. Blood flow to the choroid plexus from the choroidal artery and CSF/blood diffusion through the arachnoid villa occurs within the capillary system. This original qualitative model is intended to show the integral cerebral anatomy of blood flow, and factors which may control the pressure and velocity of incoming blood to the brain parenchyma.

Figure 18: An early model of blood flow to the brain incorporating many prominent cerebral physiological components.
Figure 19: A sketch of initial steps to build a tank and flow model of CBFV throughout the brain, relating to the choroids plexus.

Figure 19 is a representative sketch of whole body anatomy effects on CBFV from a position which would facilitate its transformation into a tank and flow model. The Circle of Willis is assigned a tank to minimize the complex math required to model it in great detail, and the entire parenchymal venous system is represented by a large distensible tank to illustrate the large volume of blood it contains. This model also provides a closer look at how the choroidal artery might be modeled according to a more physiological outlook.