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Research Article

The Cerebral Windkessel as a Dynamic Pulsation Absorber

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Abstract

The cerebral windkessel is the suppression of the arterial pulse in the cranium which renders capillary blood flow smooth. Arterial pressure and flow are normally synchronous, and (counterintuitively) the intracranial pressure (ICP) pulse slightly precedes the arterial blood pressure (ABP) pulse. Transfer function analysis of the ABP pulse to the ICP pulse shows a local minimum of amplitude response (a notch) at the heart rate, and abnormal intracranial dynamics attenuates the notch and shifts phase. I propose that these counterintuitive aspects of intracranial pulsatility may be understood by treating the cerebral windkessel as a designed system. On that basis, I here apply principles of reverse-engineering to model the ICP pulse first as a simple harmonic oscillator, then as a forced harmonic oscillator with one or two degrees of freedom. By including a model of the intra- and extra-capillary pathways, I show that ABP-ICP dynamics are characteristic of a dynamic pulsation absorber—a system of vibration suppression widely used in engineering. MRI flow imaging shows that this is accomplished by an arterial-cerebrospinal fluid (CSF)-venous pump. During systole, CSF links arterial expansion to venous compression. During diastole, CSF links venous expansion to arterial relaxation. Arterial pulsations pass through the CSF to the veins, and this transposition of the arterial pulse by venous compression and relaxation provides an elastic force that continuously opposes the radial motion of the capillary walls. This maintains the resonant dynamics necessary for efficient perfusion and the anti-resonant dynamics necessary for capillary protection. Maintenance of anti-resonant dynamics (crucial for preventing cerebral edema and capillary damage) requires a system of autoregulation of intracranial pulsatility, which the cerebral windkessel provides.

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INTRODUCTION

Nearly all cerebrospinal fluid (CSF) flow and cerebral arterial and venous blood flow is pulsatile [1-4]. Capillary blood flow is nearly smooth [5,6,7]. The pulsatility of the CSF closely resembles the pulsatility of the intracranial veins [8,9], both of which have some characteristics of an arterial pulse, including, under some circumstances, a dicrotic notch [10].

Many aspects of the pulsatility of intracranial blood and CSF are difficult to understand, particularly because the pulsatile flow occurs in a rigid cranium which places obvious constraints on pulsatile dynamics. How is it that capillary blood flow is smooth, whereas the blood flow in the intracranial arteries and veins—sometimes only millimeters away from the capillaries— is quite pulsatile [3]? Why does the pulsatility of the veins resemble the pulsatility of the CSF [9]? Why do the CSF and venous pressure pulse waveforms have some characteristics of an arterial pulse [10]? Why does the intracranial pressure (ICP) pulse normally *precede* the arterial blood pressure (ABP) pulse, but lag with intracranial hypertension [1,6,11-14] (fig 1)?

I propose that a useful approach to understanding these counterintuitive aspects of intracranial pulsatility is to consider the dynamics of the cerebral windkessel as that of a designed system. Such a system manifests design principles that accomplish specified goals, which for the cerebral windkessel is the buffering of arterial pulsatility—an unwanted 'vibration'—in cerebral blood flow, while at the same time maintaining optimal cerebral blood flow and minimizing energy dissipation. This approach to exploring intracranial pulsatility entails reverse engineering of the cerebral windkessel, in accordance with established engineering principles of vibration control.

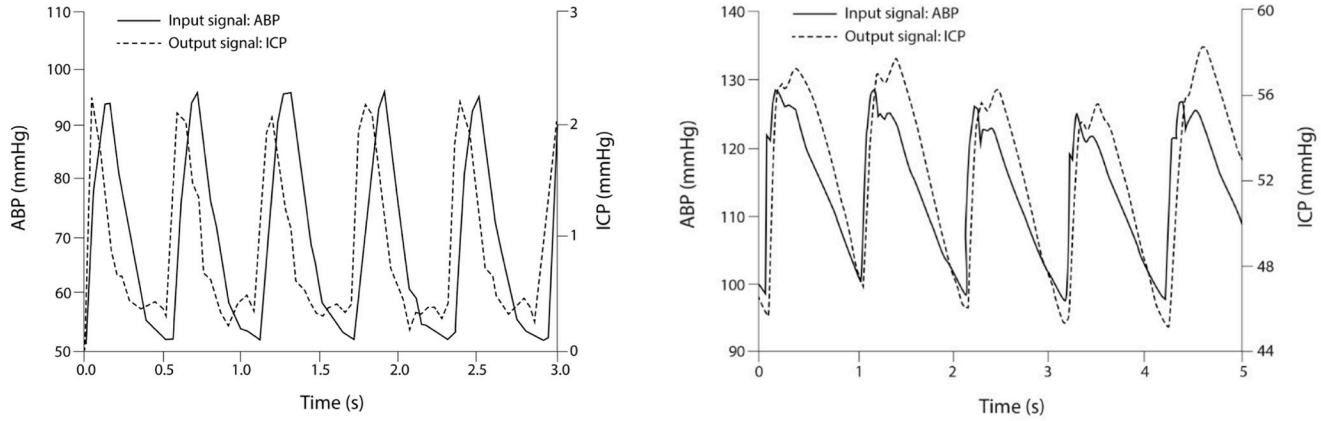

Figure 1. Phase relationships between the arterial pulse and the ICP pulse. Left: During normal dynamics, the ICP (dotted line) pulse slightly *precedes* the carotid arterial pulse (solid line) [1,6,11-14]. Wagshul et al found a lead of ICP with respect to arterial pressure averaging one-sixth of the cardiac cycle. **Right:** With elevation of ICP, the ICP pulse lagged the carotid arterial pulse. Such phase relationships cannot be explained if the ICP pulse is the transmission of the arterial pulse through the cranium, either as a bolus of blood through the capillaries or as a transmitted compression wave. The phase lead of ICP in normal dynamics and phase lag of ICP with intracranial hypertension suggest that the ICP pulse is a standing wave in the cranium that is excited by the arterial pulse. Standing waves can have leading or lagging phase relationships with exciting waves. Reprinted from Wagshul et al [14] with permission. **doi:**10.5048/BIO-C.2019.3.f1

CONSERVATION OF MASS AND ENERGY IN THE CRANIUM

The Monroe-Kellie doctrine, which is the traditional framework by which intracranial pulsatility is understood, is an assertion of mass conservation. The cranium contains three incompressible fluids, and an increase in one must equal a decrease in another, or intracranial pressure will rise. However, as a model of intracranial dynamics, the Monroe-Kellie principle of mass conservation is incomplete.

Energy, as well as mass, is conserved in the cranium. The energy inflow into the cranium must equal the energy outflow. The flux of energy associated with blood flow through the cranium entails exchange of inertial kinetic energy and elastic potential energy during the cardiac cycle, and like the flux of mass, it entails physiological constraints. The energy flux must not damage delicate tissues, and energy associated with blood flow must efficiently propel blood.

Energy flux in the cranium may be constant or change with time. Time-changing flux is pulsatile, and pulsatile blood flow endangers capillary beds and poses obstacles to cerebral blood flow. The means by which vascular pulsations are buffered and by which blood flow is optimized is the windkessel [1,14] which is present in all organs [7,15,16].

Because the vasculature is encased in a rigid cranium, the cerebral windkessel has unique properties. The purpose of this paper is to review the empirical evidence for the cerebral windkessel, to examine and model its properties in detail, and to explore its physiological implications.

THE EVIDENCE FOR THE CEREBRAL WINDKESSEL

Over the past two decades, investigators have studied the suppression of pulsatility in the cranium in some detail, and several characteristics of the cerebral windkessel have emerged.

Intracranial pulse amplitude is minimal in normal dynamics.

It is well known that the amplitude of the ICP pulse is at a minimum in normal dynamics, compared with the amplitude of the pulse in intracranial hypertension [1,17]. However, intracranial *hypotension* also raises ICP pulse pressure [18]. Normal ICP represents minimal pulse amplitude compared to both high and low mean ICP [19].

Our traditional understanding of pressure-volume relationships in the cranium does not provide an explanation for the paradoxical increase in ICP pulse amplitude at below-normal ICP, which should be smaller than the amplitude at normal ICP, in accordance with our understanding of the pressure-volume response of the cranium in a state of high compliance. It is remarkable that normal ICP pulse pressure is an extremum of low amplitude, compared with states of abnormally low compliance and abnormally *high* compliance in the cranium.

Brain expansion in systole with normal dynamics is minimal as well. Greitz measured brain expansion and arterial, CSF and venous volume displacement in normal controls during systole and diastole using flow MRI [3]. He found that brain expansion (the microvascular volume displacement) was ordinarily at

a minimum and was only two percent of arterial expansion. He found that in hydrocephalus, this brain expansion increases markedly. Brain expansion in the cardiac cycle is caused by microvascular volume shifts, so this represents direct measurement of the cerebral windkessel.

The arterial pulse diverts through the CSF to the veins.

Many investigators have observed volume conduction of the arterial pulse through the CSF to the veins, bypassing the capillaries. Bateman has demonstrated arterial-CSF linked venous compression in systole and re-expansion of intracranial veins in diastole [20-23]. Foltz has called this CSF-mediated venous compression and re-expansion that is synchronous with the cardiac cycle "venous volume venting" [18].

Normal pressure and flow waveforms in the cranium are nearly synchronous.

Using flow MRI, Greitz found that pulsatile CSF flow (at the foramen magnum), arterial flow, and venous flow are normally synchronous throughout the cardiac cycle and are in phase with the arterial pulse [3].

The normal ICP pulse slightly precedes the arterial pulse.

As noted, the normal ICP pulse slightly leads the ABP pulse. This was first demonstrated by Nitta [11], and has been observed by several investigators [6,11-14]. The lead is evident in the time domain (fig 1), and transfer function analysis shows a moderate leading phase of about 60 degrees at the heart rate frequency, which persists across most of the windkessel notch [14,24]. This of course is profoundly counterintuitive. In the cranium, it would seem that the output (the ICP pulse) precedes the input (the ABP pulse). This phenomenon is only explainable if we gain a deeper understanding of the ICP pulse and of the dynamics of the cranium.

Transfer function analysis of ABP pulse to ICP pulse shows a local minimum of amplitude response (the windkessel notch) at the heart rate.

Waveforms such as the ABP waveform and the ICP waveform can be decomposed mathematically into component sine waves of varying frequency, amplitude and phase, the sum of which is the original waveform. This process of decomposition of a waveform into its constituent harmonic components is called Fourier analysis. Notably, in an oscillator the harmonic motions represented by the Fourier components of the waveform are actual physical modes of oscillation in the system; real objects oscillate with a superposition of modes of vibration of discrete frequency, amplitude and phase.

Input of a waveform into a system produces an output which is also a waveform, and the ratio of input to output for each Fourier component (each sine wave) is called the transfer function and can provide important information about the system. The amplitude transfer function describes the amplitude response of the system for each Fourier component, and the phase transfer function describes the phase shifts the system imposes on the Fourier components of the input waveform to yield the output waveform. Analysis of waveforms without Fourier decomposition is called analysis in the 'time domain', and analysis of the waveforms by Fourier components is called analysis in the 'frequency domain'.

Transfer function analysis has been done on the cranium by several investigators, using the arterial pulse as the input and the ICP pulse as the output. It has shown a consistent local suppression of amplitude response—a notch—at the frequency of the heart rate [14,24-27]. This represents minimal pulsatility at normal dynamics. The notch is surrounded by two high amplitude peaks, roughly symmetrical, at higher and lower frequencies.

I will discuss transfer function analysis of the cerebral windkessel in more detail in the discussion of the mathematics of the damped windkessel.

Abnormal intracranial dynamics attenuates the notch

While the windkessel notch appears to be a feature of normal intracranial dynamics, several investigators have shown ablation of the windkessel notch in animals with elevated ICP [14,27] and restoration of the notch with restoration of normal ICP [14]. Ablation of the windkessel notch has been observed in hydrocephalus in animals [27] and humans [26].

Abnormal intracranial dynamics shifts phase, in accordance with the mathematical description of impedance phase.

Wagshul et al [14] (fig 1) have demonstrated changes in phase in the time domain and in the phase of the fundamental (heart rate) harmonic in the frequency domain associated with alteration of ICP. The phase shift is a lead of the ICP with intracranial hypotension and a lag of the ICP with intracranial hypertension. This demonstrates that the phase relationship between the ICP pulse and the ABP pulse is not fixed but varies in a consistent way with alterations of intracranial dynamics.

THE ICP PULSE AS A HARMONIC OSCILLATOR WITH A SINGLE DEGREE OF FREEDOM

The windkessel is an essential feature of capillary circulation [7,15], and it is clear from multiple lines of evidence that it exists in the cranium [1,3,5,6,20-27]. The cerebral windkessel has distinctive features, and careful consideration of the nature of the ICP pulse and an exploration of the oscillatory properties of the cranium—including some properties that are profoundly counterintuitive— suggests that a specific kind of dynamics underlies it.

Fundamental to our understanding of windkessel dynamics is an understanding of the physical nature of the ICP pulse itself.

The ICP pulse

In ordinary clinical practice, the ICP pulse is generally understood as the propagation of the arterial pulse through the cerebral vasculature. Although this seems intuitively correct,

it is mistaken. The cerebral windkessel effect minimizes the propagation of the arterial pulse through the microvasculature, so there is no 'bolus' of blood traversing the brain parenchyma that would give rise to a pressure wave. This is clearly seen at surgery when the cortex is incised. At the capillary level, blood flows smoothly as a gentle seepage, not in a pulsatile fashion. Furthermore, as noted above, the normal ICP pulse slightly *precedes* the arterial pulse [1,6,11-14], obviously ruling out the hypothesis that the ICP pulse is the propagation of the arterial bolus of blood through the microvasculature.

Some investigators have instead regarded the ICP pulse as the nearly instantaneous transmission of the arterial pressure compression wave through the cranium, similar to the propagation of a wave in water or a sound wave in air [28]. There are problems with this view as well [6].

The amplitude of a traveling pressure wave decreases on the inverse of the distance from the source [29]. If the ICP pulse is the transmitted arterial wave, the amplitude of the ICP pulse should vary inversely with the distance from a strong source of the arterial pulse—i.e. the amplitude of the transmitted ICP pulse in the cranium should vary considerably with location. For example, the amplitude of an ICP pulse measured in the brain parenchyma one centimeter from the carotid bifurcation should be ten times larger than the amplitude of an ICP pulse in the brain parenchyma in the centrum semiovale, ten centimeters away. This effect is not observed. The amplitude of the ICP pulse is roughly the same throughout the cranium, regardless of the distance from a strong arterial source.

In addition, traveling pressure waves reflect off surfaces, which is the principle on which ultrasonography is based. Given the many complex surfaces in the cranium, the ICP pulse (if it were a transmitted wave) would be expected to be 'granular' with respect to location, with hyperechoic and hypoechoic regions near surfaces. Such an effect is not observed.

Furthermore, the transmission of pressure wave in water (the contents of the cranium are mostly water) is very fast—4,900 feet per second [6]. Thus, if the ICP pulse were a transmitted pressure wave, it should be synchronous with the arterial wave that generated it.

However, the ICP wave is often quite *asynchronous* with the arterial wave. Investigators have found that the ICP pulse can occur significantly after the arterial pulse [6,11,14], by an interval that would not be consistent with a pressure wave traveling at 4900 feet per second in the cranium.

And of course, as noted above, many investigators have found that the ICP pulse can occur *before* the arterial pulse [1,6,11-14]. Clearly, the ICP pulse cannot be a transmitted arterial wave if the ICP pulse *precedes* the arterial pulse.

If the ICP pulse is not a bolus of blood traversing the microvasculature nor transmitted pressure wave, what is it? The cranium is a chamber with elastic content (the displaceable venous blood [17]) that is that is rhythmically driven by the heartbeat. Such dynamics gives rise to a *standing wave*, which differs considerably from a transmitted wave.

A standing wave is a stationary wave that oscillates but does not travel beyond the confines of the cavity that contains it. It consists of two superimposed waves—a wave excited by an external force and a wave reflected by the elastic contents of the cavity. Elastic cavities—such as hollow musical instruments—typically produce standing waves inside their chambers. The sound wave inside a violin or a clarinet is a standing wave.

It is important to recognize, furthermore, that the ICP wave is evoked in response to the ABP wave, but does not represent the ABP wave itself. The ABP pulse excites an ICP wave that is equal and opposite to it, in accordance with Newton's third law. The ICP pulse is a standing wave generated in the cranial cavity that is excited by the arterial pulse.

The ICP standing wave is the composite of three forces: 1) inertial force, which is generated by the opposition by the cranial contents to accelerated motion 2) the elastic force, which is generated by the opposition by the cranial contents to unaccelerated motion 3) resistive/damping force, which dissipates energy and is generated by the opposition by the cranial contents to all motion. Inertial force is generated by the ABP pulse and the ICP pulse, which are equal in mass. Elastic force is generated primarily from the compression and re-expansion of cerebral veins. Resistive/damping force is the combination of viscous resistance of fluids and structural damping of tissues. These three forces are generated by the cranial contents in reaction to the ABP pulse, and their sum is the ICP pulse. The ICP pulse is equal and opposite to the ABP pulse, in accordance with Newton's third law.

What does it matter that the ICP pulse is a standing wave, rather than a bolus propagated through the microvasculature or a transmitted wave? Standing waves have complex dynamics not inherent to transmitted waves. For example, the phase lead of the ICP pulse with respect to the ABP pulse [14] can be readily explained by consideration of the phase transfer function of the ABP pulse and the ICP pulse (discussed in more later in this review). The ABP pulse is composed of individual harmonics, which are sine waves (modes of oscillation) that have a frequency, amplitude and a phase. The cranium has its own natural spectrum of harmonics, which are not identical to the harmonics of the ABP pulse. The ABP pulse is an energy pump, and the cranium transfers the energy of the ABP pulse to the ICP pulse. The energy is transferred in the form of modes of oscillation of various frequencies, amplitudes and phases. The cranium has natural modes of oscillation that differ in frequency, amplitude and phase from the ABP spectrum, and the ABP spectrum excites and suppresses various modes of oscillation in the cranium. The dynamics are such that the cranium amplifies the leading phase components of the ABP pulse and shifts phase of the fundamental (heart beat) frequency to positive, thereby yielding a leading ICP pulse. Leading and lagging phases are characteristic of cavity resonators in the steady state driven by external pulsations.

The cranium is a cavity resonator—an elastic chamber that contains standing waves set in rhythmic motion by the arterial pulse. Cavity resonators exhibit such characteristics as phase, impedance, reactance, resonances, and anti-resonances, and have the potential to influence cerebral physiology in non-intuitive ways.

The ICP pulse and kinetic and potential energy

To understand the dynamics of the ICP pulse, an extension of the Monroe-Kellie doctrine is necessary. The Monroe-Kellie doctrine is a statement of mass conservation: mass entering the cranium must equal mass leaving the cranium, or pressure will rise. But whereas mass is conserved in the cranium, energy is also conserved. The ICP pulse is a cyclic exchange of kinetic and potential energy between the inertial and elastic forces in the cranium. It is only by the study of this energy exchange that the ICP pulse can be properly understood. Energy conservation in the cranium mirrors mass conservation: energy that enters the cranium is equal to energy that p19leaves the cranium, just as mass that enters the cranium is equal to mass that leaves the cranium.

The total energy associated with normal intracranial pulsatility remains constant; blood and CSF oscillate *in* the cranium via the exchange of kinetic and potential energy, and energy and mass flow *through* the cranium. The ICP pulse is the rhythmic steady state exchange of kinetic and potential energy.

Application of mathematics of harmonic motion to intracranial dynamics

It is useful to begin the exploration of windkessel dynamics with this question: to what extent is the application of the mathematics of harmonic motion applicable to a system as complex as the cranium and to phenomenon such as the ICP pulse?

As we will see, this simplification of pulsatile intracranial dynamics to harmonic motion is physically justified. Oscillatory motion in nature has a remarkable property: *for small amplitude and energy, which certainly applies to the ICP pulse, the mathematics of simple harmonic motion describes the dynamics quite well*. Simple harmonic motion in a vast variety of physical systems consists of relatively small periodic displacement of the system from equilibrium, followed by restoration and overshoot of that equilibrium. This is in a sense a 'potential well' in which the system is confined, analogous to a marble oscillating in the bottom of a round bowl. The restoring force is provided by energy exchange between potential and kinetic energy.

Nearly all potential wells encountered in nature are essentially parabolic for small displacements. The mathematical justification for this is easily seen. For a simple harmonic oscillator consisting of an oscillating mass and spring, the restoring (elastic) force for the simple harmonic motion is

$$F = kx$$

Where F is the elastic force, k is the spring constant (the elastance) and x is the displacement of the oscillator. The potential energy U_{SHM} of the simple harmonic motion is a function of displacement and is the work expended to compress or stretch the spring, and is given by:

$$U_{SHM} = \int_0^x kx\,dx = \frac{kx^2}{2}$$

Now consider the potential energy U_{ICP} of the ICP pulse, which is physically more complex than a simple harmonic oscillator. The potential energy of the ICP pulse is a function of displacement and can be expanded according to Taylor's theorem, which states that any function $f(x)$ that is continuous and possesses derivatives of all orders at x=a can be expanded in a power series in $(x-a)$ close to the region of $x=a$. All potential wells involving small displacements (e.g. the ICP pulse) can be described by functions that can be expanded in a power series.

Taking the equilibrium position of the ICP pulse as $x_{ICP}=0$, we expand the function $U_{ICP}(x_{ICP})$ for the potential energy of the ICP pulse around $x_{ICP}=0$:

$$U_{ICP}(x_{ICP}) = U_{ICP}(0) + x_{ICP}\left(\frac{dU_{ICP}}{dx_{ICP}}\right)_{x_{ICP}=0} +$$

$$\frac{x_{ICP}^{\ 2}}{2!}\left(\frac{d^2U_{ICP}}{dx_{ICP}^{\ 2}}\right)_{x_{ICP}=0} + \frac{x_{ICP}^{\ 3}}{3!}\left(\frac{d^3U_{ICP}}{dx_{ICP}^{\ 3}}\right)_{x_{ICP}=0} + \ldots$$

$U_{ICP}(0)$ is a constant and has no physical relevance to the ICP pulse, because the potential energy can be measured from any initial position and can be arbitrarily set to zero.

$$x_{ICP}\left(\frac{dU_{ICP}}{dx_{ICP}}\right)_{x_{ICP}=0}$$

is zero because the slope of the displacement curve of the ICP pulse at the bottom of the potential well is zero. We consider the first non-zero term of the Taylor expansion, and we note that the second derivative of the potential energy

$$\left(\frac{d^2U_{ICP}}{dx^2{}_{ICP}}\right)_{x_{ICP}=0}$$

evaluated at $x=0$ is a constant. We discount the higher order terms (with large factorials in the denominators), which is appropriate as long as the displacement x_{ICP} of the pulse is small. The potential energy of the ICP pulse is

$$U_{ICP}(x_{ICP}) \cong \frac{x^2{}_{ICP}}{2!}\left(\frac{d^2U_{ICP}}{dx^2{}_{ICP}}\right)_{x_{ICP}=0}$$

As long as the ICP pulse is relatively small, the constant

$$\left(\frac{d^2U_{ICP}}{dx_{ICP}^{\ 2}}\right)$$

for the ICP pulse is essentially the spring constant for a simple harmonic oscillator:

$$\left(\frac{d^2U_{ICP}}{dx_{ICP}^{\ 2}}\right) \cong k$$

Thus, the potential energy of the ICP pulse is approximately equal to the potential energy of a simple harmonic oscillator:

$$U_{ICP} \cong \left(\frac{d^2U_{ICP}}{dx^2{}_{ICP}}\right)\frac{x^2{}_{ICP}}{2} \cong k\frac{x^2}{2} \cong U_{SHM}$$

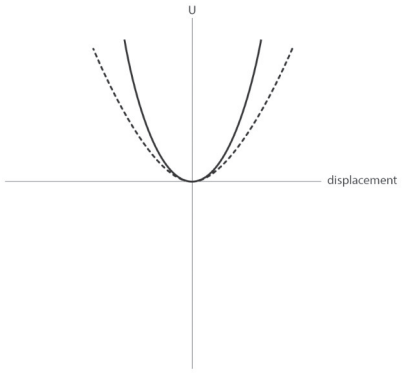

Figure 2. The ICP pulse and simple harmonic motion. The ICP pulse consists of the rhythmic exchange of inertial kinetic energy and elastic potential energy during the cardiac cycle. Kinetic energy of the cranial contents peaks in mid-systole and mid-diastole, and potential energy peaks in the transitions between systole and diastole and diastole and systole. This exchange of energy is oscillation in a potential well, akin to a marble oscillating in a round bowl. This figure shows the parabolic curve of the mathematical description of potential energy of a simple harmonic oscillator (solid line) and the schematic curve of potential energy of an ICP pulse (dotted line). The abscissa is the displacement of the oscillation and the ordinate is the potential energy U. For small displacement, the mathematical description of a simple oscillator is superimposable on the curve of the ICP pulse, meaning the potential energy well is nearly parabolic. **doi:**10.5048/BIO-C.2019.3.f2

Conceptually, we can say that a graph of the potential energy well of the ICP pulse plotted against displacement of the pulse is a reasonably good fit to the parabolic curve of a simple harmonic oscillator when the ICP pulse is small (fig 2).

This approximation of nature to simple harmonic motion applies to a large range of systems, including gravitational waves, water waves, masses attached to springs, oscillations of atoms in lattices, the flow of electrons in circuits, and quantum mechanical behavior of subatomic particles. The mathematics of simple harmonic motion can be applied with reasonable accuracy to the dynamics of the ICP pulse, because, for small displacement, all systems in oscillatory motion in a potential well conform approximately to simple harmonic motion.

We will use this principle to explore the dynamics of the cerebral windkessel.

Correspondence between the cerebral windkessel and mechanical and electrical oscillators

The subtleties of forced harmonic motion have been worked out in considerable detail for a variety of physical systems. There is a correspondence between the dynamics of a mechanical vibration absorber, a parallel "wavetrap" electrical circuit, and the cerebral windkessel, which, as we will see, is the organizing principle of pulsatile intracranial dynamics. These systems are described by analogous differential equations.

Pressure, flow and volume

Systems in forced oscillatory motion are driven to periodic displacement from equilibrium and return to equilibrium. The oscillatory motion occurs because of overshoot: the oscillator overshoots the equilibrium position, and restoring forces push it back to equilibrium, only to have the oscillator overshoot again. The externally applied force interacts with this displacement and restoration/overshoot in complex ways. This is common to all forced oscillators, including the ICP pulse.

All forced oscillators exhibit three interrelated variables: force, velocity, and displacement. In linear mechanical systems, force, velocity and displacement refer to the forces acting on the mass in oscillation, to the velocity of the mass, and to the displacement of the mass, respectively. In torsional mechanical systems, torque, angular velocity and angular displacement are the force, velocity and displacement variables. In electrical systems, voltage, current and charge are the force, velocity and displacement variables.

In the dynamics of the ICP pulse, pressure is the force variable, flow is the velocity variable, and volume (of the pulse) is the displacement variable. A proviso is noted regarding flow: for the purposes of the windkessel, flow refers to the radial motion of capillary walls and the radial motion of the cranial contents, not specifically to the longitudinal flow of blood and CSF, as flow is commonly understood. Longitudinal flow of blood and CSF of course bear relation to radial motion of capillaries and cranial contents, but the relationship is complex and is beyond the scope of our discussion. By "flow" in windkessel dynamics I will mean the radial velocity of expansion and relaxation of the vasculature and of the contents of the cranium. As will become evident, radial expansion of the vasculature during the cardiac cycle is of great importance in intracranial physiology.

It is important to note that this discussion applies only to the pulsatile forces and flow in the cranium. Smooth bulk flow of blood and CSF are obviously of great physiological interest, and are related to pulsatile flow (radial motion) of fluids and tissues, as we will see. But impedance refers to opposition to flow in a pulsatile system, and in this discussion we will limit the analysis of impedance to radial pulsatility of the cranial contents. This is appropriate, because the windkessel mechanism is a system that suppresses radial motion of the microvasculature.

The ICP pulse may be approximated, with increasing accuracy to the physical reality of the pulse, as a simple harmonic oscillator with one degree of freedom, or as a forced harmonic oscillator with damping with one degree of freedom, or as an undamped windkessel with two degrees of freedom, and or as a damped windkessel with two degrees of freedom. Degrees of freedom refer to the pathways through which the pulse can travel and specifies the number of equations necessary to describe the motion. Each level of increasingly accurate approximation and increasing complexity will be examined in the discussion that follows.

The simple harmonic oscillator

We begin the discussion of the dynamics of the cerebral windkessel by applying the mathematics of simple harmonic motion with one degree of freedom to the ICP pulse. We can think of the (nearly) harmonic motion of the ICP pulse as oscillation of the cranial contents induced by a radial displacement (expansion). The cranial contents relax, overshoot, and rebound in a periodic motion. We will discount the effect of the heartbeat for now and consider only the natural unforced 'vibration' of the cranial contents. We will assume no damping, so the vibrations of the cranial contents continue unabated. The equation of motion for the ICP pulse, to a first order of approximation, is analogous to the equation of motion for a mass m attached to a massless spring with spring constant k displaced to x and left to oscillate. There is a rhythmic exchange of kinetic and potential energy by the inertial and elastic forces in the cranium. The force of the elastic displacement and the inertial force are the only two forces acting, and they are equal and opposite in direction. The equation of motion of this simple unforced ICP pulse is

$$m_{ICP}\ddot{x}_{ICP} = -k_{ICP}x_{ICP}$$

where m_{ICP} is the mass of the ICP pulse (the mass of the fluid displaced), $\ddot{x}_{ICP}$ is the acceleration of the ICP pulse with respect to time, x_{ICP} is the displacement (volume) of the pulse and k_{ICP} is the elastance of the ICP pulse. The equation may be written as

$$\ddot{x}_{ICP} = -\omega^2 x_{ICP}$$

Where ω^2 may be understood as the restoring force per unit mass per unit displacement and

$$\omega = \sqrt{\frac{k_{ICP}}{m_{ICP}}}$$

represents the natural frequency of oscillation of the ICP pulse, expressed in angular units (radians/sec).

If we assume no damping, the ICP pulse will oscillate indefinitely at frequency ω, undergoing displacement, restoration, overshoot, etc. with period

$$T = \frac{2\pi}{\omega}$$

The solution to the differential equation for the motion of the ICP pulse is

$$x_{ICP} = x_0 \cos(\omega t + \varphi)$$

where x_{ICP} is a cosine wave with maximum volume displacement x_0, and φ is the phase angle of the displacement.

Forced harmonic motion and the ICP pulse

In the next level of accuracy and complexity, we will consider the ICP pulse as a forced harmonic oscillator with damping with one degree of freedom. The ICP pulse is driven by the arterial pulse and is equal and opposite to it. As such, the ICP pulse is the reaction of the cranial contents to the arterial pulse and is forced periodic motion. It is the composite of three forces:

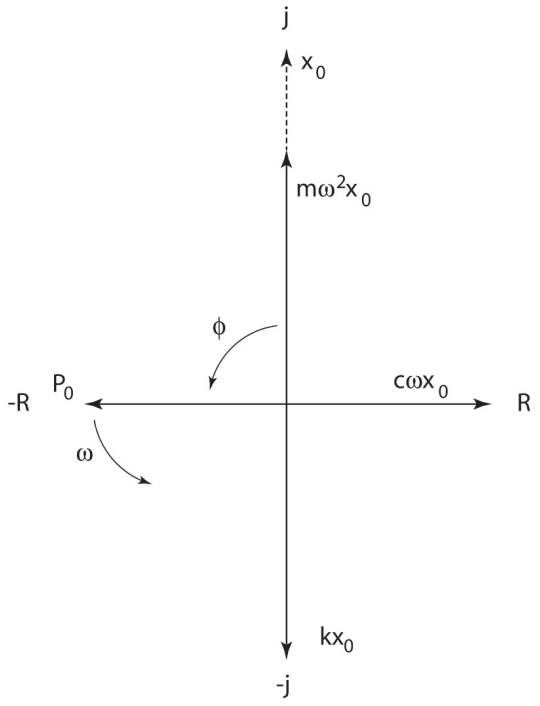

Figure 3. Phasor representation of the ICP pulse. The motion of the ICP pulse is driven by four forces, which can be depicted on the complex plane. Maximal displacement volume x_0 is a phasor pointing along the positive j axis at time t. Elastic force $-k_{ICP}x_{ICP}$ with amplitude $k_{ICP}x_0$ opposes displacement and is depicted as a phasor on the $-j$ axis. Damping force $c_{ICP}\dot{x}_{ICP}$ with amplitude $c_{ICP}\omega x_0$ is a quarter cycle ahead of elastic force, on the positive real axis. Inertial force $m_{ICP}\ddot{x}_{ICP}$ with magnitude $m_{ICP}\omega^2 x_0$ is a quarter cycle ahead of damping force on the $+j$ axis. The force of the arterial pulse $P_0 \sin\omega t$ is a phasor of magnitude P_0 located φ degrees ahead of the displacement.The phasors turn together counterclockwise at frequency ω, and the force can be taken as the sinusoidal reflection of each phasor on either the real (cosine) or imaginary (sine) axis. The ICP pulse is the sum of the inertial, damping and elastic forces, and is equal in magnitude and opposite in direction to the arterial pulse, by Newton's third law. The equation of motion for the system is $m_{ICP}\ddot{x}_{ICP} + c_{ICP}\dot{x}_{ICP} + k_{ICP}x_{ICP} = P_0 \sin\omega t$, which can be solved by applying Newton's third law to the horizontal and vertical components of the phasors (see text).
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inertial force, damping force and elastic force. The equation of motion for the ICP pulse, understood in its simplest form as a damped single-degree-of-freedom oscillator undergoing forced harmonic motion, is

$$m_{ICP}\ddot{x}_{ICP} + c_{ICP}\dot{x}_{ICP} + k_{ICP}x_{ICP} = P_0 \sin\omega t$$

where x_{ICP} is the displacement volume variable, m_{ICP} is the mass of fluid and tissue displaced by the pulse, c_{ICP} is the damping of the cranial contents, k_{ICP} is the intracranial elastance and P_0 is the maximal pressure of the ICP pulse.

There are several ways to solve this equation and explore the dynamics it represents, and for our purposes phasor

representation on the complex plane is most helpful (fig 3). We note that all four forces in the cranium (the arterial pulse and the three forces that comprise the ICP pulse) are roughly sinusoidal, in accordance with our analogy of the ICP pulse to forced harmonic motion.

The displacement volume of the pulse varies as a sinusoid and takes the form

$$x_{ICP} = x_0 \sin\left(\omega t - \varphi\right)$$

where x_0 is the maximal displacement (volume) of the ICP pulse, and φ is the phase of the displacement with respect to the force.

To solve for the displacement of the ICP pulse, we use the principle that harmonic motion can be represented as rotating vectors. To do so we use Euler's formula:

$$e^{j\omega t} = \cos\omega t + j\sin\omega t$$

and we can take either the real part $\cos\omega t$ or the imaginary part $j\sin\omega t$ as representing the waveform of the ICP pulse.

The four forces in the cranium will then be phasors rotating on the complex plane with frequency ω, which is the heart rate, and the representation of the force will be the sinusoidal reflection of the phasor on an axis. We denote the maximal displacement volume x_0 as a phasor pointing along the positive j axis at time t.

The elastic force $-k_{ICP}x_{ICP}$ is always opposing the displacement from equilibrium and will be a phasor of magnitude $k_{ICP}x_0$ pointing down the negative j axis, opposite the displacement phasor.

The damping force $c_{ICP}\dot{x}_{ICP}$ is proportional to and directed opposite to the velocity (flow), and is the first time derivative of the displacement. Differentiation on the complex plane multiplies by ω and rotates the phasor a quarter cycle counterclockwise, so the damping force phasor is along the positive real axis and has magnitude $c_{ICP}\omega x_0$.

The acceleration in the inertial force $m_{ICP}\ddot{x}_{ICP}$ is the first time derivative of the velocity and is rotated a quarter cycle counterclockwise from the damping force phasor. The inertial force phasor is along the positive j axis and has magnitude $m_{ICP}\omega^2 x_0$.

The force of the arterial pulse $P_0\sin\omega t$ is a phasor of magnitude P_0 located φ degrees ahead of the displacement.

To solve for displacement magnitude and phase, consider that by Newton's third law the phasor sum of forces in the cranium must at all times be zero, which means that the following equations hold:

Vertical:

$$k_{ICP}x_0 - m_{ICP}\omega^2 x_0 - P_0\cos\varphi = 0$$

Horizontal:

$$c_{ICP}\omega x_0 - P_0\sin\varphi = 0$$

Solving for displacement and phase, displacement magnitude is

$$x_0 = \frac{P_0}{\sqrt{\left(c_{ICP}\omega\right)^2 + \left(k_{ICP} - m_{ICP}\omega^2\right)^2}}$$

The dynamics can be made more clear by the introduction of dimensionless variables. The forced frequency ratio

$$\frac{\omega}{\omega_n}$$

is the ratio of heart rate to natural frequency of the ICP pulse and the damping ratio

$$\frac{c_{ICP}}{c_0}$$

is the ratio of intracranial damping to critical damping. Critical damping is the largest value of damping at which transient ICP oscillations would stop if the arterial pulse stopped and represents the inflection point between underdamping and overdamping in the cranium. The displacement magnitude can be expressed as

$$x_0 = \frac{\frac{P_0}{k_{ICP}}}{\sqrt{\left(1 - \frac{\omega^2}{\omega_n{}^2}\right)^2 + \left(2\frac{c_{ICP}}{c_0}\cdot\frac{\omega}{\omega_n}\right)^2}}$$

This equation for displacement magnitude is of great relevance in the understanding of intracranial pulsatility. Note that the displacement becomes very large when the heart rate ω nears the natural frequency ω_n, which is characteristic of a single degree of freedom system driven near its natural frequency. The displacement variable x_0 relevant to the windkessel is the radial displacement of capillary walls.

The normal synchrony of arterial pressure and flow waveforms [30] implies that for arterial blood flow in the cranium the fundamental natural frequency of the vascular tree is equal to the heart rate, as will be discussed below. This is necessary to optimize the efficiency of cerebral blood flow; asynchronous pressure and flow causes reflection of kinetic energy back to the left ventricle. However, this synchrony of arterial pressure and flow (when $\omega = \omega_n$) can cause maximal radial displacement of capillary walls, with potential to damage the microvasculature.

The displacement phase is

$$\varphi = \tan^{-1}\left(\frac{c_{ICP}\omega}{k_{ICP} - m_{ICP}\omega^2}\right)$$

or

$$\varphi = \tan^{-1}\left(\frac{2\frac{c_{ICP}}{c_0}\cdot\frac{\omega}{\omega_n}}{1 - \frac{\omega^2}{\omega_n{}^2}}\right)$$

Note that at $\omega = \omega_n$, the displacement phase is a quarter cycle behind the external force. In the cranium, in which the natural frequency of the vasculature is normally equal to the heart rate (i.e. the pressure and flow are synchronous) [30], the displacement volume waveform lags the pressure waveform and the flow waveform by a quarter cycle.

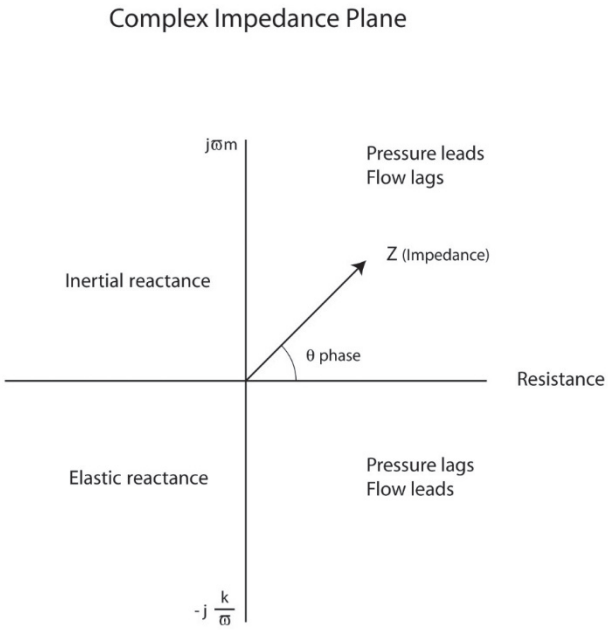

Figure 4. ICP pulse impedance on the complex plane. Impedance is the ratio of pressure to flow, and can be represented on the complex plane. Pressure pulse impedance is the sum of intracranial reactance and resistance. Reactance is the sum of inertial reactance ωm_{ICP}, which is the opposition to motion due to the mass of the cerebral systolic stroke volume, and elastic reactance $\frac{k_{ICP}}{\omega}$ which is the opposition to motion due to systolic compression and diastolic refilling of cerebral veins. Inertial reactance (increased by tachycardia) is represented on the $+j$ axis and elastic reactance (increased by bradycardia) is represented on the $-j$ axis. Damping is represented on the positive real axis. When inertial reactance and elastic reactance are balanced, impedance is purely resistive, and is an extremum of either suppression or augmentation, depending on the geometry of the oscillator. Normal dynamics in the capillaries corresponds to anti-resonance, which is an extremum of suppression. This protects capillary beds from pulsatility. Impedance has a phase as well as an amplitude. Impedance phase gives the timing relationship between pressure and flow. A positive impedance angle implies an excess of inertial reactance, and the pressure pulse leads the flow pulse. A negative impedance angle means an excess of elastic reactance, and the pressure pulse lags the flow pulse. An impedance angle of zero corresponds to synchrony (resonance or anti-resonance) between pressure and flow pulses.
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Impedance magnitude and phase of the ICP pulse

Because the ICP pulse velocity flow variable $\dot{x}_{ICP}$ is the first time derivative of the ICP volume displacement x_{ICP}, and differentiation on the complex plane is multiplication by ω and rotation counterclockwise by a quarter cycle, the maximal velocity of the ICP pulse can be written as

$$x_0\omega = \frac{P_0}{\sqrt{{c_{ICP}}^2 + \left(\omega m_{ICP} - \frac{k_{ICP}}{\omega}\right)^2}}$$

The radical in the denominator is the impedance $\vec{Z}_{ICP}$ of the ICP pulse, which is the instantaneous ratio between the ICP pulse pressure and the ICP pulse velocity:

$$\vec{Z}_{ICP} = \frac{\vec{P}_{ICP}}{\vec{F}_{ICP}}$$

where $\vec{P}_{ICP}$ is ICP (force), $\vec{F}_{ICP}$ is the flow (velocity) of the ICP pulse and $\vec{Z}_{ICP}$ is impedance, each expressed in complex.

The impedance to flow, as a complex number, has a magnitude and a phase, and can be represented as a vector on the complex plane (fig 4).

In polar form, impedance is

$$\vec{Z}_{ICP} = \frac{\vec{P}_{ICP}}{\vec{F}_{ICP}} = Z_{ICP}\angle\theta$$

where Z_{ICP} is the magnitude of the impedance of the ICP pulse and θ is the impedance phase of the ICP pulse. Note that the impedance phase θ is different from displacement phase φ.

Impedance magnitude is

$$Z_{ICP} = \sqrt{{c^2}_{ICP} + {X^2}_{ICP}}$$

ICP pulse impedance Z_{ICP} has two components: damping c_{ICP} and reactance X is

$$X = X_I - X_E = \omega m_{ICP} - \frac{k_{ICP}}{\omega}$$

Damping

Damping c_{ICP} of the ICP pulse is the opposition to flow that dissipates energy. Damping is the resistive force in the cranium, comprised of frictional energy loss due to the flow of blood and CSF and of structural damping as the cranial contents oscillate. In the vasculature, which generates much of the resistance to pulsatile motion, resistance is the dissipation of energy that is lost to work of pumping blood. Damping can take three forms: resistance associated with inertia, resistance associated with elastance, and resistance associated with the smooth flow of blood.

Resistance associated with inertia is viscous resistance to the motion of blood in vessels. Inertial resistance is similar to the kind of resistance that is ordinarily considered in the study of resistance to non-pulsatile blood flow. Inertial resistance is related, for example, to peripheral vascular resistance. It is the resistance encountered by blood moving through vessels, and it accounts for a significant portion of the resistance to pulsatile blood flow as well.

Resistance associated with elastance in systems other than the cranium is typically due to structural damping. Structural damping is energy loss associated with the to-and-fro movements of the elastic cranial contents during the cardiac cycle. It differs from viscous damping in that it is not resistance due to the flow of blood, but to dissipation of energy in the cranium due to motions of elastic intracranial structures.

However, resistance due to elastance in the cranium is not entirely, or even primarily, due to structural damping. By the Monroe Kellie doctrine, the contents of the cranium are incompressible, and compliance to arterial pulsations can only be achieved by displacement of cranial contents. Intracranial elastance is achieved almost entirely by the displacement of venous blood, which has been termed "fast-volume-venting" [18]. It is likely that much of the resistance associated with elastance in the cranium is viscous resistance of vascular flow, akin to the resistance associated with smooth flow of blood.

In the analysis of the damping component of intracranial impedance, we will assume that the resistance force varies directly with the velocity of flow and is in opposition to it, as it does in a simple harmonic oscillator. It is likely, however, that the resistance to pulsatile flow in the cranium is non-linear, and varies on the square of velocity, which is characteristic of resistance caused by turbulent damping in fluid. [31]

Reactance

Reactance is opposition to flow that stores and releases energy. There are two kinds of reactance: inertial reactance, which stores and releases kinetic energy, and elastic reactance, which stores and releases potential energy.

Inertial reactance

Inertial reactance X_I is the product of mass m_{ICP} and frequency ω. Inertial reactance is given by

$$X_I = \omega m_{ICP}$$

In simple harmonic motion, the mass is constant and is assumed to be concentrated in one place. In the cranium, the pulsatile mass is distributed, and it is not constant. The mass of the intracranial pulse is equal to the volume of fluid displaced by the arterial pulse, or about 5 cc of blood with each heartbeat (roughly equal to the pulsatile half of the stroke volume of cerebral blood flow). The mass is not constant—during the cardiac cycle, it varies continuously, like the fluid displaced by a piston. As noted above, however, simplification to simple harmonic motion (with fixed lumped parameters) is justifiable in order to describe the dynamics, as long as the amplitude and energy of the ICP pulse is not too high. Furthermore, as we will see, there are two masses to be considered in the analysis of intracranial pulsations—the mass of the vascular pulse, and the mass of the extravascular fluid displaced by the vascular pulse. Because the latter is just the volume displaced by the former, we consider these masses to be equal. This ratio of the mass of the intracranial pulse to the mass of the vascular pulse, called the mass ratio μ, will be assumed to be one. This will be important in the analysis of the cerebral windkessel.

It should be noted that inertial reactance is the product of the mass of the pulse *and* the heart rate. Inertial reactance is the opposition to accelerated flow, and the more frequent the pulses, the more frequent the accelerations, and the more opposition is offered.

Elastic reactance

Elastic reactance X_E is the ratio of elastance k_{ICP} (the inverse of intracranial compliance) and frequency ω (heart rate). It is given by

$$X_E = \frac{k_{ICP}}{\omega}$$

In simple harmonic motion, elastic force is the product of the spring constant (elastance) and the displacement. Displacement in the cranium is volume, so the elastance of the intracranial pulse is proportional to the volume of the pulse. By the Monroe-Kellie doctrine, the cranial contents are incompressible, so most of the elastance of the cranium is from displacement of venous blood [18]. With systole and diastole, cerebral veins are compressed and refill. CSF exits the foramen magnum in systole and returns in diastole, although this displacement is made possible by the compression and relaxation of spinal veins. Thus, nearly all of the cardiac-cycle-related elastance in the cranium is due to displacement of venous blood. Intracranial elastance is a function of cerebrovenous pressure.

Elastic reactance

$$\frac{k_{ICP}}{\omega}$$

is the ratio of the elastance and the heart rate. Elastic reactance is the opposition to unaccelerated flow, and the less frequent the pulses, the more opposition is offered.

Total reactance

Total reactance is given by

$$X = \omega m - \frac{k}{\omega}$$

Total reactance is the *difference* between inertial and elastic reactance. This is because impedance is a complex variable, and reactance is represented on the imaginary axis. The total reactance is the difference between the inertial reactance (on the $+j$ axis) and elastic reactance (on the $-j$ axis).

Impedance magnitude

Impedance magnitude is related to the resistance and to the difference between inertial reactance and elastic reactance, expressed in complex:

$$|Z| = \sqrt{{c_{ICP}}^2 + \left(\omega m_{ICP} - \frac{k_{ICP}}{\omega}\right)^2}$$

Impedance phase

Impedance phase θ (theta) is the timing relationship between the pressure pulse and the flow pulse. It is given by the angle on the complex plane that the impedance vector (representing a complex number) makes with the positive real axis:

$$\theta = \tan^{-1}\left(\frac{\omega m_{ICP} - \frac{k_{ICP}}{\omega}}{c_{ICP}}\right)$$

Positive impedance phase means that the ICP pulse leads the flow pulse. Negative phase means that the ICP pulse lags the flow pulse.

When ICP pulse impedance is dominated by inertial reactance, ICP leads flow. When ICP impedance is dominated by elastic reactance, ICP lags flow. Damping tends to mitigate phase shifts caused by imbalance of reactances.

Flow phase

It is important to keep in mind, when measuring phase relationships in the cranium, whether the variable measured is pressure (force), flow (velocity), or volume (displacement). Pressure is measured by ICP monitoring. Flow is measured by flow sensitive MRI or by Doppler. Pressure and flow phase are given by the impedance phase, and changes in heart rate, inertia, elastance or damping alter the impedance phase and have inverse effects on the timing of the pressure and flow pulses:

$$\vec{P}_{ICP} = \vec{F}_{ICP} \angle \theta$$

$$\vec{F}_{ICP} = \frac{\vec{P}_{ICP}}{\angle \theta}$$

$$\vec{F}_{ICP} = \vec{P}_{ICP} \angle -\theta$$

where $\vec{P}_{ICP}$ is pressure, $\vec{F}_{ICP}$ is flow, θ is the impedance angle, with the relationship between pressure and flow expressed in polar form. Positive reactance causes flow to lag pressure, and negative reactance causes flow to lead pressure. This effect is readily demonstrable in syrinxes, in which flow within the syrinx has been found to lead flow in the adjacent subarachnoid space by a quarter cycle [1,32]. This is understandable if we assume that the CSF flow in the subarachnoid space is synchronous with the arterial pulse, and thus has an impedance phase of zero. The syrinx is a 'capacitor' with high elastance and very little inertance, analogous to a capacitor in an electrical circuit. Just as current leads voltage by a quarter cycle in a capacitor, flow leads pressure by a quarter cycle in a syrinx.

Displacement volume phase

Displacement, as well as pressure and flow, is a variable in the cranium. It refers to oscillatory changes in volume and is analogous to linear displacement in a mass-spring oscillator and to charge in an electrical circuit.

The phase of volume displacement of the ICP pulse is *not* the same as the impedance phase. The displacement phase φ (phi) is derived above (for the forced damped oscillator), and is given by

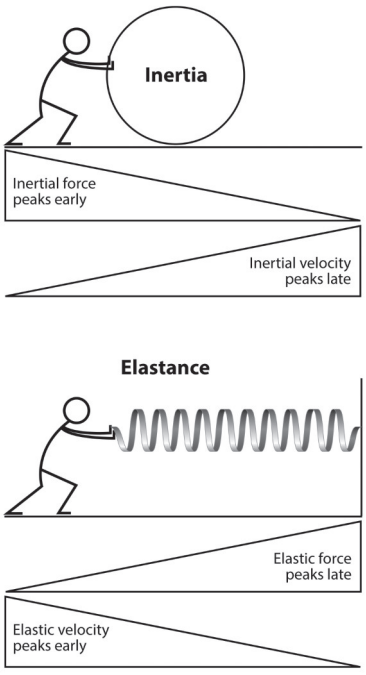

Figure 5. An intuitive understanding of phase in the cranium. Imagine a man pushing a heavy mass (top). It's hard at first, then gets easier. Now imagine a man pushing a heavy spring (bottom). It's easy at first, then gets harder. Inertial force (mass) peaks early, and elastic force (spring) peaks late. Velocity is opposite. Velocity in an inertial system peaks late, and velocity in an elastic system peaks early. Now imagine the man pushing a mass-spring against a wall in forced harmonic motion. He maintains a velocity that can be represented as a sinusoid and maintains a steady rhythmic motion. Pushing against the wall is 'systole' and pulling away from the wall is 'diastole'. If the mass and spring are balanced, the sinusoidal force he applies to the mass-spring is synchronous with the velocity, meaning that systolic force and velocity peak in mid-systole, and diastolic force and velocity peak in mid-diastole.

Suppose the mass is very big, and the spring is very small. The man executes the same motion with the same sinusoidal velocity. He will exert a lot of force in early systole and a lot of force in early diastole to get the mass moving in different directions. Velocity will be opposite—slow early and fast late. When inertia dominates, force is early, and velocity is late.

Suppose next that the mass is very small, and the spring is very big. The man executes the same motion with the same sinusoidal velocity. He will exert a lot of force in late systole and late diastole to compress and stretch the big spring. Velocity will be opposite—fast early and slow late. When elastance dominates, velocity is early, and force is late.

Now suppose the man changes the frequency of the cycle. Inertia opposes accelerated motion, so when change is more frequent, inertial opposition dominates. Elastance opposes unaccelerated motion, so when change is less frequent, elastic opposition dominates.

The total force the man applies to the harmonic motion is the sum of the inertial forces and elastic forces. Excessive inertia makes the force peak early and velocity peak late. Excessive elastance makes the velocity peak early and force peak late. High frequency accentuates inertia, and low frequency accentuates elastance.

Inertial reactance is the product of inertial and frequency. Elastic reactance is the ratio of elastance to frequency. In the cranium, synchrony between pressure and flow is a balance of inertial reactance and elastic reactance. Excess inertial reactance makes pressure lead flow, and excess elastic reactance makes flow lead pressure. If flow is taken as the reference, excessive inertial reactance makes ICP lead and excessive elastic reactance makes ICP lag.

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$$\varphi = \tan^{-1}\left(\frac{c_{ICP}\omega}{k_{ICP} - m_{ICP}\omega^2}\right)$$

or

$$\varphi = \tan^{-1}\left(\frac{2\frac{c_{ICP}}{c_0}\cdot\frac{\omega}{\omega_n}}{1-\frac{\omega^2}{\omega_n{}^2}}\right)$$

Positive φ means that the ICP pulse leads the displacement. Negative φ means that displacement leads the ICP pulse. Note that at $\omega = \omega_n$ (which represents normal dynamics), the displacement phase is a quarter cycle behind the ICP pulse. In the cranium, in which the natural frequency of the vasculature is normally equal to the heart rate (i.e. the pressure and flow are synchronous), the displacement volume waveform lags the ICP waveform and the flow waveform by a quarter cycle.

Displacement volume can be measured by pulse oximetry or by MRI. Pulse oximetry measures absorption of light by oxygenated hemoglobin in arteriolar blood volume during the cardiac cycle. Fluctuation of absorption of light in arterioles during the cardiac cycle is a method of measuring vascular pulsatility, and the fluctuation with the cardiac cycle is dependent on the *volume* (i.e. displacement) of oxygenated blood in the tissue.

An example of an experimental measurement of the phase of the displacement variable in the cranium can be inferred from the work of Greitz [3]. Greitz measured two different variables in normal subjects using flow MRI. He measured velocity of blood/CSF flow, and he measured volume change in intracranial spaces, which is a displacement variable. The velocity of blood flow in the carotid artery began its upstroke at 0.1 of the cardiac cycle (taking the R wave as the beginning of the cycle). ICP was not directly measured, but assuming synchrony of the ABP and arterial flow pulses (32), the carotid blood velocity upstroke at 0.1 of the cycle would imply that the ICP pulse happened at -0.07 of the cardiac cycle (using Wagshul et al's [14] finding that the ICP pulse leads the ABP pulse by 60 degrees in normal dogs, which is about 0.17 of the cardiac cycle). The first upstroke of the brain volume displacement in Greitz' patients was at 0.2 of the cardiac cycle, which would put the brain expansion (volume displacement variable) at a phase lag behind the ICP pulse of 27% of the cardiac cycle, which is approximately the quarter cycle lag predicted by windkessel theory.

For an intuitive explanation of phase in the cranium, see fig 5.

Resonance and anti-resonance

When the inertial reactance equals the elastic reactance, the phase is zero, and the impedance is at a minimum (in a single degree of freedom system). This is resonance. In systems with two or more degrees of freedom, in addition to low impedance resonance, there can be (depending on the geometry of the system) high impedance resonance, in which at zero phase there is a local maximum of impedance. This is sometimes called anti-resonance, which is the term I will use for it. As will be seen below in the discussion of two degree of freedom systems, anti-resonance has great relevance to the cerebral windkessel.

Heart rate and frequency

In the cranium, the variable ω is the heart rate, measured as angular frequency in radians. Because the ABP pulse and the ICP pulse are not simple sine waves, Fourier analysis of the ABP/ICP pulse gives a range of harmonic frequencies and corresponding amplitudes and phases. The fundamental harmonic of the ICP pulse and the ABP pulse is the heart rate, and second, third etc. harmonics are multiples of the heart rate.

For our analysis of the ICP pulse as a forced damped harmonic oscillator, we have simplified the discussion to that of a system with a single degree of freedom driven by a single frequency of input. In reality, the ICP pulse is a distributed parameter system (with infinite degrees of freedom) driven by an arterial pulse with a fundamental frequency and harmonics. However, as noted above, simplification of the ICP pulse to a harmonic oscillator is physically justifiable, given the correspondence of complex periodic motion found in nature with the mathematics of simple harmonic motion for relatively small amplitudes of displacement. Furthermore, as we will see below, we can focus our analysis of pulsatile dynamics on the fundamental frequency of the heart rate and the neighboring harmonics, which carry much of the energy of the ICP and ABP pulse.

Energy of the ICP pulse

Energy considerations of the ICP pulse are of great importance in understanding intracranial dynamics, because pulsatile motion in the cranium represents oscillation of the cranial contents in a potential energy well, in which total energy stays constant and there is a continuous exchange of potential and kinetic energy between the elastic and inertial forces in the cranium. Furthermore, there is dissipation of energy associated with damping force. The energy of the ABP pulse or the ICP pulse is proportional to the square of the amplitude of the pulse.

Of course, when we refer to conservation of energy we are considering only the energy associated with the motion of cranial contents, not the energy associated with metabolic processes in the cranium (which is conserved as well, but is beyond the scope of this discussion). In addition, when we are discussing velocity and flow in the cranium associated with pulsatility, we are referring to *radial motion of the intracapillary and extracapillary spaces*, not specifically to flow in vessels or CSF pathways per se. The net flow of blood and CSF is related to the radial motion of vessel walls and CSF spaces, of course, but the relationship is more complex than can be addressed in this discussion.

Kinetic energy T_{ICP} is associated with ICP pulse inertia, and is the work required to impart velocity to the mass of the pulse (which varies up to 5 grams or so) over one cycle. In a simple

harmonic oscillator, inertial force is equal and opposite (by Newton's third law) to the elastic force. The kinetic energy of the pulse is

$$T_{ICP} = \frac{m_{ICP} v_{ICP}^{\ 2}}{2}$$

Where m_{ICP} is the mass of the ICP pulse and v_{ICP} is the velocity of the pulse.

The potential energy U_{ICP} is the energy stored and released by the elastic contents of the cranium with each cardiac cycle, and is equal to the work of 'loading' and unloading the intracranial elastance (mostly the work of compressing veins). The potential energy is

$$U_{ICP} = \int_0^x k_{ICP} x_{ICP}\, dx_{ICP} = \frac{k_{ICP} x_{ICP}^{\ 2}}{2}$$

In the harmonic oscillator without damping, energy is conserved, so by Newton's second law:

$$m_{ICP} \frac{dv_{ICP}}{dt} = -k_{ICP} x_{ICP}$$

Multiplying this by $dx_{ICP} = \frac{dx_{ICP}}{dt} dt$ gives

$$m_{ICP} v dv = -k_{ICP} x_{ICP} dx_{ICP}$$

So

$$d\left(\frac{m_{ICP} v_{ICP}^{\ 2}}{2}\right) = -d\left(\frac{k_{ICP} x_{ICP}^{\ 2}}{2}\right)$$

Integrating:

$$\frac{m_{ICP} v_{ICP}^{\ 2}}{2} + \frac{k_{ICP} x_{ICP}^{\ 2}}{2} = T_{ICP} + U_{ICP} = E_{Total}$$

The total energy E_{ICP} in reactance in the cranium is constant during normal dynamics, and consists of the exchange of kinetic energy T_{ICP} carried by inertia and potential energy U_{ICP} carried by elastance.

The storage and release of kinetic and potential energy in the cranium are 180 degrees out of phase, with kinetic energy of the ICP pulse (associated with radial displacement of walls of vessels and ventricles and cisterns) at a maximum in mid systole and mid diastole and potential energy at a maximum at the transitions between systole and diastole. Again, it is noted that the kinetic and potential energy of the ICP pulse are related to radial expansion of intracranial spaces, not specifically to the bulk longitudinal flow of blood and CSF, with which radial oscillation of intracranial vessels and spaces bears a complex relationship.

Resonance, anti-resonance and intracranial dynamics

The arterial pressure pulse and the arterial flow pulse are normally synchronous, and synchrony of pressure and flow is necessary for optimally efficient cerebral blood flow (see below).

Synchrony between pressure and flow represents resonance, of which there are two types: low impedance resonance and high impedance resonance. Low impedance resonance is a condition in which there is a maximal amplitude response, and in the capillaries this would risk excessive capillary pulsatility, edema and loss of capillary integrity. This low impedance resonance is characteristic of a single degree of freedom oscillator, although it can occur in oscillators of higher degrees of freedom.

High impedance resonance is a condition of synchrony between pressure and flow in which there is suppression of flow, which means minimal amplitude response. This is characteristic of systems of two (or more) degrees of freedom.

As noted above, for clarity, I will refer to low impedance resonance as "resonance" and high impedance resonance as "anti-resonance," to emphasize the suppressive nature of the anti-resonance characteristic of the cerebral windkessel. The need for protection of capillary beds and the need for optimally efficient cerebral blood flow suggests that the cerebral windkessel is a system of anti-resonant oscillation tuned to the anti-resonant frequency (the heart rate) that simultaneously permits resonant flow in large arteries and veins. This presents a paradox, and we will examine this paradox in detail in the discussions that follow.

For the discussion that follows, I am indebted to J.P. Den Hartog for his lucid exploration of dynamic vibration absorption and from whose work this mathematical analysis is derived [33].

THE UNDAMPED WINDKESSEL WITH TWO DEGREES OF FREEDOM

Intracapillary and extracapillary spaces

The cranium is a geometrically complex non-linear system in which inertia and elastance are distributed continuously throughout the contents and which is driven by a quasi-periodic non-harmonic external force. Its dynamics are functions of spatial variables as well as time. A rigorous description of such dynamics is ponderous and necessarily inexact. Furthermore, rigorous examination of such complex dynamics tends to obscure the simpler fundamental properties of the system.

Because the cranium is a distributed parameter system, and the equations of motion we have used are of a lumped parameter system, it is helpful to specify the portions of the vasculature represented by the mathematical simplifications necessary to this analysis. Since this is a model of the cerebral windkessel, we will infer that the cerebral *micro*vasculature, especially the capillaries, are the space in which the pulsation absorption system is most effective. The windkessel also involves the intracranial arteries and veins, in very important ways, but the windkessel is most evident in flow in the microvasculature. While the arteries also have a windkessel mechanism, the arteries and veins in the cranium are an essential part of the *absorber*, and will be included in a different space (the absorber space) than the capillaries

We thus far have examined the properties of the ICP pulse as a simple harmonic oscillator and a forced harmonic oscillator with damping. Both systems have one degree of freedom.

With windkessel dynamics, we begin the discussion of intracranial dynamics as a system with two degrees of freedom. In the next level of accuracy and complexity, we will consider the ICP pulse as a manifestation of the dynamics of an undamped windkessel. As noted above, conceptually this entails two different pathways through which pulsations may travel: the intracapillary space and the extracapillary space.

Radical simplification of pulsatile intracranial dynamics has the advantage of (relative) clarity, and, as noted above, it is justified as long as the displacement and the energy of the pulsatility is relatively small, which is certainly the case with the ICP pulse. As will be seen, even quantitative estimates of intracranial pulsatility can be made that have modest agreement with experiment, as long as proper caution is taken in interpretation.

The windkessel is a two-degree-of-freedom oscillator

We divide the cranium conceptually into an intracapillary space and an extracapillary space, understanding the intracapillary space to mean the lumina of the capillaries and the extracapillary space to mean the remainder of the cranial contents, to include the brain parenchyma, the ISF and CSF, and the large intracranial arteries and veins.

The equations of motion for the intracapillary and extracapillary spaces are:

For the intracapillary space

$$M_{IC}\ddot{x}_{IC} + \left(K_{IC} + k_{EC}\right)x_{IC} - k_{EC}x_{EC} = P_0 \sin\omega t$$

where M_{IC} is the mass of the intracapillary (intravascular) pulse, x_{IC} is the displacement volume of the intracapillary space, K_{IC} is the elastance of the intracapillary space, k_{EC} is the elastance of the extracapillary space, and x_{EC} is the displacement volume of the extracapillary space.

The equation of motion for the extracapillary space is

$$m_{EC}\ddot{x}_{EC} + k_{EC}(x_{EC} - x_{IC}) = 0$$

where m_{EC} is the mass of the ICP pulse in the extracapillary space.

Because the forced pulsations lack the first derivatives $\dot{x}_{IC}$ and $\dot{x}_{EC}$, and a sine function twice differentiated remains a sine function, the forced pulsations will take the form:

$$x_{IC} = a_{IC}\sin\omega t$$

$$x_{EC} = a_{EC}\sin\omega t$$

Division by $\sin\omega t$ transforms the equations of motion from differential equations to algebraic equations.

For the intracapillary space

$$a_{IC}\left(-M_{IC}\omega^2 + K_{IC} + k_{EC}\right) - a_{EC}k_{EC} = P_0$$

And for the extracapillary space

$$-k_{EC}a_{IC} + a_{EC}\left(-m_{EC}\omega^2 + k_{EC}\right) = 0$$

To simplify by dimensionless quantities, we define:

$$x_{STIC} = \frac{P_0}{K_{IC}}$$

= static radial displacement of capillary wall (i.e. the radial displacement of the capillary wall if it were subjected to constant pressure P_0)

$$\omega_{EC}{}^2 = \frac{k_{EC}}{m_{EC}} =$$

the square of natural frequency of extracapillary space

$$\Omega_{IC}{}^2 = \frac{K_{IC}}{M_{IC}} =$$

the square of natural frequency of intracapillary space

$$\mu = \frac{m_{EC}}{M_{IC}} =$$

the mass ratio of extracapillary pulse to intracapillary pulse

$\omega =$ the angular heart rate in radians

The equation of motion for the intracapillary space becomes

$$a_{IC}\left(1 + \frac{k_{EC}}{K_{IC}} - \frac{\omega^2}{\Omega_{IC}{}^2}\right) - a_{EC}\frac{k_{EC}}{K_{IC}} = x_{STIC}$$

and for the extracapillary space

$$a_{IC} = a_{EC}\left(1 - \frac{\omega^2}{\omega_{EC}{}^2}\right)$$

Solving for a_{IC} and a_{EC}:

Capillary wall displacement =

$$\frac{a_{EC}}{x_{STIC}} = \frac{1}{\left(1 - \frac{\omega^2}{\omega_{EC}{}^2}\right)\left(1 + \frac{k_{EC}}{K_{IC}} - \frac{\omega^2}{\Omega_{IC}{}^2}\right) - \frac{k_{EC}}{K_{IC}}}$$

Extracapillary space displacement =

$$\frac{a_{EC}}{x_{STIC}} = \frac{1}{\left(1 - \frac{\omega^2}{\omega_{EC}{}^2}\right)\left(1 + \frac{k_{EC}}{K_{IC}} - \frac{\omega^2}{\Omega_{IC}{}^2}\right) - \frac{k_{EC}}{K_{IC}}}$$

The displacement of the capillary wall

$$\frac{a_{IC}}{x_{STIC}}$$

at anti-resonance (when $\omega = \omega_{EC}$) is zero. In the undamped windkessel model, this is when the heart rate equals the natural frequency of the extracapillary space. *At anti-resonance, the extracapillary space 'absorbs' the intracapillary pulsations, and renders the capillary blood flow pulseless.* Note that pulsation absorption is dependent on heart rate, elastance, inertia and damping.

To gain deeper physical insight into the windkessel, consider the amplitude of extracapillary pulsations. Because

$$x_{STIC} = \frac{P_0}{K_{IC}}$$

extracapillary displacement at anti-resonance is given by

$$a_{EC} = -\frac{K_{IC}}{k_{EC}} x_{STIC} = -\frac{K_{IC}}{k_{EC}} \cdot \frac{P_0}{K_{IC}} = -\frac{P_0}{k_{EC}}$$

When at anti-resonance (i.e. when the windkessel is effective), the extracapillary space is undergoing the motion

$$a_{EC} = -\frac{P_0}{k_{EC}} \sin \omega t$$

The pressure (i.e. elastic force) P_{EC} that the extracapillary space exerts externally on the capillary walls varies as

$$P_{EC} = -P_0 \sin \omega t$$

with the negative sign in $-P_0$ indicating that the pressure is external to the capillary walls.

Simultaneously, the pressure P_{IC} that the arterial pulse exerts internal to the capillary walls is

$$P_{IC} = P_0 \sin \omega t$$

The instantaneous net pressure across the capillary walls at any moment during the cardiac cycle is

$$P_{IC} + P_{EC} = P_0 \sin \omega t - P_0 \sin \omega t = 0$$

Thus, at anti-resonance, the *pressure in the extracapillary space is continuously equal to and opposed to the pressure in the intracapillary space, and the transmural pressure across the capillary wall is zero.* The capillaries are protected from arterial pulsatility (fig 6).

This is the essence of the windkessel: it entails a continuous reciprocating balance of intracapillary and extracapillary pressures so as to render capillary walls motionless, and to render capillary flow pulseless (in the radial direction) (fig 7).

Note especially that the windkessel dynamics depends critically on anti-resonance. The natural frequency of oscillation of the intracapillary and extracapillary spaces must be equal to the heart rate for capillary wall motion to be suppressed. This is characteristic of the undamped model of the windkessel. As we will see below, damping alters the condition of anti-resonance somewhat, although the basic principles of the windkessel and pulsation absorption remain the same.

To clarify the windkessel conditions at anti-resonance, consider the dynamics for anti-resonance, in which $\omega_{EC} = \Omega_{IC}$ (the natural frequency of the extra-and intracapillary spaces are equal) and

$$\frac{k_{EC}}{m_{EC}} = \frac{K_{IC}}{M_{IC}} = \omega_{EC}^2 = \Omega_{IC}^2$$

(the corresponding mass and elastance ratios correspond to anti-resonance) and

$$\frac{k_{EC}}{K_{IC}} = \frac{m_{EC}}{M_{IC}}$$

and μ, defined as the mass ratio of the extra- to intracapillary space, is given by

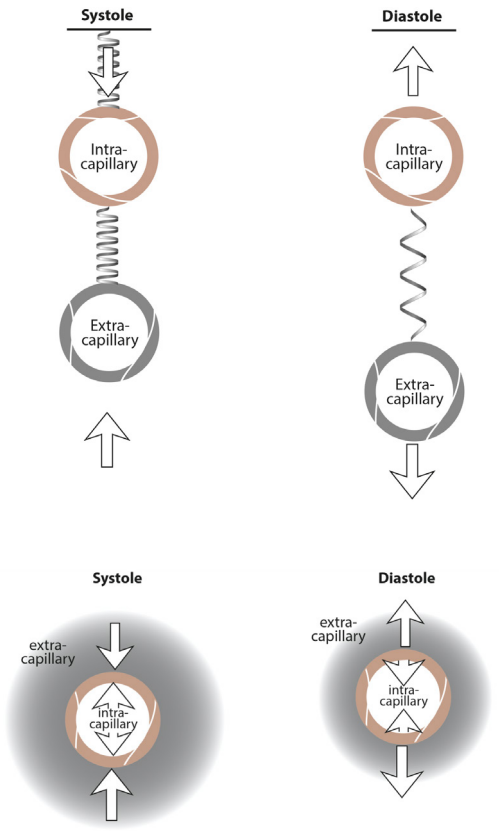

Figure 6. The cerebral windkessel is a dynamic pulsation absorber. When a system is driven by an external force at resonance with the system's natural frequency of oscillation, it will respond with a large amplitude of displacement that is often damaging. Cerebral blood flow has synchronous pressure and flow, which represents resonant perfusion. This endangers capillaries because of the large displacement of vascular walls inherent to resonant dynamics. Large amplitude resonant responses can be prevented by the use of dynamic vibration absorption, which is a method widely used in engineering to protect against damage caused by resonant vibrations. **Upper:** depicts blood flow in the intracapillary space driven at its resonant frequency (the heart rate). The extracapillary space, which is most of the cranial contents, is analogous to an absorber mass on a spring, in that it oscillates naturally in opposition to the heart beat that is driving the intracapillary space. When the heart beat pushes down, the extracapillary mass is pushing up. When the heart beat pulls up, the extracapillary mass is pulling down. The arterial and elastic forces on the intracapillary mass are always equal and opposite, and the pulse is removed from the intracapillary mass. **Lower:** extends the analogy to a somewhat more realistic geometry, in which the intracapillary space is a sphere encased in a spherical extracapillary space. The natural frequency of both spaces are equal to each other and to the heart rate. The "spring" force is provided by compression and relaxation of cerebral veins by the reciprocal transposition of the pulse through the CSF between the arteries and the veins. During systole, expansion of the intracapillary space is prevented by the elastic force of the extracapillary pressure pushing inward. During diastole, relaxation of the intracapillary space is prevented by the elastic force of the extracapillary pressure pulling outward. This prevents radial motion in the intracapillary space and protects capillary walls from the arterial pulse. The dynamic between the intra-and extra-capillary space is anti-resonant and depends on the balance of pressures inside and outside the capillaries. Proper function of the cerebral windkessel also depends on proper direction of forces, which is accomplished by hydraulic linkage provided by the CSF, which solves the geometrical problem inherent to pulsation absorption in a resonator (the cranium) with complex anatomy. **doi:**10.5048/BIO-C.2019.3.f6

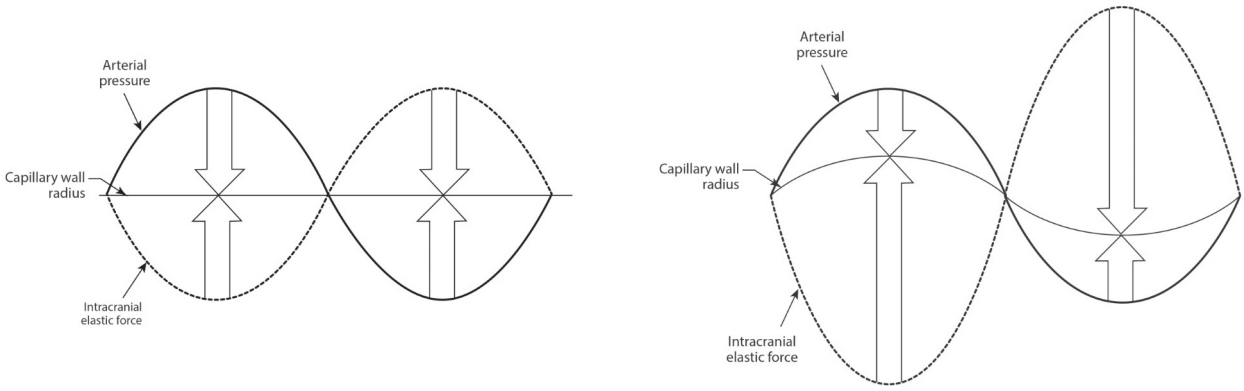

Figure 7. The windkessel is a continuous reciprocating balance of capillary transmural pressures. Left: a schematic representation of the transmural forces generated by the cerebral windkessel during one cardiac cycle. The central baseline is the capillary radius, which represents capillary wall stress. The solid sinusoid represents the internal arterial pressure on the capillary wall. The dotted sinusoid represents the external pressure exerted by the extracapillary fluids and tissues on the capillary wall. When the windkessel is working properly (i.e. at anti-resonance), the internal intracapillary and external extracapillary pulse pressures are continuously balanced and the transmural capillary pressure is zero. This protects capillaries from arterial pulsatility. **Right:** a schematic representation of the transmural forces generated by an impaired cerebral windkessel during one cardiac cycle. The intracranial elastic force is increased due to cerebral edema or an intracranial mass lesion. The arterial pressure (solid sinusoid) is no longer balanced by the extracapillary pressure (dotted sinusoid), which is high due to the increased intracranial elastance. This imbalance causes abnormally high motion stress in capillary walls, predisposing to cerebral edema and catastrophic loss of capillary integrity. This normal and abnormal windkessel motion has been observed using flow MRI [3], and the frequency domain signature of these dynamics has been observed in normal animals and in intracranial hypertension [14,24], and in experimental [27] and human [26,28] hydrocephalus. **doi**:10.5048/BIO-C.2019.3.f7

$$\mu = \frac{m_{EC}}{M_{IC}}$$

It is noteworthy that the inertia in the cranium is not fixed, but varies with time during the cardiac cycle, because the intracapillary pulse is a piston that displaces a similar amount of extracapillary fluid. Although this complicates the analysis somewhat, it greatly simplifies it as well, because the mass of the intracapillary pulse and the extracapillary pulse are always the same: the latter is just the fluid displaced by the former. μ in the cranium is always equal to 1. This is tantamount to an assertion of the Monroe Kellie doctrine.

At anti-resonance, the equations of displacement are for the intracapillary space:

$$\frac{x_{IC}}{x_{STIC}} = \frac{1 - \frac{\omega^2}{\omega_{EC}{}^2}}{\left(1 - \frac{\omega^2}{\omega_{EC}{}^2}\right)\left(1 + \mu - \frac{\omega^2}{\omega_{EC}{}^2}\right) - \mu} \sin \omega t$$

and for the extracapillary space:

$$\frac{x_{EC}}{x_{STIC}} = \frac{1}{\left(1 - \frac{\omega^2}{\omega_{EC}{}^2}\right)\left(1 + \mu - \frac{\omega^2}{\omega_{EC}{}^2}\right) - \mu} \sin \omega t$$

Note that the denominators of the displacement of the intracapillary and extracapillary spaces are equal. When multiplied out, the denominator yields a quadratic equation in

$$\frac{\omega^2}{\omega_{EC}{}^2}$$

which has two roots. This means that for two values of frequency the denominator of the extracapillary displacement and intracapillary displacement become zero, and thus the amplitude of the pulsations becomes unbounded. These unbounded responses are the two resonant (rather than anti-resonant) frequencies of the combined intra- and extracapillary spaces.

What the pulsation absorber mechanism does to the pulsatile dynamics is that it shifts the high amplitude single resonant response of the single-degree-of-freedom intracapillary space to a double resonant response split around the central (heart rate) frequency, with two resonant responses which are off (one above and one below) the heart rate frequency (fig 8). At the heart rate frequency, sometimes called the 'notch', the radial motion of the capillary walls is suppressed.

It is noteworthy that high-amplitude resonant responses don't *disappear* with the windkessel. Rather, the normal resonance at the heart rate frequency is split and shifted to frequencies above and below the heart rate. Teleologically, this is advantageous for intracranial dynamics, because while two new resonances are created by the windkessel, they are at frequencies away from the heart rate which transfer a much smaller portion of the energy of arterial pulsatility than is transferred at the fundamental of the heart rate. The windkessel shifts the resonance responses to frequencies that pose less risk to the microcirculation because they carry less energy than the heart rate frequency.

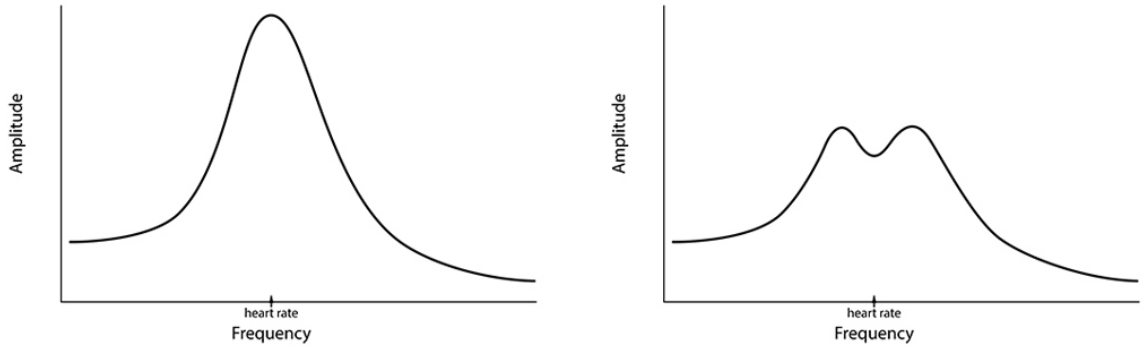

Figure 8: Resonant and anti-resonant responses. Left: A single-degree-of-freedom oscillator shows a resonant response. Schematic graph of the frequency response of a single degree of freedom oscillator. At the resonant frequency, which is a balance of inertial and elastic reactance, there is a high amplitude response. A system driven at this resonant frequency will have maximal efficiency of power, but the high amplitude of oscillations may cause damage. This would occur in the cerebral capillaries, without the windkessel. **Right:** A two-degree-of-freedom oscillator (notch filter) shows an anti-resonant response. At the anti-resonant frequency, there is a low amplitude response, sometimes called a 'notch'. A system driven at this anti-resonant frequency will be protected from damage caused by high amplitude oscillations. The two peaks at higher and lower frequencies represent resonant frequency responses characteristic of this kind of system. This is a dynamic vibration absorber, which is a mechanical analogue of the cerebral windkessel. **doi:**10.5048/BIO-C.2019.3.f8

The frequencies of the split resonances can be determined by setting the denominator to zero:

$$\left(1-\frac{\omega^2}{\omega_{EC}{}^2}\right)\left(1+\mu-\frac{\omega^2}{\omega_{EC}{}^2}\right)-\mu=0$$

$$\left(\frac{\omega}{\omega_{EC}}\right)^4-\left(\frac{\omega}{\omega_{EC}}\right)^2(2+\mu)+1=0$$

The solutions in terms of the ratios of the resonant frequencies to the natural frequency of the extracapillary space in the undamped windkessel model are

$$\left(\frac{\omega}{\omega_{EC}}\right)^2=\left(1+\frac{\mu}{2}\right)\pm\sqrt{\mu+\frac{\mu^2}{4}}$$

Calculation of resonant peaks in Wagshul et al example

The two resonant peaks predicted by windkessel theory have been observed and studied empirically [14]. Wagshul et al studied the frequency response of the ICP to the ABP in dogs, and they observed a region of pulsation suppression around the heart rate encompassing the first two harmonics, as well as high frequency resonant peak from 6-15Hz (fig 9). There is a peak in the lower frequencies as well, although potential artifacts in the low frequency range of the transfer function make discernment more difficult. Both high frequency and low frequency peaks are associated with phase transitions (the lower peak from lagging to leading phase, and the higher peak from leading to lagging), which is characteristic of two degree of freedom pulsation absorbers [34] and supports the view that the peaks are resonant responses and not just artifacts.

Based on windkessel theory, the location of the resonant peaks in the cranium can be estimated. This estimation is based on an undamped windkessel model, in which the natural frequency of oscillation of the extracapillary space (the absorber) is equal to the natural frequency of the intracapillary space. As we will see, this is not true for the damped windkessel model, but it is a reasonable approximation in the simplified dynamics of the undamped windkessel.

At anti-resonance, note that $\omega_{EC}=\Omega_{IC}$ (for the *undamped* windkessel) so for this analysis

$$\frac{\omega_{Peak}}{\omega_{EC}}=\sqrt{\left(1+\frac{\mu}{2}\right)\pm\sqrt{\mu+\frac{\mu^2}{4}}}$$

is

$$\frac{\omega_{Peak}}{\Omega_{IC}}=\sqrt{\left(1+\frac{\mu}{2}\right)\pm\sqrt{\mu+\frac{\mu^2}{4}}}$$

$$\omega_{Peak}=\Omega_{IC}\sqrt{\left(1+\frac{\mu}{2}\right)\pm\sqrt{\mu+\frac{\mu^2}{4}}}$$

The mass ratio μ, which represents the ratio of the mass of the extracapillary pulse to the intracapillary pulse, is 1, because the former is just the fluid displaced by the latter.

Given that blood flow is resonant and $\omega=\Omega_{IC}$ (the heart rate is equal to the resonant frequency of the intracapillary space), it follows that

$$\omega_{Peak}=\omega\sqrt{(1.5)\pm\sqrt{1.25}}$$

$$\omega_{Peak\,HIGH}=\omega\sqrt{2.62}=1.62\omega$$

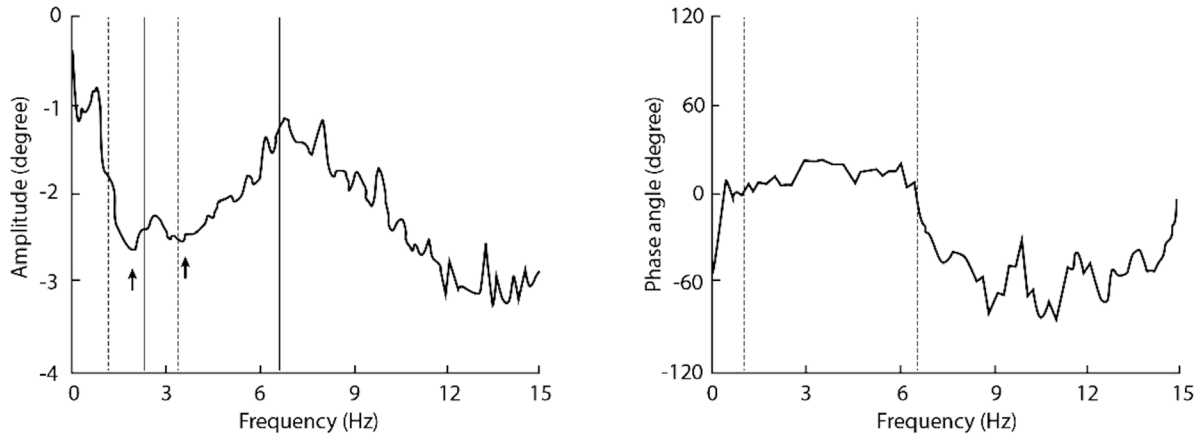

Figure 9: ABP to ICP amplitude (Left) and phase (Right) transfer functions (reprinted from Wagshul et al [14] with minor modifications, with permission). Wagshul et al [14] measured the ABP to ICP amplitude transfer function in dogs. Consistently there was a notch in the transfer function spanning the region of the heart rate and the second harmonic (arrows), with two high amplitude peaks at higher and lower frequencies forming the walls of the notch. This transfer function is characteristic of the cerebral windkessel, which is a two degree of freedom dynamic pulsation absorber driven near its natural frequency. Theory predicts peaks at 1.24 Hz and 3.24 Hz associated with the heart rate harmonic (dotted lines), and peaks at 2.48 Hz and 6.48 Hz associated with the second harmonic (solid lines). The 1.24 Hz peak and the 6.48 Hz peak correspond reasonably well with the observed peaks, and the 2.48 Hz and 3.24 Hz peaks appear to be attenuated in the notch, although there is a small peak in that region. The predicted peaks at 1.24 Hz and 6.48 Hz fall slightly medial to the observed peaks, which can be explained as the consequence of damping in the actual cerebral windkessel, which is not accounted for in the mathematical prediction of the peaks and which would widen the notch.

The phase transfer function in dogs shows phase transition (dotted lines) from lagging to leading ICP phase at the frequency of the lower peak with a phase transition from leading to lagging at the higher peak. The phase transitions correlate reasonably well with the location of the lower (1.28 Hz) and upper (6.48 Hz) resonance peaks predicted by windkessel theory. The phase transition at the location of each peaks supports the view that these peaks are resonant responses. In the notch the phase is slightly positive, which correlates with an ICP pulse that leads the ABP pulse in the time domain (Fig 1). **doi:**10.5048/BIO-C.2019.3.f9

$$\omega_{Peak\,LOW} = \omega\sqrt{.38} = 0.62\omega$$

We assume that the windkessel notch spans the fundamental and 2nd harmonic, so
For the 2 Hz harmonic of the notch,

Upper peak $\omega_{Peak\,2\,Hz} = 3.24$

Lower peak $\omega_{Peak\,2\,Hz} = 1.24$

For the 4 Hz Harmonic of the notch,

Upper peak $\omega_{Peak\,4\,Hz} = 6.48$

Lower peak $\omega_{Peak\,4\,Hz} = 2.48$

The middle peaks (2.48 Hz and 3.24 Hz) fall within the notch and are suppressed.

The upper peak of the second harmonic and the lower peak of the fundamental are the high and low peaks defining the notch, and are at

Notch (2-4 Hz) upper peak 6.48

Notch (2-4 Hz) lower peak 1.24

The observed and predicted peaks are shown in fig 9. The frequencies of the observed peaks correlate reasonably well with the frequencies predicted by windkessel theory.

Note that this estimate is derived from the undamped model of the windkessel. Damping in the windkessel widens the notch (see below). In fig 9, the predicted peaks fall slightly closer to the central frequency than the observed peaks, consistent with fact that the equation derived from the undamped windkessel slightly underestimates the width of the notch.

Quality factor and bandwidth

The quality factor and the bandwidth of the windkessel describe the width of the notch. Damping will diminish the amplitude of the peaks, increase the amplitude of the response at the anti-resonant frequency, and widen the effective notch, as we will see below. The width of the notch is measured by the bandwidth, which is defined as the full width of the notch at half-maximum power. The bandwidth is related to the inverse of the quality factor Q, which is a measure of the sharpness of the notch. The quality factor at resonance is the ratio between energy stored and energy dissipated per cardiac cycle

$$Q = \frac{E_{Stored}}{E_{Dissipated}}$$

and is given by

$$Q = \frac{X}{c}$$

where X is the inertial reactance or the elastic reactance (which are equal at resonance), and c is the damping.

The relationship between Q and the width of the notch $\Delta\omega$ (i.e. the bandwidth) in normal dynamics is:

$$\Delta\omega = \frac{\omega_{anti-resonant}}{Q}$$

Where $\Delta\omega$ is the width of the notch, Q is the quality factor, and $\omega_{anti-resonant}$ is the central frequency of the notch. Q is the ratio of energy stored to energy dissipated by intracranial pulsations per cardiac cycle, and describes the 'sensitivity' or narrowness of the notch. In a damped absorber, which is a more accurate approximation of the cerebral windkessel, the windkessel notch is wider than is predicted by the mathematics of the undamped model.

THE DAMPED WINDKESSEL WITH TWO DEGREES OF FREEDOM

The windkessel is a two-degree-of-freedom oscillator with damping

With undamped windkessel dynamics, we began the discussion of intracranial dynamics as a system with two degrees of freedom. The idealized undamped absorber model is useful because it clarifies the essential dynamics of the cerebral windkessel—the anti-resonant suppression of the heart rate frequency in the cranium.

The cerebral windkessel is a damped system, however, and damping alters the dynamics in complex but important ways. In the next level of accuracy and complexity, we will consider the ICP pulse as a manifestation of the dynamics of a damped windkessel. We assume a linear system with viscous damping c in the extracapillary space.

For the intracapillary space, the equation of motion with damping c_{EC} in the extracapillary space is

$$M_{IC}\ddot{x}_{IC} + K_{IC}x_{IC} + k_{EC}(x_{IC} - x_{EC}) + c_{EC}\left(\dot{x}_{IC} - \dot{x}_{EC}\right) = P_0 \sin\omega t$$

For the extracapillary space

$$m_{EC}\ddot{x}_{EC} + k_{EC}(x_{EC} - x_{IC}) + c_{EC}\left(\dot{x}_{EC} - \dot{x}_{IC}\right) = 0$$

We are concerned with the forced steady state oscillations. The displacements of the spaces are harmonic motions that can be represented by phasors on the complex plane rotating with heart rate frequency ω, taking the rotating projection on the real axis $(\cos\omega t)$ as the displacement of the wall of the space. The equations for the two degree of freedom damped windkessel can be solved by representing the phasors as complex numbers.

For the intracapillary space:

$$-M_{IC}\omega^2 x_{IC} + K_{IC}x_{IC} + k_{EC}(x_{IC} - x_{EC}) + j\omega c_{EC}\left(x_{IC} - x_{EC}\right) = P_0$$

For the extracapillary space:

$$-m_{EC}\omega^2 x_{EC} + k_{EC}(x_{EC} - x_{IC}) + j\omega c_{EC}\left(x_{EC} - x_{IC}\right) = 0$$

where x_{IC} and x_{EC} are complex numbers representing displacement of the walls of the intracapillary and extracapillary spaces.

Simplifying:

$$-M_{IC}\omega^2 x_{IC} + K_{IC}x_{IC} + k_{EC}(x_{IC} - x_{EC}) + j\omega c_{EC}\left(x_{IC} - x_{EC}\right) = P_0$$

$$-m_{EC}\omega^2 x_{EC} + k_{EC}(x_{EC} - x_{IC}) + j\omega c_{EC}\left(x_{EC} - x_{IC}\right) = 0$$

We solve for the displacement of the intracapillary space by substitution of x_{EC} in terms of x_{IC}:

$$x_{IC} = P_0 \frac{\left(k_{EC} - m_{EC}\omega^2\right) + j\omega c_{EC}}{\left[\left(-M_{IC}\omega^2 + K_{IC}\right)\left(-m_{EC}\omega^2 + k_{EC}\right) - m_{EC}\omega^2 k_{EC}\right] + j\omega c_{EC}\left[-M_{IC}\omega^2 + K_{IC} - m_{EC}\omega^2\right]}$$

This equation is a phasor representation on the complex plane of the displacement of the wall of the intracapillary space. The intracapillary displacement has two components—one real component in phase with the arterial pulse pressure and one imaginary component leading it by 90 degrees. We carry out the complex division by multiplying by the complex conjugate of the denominator and then rearranging terms.

For the motion of the capillary wall:

$$\frac{{x_{IC}}^2}{{P_0}^2} = \frac{\left(k_{EC} - m_{EC}\omega^2\right)^2 + \omega^2 {c_{EC}}^2}{\left[\left(-M_{IC}\omega^2 + K_{IC}\right)\left(-m_{EC}\omega^2 + k_{EC}\right) - m_{EC}\omega^2 k_{EC}\right]^2 + \omega^2 {c_{EC}}^2\left[-M_{IC}\omega + K_{IC} - m_{EC}\omega^2\right]^2}$$

To simplify and clarify the dynamics, we write it in dimensionless form using the following identities:

$$\mu = \frac{m_{EC}}{M_{IC}}$$

= mass ratio of extracapillary pulse to intracapillary pulse = 1

$$\omega_{EC}^2 = \frac{k_{EC}}{m_{EC}}$$

= square of natural frequency of extracapillary space

$$\Omega_{IC}^2 = \frac{K_{IC}}{M_{IC}}$$

= square of natural frequency of intracapillary space

$$f = \frac{\omega_{EC}}{\Omega_{IC}}$$

= natural frequency ratio of extracapillary to intracapillary space

$$g = \frac{\omega}{\Omega_{IC}}$$

= forced frequency ratio

$$x_{STIC} = \frac{P_0}{K_{IC}}$$

= static displacement of capillary walls

c_{EC} = damping in the extracapillary space

$$c_c = 2m_{EC}\Omega_{IC}$$

= critical damping, which is damping at which the wall returns to its static position most quickly

The amplitude ratio of the radial displacement of the capillary wall in dimensionless variables is

$$\left(\frac{x_{IC}}{x_{STIC}}\right)^2 = \frac{\left(2\frac{c_{EC}}{c_c}gf\right)^2 + \left(g^2 - f^2\right)^2}{\left(2\frac{c}{c_c}gf\right)^2\left(g^2 - 1 + \mu g^2\right)^2 + \left[\mu f^2 g^2 - \left(g^2 - 1\right)\left(g^2 - f^2\right)\right]^2}$$

To gain deeper insight into damping, consider that at zero damping there are two unbounded resonant peaks straddling the central frequency, as we have already shown with the undamped windkessel. At infinite damping, the intra-and extracapillary spaces are essentially stuck together and oscillate as a single degree of freedom system, with high amplitude response at the central frequency. So there must be a value for damping between zero and infinity at which damping is optimal, in that the windkessel suppresses the heartbeat most efficiently.

To determine this value for damping, we consider that for all values of damping the amplitude response curve passes through the same two points, one above and one below the central (anti-resonant) heart beat frequency [35]. The most favorable damping is that at which these two resonant peaks are symmetrical and at which a tangent has a slope of zero.

This greatly simplifies the search for the equation for the optimally damped windkessel, because we need only find an expression in which the intracapillary displacement is independent of damping, and that will be the optimally damped windkessel. So what are the values of the natural frequency ratio

$$f = \frac{\omega_{EC}}{\Omega_{IC}}$$

and the forced frequency ratio

$$g = \frac{\omega}{\Omega_{IC}}$$

for which the displacement of the capillary wall is independent of damping?

Consider:

$$\left(\frac{x_{IC}}{x_{STIC}}\right)^2 = \frac{A\left(\frac{c_{EC}}{c_c}\right)^2 + B}{C\left(\frac{c_{EC}}{c_c}\right)^2 + D}$$

The amplitude of capillary wall motion does not depend on damping if

$$\frac{A}{C} = \frac{B}{D}$$

because the numerator can them be factored out to eliminate the damping ratio and capillary wall motion only depends on the four remaining terms. Written out in the four terms of the dimensionless windkessel equation, the capillary wall motion is independent of damping if:

$$\left(\frac{1}{g^2 - 1 + \mu g^2}\right)^2 = \left(\frac{g^2 - f^2}{\mu f^2 g^2 - \left(g^2 - 1\right)\left(g^2 - f^2\right)}\right)^2$$

We can take the square root of both sides (but we must add a $\pm$ sign to the right side). Taking the minus sign,

$$\mu f^2 g^2 - \left(g^2 - 1\right)\left(g^2 - f^2\right) = -\left(g^2 - f^2\right)\left(g^2 - 1 + \mu g^2\right)$$

which reduces to

$$\mu f^2 g^2 = -\mu g^2\left(g^2 - f^2\right)$$

or

$$f^2 = -g^2 + f^2$$

So that

$$g^2 = 0$$

This is trivially true, because if g or ω are zero, the capillary wall doesn't move and the damping is irrelevant.

If we take the plus sign in the simplification, we get

$$g^4 - 2g^2\frac{1 + f^2 + \mu f^2}{2 + \mu} + \frac{2f^2}{2 + \mu} = 0$$

which is quadratic in g^2, which has two solutions (the roots are g_1^2 and g_2^2) and will provide the frequency and amplitude of the two peaks at which the damped windkessel is optimal.

It is not necessary to solve for g^2 because we can simplify the equation by noting that all damping curves pass through the two resonance peaks and setting damping equal to infinity. The equation reduces to

$$\frac{x_{IC}}{x_{STIC}} = \frac{1}{1 - g^2\left(1 + \mu\right)}$$

Substituting g_1^2 and g_2^2:

$$\frac{1}{1 - g_1^2\left(1 + \mu\right)} = \frac{1}{1 - g_2^2\left(1 + \mu\right)}$$

Correcting for phase reversal and normalizing amplitude, this simplifies to

$$g_1^2 + g_2^2 = \frac{2}{1 + \mu}$$

Noting that the negative coefficient of the middle term in a quadratic equation is the sum of the roots, we obtain

$$g_1^2 + g_2^2 = \frac{2\left(1 + f^2 + \mu f^2\right)}{2 + \mu}$$

Substituting:

$$f = \frac{1}{1 + \mu}$$

Remember that for the cranium the mass ratio μ of the intracapillary pulse and the extracapillary pulse is 1. Therefore for the cerebral windkessel, the frequency ratio f (the ratio between the natural frequency of the extracapillary space ω_{EC} and the natural frequency of the intracapillary space Ω_{IC}) is

$$f = \frac{\omega_{EC}}{\Omega_{IC}} = \frac{1}{1+\mu} = \frac{1}{2}$$

This means that *for a damped windkessel, optimal pulsation absorption occurs when the natural frequency of the extracapillary space is half the heart rate* (the heart rate is equal to the natural frequency of the intracapillary space). Notice that since the natural frequency of the extracapillary space is given by

$$\omega_{EC} = \sqrt{\frac{k_{EC}}{m_{EC}}}$$

and the natural frequency of the extracapillary space is half that of the intracapillary space and half the heart rate, the lower natural frequency of the extracapillary space implies a dominance of inertia over elastance in the extracapillary space, as compared to the balance of inertia and elastance in the intracapillary space.

So the most effective *damped* windkessel absorber (which corresponds most closely to the cranium) is an intracapillary space with equal inertial and elastic reactance, and an extracapillary space with a high ratio of inertial reactance to elastic reactance. This facilitates optimally efficient blood flow and suppression of capillary pulsation.

By the impedance phase equation for the extracapillary space

$$\theta = \tan^{-1}\left[\frac{\omega m_{EC} - \frac{k_{EC}}{\omega}}{c_{EC}}\right]$$

the impedance phase of the extracapillary space for the most effective damped windkessel (i.e. a windkessel in which ωm_{EC} is greater than

$$\frac{k_{EC}}{\omega}$$

) is positive. That is, *the windkessel is most effective when the ICP pulse leads the ABP pulse*. It is noteworthy that this result correlates well with the consistent finding that the ICP (extracapillary) pulse leads the ABP (intracapillary/intravascular) pulse in normal dynamics [14] and (fig 1).

Transfer function analysis of the cerebral windkessel

The cerebral windkessel is a notch filter, which is a type of frequency sensitive filter that transforms an input (the ABP pulse) into an output (the ICP pulse). This transformation can be analyzed using the transfer function, which is a mathematical technique used to characterize the manner in which a system such as the cranium modifies an input signal (the ABP waveform) to yield an output signal (the ICP waveform). Transfer function analysis is of particular value in the study of the cerebral windkessel, because it reveals dynamics such as resonance, anti-resonance, phase, notch, resonant peaks, etc. that are not readily discernable by inspection of the ABP and ICP waveforms in the time domain. The empirical study of the cerebral windkessel has been based almost entirely on analysis of the transfer function of the ABP pulse to the ICP pulse [14,24-27]. A rigorous discussion of the mathematical basis for transfer function is beyond the scope of this review, but it is appropriate to review the basic principles of transfer functions as apply to the experimental identification and measurement of the windkessel notch.

The transfer function (as relevant to analysis of the windkessel) is a linear mapping of the Laplace transform of the input of the windkessel (e.g. the ABP pulse) to the Laplace transform of the output of the windkessel (e.g. the ICP pulse).

The Laplace transform is a mapping of a real function $f(t)$ in the time domain to a complex function $F(s)$ in the frequency domain. s is a complex variable given by $s = \sigma + j\omega$

The Laplace transform is defined as

$$F(s) = \int_0^\infty f(t)e^{-st}dt$$

For readers familiar with Fourier series, I note that the Laplace transform is similar to the Fourier transform, in that it identifies the frequency components of a signal. The Fourier transform of a waveform is essentially the evaluation of the Laplace transform of the waveform on the imaginary ($j\omega$) axis, neglecting the real (σ) axis.

For this discussion, we assume linear intracranial pulsatile dynamics and we assume that the ICP pulse is of constant frequency (constant heart rate) and thereby we neglect transient effects. The ICP pulse understood in this way is a linear time-invariant system. This is a reasonable approximation for pulsatile intracranial dynamics, and methods can be applied to the transfer function analysis to correct for empirical deviations from linear time-invariance (such as fluctuations of heart rate) [14].

The transfer function $H(s)$ of the cerebral windkessel thus understood is the linear mapping of the Laplace transform $X(s)$ of the input (the ABP) to the Laplace transform $Y(s)$ of the output (the ICP):

$$H(s) = \frac{Y(s)}{X(s)} = \frac{ICP(s)}{ABP(s)}$$

This means that transfer function analysis of the cerebral windkessel provides the frequency-by-frequency ratio of input and output magnitude and phase of each sinusoidal component of the ABP and the ICP.

The mapping of input to output can be examined in more detail. If we express the ABP as a function $x(t)$, it can also be expressed in terms of frequency ω:

$$ABP(t) = x(t) = Xe^{j\omega t} = |X|e^{j(\omega t+\theta_{ABP})}$$

where

$$X = |X|e^{j\theta_{ABP}}$$

$|X|$ corresponds to the amplitude of each frequency component of the ABP pulse and θ_{ABP} corresponds to the phase of each frequency component of the ABP pulse.

Similarly, ICP can be expressed as a function $y(t)$:

$$ICP(t) = y(t) = Ye^{j\omega t} = |Y|e^{j(\omega t+\theta_{ICP})}$$

for

$$Y = |Y|e^{j\theta_{ICP}}$$

Where $|Y|$ corresponds to the amplitude of each frequency component of the ICP pulse and θ_{ICP} corresponds to the phase of each frequency component of the ICP pulse.

The response of the ICP pulse to each sinusoidal mode of oscillation of the ABP pulse entails a change in amplitude and phase of the sinusoid. If we denote each mode of oscillation of the ABP as $ABP(\omega)$ and each mode of oscillation of the ICP as $ICP(\omega)$, the amplitude change is the gain $G(\omega)$:

$$G(\omega) = |H(j\omega)| = \frac{|Y|}{|X|} = \frac{|ICP(\omega)|}{|ABP(\omega)|}$$

The phase change for the sinusoid of frequency ω is

$$\theta(\omega) = \arg[H(j\omega)] = \theta_{ICP} - \theta_{ABP}$$

Setting the ABP pulse as the reference phase, the phase of the sinusoidal component of the ICP pulse θ_{ICP} at each mode of oscillation ω is the phase transfer function $\theta(\omega)$ evaluated at that frequency.

This raises a question that is important in the physical interpretation of the transfer function of the cerebral windkessel. In what way are the phase transfer function $\theta(\omega)$ and the impedance phase θ_{ICP} of the ICP pulse related?

Consider the nature of the ICP pulse. The ICP pulse, as noted above, is a standing wave excited in the cranium by the ABP pulse. The ABP and the ICP pulse are, by Fourier's theorem, the spectrum of individual modes of oscillation (frequencies) that sum to produce the waveforms. This represents the physical reality of the pulse, not merely a mathematical device. The cranium has its own natural modes of oscillation, each with its own natural frequency, and its own amplitude and phase response. The ABP pulse is essentially an energy pump, and transmits energy in the form of a spectrum of oscillations of different frequency, amplitude and phase into the cranium. The cranium in turn is excited by this energy. However, the cranium has its own natural modes of oscillation, which vary in frequency, phase and amplitude.

The transfer function reflects the fact that specific modes of oscillation in the ABP pulse excite specific modes of oscillation in the ICP pulse. The energy transfer from the ABP to the ICP is work, and the work done by a harmonic force (the ABP pulse) on a harmonic oscillator (the ICP pulse) has several characteristics:

1. The work done by a harmonic force acting on a harmonic velocity (or displacement) of *different* frequency is zero over a time interval comprising an integer number of force and displacement cycles.
2. The work done by a harmonic force in phase with a harmonic *displacement* of the same frequency is zero over a cardiac cycle.
3. The work done by a harmonic force in phase with a harmonic *velocity* of the same frequency is $\pi P_0 v_0$ over a cardiac cycle (where P_0 is the force of the ABP pulse and v_0 is the velocity).

This means that the modes of oscillation of the ABP pulse only do work on (i.e. augment) modes of the ICP pulse that have velocities that are in phase with the modes of the ABP pulse. Thus the cranium is *selective* in its augmentation and suppression of the ABP pulse, and the ICP pulse is a function of this selectivity. The transfer function is a measure of this selectivity, both with respect to amplitude and with respect to phase. The velocity phase of the modes of the ICP pulse are in turn a function of the impedance phase θ_{ICP} of the ICP, because the phases of the modes of oscillation of the cranium are determined by the impedance phase θ_{ICP} of the cranial contents.

Thus, the cranium will selectively accentuate and suppress the ICP response to modes of the ABP pulse depending on the correspondence of the impedance phase θ_{ABP} of the ABP modes and the impedance phase θ_{ICP} of the ICP modes.

The shape of the ICP waveform is determined by the interplay between ABP and ICP impedance. This interplay determines the manner in which the energy of the ABP pulse is transferred to the ICP pulse. If the intracranial impedance has an inertial bias, which it normally appears to have, this inertial bias (i.e. positive impedance phase θ_{ICP}) will cause the ICP waveform to be dominated by input from inertially biased modes of oscillation of the ABP pulse of the same impedance phase. In the steady state, this inertial bias will shape the ICP waveform, which will cause the waveform of the ICP to lead the waveform of the ABP both in the time domain (fig 1) and in the frequency domain (fig 9). Thus, the phase transfer function $\theta(\omega)$ correlates with the impedance angle θ_{ICP} of the ICP pulse, evaluated in the frequency domain and the time domain, respectively.

Leading phase at anti-resonance in damped windkessel

As noted, several investigators have found that the normal ICP pulse leads the arterial pulse [1,6,11-14]. Wagshul et al [14] used transfer function analysis to measure the average lead at roughly 60 degrees. The ICP pulse is a standing wave excited in the cranium by the ABP pulse, and therefore has a phase relation to the ABP which may lead or lag depending on the balance of intracranial inertia and elastance. So it is clear *how* the ICP can lead the ABP. But the question remains: *why* does the ICP pulse lead the ABP? Does it serve a physiological purpose? Consideration of the conditions necessary for optimal pulsation absorption in a damped windkessel suggests that it may serve a purpose.

In windkessel theory, the heart rate is at the natural frequency of oscillation of the intracapillary space. Blood flow in large arteries is in phase with arterial blood pressure, because this

optimizes blood flow. This is supported by the observation that arterial pressure and flow are normally synchronous [30], which suggests resonance [1,6].

Consider the frequency ratio f at which a damped windkessel is most effective. At anti-resonance, the heart rate ω is equal to the natural frequency of the intracapillary space Ω_{IC}, and for optimal effectiveness of pulsation absorption the natural frequency of the extracapillary space ω_{EC} must be half that of the heart rate:

$$f = \frac{\omega_{EC}}{\Omega_{IC}} = \frac{1}{2}$$

So:

$$\omega = \Omega_{IC} = \sqrt{\frac{K_{IC}}{M_{IC}}} = 2\omega_{EC} = 2\sqrt{\frac{k_{EC}}{m_{EC}}}$$

To examine the effect this modification of the damped windkessel has on the phase of the extracapillary space, consider the general impedance equation for the ICP pulse as a simple single-degree-of-freedom forced harmonic oscillator with mass m_{EC}, elastance k_{EC}, and damping c_{EC} driven at a frequency ω which is not necessarily its natural frequency. The ICP impedance phase is

$$\theta_{EC} = \tan^{-1}\left[\frac{\omega m_{EC} - \frac{k_{EC}}{\omega}}{c_{EC}}\right]$$

Consider the substitution:

$$m_{EC} = \frac{k_{EC}}{\omega_{EC}^{\,2}}$$

Substituting, we get

$$\theta_{EC} = \tan^{-1}\left[\frac{\frac{\omega k_{EC}}{\omega^2{}_{EC}} - \frac{k_{EC}}{\omega}}{c_{EC}}\right]$$

Rearranging:

$$\theta_{EC} = \tan^{-1}\left[\frac{k_{EC}\left(\frac{\omega}{\omega^2{}_{EC}} - \frac{1}{\omega}\right)}{c_{EC}}\right]$$

Multiplying by $\frac{\omega}{\omega}$

$$\theta_{EC} = \tan^{-1}\left[\frac{k_{EC}\left(\left[\frac{\omega}{\omega_{EC}}\right]^2 - 1\right)}{\omega c_{EC}}\right]$$

This gives the general relationship between phase and frequency ratio for an oscillator driven at ω. To return to the analogy of the windkessel, within the center of the notch there is not a phase transition, and the ICP pulse behaves as a simple single-degree-of-freedom oscillator with respect to phase. If the heart rate ω is greater than the natural frequency ω_{EC} of the extracapillary space, the impedance phase θ_{EC} is positive. That is, the ICP leads the ABP. If the heart rate ω is less than the natural frequency of the extracapillary space ω_{EC}, the impedance phase θ is negative—the ICP lags the ABP.

Now, we can apply this analogy to the normal cerebral windkessel. Because

$$f = \frac{\omega_{EC}}{\Omega_{IC}} = \frac{1}{1+\mu} = \frac{1}{2}$$

Therefore

$$\omega = \Omega_{IC} = 2\omega_{EC}$$

The natural frequency of the extracapillary space in an optimally damped cerebral windkessel is half that of the heart rate (and half the natural frequency of the intracapillary space).

So the equation giving the general relationship between phase and frequency ratio for an optimally damped oscillator with a mass ratio μ of 1 driven at ω becomes

$$\theta_{EC} = \tan^{-1}\left[\frac{3k_{EC}}{\omega c_{EC}}\right]$$

which is a positive impedance phase for all values of k_{EC}, ω, and c. Applied to the windkessel, this means that *when the windkessel is most effective (i.e. in the region of anti-resonance), the ICP pulse will lead the ABP pulse.*

The general equation for phase and frequency ratio

$$\theta_{EC} = \tan^{-1}\left[\frac{k_{EC}\left(\left[\frac{\omega}{\omega_{EC}}\right]^2 - 1\right)}{\omega c_{EC}}\right]$$

also explains the progressive phase lag of ICP with intracranial hypertension identified by several investigators [11,14]. Intracranial hypertension increases intracranial elastance k_{EC}, raising the square of the natural frequency ω_{EC} of the extracapillary space proportionately to k_{EC}:

$$\omega_{EC}^{\,2} = \frac{k_{EC}}{m_{EC}}$$

Since

$$\theta_{EC} = \tan^{-1}\left[\frac{k_{EC}\left(\left[\frac{\omega}{\omega_{EC}}\right]^2 - 1\right)}{\omega c_{EC}}\right] =$$

$$\tan^{-1}\left[\frac{k_{EC}\left(\frac{\omega^2}{k_{EC}/m_{EC}}-1\right)}{\omega c_{EC}}\right]=\tan^{-1}\left[\frac{\omega^2 m_{EC}-k_{EC}}{\omega c_{EC}}\right]$$

if the heart rate ω remains the same and k_{EC} increases, the numerator becomes less positive, making the impedance phase θ progressively less positive, resulting in corresponding shifts of ICP phase to negative as the elastance (and ICP) increase.

Intuitively, the normal phase lead of ICP may be understood as follows. Damping (resistance) in the cranium suppresses motion of the extracapillary fluid pulse. This is frictional loss, because damping is (by definition) energy loss by dissipation. To overcome this frictional suppression of the extracapillary pulse, which is a suppression of the pulsation absorber effect of the windkessel, an increase in mass of the pulse is necessary, because inertia mitigates impediment due to frictional force. This principle is widely recognized in ballistics: a heavier projectile is more accurate than a light projectile because it is less deflected by wind friction. A damped windkessel is more effective if there is an inertial bias in its impedance, which helps overcome damping. The phase lead of ICP with respect to ABP represents an optimally effective damped windkessel. This is apparent conceptually and can be demonstrated mathematically.

Stress in brain tissue associated with windkessel function and impairment

The process of windkessel pulsation absorption necessarily entails motion in the extracapillary space. It is in fact the extracapillary motion that *absorbs* the radial pulsatility from the capillaries. Normal function of the windkessel causes motion stress in the brain, and abnormal function of the windkessel can, conceptually, either increase or decrease that stress. Conditions such as arteriosclerosis and hydrocephalus can impair the windkessel, and it is useful to consider the alterations of the stress on functional neurological tissue occasioned by these impairments.

For the calculation of stress on brain tissue associated with the windkessel, we first need to determine the optimal damping ratio. This can be done for a dynamic vibration absorber [36], which gives for optimal damping

$$\left(\frac{c}{c_c}\right)^2=\frac{\mu\left(3-\sqrt{\frac{\mu}{\mu+2}}\right)}{8(1+\mu)^3}$$

and

$$\left(\frac{c}{c_c}\right)^2=\frac{\mu\left(3+\sqrt{\frac{\mu}{\mu+2}}\right)}{8(1+\mu)^3}$$

Averaging between these two peaks gives the optimum damping for the cerebral windkessel:

$$\left(\frac{c}{c_c}\right)^2=\frac{3\mu}{8(1+\mu)^3}$$

For μ=1 the optimal damping ratio is:

$$\frac{c}{c_c}=0.22$$

To determine the stress in the brain tissue in the damped windkessel model, we will make use of the fact that at resonance the phase angle between the pressure and flow is near zero and the phase angle between pressure and the displacement is -90 degrees (at resonance, displacement lags pressure and flow by a quarter cycle).

Therefore the work done per cycle by force P_0 is

$$W=\pi P_0 x_{IC}\sin 90^0=\pi P_0 x_{IC}$$

This holds approximately true for modest deviations from resonance, because the sine changes rather little with deviations around 90 degrees.

The work done by damping is proportional to the product of the damping force and the displacement. The damping force is exactly in phase with the velocity (by definition) and thus leads the displacement by a quarter cycle near resonance

$$W_{dissipated}=\pi\left(c_{EC}\omega x_{rel}\right)x_{rel}=\pi c_{EC}\omega {x_{rel}}^2$$

where x_{rel} is the relative displacement amplitude between the intracapillary space and the extracapillary space and is a measure of the pulsatile stress on the brain.

Bringing the two equations together we get

$$\pi P_0 x_{IC}=\pi c_{EC}\omega {x_{rel}}^2$$

which is

$$x_{rel}^2=\frac{P_0 x_1}{c_{EC}\omega}$$

In dimensionless variables this is:

$$\left(\frac{x_{rel}}{x_{STIC}}\right)^2=\frac{x_{IC}}{x_{STIC}}\frac{1}{2g\frac{c_{EC}}{c_c}}$$

This equation is quite valuable for applying windkessel theory to hydrocephalus. To the left of the equal sign,

$$\left(\frac{x_{rel}}{x_{STIC}}\right)^2$$

is the (square) of the displacement amplitude between the intracapillary space and the extracapillary space, which is a measure of the stress on the elastance elements in the extracapillary space—effectively, the strain on the brain tissue through which

arterial pulsations are absorbed. To the right of the equal sign are two terms.

$$\frac{x_{IC}}{x_{STIC}}$$

is the displacement of the capillary walls, and

$$\frac{1}{2g\frac{c_{EC}}{c_c}}$$

which is a term of proportionality between strain on brain tissue and capillary pulsatility. g is the ratio between the heart rate and the natural frequency of the intracapillary space, assumed to be near 1.

$$\frac{c_{EC}}{c_c}$$

is the damping ratio, and correlates with resistance in the extracapillary tissues, most notably resistance in the CSF spaces.

Conceptually, brain tissue stress can be expressed as:

$$\text{Brain tissue stress} \propto \text{Capillary wall motion} \bullet \frac{1}{Damping}$$

That is, pulsatile brain tissue stress is proportional to capillary wall motion and to the inverse of damping. Brain tissue stress is inversely proportional to damping. The meaning of the equation is that damping (high resistance in CSF pathways) *mitigates* brain tissue stress caused by windkessel dysfunction. The motion of the brain tissue is part of the absorber component of the windkessel, and with impairment of the windkessel the absorber (brain tissue) may move with greater amplitude or less amplitude *depending on the damping*, placing more or less stress on brain tissue. If the damping in the absorber (e.g. resistance in the CSF spaces) is low, then there is high stress on the brain tissue with windkessel impairment. If the damping in the CSF spaces is high, this mitigates the strain on the brain by effectively "gluing" the extracapillary space to the intracapillary space. This gluing together of the intra- and extra-capillary spaces is very dangerous for the capillaries, because it may catastrophically disable the windkessel, but it tends to reduce somewhat the motion stress that windkessel dysfunction causes on the brain tissue itself.

Physiologically, this implies that windkessel dysfunction due to high resistance in the CSF spaces can cause substantial capillary damage and cerebral edema, but the pulsatile stress on the brain is mitigated by the high resistance. Low resistance in the CSF spaces, on the other hand, protects the capillaries but magnifies pulsatile stress on the brain caused by windkessel dysfunction.

Brain stress and hydrocephalus

It is interesting to apply windkessel-related brain stress to communicating hydrocephalus. Proper functioning of the windkessel depends on appropriate ratios of inertia, elastance, damping and heart rate. There is good evidence that experimental hydrocephalus and NPH are associated with windkessel impairment [26,27]. Conceptually, this windkessel impairment in hydrocephalus may take two forms: excessive elastance and excessive resistance. In hydrocephalus, extracapillary elastance and/or damping may be elevated, causing an imbalance in absorber dynamics and impairment of the windkessel.

In hydrocephalus caused by obstruction of the CSF spaces (for example, from subarachnoid hemorrhage or meningitis), there is significant increase in damping (resistance) in the cranium. This increased resistance is particularly damaging to the windkessel, because the absorber mechanism depends on free flow of CSF to link arterial pulsations to venous pulsations. Without free flow of CSF, the intracapillary space and the extracapillary space are essentially "glued" together and the flow of pulsations in the cranium loses its two-degree-of-freedom character and behaves more like a single-degree-of-freedom oscillator, with high amplitude resonance at synchrony between the capillary pressure and flow. This is quite dangerous and may cause brain edema, intracranial hypertension and loss of capillary integrity, requiring CSF diversion to lower elastance by lowering ICP and to lower resistance by providing an additional parallel pathway for pulsations. This restoration of normal elastance and resistance helps restore normal windkessel dynamics.

Counterintuitively, high damping in the CSF spaces in obstructive hydrocephalus is relatively *protective* of brain tissue (although it is destructive of capillary beds). With high

$$\frac{c_{EC}}{c_c}$$

the term of

$$\frac{1}{2g\frac{c_{EC}}{c_c}}$$

proportionality is small, and the strain on brain tissue

$$\frac{x_{rel}}{x_{STIC}}$$

caused by capillary windkessel impairment

$$\frac{x_{IC}}{x_{STIC}}$$

is relatively small. The increase in capillary pulsatility with windkessel impairment in obstructive hydrocephalus is life-threatening due to edema and capillary disruption, but pulsatile brain tissue stress is mitigated by the same increase in resistance that endangers the capillaries.

Now consider the application of windkessel-related brain stress to NPH. The cause of NPH is not well established, but it is clear that ordinarily there is no significant obstruction to CSF flow within the CSF spaces. NPH appears to be associated with arteriosclerosis, and Greitz, Bateman and others [2,3,21] have suggested that restriction of subarachnoid arterial pulsatility due to arteriosclerosis plays a role. NPH has been shown to be associated with windkessel dysfunction [26].

Windkessel impairment in NPH is of a different nature than windkessel impairment in obstructive hydrocephalus. In NPH, the CSF spaces are open. In obstructive hydrocephalus, the CSF spaces are obstructed. The equations show that open CSF

spaces, in the setting of windkessel dysfunction, cause considerable stress on brain tissue. In NPH the windkessel dysfunction is less destructive of capillary beds (the two-degree-of-freedom nature of the cerebral windkessel remains intact—the extracapillary space is not "glued" to the intracapillary space.) However, in NPH the motion stress on brain tissue may be *greater* than in communicating hydrocephalus caused by CSF pathway obstruction, because with low

$$\frac{c}{c_c}$$

the term of proportionality

$$\frac{1}{2g\frac{c_{EC}}{c_c}}$$

is large, and the strain on brain tissue

$$\frac{x_{rel}}{x_{STIC}}$$

caused by capillary windkessel impairment

$$\frac{x_{rel}}{x_{STIC}}$$

is proportionately large.

The windkessel impairment in NPH is not life-threatening, but there is considerable strain on brain tissue with windkessel impairment. This would be expected to be most severe in the tissue adjacent to the CSF pathways, such as the periventricular white matter, through which pathways that mediate gait and urinary continence project.

Conceptually, from the perspective of windkessel theory, obstructive hydrocephalus stresses capillaries but protects brain tissue from motion stress. NPH protects capillaries but stresses brain tissue. This theoretical perspective is consistent with the life-threatening manifestations of obstructive hydrocephalus, and the gait apraxia and urinary incontinence, without threat to life, that are characteristic of NPH.

In windkessel theory, NPH would be particularly responsive to 'tuning' of the programmable shunt to obtain optimal intracranial elastance. Unlike the dynamics of obstructive hydrocephalus, in which the normal two-degree-of-freedom windkessel is 'glued' together to create a life-threatening one-degree-of-freedom resonant system, NPH preserves the two-degree-of-freedom absorber mechanism of the windkessel, but 'detunes' it by increasing elastance. This may account for the responsiveness of NPH to tuning with programmable valves, as compared with obstructive hydrocephalus, which is not as responsive to valve adjustment.

Energy, power and cerebral blood flow

Work is done by the heart in propelling cerebral blood flow, and there are obvious physiological reasons why cerebral perfusion should be maximally efficient. Milnor has remarked [7] that in a young healthy animal the arterial tree is a remarkably efficient diffuser, meaning that there is vanishingly little reflectance of kinetic energy retrograde through the arterial tree back to the left ventricle. Virtually all of the energy of circulation is used to propel blood through the microvasculature. Milnor notes that the degree of efficiency achieved by the arterial perfusion is greater than has been accomplished in most sophisticated engineering applications.

Yet the heart is a pulse pump, which presents a problem for efficient perfusion. Consider a simple model of the cerebral circulation, in which the pressure is:

$$P = P_0 \sin\left(\omega t + \varphi\right)$$

Note that the phase angle φ represents the displacement phase between the pressure and the displacement, not the impedance phase angle between the pressure and the flow.

It is helpful to derive the work done per unit time (the power) associated with cerebral perfusion. The displacement of the blood distally in the cerebral arterial tree is x. The work done by a small displacement is force multiplied by distance

$$W = Pdx = P\frac{dx}{dt}dt$$

ωt varies from 0 to 2π, and thus t varies from 0 to $\frac{2\pi}{\omega}$.

The work done by one cardiac cycle is

$$W = \int_0^{\frac{2\pi}{\omega}} P\frac{dx}{dt}dt = \frac{1}{\omega}\int_0^{2\pi} P\frac{dx}{dt}d\left(\omega t\right) =$$

$$P_0 x_0 \int_0^{2\pi} \sin\left(\omega t + \varphi\right)\left(\cos\omega t\right)d\left(\omega t\right)$$

$$x_0 \int_0^{2\pi} \cos\omega t\left[\sin\omega t\cos\varphi + \cos\omega t\sin\varphi\right]d\left(\omega t\right)$$

$$= P_0 x_0 \cos\phi \int_0^{2\pi} \sin\omega t\cos\omega t d\left(\omega t\right) + P_0 x_0 \sin\phi \int_0^{2\pi} \cos^2\omega t d\left(\omega t\right)$$

The first integral is zero and the second is π, so the work done in a cardiac cycle is

$$W = \pi P_0 x_0 \sin\varphi$$

Note that the work available to propel blood is a function of the displacement phase φ. If the heart beat is out of phase (out of sync) with the cranial contents, cerebral blood flow is impaired.

An equivalent way to characterize the work done per unit time by cerebral blood flow is the power factor, which is given by the cosine of the impedance phase for the intracapillary (intravascular) space

$$\text{Power factor} = \cos\left[\tan^{-1}\left(\frac{\omega m_{IC} - \frac{k_{IC}}{\omega}}{c_{IC}}\right)\right]$$

Power factor is the ratio of active (effective) power to apparent power (the algebraic sum of pressure and flow). When the power factor is 1 (at resonance and anti-resonance), the work of cerebral blood flow is optimized, with minimal reflection of kinetic energy back to the left ventricle.

The work done by the heart on cerebral blood flow is proportional to the sine of the displacement phase φ between the pressure and displacement of the blood, and is proportional to the cosine of the impedance phase θ between the pressure and the flow. This concept is important in understanding the role of resonance and of the windkessel in cerebral blood flow. Cerebral blood flow is maximally efficient when pressure and flow are in phase and pressure and displacement of blood are a quarter cycle (displacement lagging) out of phase, which represents resonance. This represents the maximal work that can be done on propelling blood distally by the left ventricle and represents optimal and efficient cerebral blood flow. However, it also entails high amplitude oscillations of vessel walls, which endangers capillaries.

It is in this principle of pulsatile flow that the nature of the problem addressed by the windkessel is clear: *the most efficient cerebral blood flow is when the ABP is synchronous with the flow pulse*. This is resonance, and it characterizes efficient blood flow. However, resonance in the cranium represents a *high* amplitude response in the microvasculature, which endangers capillaries. This would seem to create an impasse. Synchrony between pressure and flow is necessary to maximize cerebral blood flow, but asynchrony—mutual cancellation actually—is necessary to protect capillaries from pulsatility.

The cerebral windkessel solves this problem by the pulsation absorption mechanism. The windkessel creates a two-degree-of-freedom system tuned to anti-resonance, in which the arterial pulse is transposed through the CSF to the veins. The power factor is one and cerebral blood flow is maximally efficient, and at the same time the pulse is short-circuited through the CSF to the veins, rendering the capillary perfusion pulseless. Remarkably, the resonant and anti-resonant characteristics of intracranial blood flow are created by the same mechanism—the arterial-CSF-venous pump. The arterial-CSF-venous pump transposes the arterial pulse efficiently (resonance) and absorbs the arterial pulsations in order to protect the capillaries (anti-resonance). Thus, the windkessel solves the impasse of pulsatile cerebral perfusion by the use of a dual purpose pulse pump: the transposition of the arterial pulse through the CSF to the veins simultaneously optimizes cerebral blood flow and protects capillaries from arterial pulsatility.

Another perspective on the way the windkessel works is that it removes the kinetic energy of radial capillary motion from the capillaries and transfers that kinetic energy to the absorber mechanism in the extracapillary space. As noted, the prime absorber mechanism in the extracapillary space is the compression and relaxation of intracranial veins. Thus, the windkessel, when working properly, transfers the kinetic energy of radial capillary pulsatile motion to venous blood flow, thereby protecting the capillaries while efficiently using the kinetic energy thus diverted to pump venous blood, maintaining optimal cerebral blood flow.

Otto Frank's hydraulic integrator

The adaptation of intracranial dynamics to pulsatile perfusion from the heart has analogues in the extracranial systemic circulation. The cerebral windkessel is analogous in important ways to the mechanism for the smoothing of blood flow in the aorta that was described by physiologist Otto Frank in the 19th century [37].

Frank noted that during systole a portion of the left ventricular stroke volume was stored in the expansion of the aorta, and that during relaxation of the aorta in diastole the stored blood returned to the circulation. Frank termed this rhythmic storage and release of a portion of the systolic stroke volume a "hydraulic integrator". He proposed that by diminishing systolic flow and augmenting diastolic flow, the aortic hydraulic integrator served to convert much of the pulsatile flow of blood at the aortic root into smoother flow more distally in the circulation. This was part of the systemic windkessel.

A closer look at pulsatile intracranial dynamics suggests that the dynamics of the hydraulic integrator in the aorta and of the cerebral windkessel in the cranium share similarities, and that the arterial-CSF-venous pump in the cranium is a type of hydraulic integrator adapted to the rigid confines of the cranial cavity.

In the cerebral arteries, a portion of the cerebral systolic stroke volume is stored in the expanded arterial walls, in the same way that a portion of the aortic stroke volume is stored in the expanded aortic walls. Because the cerebral arteries, unlike the aorta, are encased in the rigid cranium, the elastance provided by the arterial walls in the cranium is insufficient to permit expansion and systolic storage of blood volume. In the cranium, arterial expansion is made possible by the easily displaced venous blood, which is the only appreciable elastance in the cranium that can accommodate rapid changes in intracranial volume [18]. The CSF serves as the hydraulic coupling between the cerebral arteries and veins, thus solving the problem that the complex geometry of the cerebral vasculature and the encasement in the rigid cranium poses for the hydraulic integrator.

In a manner analogous to the aortic circulation, the cerebral windkessel is 'charged' by arterial expansion and venous ejection in systole and 'discharged' by arterial relaxation and venous filling in diastole. The arterial pulse thus circumvents the capillary circulation. CSF provides the necessary hydraulic linkage for this system to work.

The windkessel and physiological regulation

The effectiveness and efficiency of the cerebral windkessel—the efficient circulation through the cranium of energy in the form of pulsations that bypasses the capillary circulation—depends critically on anti-resonance. Because anti-resonance is achieved by a specific ratio of variables in the cranium, it is likely that there are physiological mechanisms, not yet understood, that maintain it. Maintenance of anti-resonance depends on maintenance of a specific impedance phase, which in turn depends on maintenance of appropriate ratios of inertia (m_{EC}, which corresponds to the stroke volume of blood), elastance (k_{EC}, which correlates with venous pressure), damping (c_{EC}, which represents vascular resistance and structural damping) and frequency (ω, which is the heart rate in radians/second).

Maintenance of this balance is necessary to a system of autoregulation of the windkessel.

For impedance to be optimal, inertial reactance (ωm_{EC}) and elastic reactance (k_{EC}/ω) must be balanced:

$$\theta = \tan^{-1}\left[\frac{\omega m_{EC} - \frac{k_{EC}}{\omega}}{c_{EC}}\right]$$

The windkessel depends on a balance of physiological variables (e.g. heart rate) that change constantly, and thus the cerebral windkessel is probably autoregulated. It is theoretically possible that there is sufficient width of the windkessel notch that physiological alterations in heart rate and other parameters don't cause significant dysfunction. Yet it seems likely that the windkessel is, to a greater or lesser extent, maintained actively. What is the physiological mechanism for windkessel autoregulation? It would seem that alteration of heart rate would be a particularly effective method of maintaining the balance of inertial and elastic reactance, because inertial reactance is directly proportional to heart rate and elastic reactance is inversely proportional to heart rate. Fine tuning of heart rate, in the setting of physiological changes in cerebral stroke volume and elastance, would maintain the balance of inertial and elastic reactance and the anti- resonance necessary for the cerebral windkessel.

Yet, how can high impedance resonance be maintained physiologically if it is so sensitive to heart rate, given that heart rate varies significantly during daily life? It is noteworthy that there is a well characterized system of autoregulation of cerebral hemodynamics that is very sensitive to heart rate—cerebral blood flow, which is the product of heart rate and cerebral stroke volume. Cerebral blood flow is known to be autoregulated and maintained to within tight parameters—50 ml/min per 100 grams of brain tissue and is preserved over a broad range of 60-150 mm Hg mean arterial perfusion pressure [38,39].

Autoregulation of the cerebral windkessel probably exists, and it may be related to the autoregulation of cerebral blood flow. Inertial reactance ωm_{EC} is the product of heart rate and cerebral stroke volume, and thus corresponds to the pulsatile component of cerebral blood flow. Autoregulation of the inertial reactance of the cerebral windkessel may be accomplished by the same physiological mechanisms that autoregulate cerebral blood flow.

Autoregulation of elastic reactance

$$\frac{k_{EC}}{\omega}$$

can be accomplished by alterations in cerebrovenous elastance, which in thin-walled veins is determined primarily by cerebrovenous pressure. Although the regulation of cerebrovenous pressure is not well understood, there is evidence that cerebrovenous dynamics are regulated in part by smooth muscle sphincters at the convergence of the cortical veins and the superior sagittal sinus [40], and that this myoendothelial sphincter is innervated by the autonomic nervous system [38,41-44]. Venous constriction raises venous pressure and increases the elastance of the ICP pulse.

Increased sympathetic tone increases heart rate and is a potent constrictor of cerebral veins [40]. Thus, intracranial venous elastance and heart rate are likely to vary together with autonomic regulation, which would tend to maintain constant elastic reactance despite changes in heart rate. Autoregulation of elastic reactance and inertial reactance, both of which depend on autonomic tone, would preserve high impedance resonance and maintain the cerebral windkessel with changing heart rate.

Thus the well-characterized autoregulation of cerebral blood flow and the theoretical but likely autoregulation of the cerebral windkessel share several important characteristics, suggesting commonality between autoregulation of cerebral blood flow and autoregulation of the flow of pulsations in the brain.

DISCUSSION

In addition to the mass circulation of blood and CSF, *energy* circulates through the cranium. Like mass, energy flux in the cranium is conserved. Kinetic energy from the left heart passes through the cranial contents and returns to the right heart. Some of this kinetic energy is in the smooth bulk flow of blood. Some of the kinetic energy is in the pulsatile motion of the cranial fluids and tissues. Within the cranium, the pulsatile energy flow is an exchange of inertial kinetic energy and elastic potential energy between fluids and tissues, as well as the dissipation of energy in the form of heat caused by damping.

Cerebral perfusion is a piston driven by a pulse pump (the left ventricle), and the ICP pulse is a rhythmic exchange of kinetic and potential energy—a standing wave—driven by the arterial pulse. This circulation of pulsatile kinetic and potential energy does not involve the net displacement of blood or CSF—vessels and fluids merely oscillate back and forth in the cranium with the heartbeat.

The details of this energy flow of intracranial pulsations are physiologically important, because cerebral blood flow entails a paradox: cerebral perfusion must be both resonant and anti-resonant. That is, in order to maximize the efficiency of cerebral perfusion, arterial pressure and flow must be synchronous, which is resonance. But resonant flow is destructive of capillaries, because it entails high amplitude oscillations of capillary walls. So safe perfusion of capillary beds requires *anti-resonance*—the suppression of pulsatility in the microvasculature. This means that cerebral perfusion entails two apparently contradictory principles: efficient cerebral blood flow must be resonant, and safe cerebral blood flow in the microvasculature must be anti-resonant.

The cerebral windkessel solves this physiological impasse, and it does so by the application of design principles of vibration control to cerebral blood flow. The cranium is a band stop filter. Arterial pulsations are reflected through the CSF to the veins, and this transposition of the pulse by venous compression and expansion provides an elastic force that continuously opposes the radial motion of the capillary walls (fig 6 and 7). This maintains the resonant dynamics necessary for efficient perfusion and the anti-resonant dynamics necessary for

capillary protection. More succinctly, the resonant CSF-venous pump is an anti-resonant pulsation absorber. Simultaneity of pressure and flow in the large intracranial vessels is resonant, and the rhythmic impedance of capillary expansion in systole and admittance of capillary expansion in diastole is anti-resonant. Resonance and anti-resonance in cerebral perfusion are accomplished by the same mechanism: the transposition of the arterial pulse through the CSF to the veins. The windkessel thus maintains efficient and safe propulsion of blood through the cerebral vasculature.

Thus, the essence of the windkessel is the CSF-venous pump, which provides transposition of the arterial pulse through the CSF to the veins *and* the elastance for pulsation absorption. The windkessel depends on a balance of physiological variables. Intracranial inertia, elastance, damping and heart rate must balance to maintain proper impedance. Imbalance of these forces threatens the microvasculature with resonant, rather than anti-resonant, perfusion, which can be rapidly destructive of thin-walled capillaries. Maintenance of this balance presupposes a system of windkessel autoregulation, which is unexplored but probably exists. There is likely commonality between windkessel regulation and regulation of cerebral blood flow and the maintenance of autonomic tone in the cerebral vasculature.

Salient equations in windkessel theory

The mathematics underlying windkessel dynamics can be summarized by a few salient relationships, given below.

The natural frequency of the ICP pulse is the square root of the ratio of intracranial elastance to the mass of the ICP pulse:

$$\omega_{ICP} = \sqrt{\frac{k_{ICP}}{m_{ICP}}}$$

Where ω_{ICP} is the natural frequency of oscillation of the cranial contents, k_{ICP} is the intracranial elastance, and m_{ICP} is the mass of the ICP pulse.

The impedance phase between intracranial pressure and flow is the arctangent of the ratio between the total reactance (the difference between inertial and elastic reactance) and the damping in the cranium:

$$\theta = \tan^{-1}\left(\frac{\omega m_{ICP} - \frac{k_{ICP}}{\omega}}{c_{ICP}}\right)$$

Where ω is the heart rate (in radians), θ is the impedance angle, and c_{ICP} is the intracranial damping, and m_{ICP} is the mass of the ICP pulse. ωm_{ICP} is the inertial reactance and

$$\frac{k_{ICP}}{\omega}$$

is the elastic reactance. Positive impedance phase causes pressure to lead flow, and negative impedance phase causes pressure to lag flow. In the frequency domain, positive impedance causes the ICP to lead the ABP in the phase transfer function, and negative impedance causes the ICP to lag the ABP.

The phase transfer function of ABP to ICP pulse is:

$$\theta(\omega) = \arg\left[H(j\omega)\right] = \theta_{ICP}(\omega) - \theta_{ABP}(\omega)$$

The phase transfer function uses the input (ABP) phase as the reference phase, and it denotes the phase relationship between modes of oscillation in the ABP pulse and the ICP pulse in the frequency domain. The phase transfer function $\theta(\omega)$ is a function of the ratio of ICP impedance phase θ_{ICP} and ABP impedance phase θ_{ABP} evaluated in the frequency domain.

The volume displacement phase of the ICP pulse is:

$$\varphi = \tan^{-1}\left(\frac{2\frac{c_{ICP}}{c_0}\cdot\frac{\omega}{\omega_{ICP}}}{1-\frac{\omega^2}{\omega_{ICP}^{\ 2}}}\right)$$

Where φ is the displacement phase of the volume of the ICP pulse relative to the pressure, c_0 is the critical damping, ω_{ICP} is the natural frequency of the cranial contents, ω is the heart rate (in radians), and c_{ICP} is the intracranial damping. During normal dynamics, the displacement phase of the pulse of the cranial contents, as measured by flow MRI, lags the ICP pulse and the flow pulse by a quarter cycle.

Resonant capillary displacement with a single degree of freedom:

$$\varphi = \tan^{-1}\left(\frac{2\frac{c_{ICP}}{c_0}\cdot\frac{\omega}{\omega_{ICP}}}{1-\frac{\omega^2}{\omega_{ICP}^{\ 2}}}\right)$$

x_0 is the maximal displacement of the pulse,

$$\frac{\omega}{\omega_n}$$

is the ratio of the heart rate frequency to the natural frequency of the oscillator, P_0 is the maximum forced pressure, k is the elastance and

$$\frac{c}{c_0}$$

the damping ratio, which is the ratio of oscillator damping to critical damping. This equation expresses the displacement for a single degree of freedom system, such as would occur in the capillaries without the windkessel. Note that when the heart rate ω approaches the capillary space's natural frequency ω_n, the displacement of the capillary walls is at a maximum extremum, which would be destructive to the microvasculature.

Anti-resonant displacement of capillary walls in an undamped windkessel with two degrees of freedom:

$$\frac{x_{IC}}{x_{STIC}} = \frac{1-\frac{\omega^2}{\omega_{EC}^{\ 2}}}{\left(1-\frac{\omega^2}{\omega_{EC}^{\ 2}}\right)\left(1+\mu-\frac{\omega^2}{\omega_{EC}^{\ 2}}\right)-\mu}\sin\omega t$$

Where x_{IC} is the capillary wall displacement, x_{STIC} is the maximal static displacement of the capillary wall, ω is the heart rate (in radians), ω_{EC} is the natural frequency of the extracapillary space (i.e. most of the cranial contents), μ is the mass ratio of the vascular and ICP pulse, which is by definition 1. The equation demonstrates that in an undamped windkessel at anti-resonance, in which the natural frequency of the extracapillary space equals the heart rate, the numerator goes to zero and the capillary walls do not move with the pulse. The actual windkessel has damping, which alters the optimal frequency ratio and inescapably permits a small amount of capillary wall motion.

The displacement of the extracapillary space (most of the cranial contents) in an undamped windkessel:

$$\frac{x_{IC}}{x_{STIC}} = \frac{1-\frac{\omega^2}{\omega_{EC}{}^2}}{\left(1-\frac{\omega^2}{\omega_{EC}{}^2}\right)\left(1+\mu-\frac{\omega^2}{\omega_{EC}{}^2}\right)-\mu}\sin\omega t$$

Where x_{IC} is the capillary wall displacement, x_{STIC} is the maximal static displacement of the capillary wall, ω is the heart rate (in radians), ω_{EC} is the natural frequency of the extracapillary space (i.e. most of the cranial contents), μ is the mass ratio of the vascular and ICP pulse, which is by definition one. The equation demonstrates that the extracapillary space in the cranium will oscillate at anti-resonance and absorb pulsations from the capillaries.

Pressure and flow impedance phase:

$$\vec{P}_{ICP} = \vec{F}_{ICP}\angle\theta$$

$$\vec{F}_{ICP} = \vec{P}_{ICP}\angle-\theta$$

Where $\vec{P}_{ICP}$ is pressure, $\vec{F}_{ICP}$ is flow, θ is the impedance angle, with the relationship between pressure and flow expressed in polar form. Inertia, elastance and heart rate have opposite effects on the timing pressure and flow pulses. Positive reactance (excess inertia) causes flow to lag pressure, and negative reactance (excess elastance) causes flow to lead pressure.

Location of resonance peaks in frequency domain:

$$\omega_{resonant\,HIGH} = 1.62\omega$$

$$\omega_{resonant\,LOW} = 0.62\omega$$

Where ω is the central frequency, $\omega_{resonant\,HIGH}$ is the frequency of the resonant peak above the windkessel notch, and $\omega_{resonantLOW}$ is the frequency below the windkessel notch. This equation is derived from the undamped windkessel model, and the actual peaks, with damping, will be farther apart (see bandwidth and quality factor)

Bandwidth and quality factor of notch:

$$\Delta\omega = \frac{\omega_{anti-resonant}}{Q}$$

Where $\Delta\omega$ is the bandwidth of the notch, Q is the quality factor, and $\omega_{anti-resonant}$ is the central frequency of the notch. Q is the ratio of energy stored to energy dissipated by intracranial pulsations per cardiac cycle. Q describes the 'sensitivity' or narrowness of the notch, and bandwidth describes the width of the notch.

Brain tissue motion stress due to windkessel:

$$\left(\frac{x_{rel}}{x_{STIC}}\right)^2 = \frac{x_{IC}}{x_{STIC}}\frac{1}{2g\frac{c_{EC}}{c_c}}$$

Where

$$\left(\frac{x_{rel}}{x_{STIC}}\right)^2$$

is the (square) of the displacement amplitude between the intracapillary space and the extracapillary space, which is a measure of the stress the brain tissue due to the windkessel.

$$\frac{x_{IC}}{x_{STIC}}$$

is the dimensionless displacement of the capillary walls, and

$$\frac{1}{2g\frac{c_{EC}}{c_c}}$$

is the proportionality between strain on brain tissue and capillary pulsatility. g is the ratio between the heart rate and the natural frequency of the intracapillary space, which is approximately 1.

$$\frac{c_{EC}}{c_c}$$

is the damping ratio, and correlates with resistance in the extracapillary tissues, most notably resistance in the CSF spaces. Conceptually, the equation can be written

$$\text{Brain tissue stress} \propto \text{Capillary wall motion} \bullet \frac{1}{Damping}$$

That is, brain tissue stress is proportional to capillary wall motion and inversely proportional to intracranial damping.

Influence of windkessel on power factor of cerebral blood flow:

$$\text{Power factor} = \cos\theta = \cos\left[\tan^{-1}\left(\frac{\omega m_{IC} - \frac{k_{IC}}{\omega}}{c_{IC}}\right)\right]$$

Where θ is the impedance angle, ω is the heart rate (in radians), c_{ICP} is the intracranial damping, and m_{ICP} is the mass of the ICP pulse. This equation expresses the fact that arterial pulse pressure and flow need to be resonant (in phase) for maximally efficient cerebral blood flow. This corresponds to anti-resonant dynamics in the capillaries, because the CSF-venous pump, which is part of the resonant flow in the arteries and veins, is also the dynamic absorber (anti-resonant) elastance of the windkessel.

Impedance angle and frequency ratio of extracapillary space and heart rate; the phase lead of ICP in normal dynamics:

$$\theta_{EC} = \tan^{-1}\left[\frac{k_{EC}\left(\left[\frac{\omega}{\omega_{EC}}\right]^2 - 1\right)}{\omega c_{EC}}\right]$$

Where θ is the impedance angle, ω is the heart rate (in radians), c_{EC} is the intracranial damping, m_{EC} is the mass of the ICP pulse, and k_{EC} is the elastance of the extracapillary space. This equation expresses the impedance angle as a function of the relation between the heart rate and the natural frequency of the extracapillary space. Damping in the cranium makes it necessary for the windkessel to have a dominance of inertia over elastance in order to optimize the windkessel. This means that the ratio of

$$\frac{\omega}{\omega_{EC}}$$

is greater than 1, which means that the impedance angle of the optimal windkessel is somewhat positive. Positive impedance angle causes the ICP pulse to lead the arterial pulse, which has been observed experimentally [1,6,11-14].

Conceptual summary of the cerebral windkessel

Our understanding of the cerebral windkessel is based on the mathematics of its dynamics, which is the dynamics of a damped dynamic pulsation absorber with two degrees of freedom. The principles derived from the mathematics of the windkessel may be summarized:

1. The intracranial pressure pulse is a standing wave in the craniospinal cavity that is excited by the arterial pulse.
2. The cerebral windkessel is a pulsation absorber oscillating at anti-resonance. The extracapillary space absorbs the pulsations from the intracapillary space.
3. The amplitude and the phase of the ICP pulse varies with the heart rate, the inertia, the elastance, and the damping of the intracranial pulse, in accordance with the mathematical description of a pulsation absorber.
4. The normal state of intracranial dynamics is anti-resonance at the fundamental frequency of the heart rate. This represents synchrony between the ICP wave and the arterial pulse. In the frequency domain, there is a notch centered at the heart rate in the arterial-ICP amplitude transfer function and a small phase angle lead, which is probably the consequence of high inertance necessary to optimize the effectiveness of the damped windkessel.
5. Anti-resonance, which is minimal amplitude response, is accomplished by the transposition of the arterial pulse through the CSF to the cerebral veins (the CSF-venous pump), which diverts the arterial pulse and protects the cerebral microvasculature from arterial pulsatility.
6. During systole, CSF links arterial expansion to venous compression. During diastole, CSF links venous expansion to arterial relaxation. The systolic/diastolic attenuation/augmentation of arterial flow and synchronous augmentation/attenuation of venous flow provides pulseless capillary perfusion.
7. Normal intracranial dynamics entails maximal efficiency of cerebral blood flow and minimal pulsatility in the cerebral microvasculature, both of which depend on normal windkessel function.
8. Windkessel dysfunction can occur as the result of changes in the balance of heart rate, inertia, elastance and damping in the cranium, with loss of anti-resonance.
9. Loss of anti-resonance causes retrograde arterial reflection of the arterial pulse, inefficiency of cerebral blood flow, increased radial pulsatility in the cerebral microvasculature, cerebrovenous hypertension (from impairment of the CSF-venous pump), cerebral edema and loss of capillary integrity. This increases intracranial elastance and predisposes to a cascade of windkessel degradation.
10. Maintenance of anti- resonance presupposes a system of physiological autoregulation of the windkessel, which entails effective ratios between heart rate and intracranial inertia, elastance, and damping.

Correspondence of windkessel theory with experiment

With these principles in mind, it is noteworthy that windkessel theory provides an explanation for several perplexing aspects of intracranial dynamics. Windkessel theory provides an explanation for

1. the mechanism by which capillary pulsatility is minimized in the cranium while synchrony of arterial pressure and flow is preserved [1,3,15,30]. (The windkessel is a dynamic pulsation absorber tuned to the heart rate).
2. why the intracranial venous waveform is synchronous with the arterial pulse and manifests some characteristics of the arterial waveform [3,8,10]. (The windkessel depends on the arterial—CSF—venous pump, which transposes the arterial pulse through the CSF to the veins.)
3. the important physiological role for CSF. CSF is the hydraulic link between arteries and veins that makes transposition of the arterial pulse to the veins, bypassing the capillaries, possible. The CSF is essential to the resonant properties of windkessel (efficient cerebral blood flow) and the anti-resonant properties of the windkessel (protection of capillary beds) [1,2].
4. the observation that normal ICP pulse amplitude is at a minimum extremum, compared to ICP pulse amplitude at both high and low ICP [18,19]. (The windkessel is a tuned dynamic pulsation absorber that is most effective at normal intracranial elastance.)

5. the quarter cycle phase lead of CSF flow in syrinxes measured by flow MRI [1,32]. (A syrinx is an elastic capacitor, and flow leads pressure by a quarter cycle when elastic reactance greatly exceeds inertial reactance.)
6. the quarter cycle phase lag in brain expansion measured by flow MRI [3]. (At resonance, volume displacement in an oscillator lags pressure and flow by a quarter cycle).
7. the windkessel notch is observed in the frequency domain by transfer function analysis of the ABP and the ICP, and windkessel theory predicts with reasonable accuracy the location of the resonance peaks that form the walls of the notch. [24-28] (In the frequency domain, dynamic pulsation absorbers are characterized by an anti-resonant notch at the central frequency with resonance peaks associated with phase transitions above and below the notch. The location resonance peaks can be predicted by the mathematical description of an undamped pulsation absorber.)
8. the normal phase lead of ICP with respect to ABP [1,6,11-14]. (With damping, the windkessel optimal pulsation absorption requires an inertial bias in the reactance, which causes a positive impedance phase and a positive phase transfer function.)
9. the phase lag of the ICP pulse associated with increasing ICP and intracranial elastance [11,14]. (Increasing ICP causes increased intracranial elastance, which shifts impedance phase to negative.)
10. the clinical presentation of NPH with gait apraxia and urinary incontinence, which is not routinely seen with other types of communicating hydrocephalus that caused by CSF space obstruction [45]. (Low of resistance in the CSF pathways, which characterizes NPH, causes high elastic strain on the windkessel pulsation absorber, which entails stress on periventricular brain tissue through which tracts mediating gait and urinary continence run.)
11. the occasional effectiveness of ETV in communicating hydrocephalus [46]. (Hydrocephalus is characterized by increased intracranial elastance, and ETV lowers elastance by connecting CSF spaces and improving compliance.)

Clinical impairment of the cerebral windkessel

Our emerging understanding of windkessel dynamics raises two obvious questions: what role does the windkessel dysfunction play in disorders of intracranial dynamics, such as hydrocephalus, stroke, head injury, etc.? And, if windkessel dysfunction is relevant to clinical practice, how can normal windkessel dynamics be restored?

The detailed discussion of windkessel dynamics in hydrocephalus and other disorders is beyond the scope of this paper. I do note that windkessel impairment has been documented empirically in experimental hydrocephalus [27], experimental intracranial hypertension [24], and in normal pressure hydrocephalus [26].

It seems likely that windkessel dysfunction plays a role in fulminant brain edema, such as can be encountered during craniotomy for brain swelling or aneurysm rupture. Severe impairment of the windkessel would, in theory, cause almost instantaneous capillary disruption and fulminant edema.

The role of windkessel dysfunction in the pathogenesis of hydrocephalus is of particular interest, because free motion of CSF is essential to windkessel function. Obstruction of the CSF spaces 'glues' the intracapillary spaces and the extracapillary spaces (the absorber) together, disabling the windkessel and threatening capillary integrity. Ventricular dilation, notably, serves to mitigate windkessel dysfunction, because it increases the volume of CSF available for coupling of arterial to venous pulsations. In this sense, ventricular dilation is an adaptation to hydrocephalus.

Shunting also alters the windkessel. Favorably, it provides decreased resistance to CSF pulsations and decreased elastance, both of which would tend to restore windkessel function disabled by obstruction to CSF pulsatility. Unfavorably, shunts drain CSF and reduce ventricular size, which diminishes the volume of CSF available for arteriovenous coupling and would tend to leave the patient dependent on the shunt for proper windkessel function.

Windkessel theory also provides a new perspective on the perplexing effectiveness of endoscopic third ventriculocisternostomy (ETV) in some patients with communicating hydrocephalus [46]. Although by traditional theory ETV should be of no value in hydrocephalus caused by obstruction to CSF flow distal the basal cisterns, windkessel theory suggests that hydrocephalus entails an impairment of the cerebral windkessel caused by an excess of elastance and resistance in the CSF pathways. Enlarging the communication between the ventricular system and the subarachnoid space connects the spaces and reduces the elastance and the resistance of the CSF pathways, which in some patients may be sufficient to restore windkessel function.

The relevance of the windkessel to the pathogenesis and management of hydrocephalus is of great interest and suggests several promising avenues of research.

Therapeutic implications of windkessel theory

How can impairment of the windkessel be treated? This question has not been addressed experimentally, although several theoretical considerations are of interest. Normal windkessel function represents a balance of heart rate, inertia, elastance and resistance. The balance is expressed conveniently in the equation for impedance phase:

$$\theta = \tan^{-1}\left[\frac{\omega m_{EC} - \frac{k_{EC}}{\omega}}{c_{EC}}\right]$$

where θ is the impedance phase, ω is heart rate, m_{EC} is the mass of the ICP pulse, k_{EC} is the intracranial elastance, and

r is the resistance/damping in the cranium. ωm_{EC} is inertial reactance, and

$$\frac{k_{EC}}{\omega}$$

is elastic reactance. For normal windkessel function, inertial reactance should balance elastic reactance, so that θ is (near) zero.

Further insight may be gained by considering the impedance angle θ_{EC} expressed in terms of the frequency ratio between the heart rate ω and the natural frequency of oscillation of the extracapillary space ω_{EC}:

$$\theta_{EC} = \tan^{-1}\left[\frac{k_{EC}\left(\left[\frac{\omega}{\omega_{EC}}\right]^2 - 1\right)}{\omega c_{EC}}\right]$$

Most disorders of intracranial dynamics are caused by an excess of elastic reactance

$$\frac{k_{EC}}{\omega}$$

in the extracapillary space, due to intracranial mass, brain swelling or stiffness. The effect of increased elastance k_{EC} on the natural frequency of the extracapillary space (essentially the cranial contents) is given by

$$\omega_{EC} = \sqrt{\frac{k_{EC}}{m_{EC}}}$$

An increase in k_{EC} will increase the natural frequency ω_{EC} of the cranium, diminish the frequency ratio

$$\frac{\omega}{\omega_{EC}}$$

and will shift the impedance angle to a more negative (less positive) number. This tends to disable the windkessel, ablate the windkessel notch, and lead to cerebral edema and loss of capillary integrity. Ablation of the notch with phase lag associated with increased ICP has been observed experimentally. [14] [figure 1]

Restoration of the balance between inertial and elastic reactance could be accomplished in several ways: decreasing elastance (k_{EC}), increasing inertia (m_{EC}), increasing heart rate ω, or increasing resistance c_{EC}.

Decreasing elastance is already standard therapy (e.g. removal of mass lesions, osmotic diuresis for edema, CSF diversion, decompressive craniectomy). Increasing inertia would entail increasing the mass of the ICP pulse, for example by increasing the left ventricular stroke volume. Increasing resistance would risk impairing the windkessel more severely by effectively clamping the intra-and extracapillary spaces together and impairing the absorber mechanism.

Of particular interest is therapeutic alteration of the heart rate (ω) to restore windkessel function. Both inertial reactance and elastic reactance are functions of heart rate, and iatrogenic increase in heart rate could restore windkessel function in the setting of an intractable increase in intracranial elastance (e.g intractable brain swelling). This observation is strictly theoretical; it is not clear what magnitude of tachycardia would be needed to restore the windkessel. Perhaps the necessary tachycardia would be beyond physiological parameters. Despite these ambiguities, it is noteworthy that windkessel theory suggests that windkessel dysfunction due to *any* imbalance of intracranial reactance, no matter how severe, could be corrected with appropriate manipulation of heart rate.

CONCLUSION

The heartbeat is a vibration, and efficient and smooth capillary blood flow is a vibration problem. Resonant arterial perfusion is necessary for efficient cerebral blood flow, and anti-resonant capillary perfusion is necessary for safe cerebral blood flow. This seems to entail mutually incompatible dynamics. This impasse is solved by the cerebral windkessel.

Despite its anatomical and mathematical complexity, the windkessel is in principle simple and elegant. The cerebral windkessel has analogues in mechanical engineering (a dynamic vibration absorber), electrical engineering (a wavetrap circuit), acoustic engineering (a cavity resonator) and systems analysis (a band stop filter). The windkessel uses resonant vascular pulsations to accomplish anti-resonant capillary wall motion. The CSF-venous pump, which is essential to normal function of the cerebral windkessel, is a pulsation absorber. Resonant vascular pulsations and anti-resonant capillary motion both depend critically on a balance of intracranial inertia, elastance, damping and heart rate. Windkessel dysfunction causes impairment of cerebral blood flow, loss of capillary integrity, and cerebral edema, which may in turn result in markedly increased intracranial elastance and a cascade of windkessel impairment.

Adjustment of heart rate or other parameters on which the windkessel depends may restore normal windkessel function. Restoration of windkessel function is possible, at least in theory, with any derangement of intracranial dynamics.

Aided by the principles of reverse engineering, we should explore the theoretical, physical, and physiological implications of this elegant system of pulsation absorption in the cranium. The difficult process of understanding the cerebral windkessel may provide new and counterintuitive insight into disorders of intracranial dynamics.

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