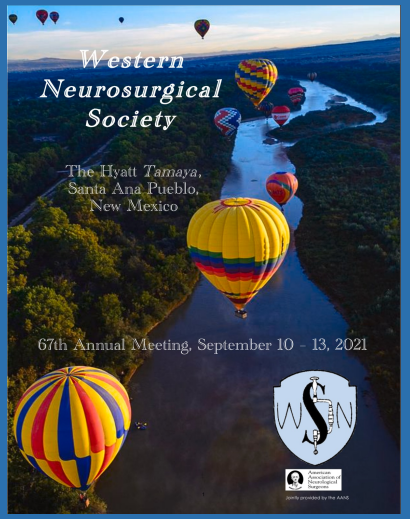
Intracranial Pressure (ICP)

From Merlin to Copernicus



Presidential Address

Marvin Bergsneider, M.D.
Professor
UCLA Department of Neurosurgery

David Geffen School of Medicine | UCLA Health

Disclosers

- Financial: none
- Other:
  - I think like an engineer, and see the world through that lens
  - I inherently question dogma
  - Linda will confirm that I've been obsessed with this theory that I began developing about 20 years ago

What is the Current Dogma?

"Textbook" ICP Theory

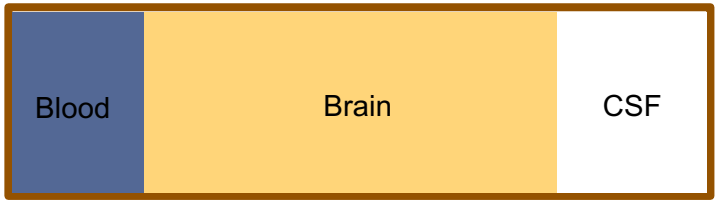
The Monro-Kellie Doctrine

Alexander Monro secundus (1733-1817)

- Premises

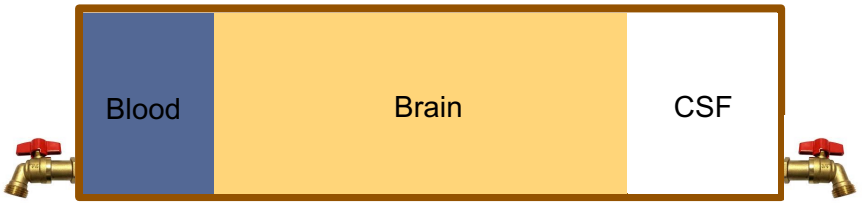
1. The intracranial cavity (compartment) has a fixed volume because the skull is rigid
2. Only 3 components fill this cavity: brain, blood, and cerebrospinal fluid (CSF)

   None of the 3 are compressible



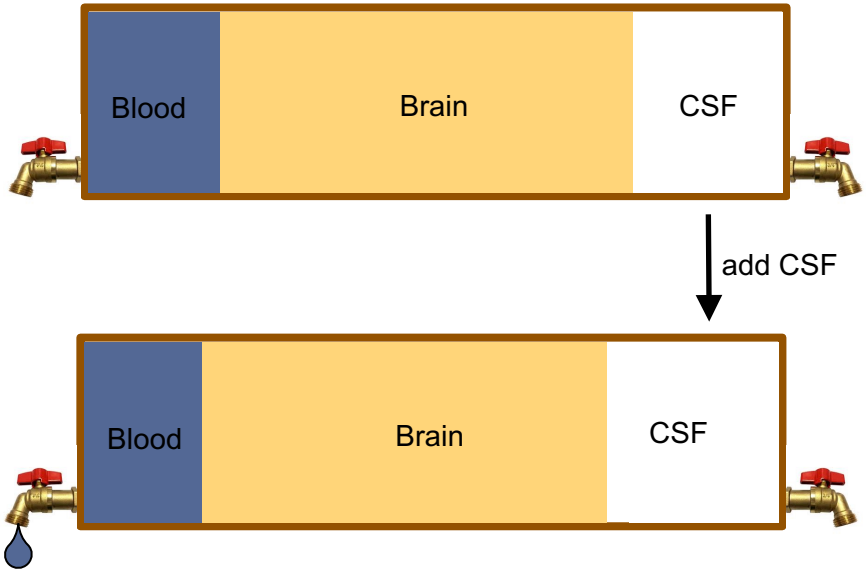
The Monro-Kellie Doctrine

- The intracranial compartment is not a closed container, however



The Monro-Kellie Doctrine

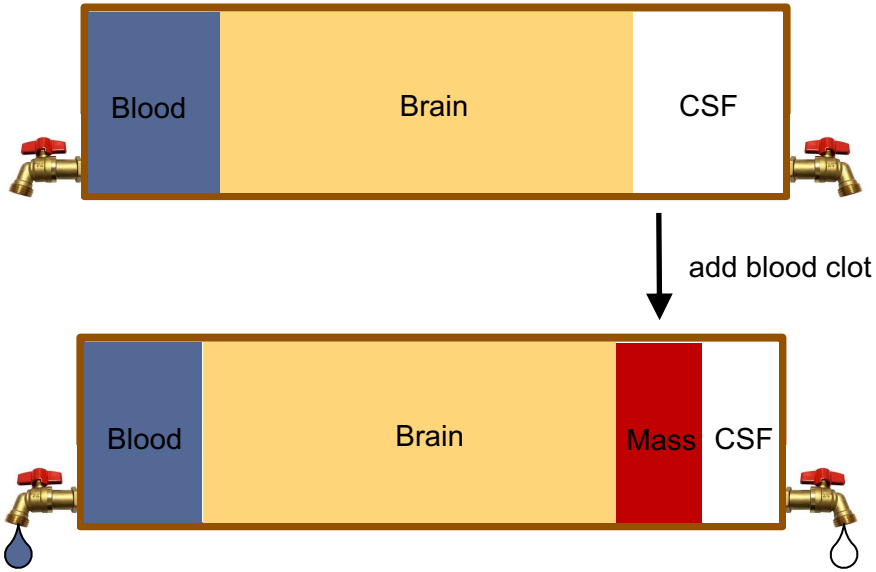
- A change in the volume of one of the components must result in reciprocal changes in one or both of the other two*



* Harvey Cushing

The Monro-Kellie Doctrine

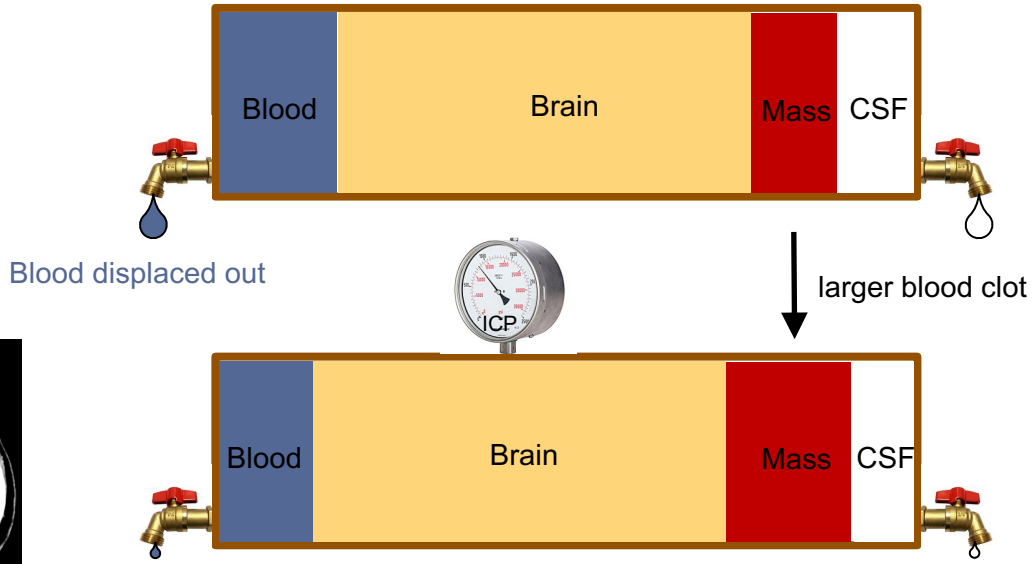
- If you add an additional component volume to the compartment (such as a blood clot), you must likewise displace one or more native components



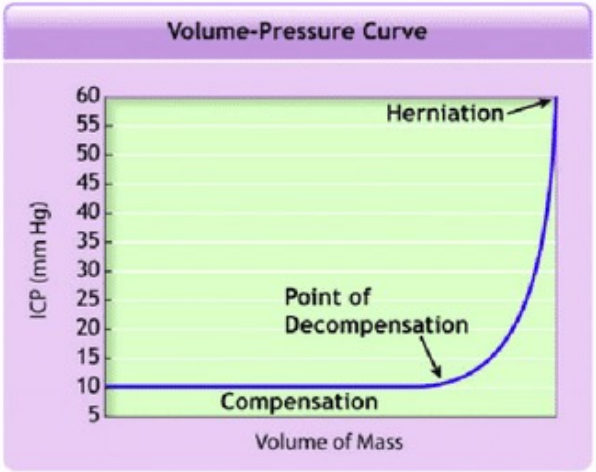
The Monro-Kellie Doctrine and ICP

- As you displace blood and CSF, the pressure inside the skull compartment will begin to rise because there's a limit how much you can squeeze out.

Intracerebral hematoma



The Famed “Pressure-Volume Curve”



- An increase in volume of one component, or the addition of a mass lesion, leads to an increase in intracranial pressure once displacement mechanisms can no longer compensate

What evidence is there to support this?

Human Studies

- Quickly inject 5 cc of saline into the CSF compartment resulted in the ICP going up a little
- Quickly inject 10 cc, ICP goes up a lot more

April 1973

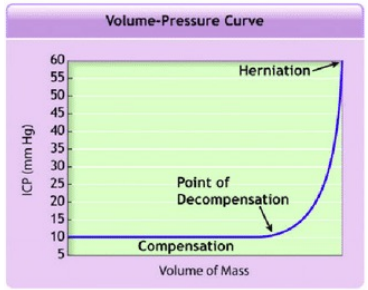
Induced Changes of Cerebrospinal Fluid Volume

Effects During Continuous Monitoring of Ventricular Fluid Pressure

J. Douglas Miller, MD; Juan Garibi, MD; John D. Pickard, MB

» Author Affiliations

Arch Neurol. 1973;28(4):265-269. doi:10.1001/archneur.1973.00490220073011



Animal Studies

- Inflate an epidural balloon a small amount in the epidural space of a cat, ICP goes up a little
- Inflate the balloon more, ICP goes up a lot more

› Neurosurgery. 1986 Jul;19(1):1-8. doi: 10.1227/00006123-198607000-00001.

Analysis of changes in intracranial pressure and pressure-volume index at different locations in the craniospinal axis during supratentorial epidural balloon inflation

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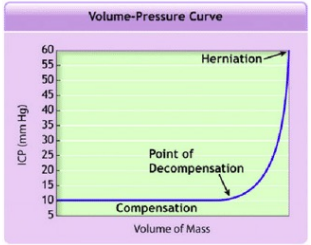
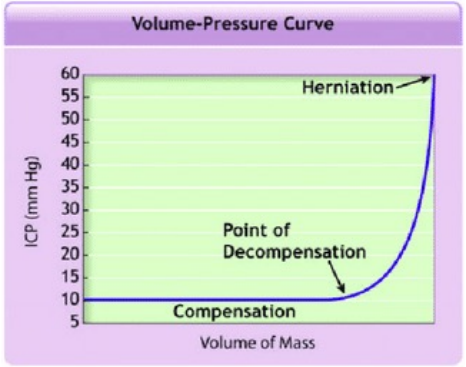


Per the “textbook” theory



Add volume

Increase pressure

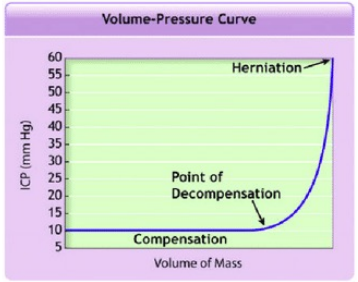
Since the 1970's, every neurosurgeon has been taught that elevated ICP is bad

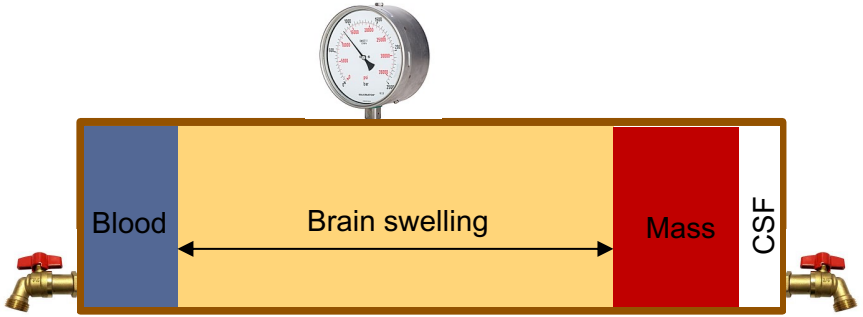
Miller JD, Becker DP, Ward JD, et al: Significance of intracranial hypertension in severe head injury. **J Neurosurg 47:**503–516, 1977

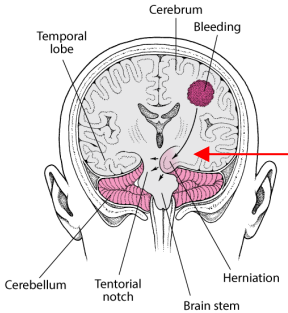
But why is it bad?

Severely Elevated ICP

1. Herniation







Herniation of brain obliterates CSF space and compresses the brainstem

Severely Elevated ICP

1. Herniation
2. Ischemia = inadequate blood flow

    For the brain = stroke

Understanding cerebral blood flow (CBF)*

Facts as Defined by Wikipedia

Cerebral perfusion pressure

From Wikipedia, the free encyclopedia

Cerebral perfusion pressure, or **CPP**, is the net pressure gradient causing cerebral blood flow to the brain (brain perfusion).

From resistance [edit]

CPP can be defined as the pressure gradient causing cerebral blood flow (CBF) such that

$$CBF = CPP/CVR$$

where:

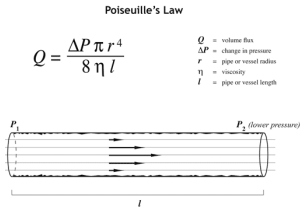
CVR is cerebrovascular resistance

If you remember High School physics, this is simply Ohm's Law of Electricity Applied to Fluid Flow

$$V = IR$$

(voltage drop = current x resistance)

$$(I = V/R)$$



for fluids

Fluid flow = pressure / resistance

$$CBF = CPP/CVR$$

How is Cerebral Perfusion Pressure (CPP) Defined?

By intracranial pressure [edit]

An alternative definition of CPP is:[1]

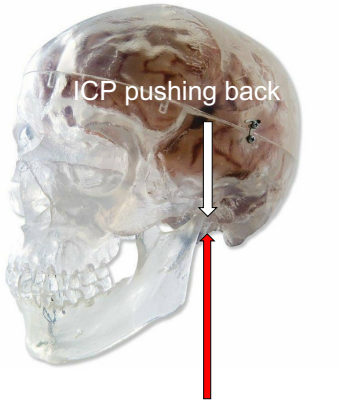
$$CPP = MAP - ICP$$

where:

MAP is mean arterial pressure

ICP is intracranial pressure

JVP is jugular venous pressure



Mean arterial blood pressure (MAP) in the internal carotid arteries driving blood flow in

Blood can only get to the brain if the blood pressure is sufficiently higher than ICP

How Does Elevated ICP Affect CBF?

- Per the textbook theory, if the ICP exceeds the blood pressure, it compresses the cerebral arteries, causing
  - Decreased blood flow (hence ischemia)
  - In the extreme, complete cessation of blood flow

- Increased ICP compresses cerebral arteries, causing increased cerebrovascular resistance (CVR).

How this Applies to Management of High ICP

If increased volume causes high ICP, then …

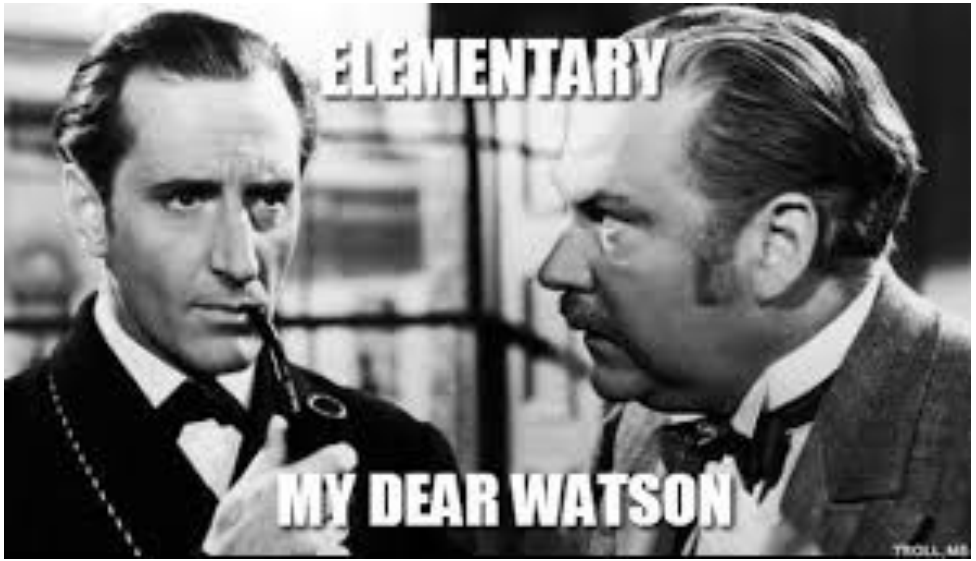
Decrease volume

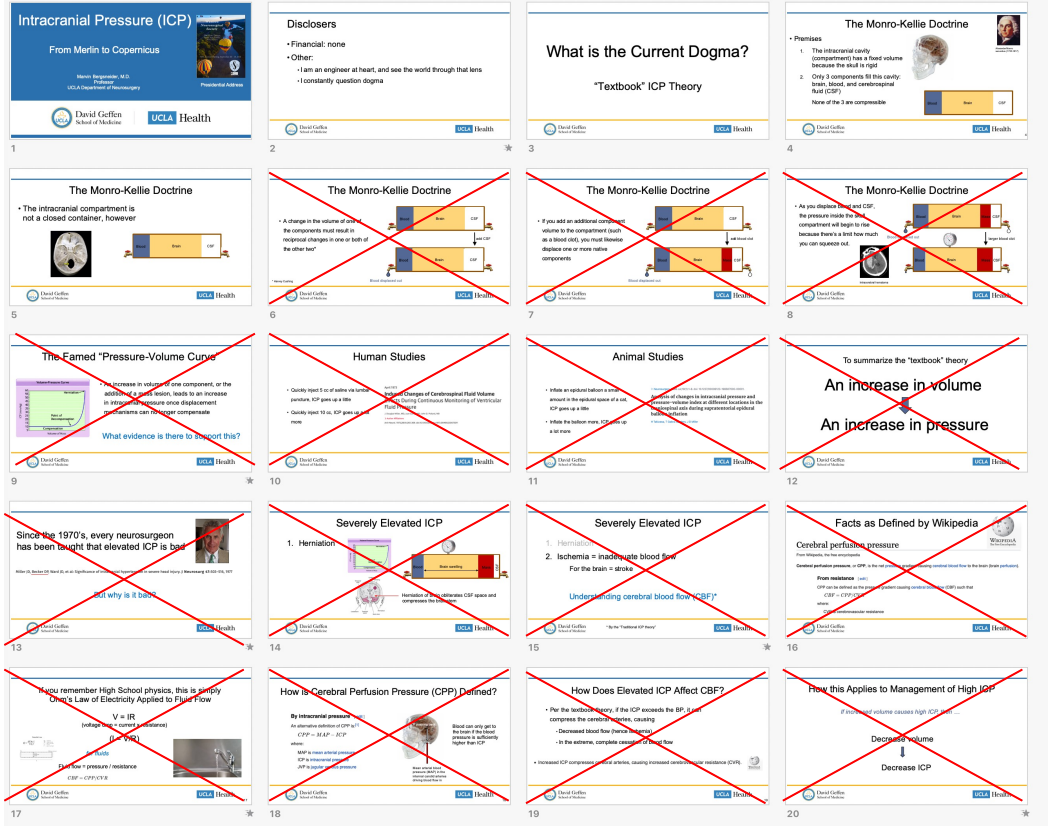
Decrease ICP

TBI Management

- Reduce cerebral blood volume (CBV)
  - Hyperventilate
  - Mannitol and hypertonic saline
- Reduce CSF volume
  - Ventriculostomy
- Remove solid volume
  - The mass (i.e. hematoma)
  - Lobectomy

What every neurosurgeon in the audience is thinking right now




Intracranial Pressure (ICP)
From Merlin to Copernicus
Disclosers
• Financial: none
• Other:
• I am an engineer at heart, and see the world through that lens
• I constantly question dogma
What is the Current Dogma?
"Textbook" ICP Theory
The Monro-Kellie Doctrine
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• The intracranial compartment is not a closed container, however
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What evidence is there to support this?
Human Studies
Animal Studies
To summarize the "textbook" theory
An increase in volume
An increase in pressure
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How is Cerebral Perfusion Pressure (CPP) Defined?
How Does Elevated ICP Affect CBF?
How this Applies to Management of High ICP
Decrease volume
Decrease ICP


RUBBISH
MY DEAR WATSON

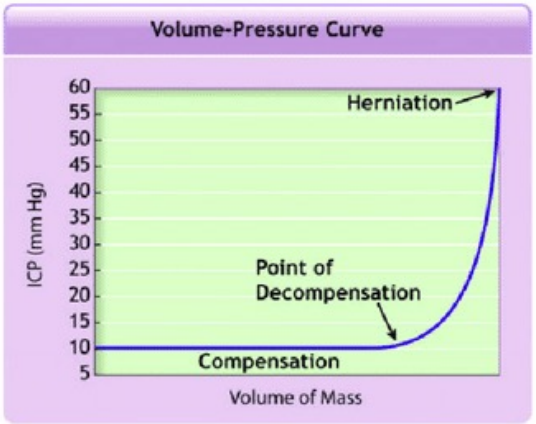

YOU MUST UNLEARN
WHAT YOU HAVE LEARNED

The Central Premise is Based on a False Association Between Volume and Pressure:

Logic Error: Association ≠ Causation

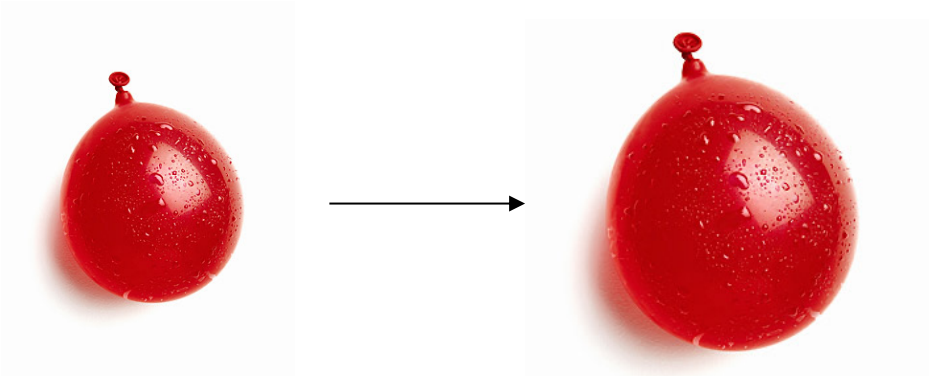
Pressure = A *force* applied over an area

Volume is not a force



The Central Premise is Nonsense

"A change in volume leads to a change in pressure" assumes that a volume can change spontaneously



Changes in volume result from changes in pressure

All “Experimental” Evidence Involved Applying a Force to Evoke a Change in Volume

April 1973

Induced Changes of Cerebrospinal Fluid Volume
Effects During Continuous Monitoring of Ventricular Fluid Pressure

J. Douglas Miller, MD; Juan Garibi, MD; John D. Pickard, MB

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The Central Premise is Magical Thinking

"~~A change in volume leads to a change in pressure~~"

Pressure must have a definable force: energy must be expended

In the “Textbook Theory”, ICP is Regarding as an Independent Force

- It can push on things
  - The brain (herniation)
  - Blood vessels (causing ischemia)

Let’s Start Over From Scratch

The Monro-Kellie Doctrine is Largely Based on Pascal's Law

- A principle in fluid mechanics that states that a *pressure* change at any point in a confined incompressible fluid is transmitted throughout the fluid such that the same change occurs everywhere

pres·sure

/ˈpreSHər/

noun

1. continuous physical force exerted on or against an object by something in contact with it.

Force is a transfer of *energy*

Pressure Must Have An Identifiable *Force*

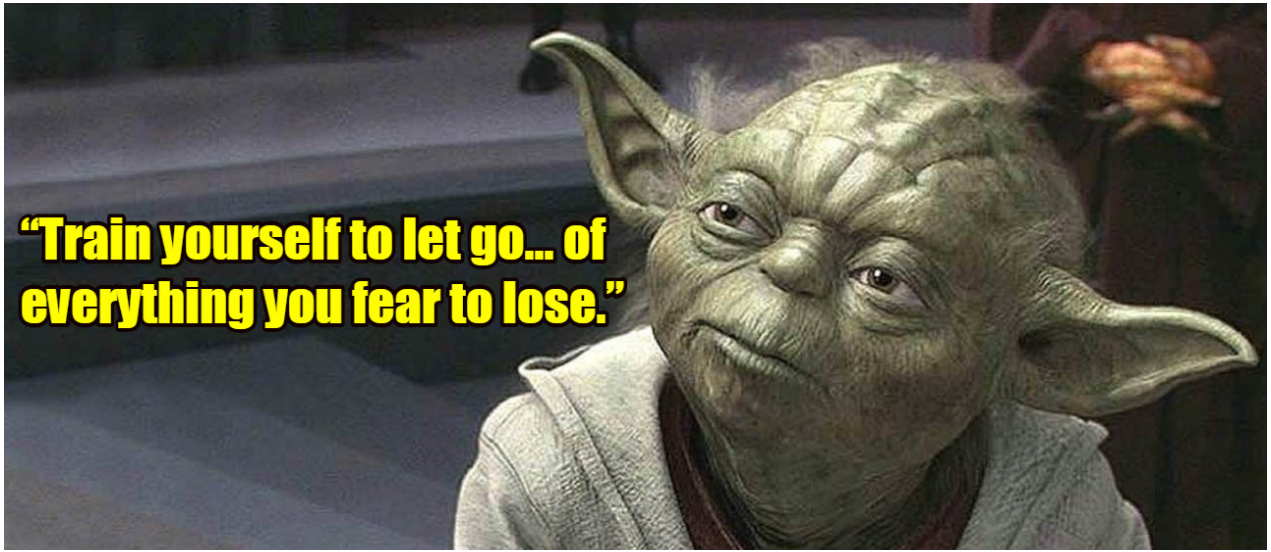
Natural Forces That Can Generate ICP

1. Blood pressure (cardiovascular energy)
2. Oncotic/osmotic pressure (gradients)

ATP-derived

3. Gravity
4. ~~Volume~~

Unnatural forces



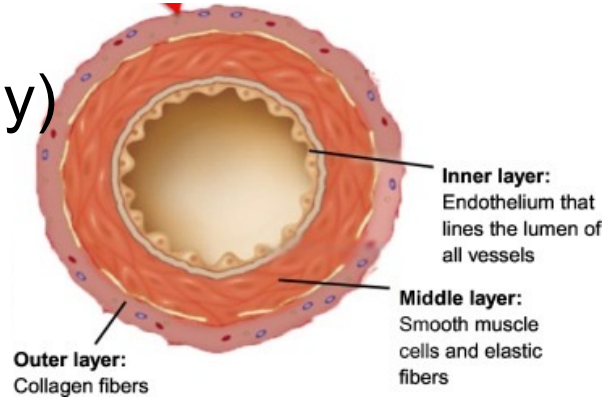
We must stop thinking of ICP as an independent pressure

Applying Pascal's Law at the Cellular Level

- I define ICP as brain tissue pressure
  - It's what you measure with a Codman ICP monitor
  - In a closed space, it is equal to CSF (ventricular) pressure

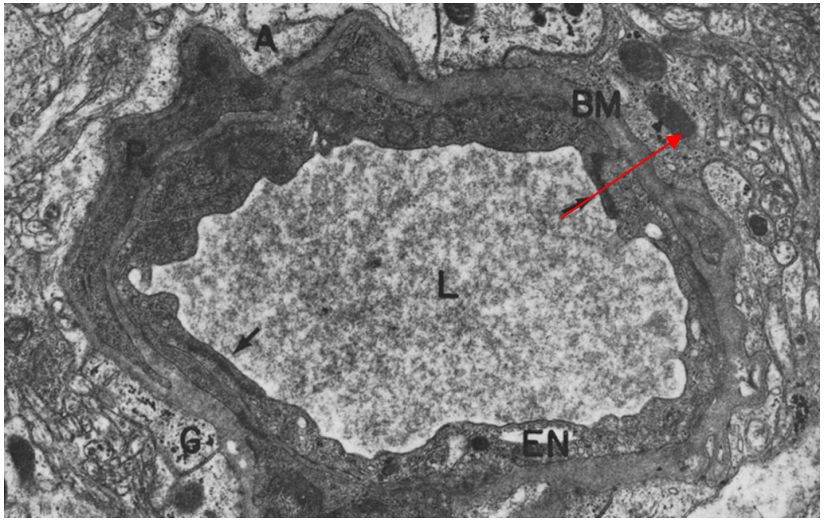
Natural Forces That Can Generate ICP

1. Blood pressure (cardiovascular energy)
2. Oncotic/osmotic pressure (gradients)
3. Gravity



BP within an artery are largely contained within the vessel

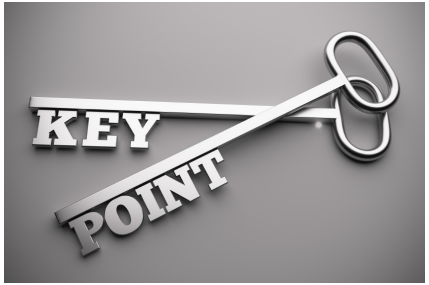
The Wall of Cerebral Capillaries is One Cell Layer Thick



Electron micrograph of a capillary wall

No muscular layer
No collagen layer

There should be a direct transmission of *capillary* blood pressure to the surrounding brain tissue

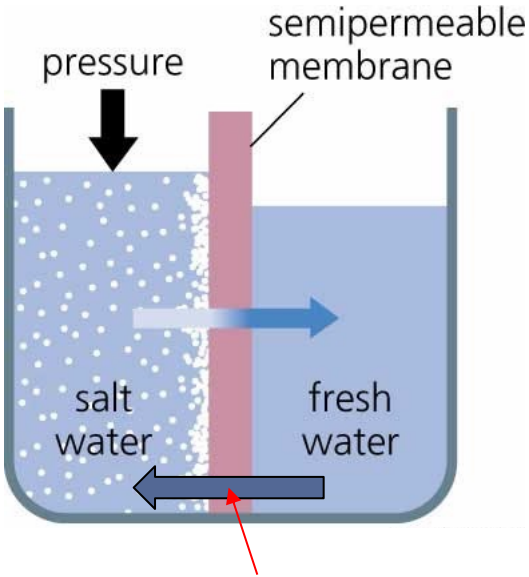


Understanding ICP Requires Understanding What Determines Cerebral Capillary Pressure

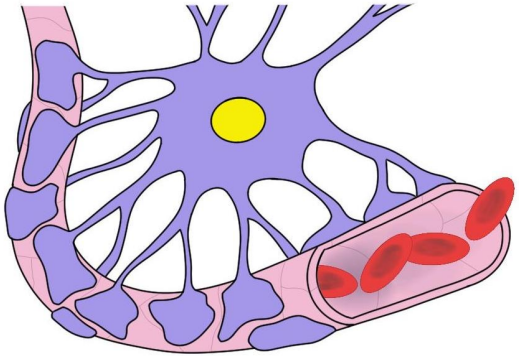
Natural Forces That Can Generate ICP

1. Blood pressure (cardiovascular energy)
2. Oncotic/osmotic pressure (gradients)
3. Gravity

Reverse Osmosis



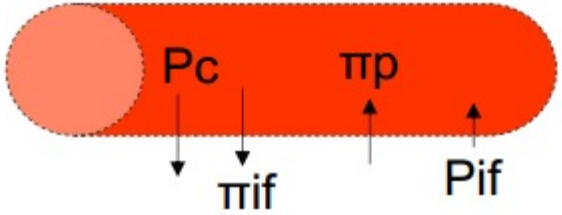
Frank-Starling Law



Blood-Brain-Barrier (BBB)

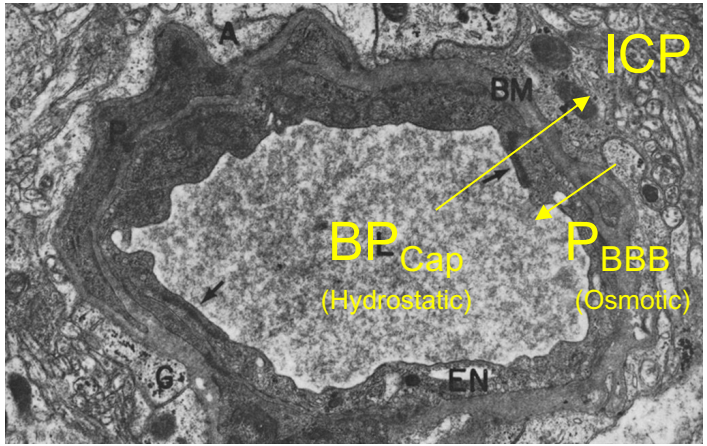
Starling Forces and Equation in trans-capillary exchange:

'Outward' forces driving filtration:
Hydrostatic pressure of blood in capillary (Pc)
Interstitial fluid colloid osmotic pressure (πif)



'Inward' forces driving absorption:
Hydrostatic pressure of interstitial fluid (Pif)
Plasma colloid osmotic pressure (πp)

Premise 2



The blood-brain-barrier (BBB) acts to lower ICP

This is modeled as a battery (DC offset)

$$ICP = BP_{Cap} - P_{BBB}$$

The Critical Importance of the BBB

- Fluid flows in a tube from high to low pressure
  - Therefore by definition $BP_{Cap} > BP_{Ven}$
- What would happen if P_{BBB} didn't exist
  - BP_{Cap} would equal ICP ($ICP = BP_{Cap} - P_{BBB}$)
  - Therefore $ICP > BP_{Ven}$
  - The venous system would collapse

The BBB osmotic pressure offset is absolutely necessary to prevent venous system collapse

To understand ICP, you must understand what affects brain capillary pressure (BP_{Cap})

What Affects Cerebral Capillary Pressure?

The brain will do what ever it takes to maintain sufficient cerebral blood flow (CBF) to provide adequate O_2 and glucose to neurons

Autoregulation: the brain arteries will vasodilate (get larger) if CBF becomes inadequate

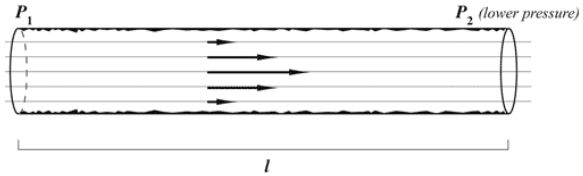
Recall that increasing the diameter of a vessel drastically lowers flow resistance

So Far We've Limited This Discussion to Blood Flow *Resistance*

Poiseuille's Law

$$Q = \frac{\Delta P \, \pi \, r^4}{8 \, \eta \, l}$$

Q = volume flux
ΔP = change in pressure
r = pipe or vessel radius
η = viscosity
l = pipe or vessel length

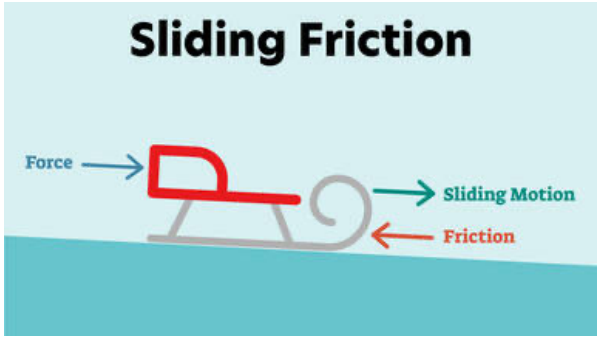


Poiseuille's Law only applies to steady (nonpulsatile) flow

Inertia

Inertia is Different than Resistance

Resistance arises from friction



Inertial Effects Have Nothing to with Resistance

It's harder to throw a bowling ball than a beach ball because the bowling ball weighs more

Greater mass = greater inertia

Blood also has mass

It takes energy to *accelerate* a mass

Acceleration Formula

$$F = ma$$

F is the force
m is the mas
a is the acceleration

It is the inertial *reactance* of the mass that impedes forward motion

It takes no energy to maintain a mass moving at constant velocity (as long as there is no friction)

Acceleration Formula

$$F = ma$$

$F = 0$ $a = 0$

F is the force
m is the mas
a is the acceleration

The electrical analogy of inertial effects is an inductor

Introducing the Variable *Impedance*

Impedance (Z) = Resistance + Reactance

Mainly affected by artery diameter

Only comes into play when blood is being accelerated due to inertial effects

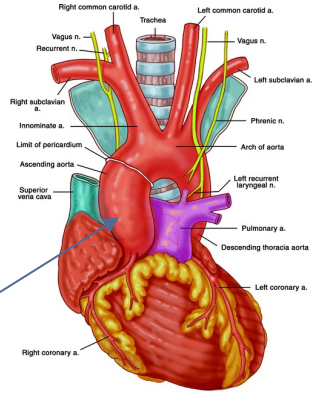
$$CBF = CPP/CVR$$

CBF = cerebral perfusion pressure (CPP)/cerebrovascular impedance

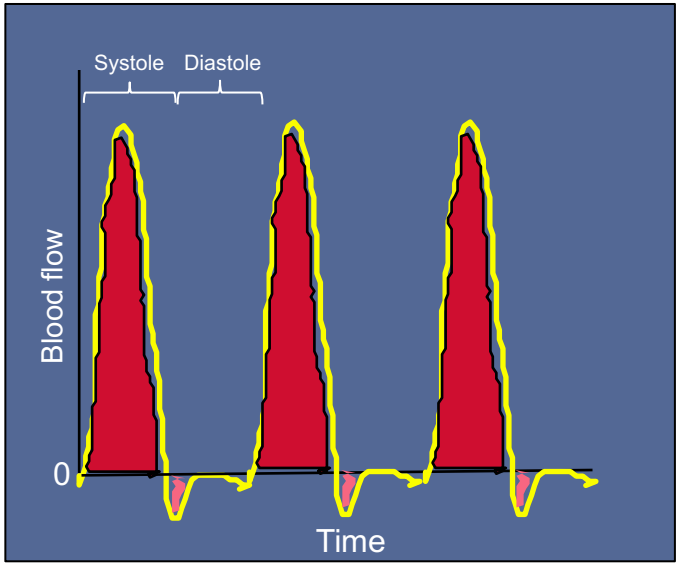
Let's Take a Detour to the Systemic (non-Cranial) Circulation

Blood flow in the aorta is rapidly accelerated during systole

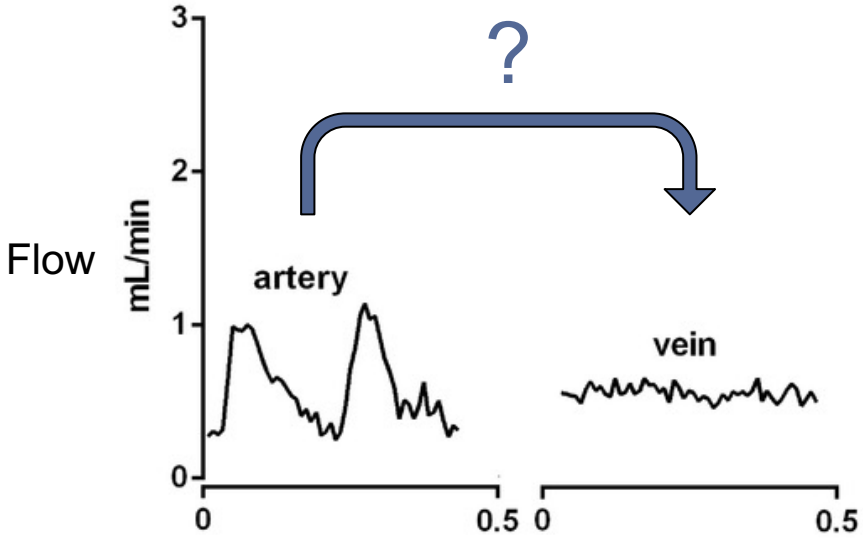
There is zero forward flow in the ascending aorta during diastole



Ascending aorta

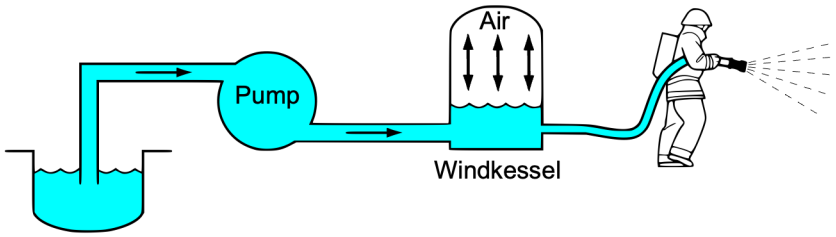


Venous flow is non-pulsatile (there is no acceleration or deceleration)

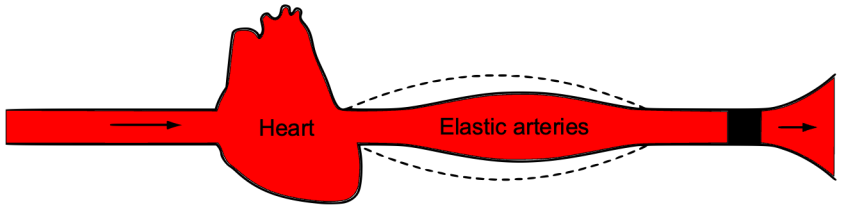


Veins, which are larger than arteries, offer low flow resistance

The Windkessel Effect



By allowing water to expand into a space temporarily, and then forcibly having it move downstream in a delayed fashion, the flow becomes progressively less pulsatile

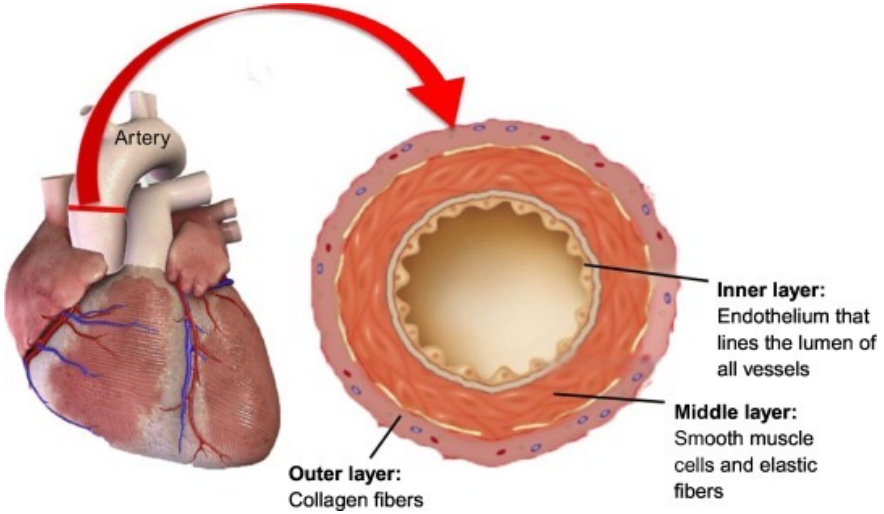


The equivalent process occurs in our cardiovascular system

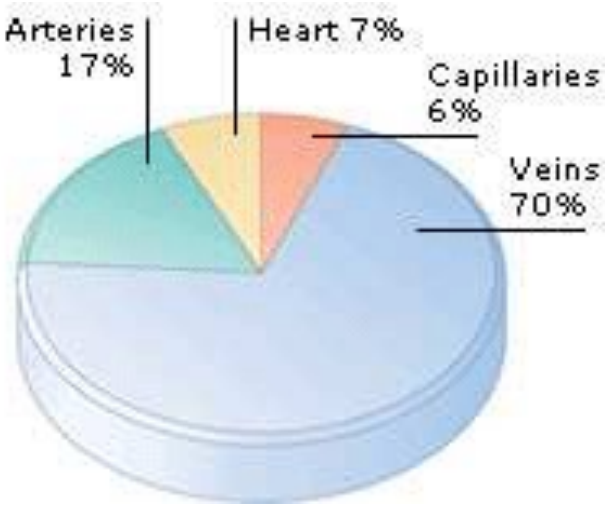
The electrical analogy of the Windkessel effect is a capacitor

The arterial muscular layer is a ”second pump”

We have an amazing *cardiovascular* system that converts pulsatile arterial flow to non-pulsatile venous flow



The Unequal Blood Volume (Mass) Distribution of the Vascular System

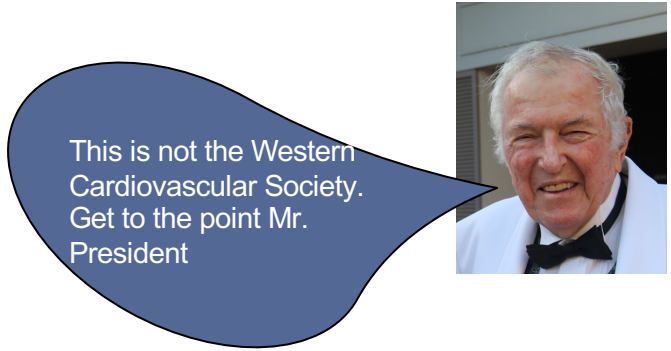


Only 17% of our blood mass is being accelerated with every heart beat

The great majority (>70%) of our blood mass flows without inertial effects (low reactance)

What would happen if you lost the Windkessel effect?

- The heart would need to accelerate the entire 5L of blood with every heart beat
  - Not possible (heart failure)
- Keeping the large venous blood mass flowing in a nonpulsatile fashion is critical to maintaining blood flow in our body
- Note, in the systemic circulation, arterial expansion is not a problem



The Intracranial Compartment Revisited

- Arterial expansion (the Windkessel Effect) within the intracranial compartment is dependent upon displacement of one or more components
  - This is the critical application of the Monro-Kellie Doctrine

It is *CSF* that exits the cranium during systole, re-enters during diastole, to make room for the arterial Windkessel Effect

On the Pulsatile Nature of Intracranial and Spinal CSF-Circulation Demonstrated by MR Imaging

August 1993 · Acta Radiologica 34(4):321-8
DOI:10.1080/02841859309173251
Source · PubMed

Authors:

D Greitz **A Franck** **B Nordell**

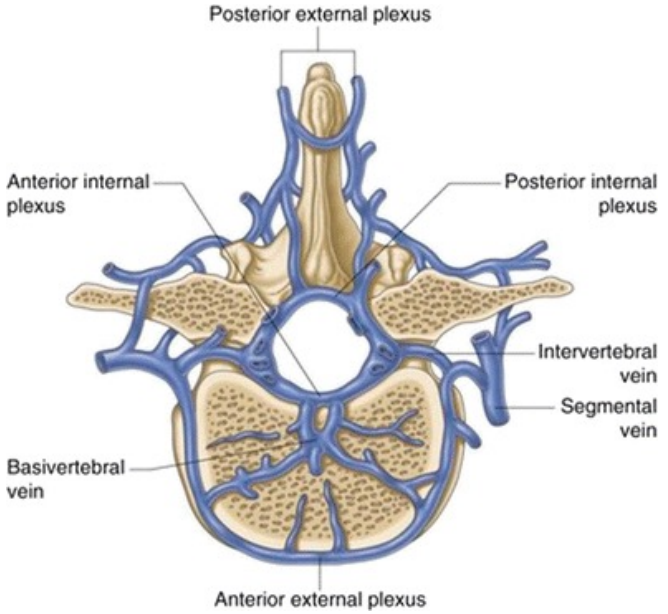
The Monro-Kellie Doctrine 2.0

- For the Windkessel effect to occur intracranially, you must maintain unimpeded *dynamic* CSF movement in and out of the cranium

But wait, where does CSF go?

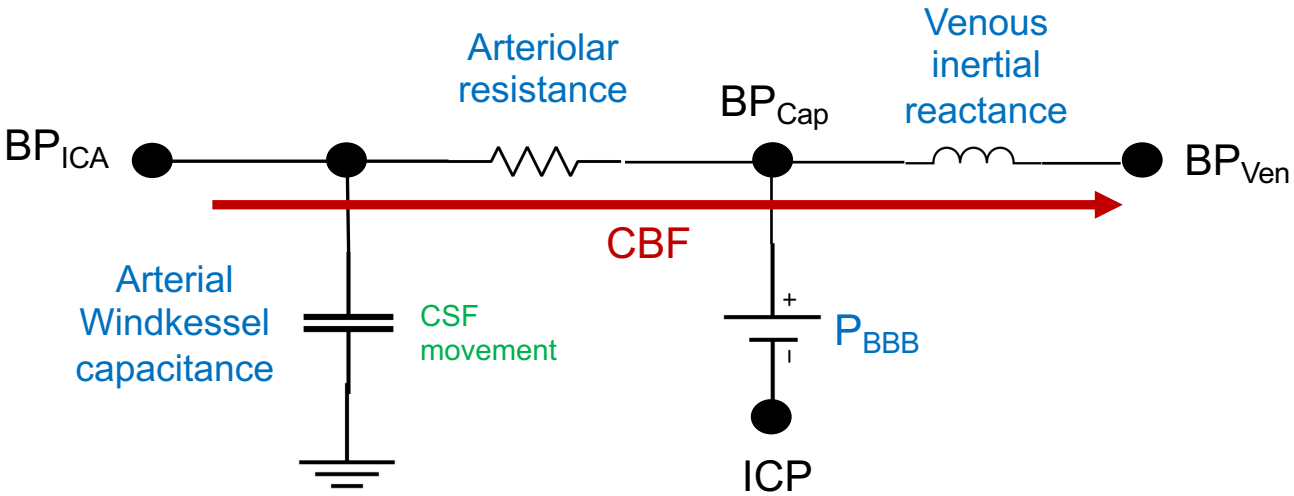
The Spinal Canal Accommodates the Windkessel Effect

- Unlike the cranial compartment, the spinal dura is surrounded by a *valveless*, epidural venous plexus
  - This allows the dural sac to expand during systole and then return back to its original position during diastole



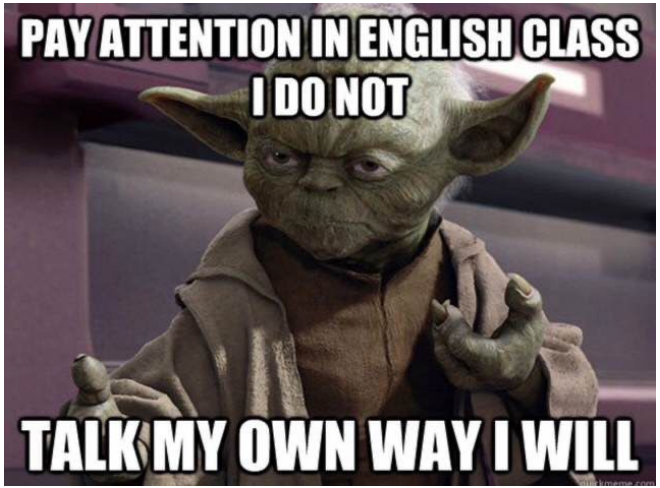
Time to Put It All Together

New ICP Model*

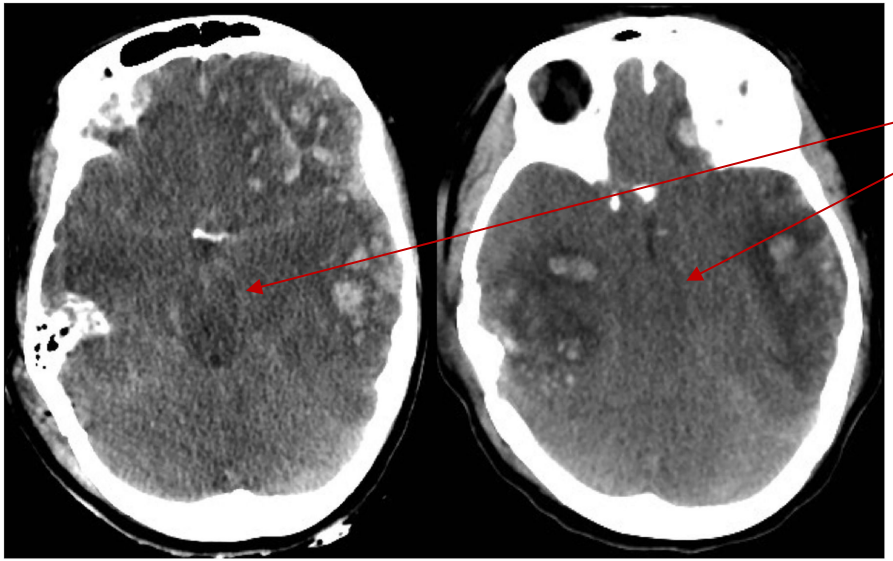


*Simplified

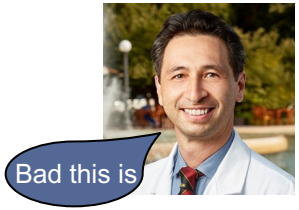
Applying the New Model



Traumatic brain injury with mass effect

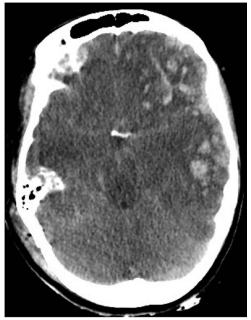


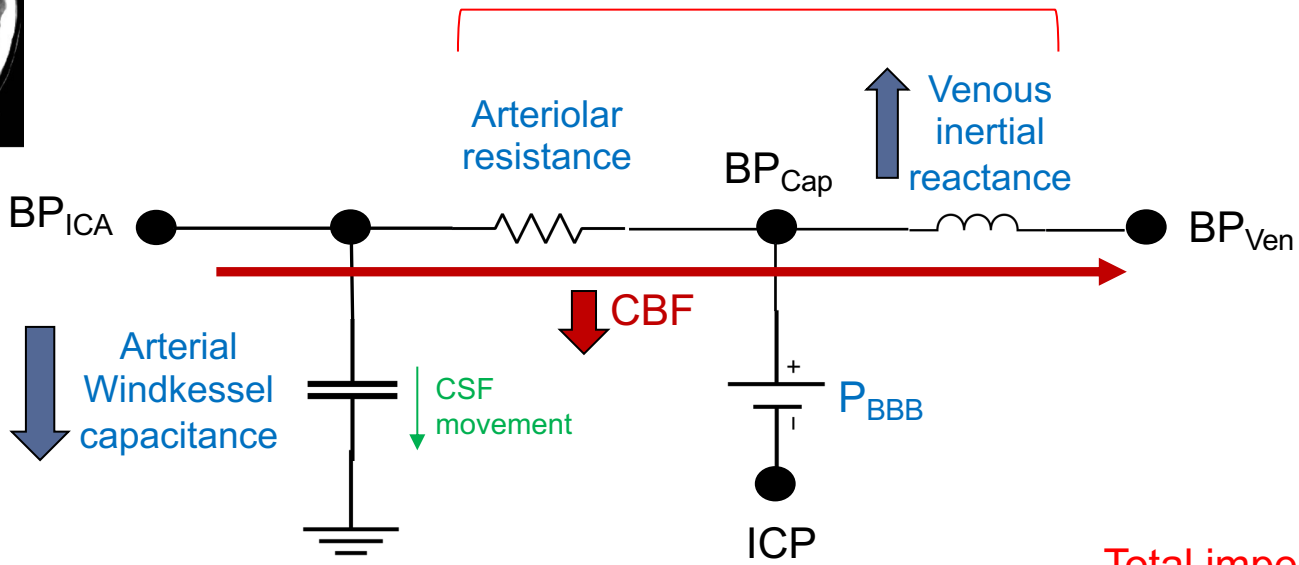
Loss of basal cisterns



Loss of dynamic CSF buffering will result in a reduction of the intracranial Windkessel effect and therefore increased venous blood pulsatility

Venous reactance



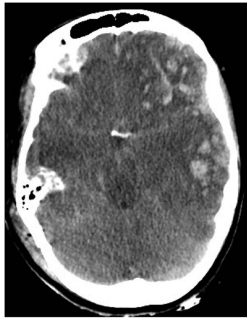


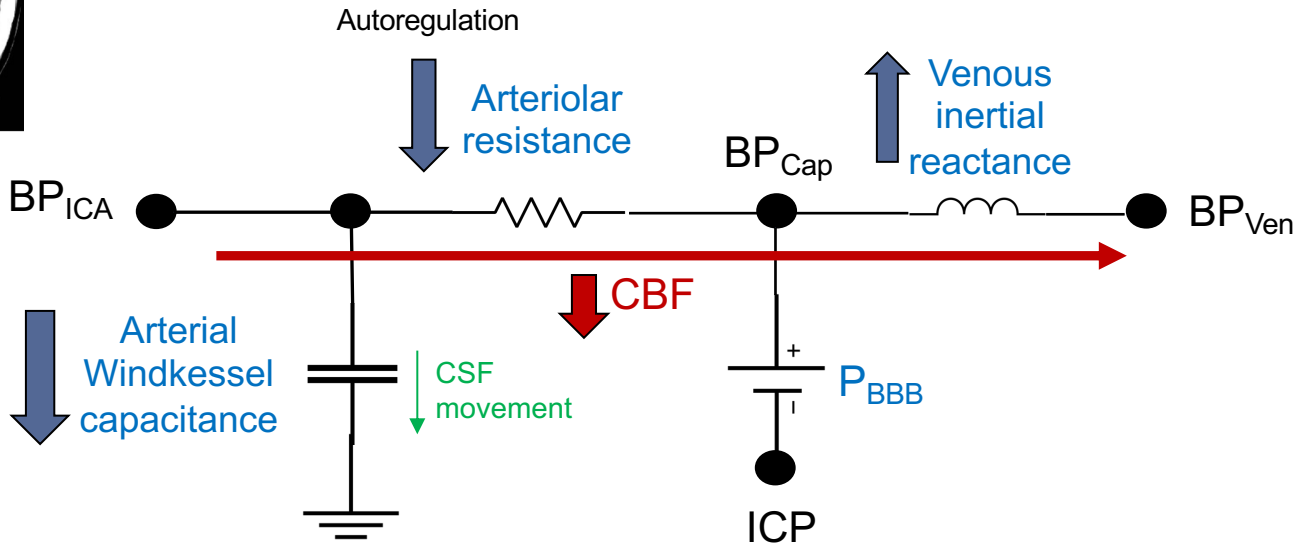
Total impedance goes up

New ICP Model

The reduction in CBF will trigger autoregulation

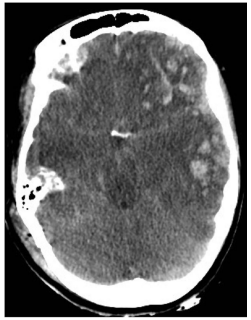
Arteriolar vasodilatation → ↓ Arteriolar resistance

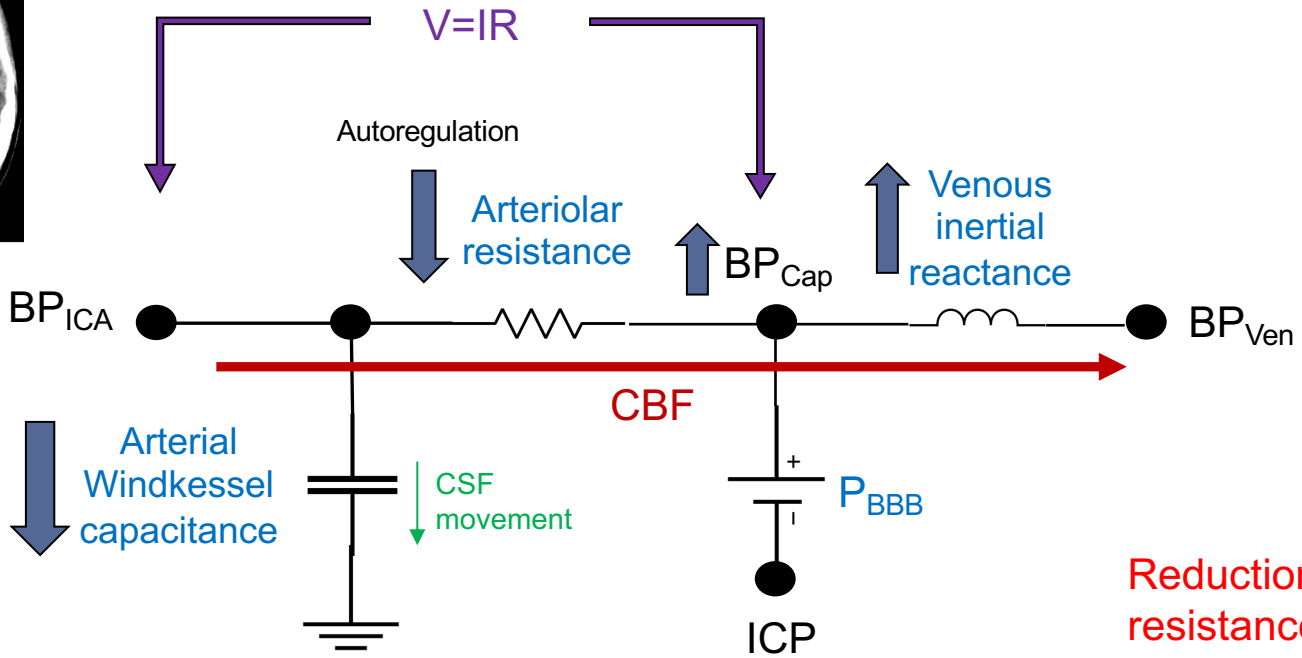




Impedance goes back to normal

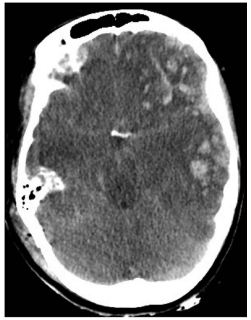
New ICP Model

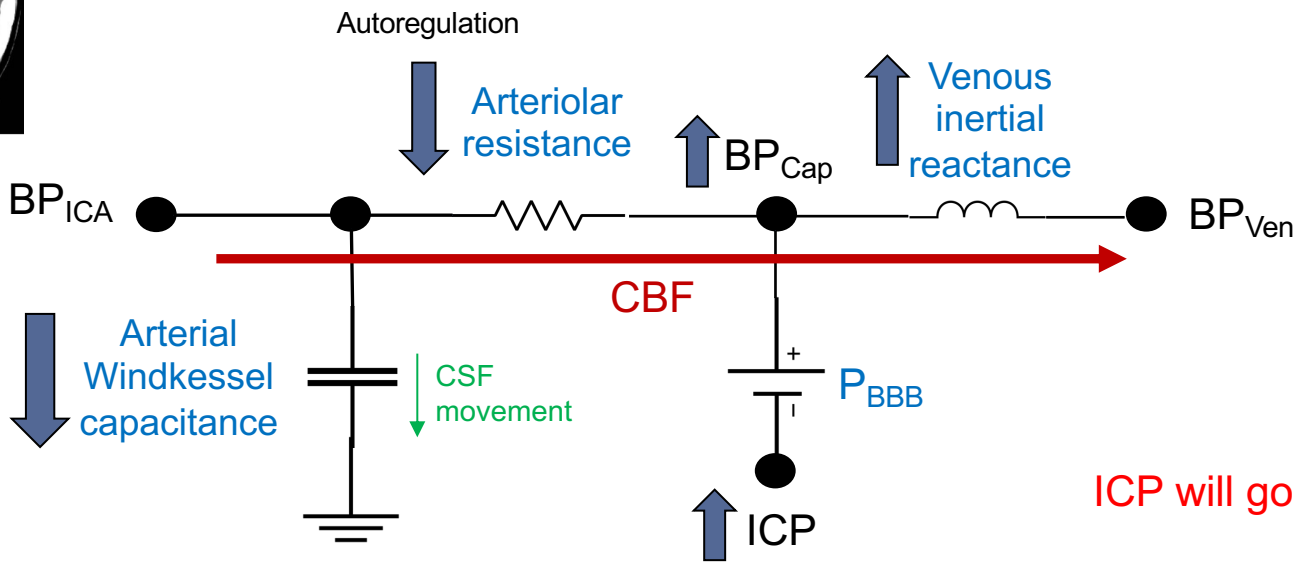




Reduction in arterial resistance results in an increase in BP_{Cap}

New ICP Model

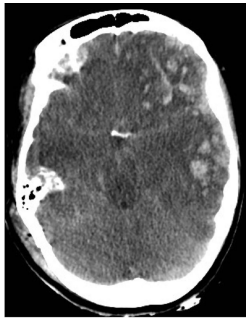




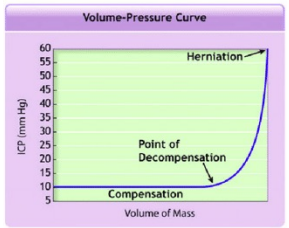
ICP will go up with BP$_{Cap}$

New ICP Model

Comparing Models



- Textbook
  - Increased volume due to brain swelling and blood clots
  - Pressure-volume curve dictates an increase in ICP

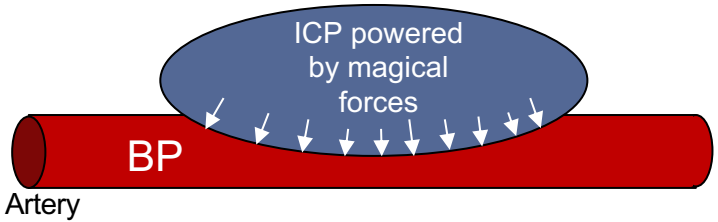


  - If ICP goes high enough, CBF drops
  - High ICP is the problem

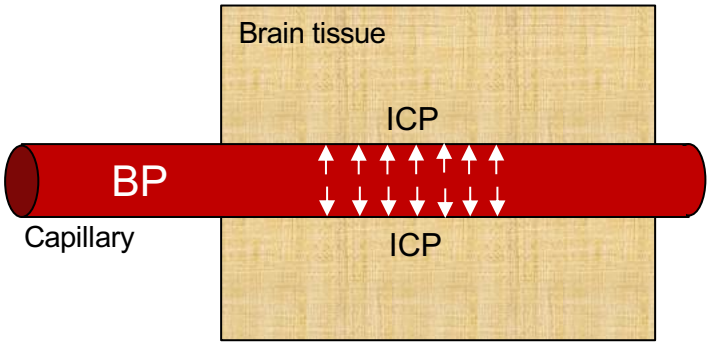
- New Model
  - New mass interferes with dynamic CSF movement
  - Increased venous reactance is compensated by arteriolar vasodilatation, resulting in increased BP_{Cap} and therefore ICP
  - Normal CBF can be maintained until autoregulation (vasodilatation) is maximized
  - High ICP is the *indicator* of a perturbed system

The Textbook Model Has It Wrong and Backwards

Textbook



New Model

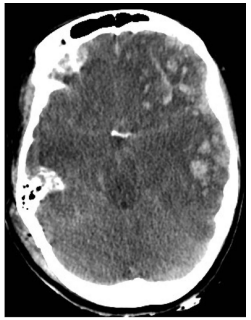


What Are the Consequences of Following the Traditional Model?

- Reduce cerebral blood volume (CBV)
  - Hyperventilate
  - Mannitol and hypertonic saline
- Reduce CSF volume
  - Ventriculostomy
- Remove solid volume
  - The mass (i.e. hematoma)
  - Lobectomy

Hyperventilation: increase the respiratory rate to lower CO_2 levels, which causes arteries to constrict

Comparing Models for Hyperventilation



- Textbook
  - You are concerned about possible herniation and/or ischemia due to high ICP
  - Because excessive “volume” is the root cause of high ICP, we need vasoconstrict the brain arteries to reduce the blood volume component

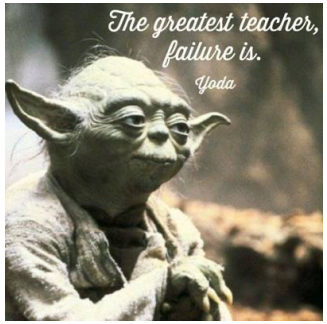
↓ Volume → ↓ ICP → ↑ CBF

Just don’t question that

Vasoconstriction → ↑ resistance → ↓ CBF

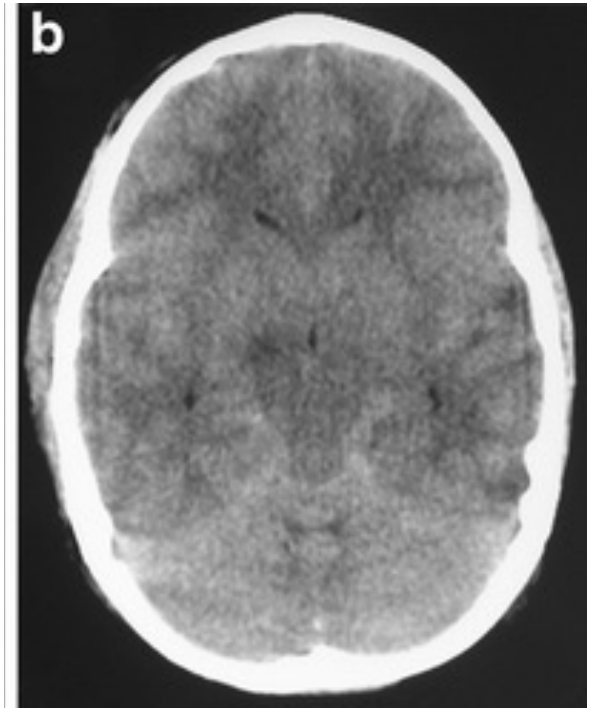
- New Model
  - The brain is attempting to maintain CBF with vasodilation (that’s why ICP went up)
  - Do not hyperventilate!
    - Although this will lower ICP, it will cause ischemia

Several TBI studies demonstrated that hyperventilation resulted in worse outcomes due to ischemia



Obrist WD, Langfitt TW, Jaggi JL, *et al.***Cerebral blood flow and metabolism in comatose patients with acute head injury: relationship to intracranial hypertension**
J Neurosurg, 61 (1984), pp. 241-253

What happens in the extreme?



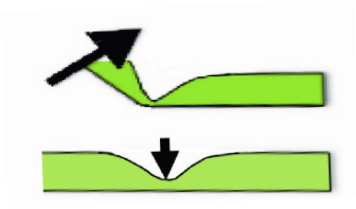
Maximum venous inertia (high reactance) should result in the systolic blood flow pulse bouncing back

Come on Bergsneider. What do you think we are, brain dead? How much venous inertia we talking about?

Brain Death after Traumatic Brain Injury

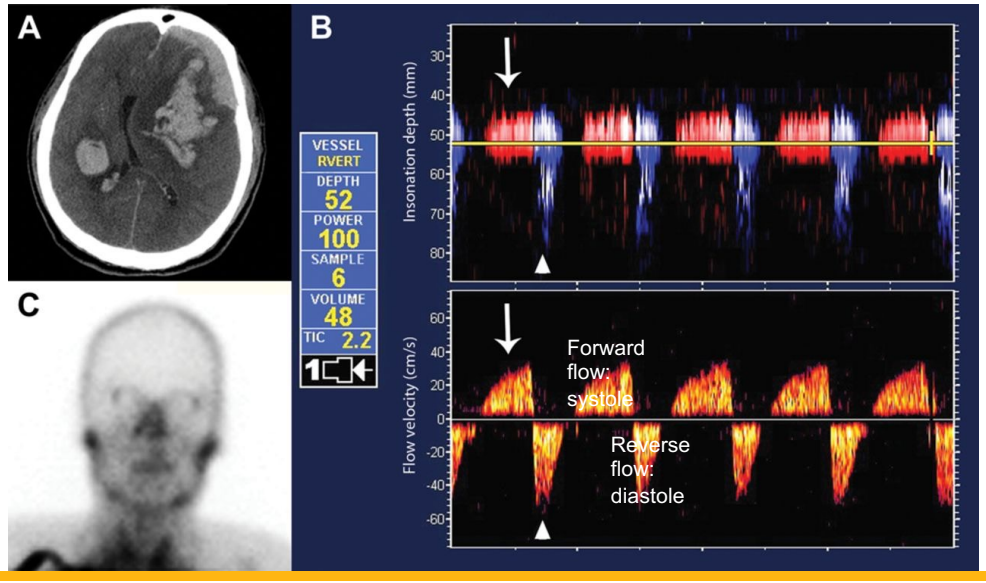
- Very high ICP's (approaching MAP)
- Cerebral angiography confirms no blood flow to the brain

Textbook theory: High ICP kinks (via herniation) or compresses cerebral arteries to the point of complete obstruction. There should be zero intracranial blood flow



What We Actually Find

The flow direction of middle cerebral artery blood flow completely reverses, flowing backwards, during diastole



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August 28, 2012; 79 (9) RESIDENT AND FELLOW SECTION

Teaching Neuro*Images*: Reverberating TCD flow pattern in brain death

Katherine E. Eder, Diogo C. Haussen, D. Eric Searls, Nils Henninger

Summary

- ICP is generated from actual physical forces (BP, osmotic)
- A fixed volume, rigid cranium filled with incompressible contents imposes major challenges to pulsatile blood flow
- It is critical to understand the interplay between dynamic CSF movement, venous inertial reactance, autoregulation, and pressure dynamics occurring at the cerebral capillary level
- High ICP is an *indicator* that the CSF-cerebrovascular system is perturbed

From Merlin to Copernicus

- Our textbook, volume-based, ICP theory created a force (ICP) lacking a physical basis. ICP is a magical force

- Understanding ICP will require abandoning accepted dogma: adopting a new concept that is grounded on sound physics, engineering, and biology principles


OVER THIS
TALK IS