Intracranial Flow (and Pressure): What can we measure?

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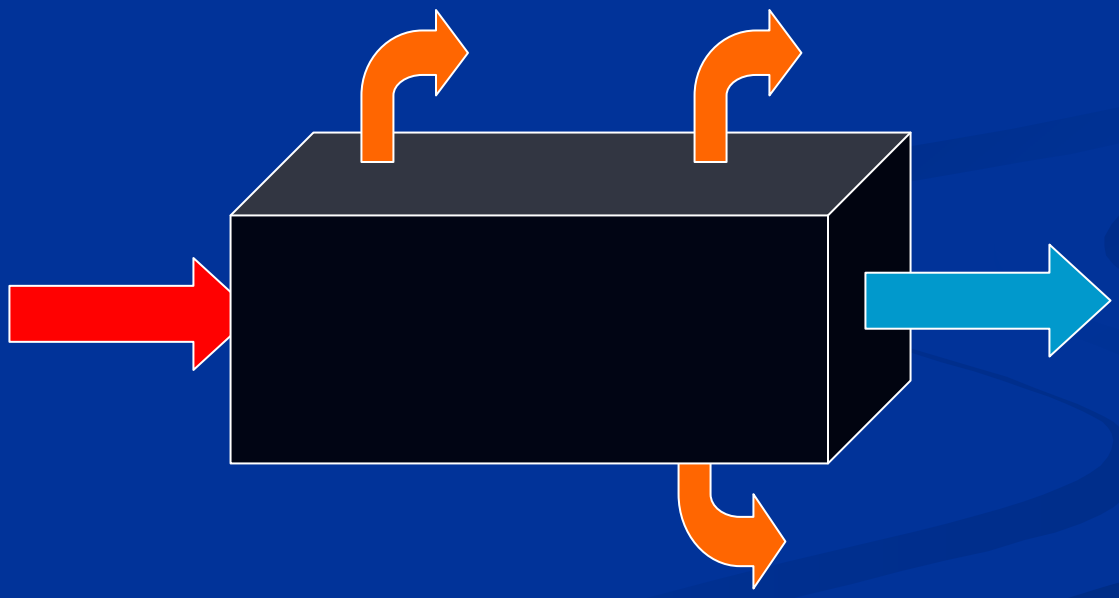
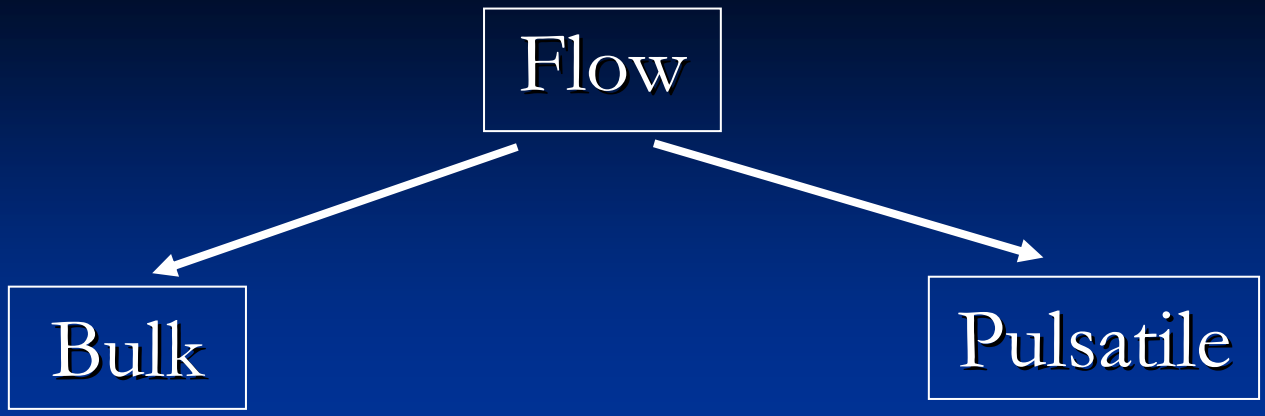
A critical component of any mathematical model of intracranial dynamics is the input/output/test-points





Examples:

CSF prod./absorp.

CBF

Physics: Modulated by resistance to flow

e.g., stenosis

Examples:

Aqueductal pulsations

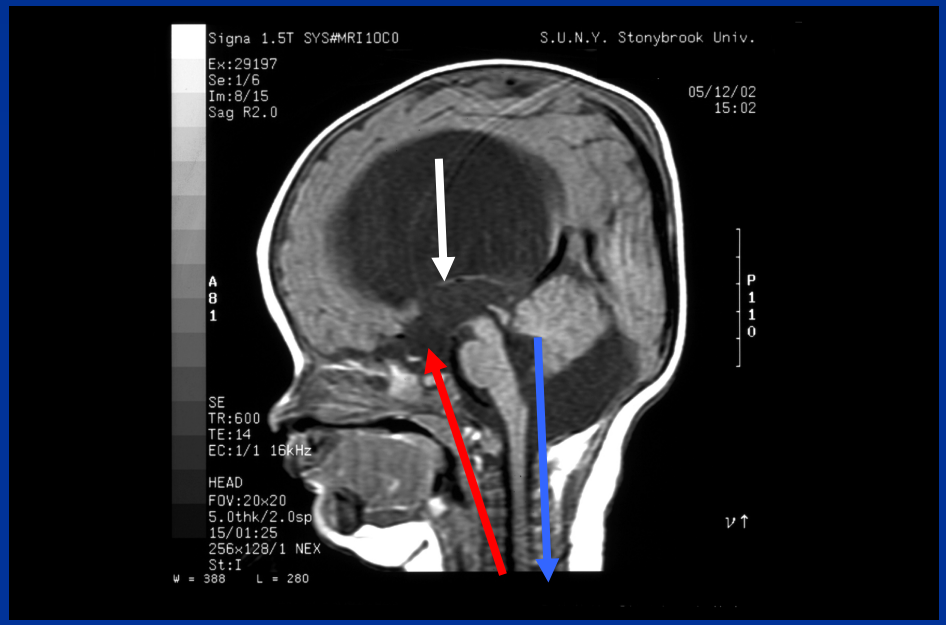
Arterial pulsations

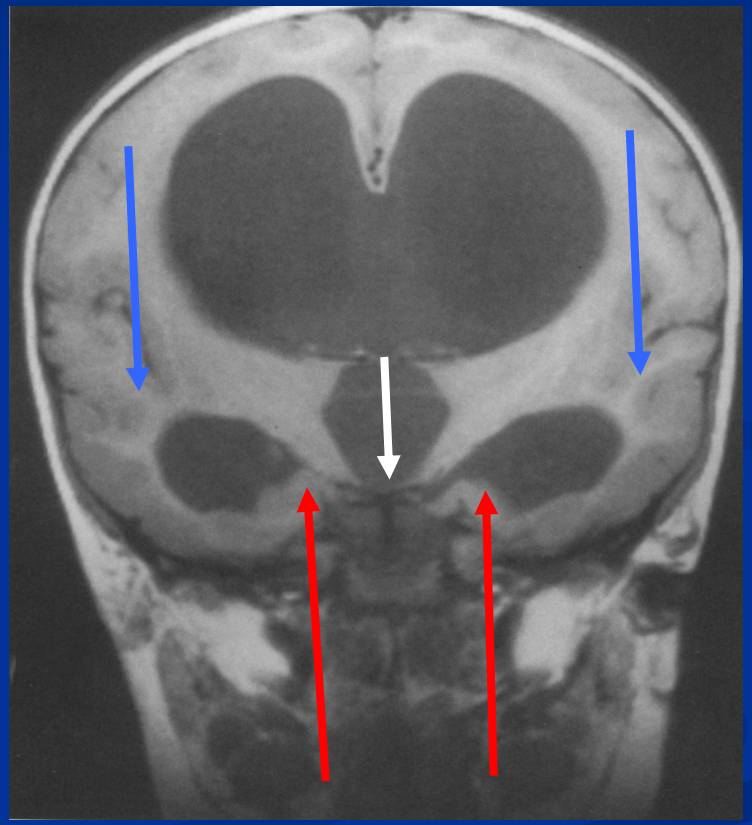
Physics: Modulated by *impedence* to flow

e.g., arteriosclerosis

Of course, these only consider the dynamics of the system.
One final important component any model will be the static components
– e.g. CSF and brain tissue spatial distributions

The closed cranium creates a complex – but coherent – flow system





Bulk Flow

Is there a connection??

Pulsatile Flow

Arterial

- PURPOSE: cerebral perfusion
- SOURCE: heart
- WITH AGE: decreases
- CHANGES IN DISEASE: diminished in NPH and AD
- MODULATED BY: vascular resistance (e.g. stenosis)

- PURPOSE: unknown
- SOURCE: heart
- WITH AGE: increases
- CHANGES IN DISEASE:
  - diminished in NPH and possibly increased in VD
  - Increased pulse *pressure* implicated in increased risk for hypertension, CVD and AD: Does pulsatile *flow* play a similar role?
- MODULATED BY: vascular compliance (e.g. arteriosclerosis)

CSF

- PURPOSE: supply of nutrients to and disposal of neurotoxins from brain
- SOURCE: arterial blood in choroid plexus
- WITH AGE: decreases
- CHANGES IN DISEASE: decreased uptake as source of ventricular dilation in HC
- MODULATED BY: CSF outflow resistance, production

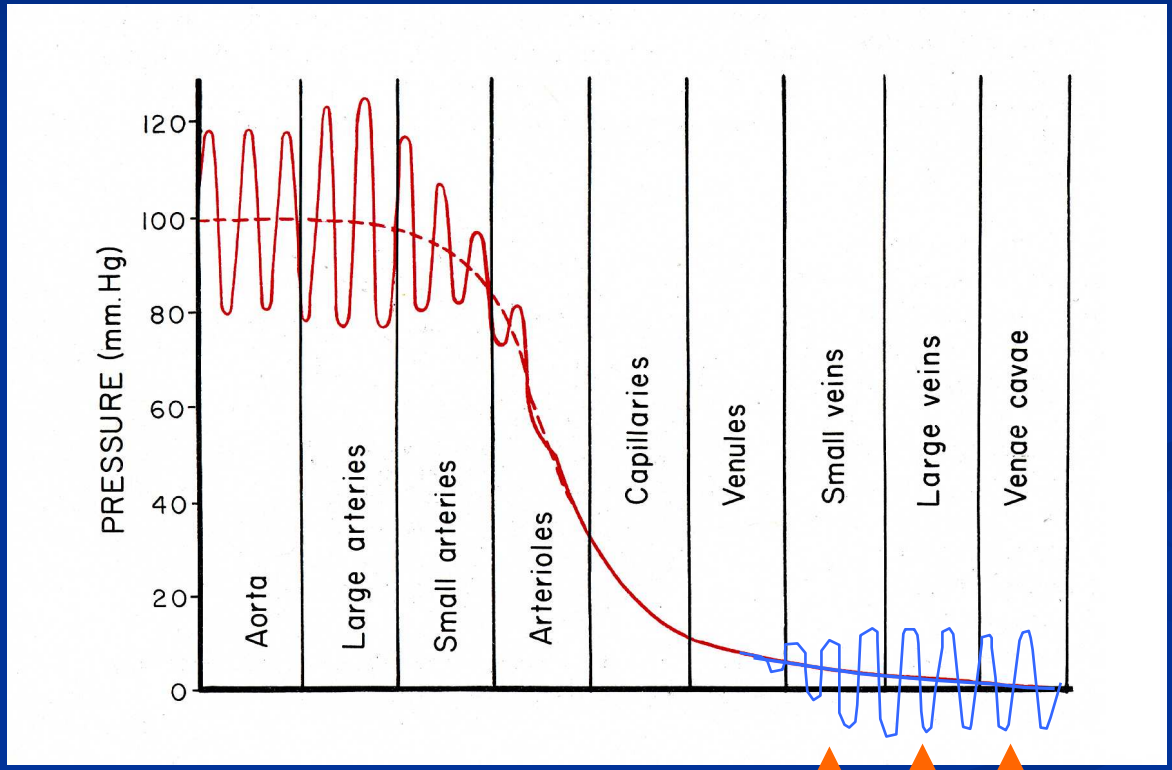
- PURPOSE: dissipation of arterial pulsations by allowing venting of CSF at CC junction
- SOURCE: arterial pulsations into closed cranium
- WITH AGE: unknown
- CHANGES IN DISEASE:
  - increased in aqueduct in NPH
  - redistribution of pulsations seen in hydrocephalus
- MODULATED BY: local intracranial compliance

Venous

- PURPOSE: allow egress of blood from cranium
- SOURCE: arterial blood through capillary trees
- AGE/DISEASE: mirrors arterial bulk flow

- PURPOSE: dissipation of arterial pulsations
- SOURCE: CSF coupling to arterial pulsations
- WITH AGE: unknown
- CHANGES IN DISEASE: possibly unevenly redistributed in NPH

The cerebral windkessel effect



Where does this come from??

Take home point

Multiple, interacting, flow pathways in the brain require a detailed assessment of *multiple* components of flow

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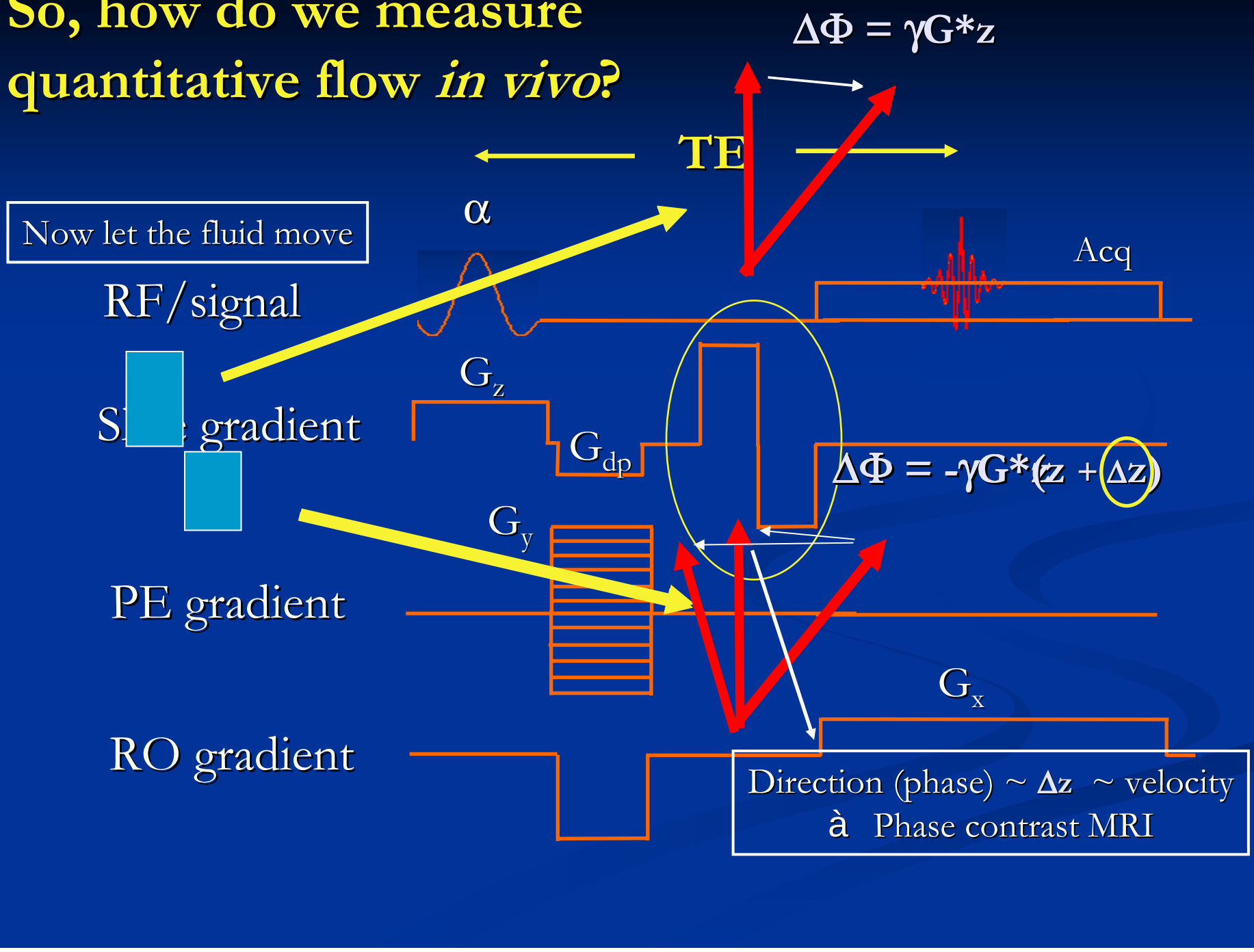
Arterial, venous, CSF – bulk, pulsatile

The important question is not (always) how much flow is there, but how is the flow distributed within the cranium

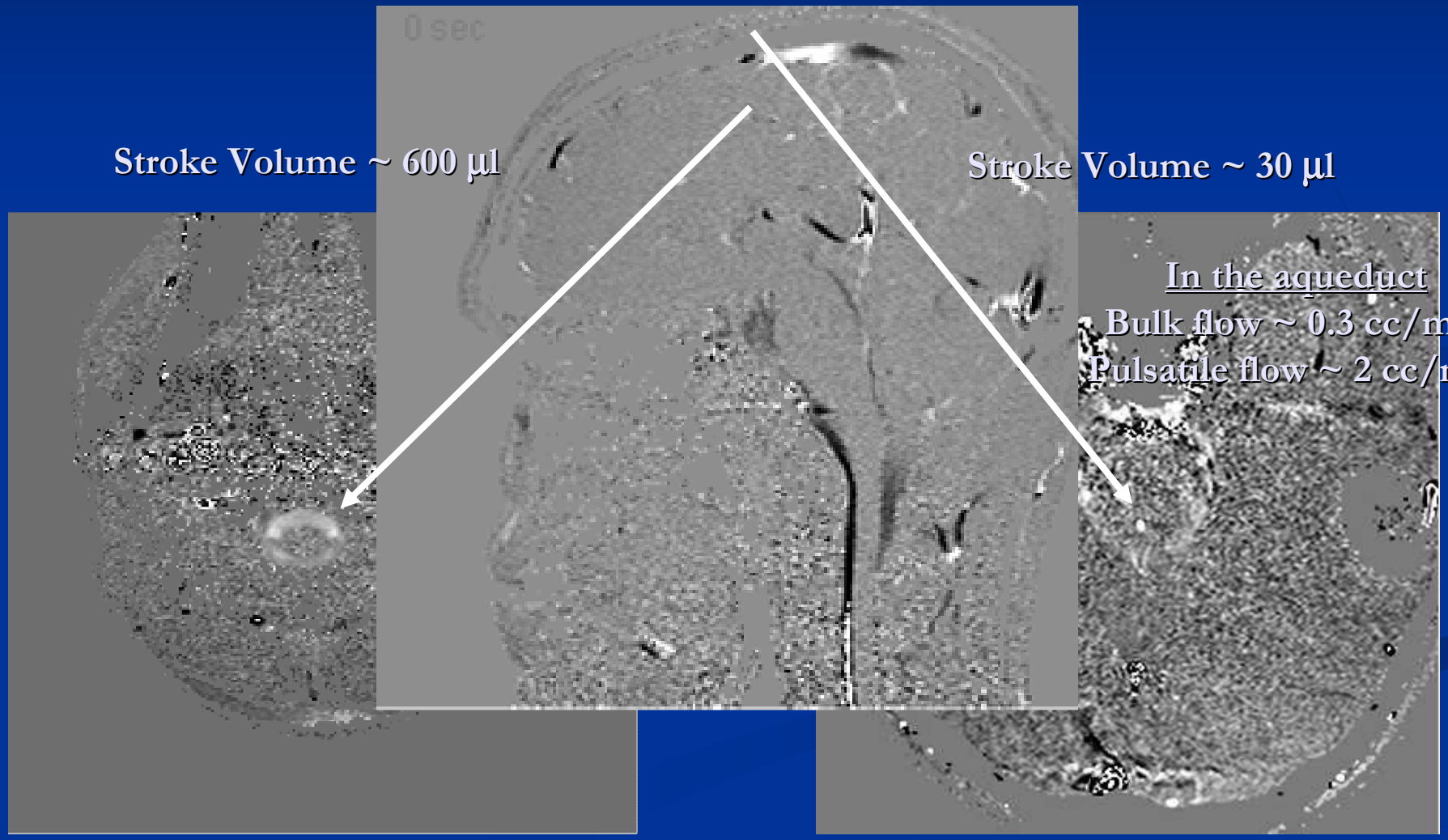
So, how do we measure quantitative flow *in vivo*?



OK, so what can we measure?

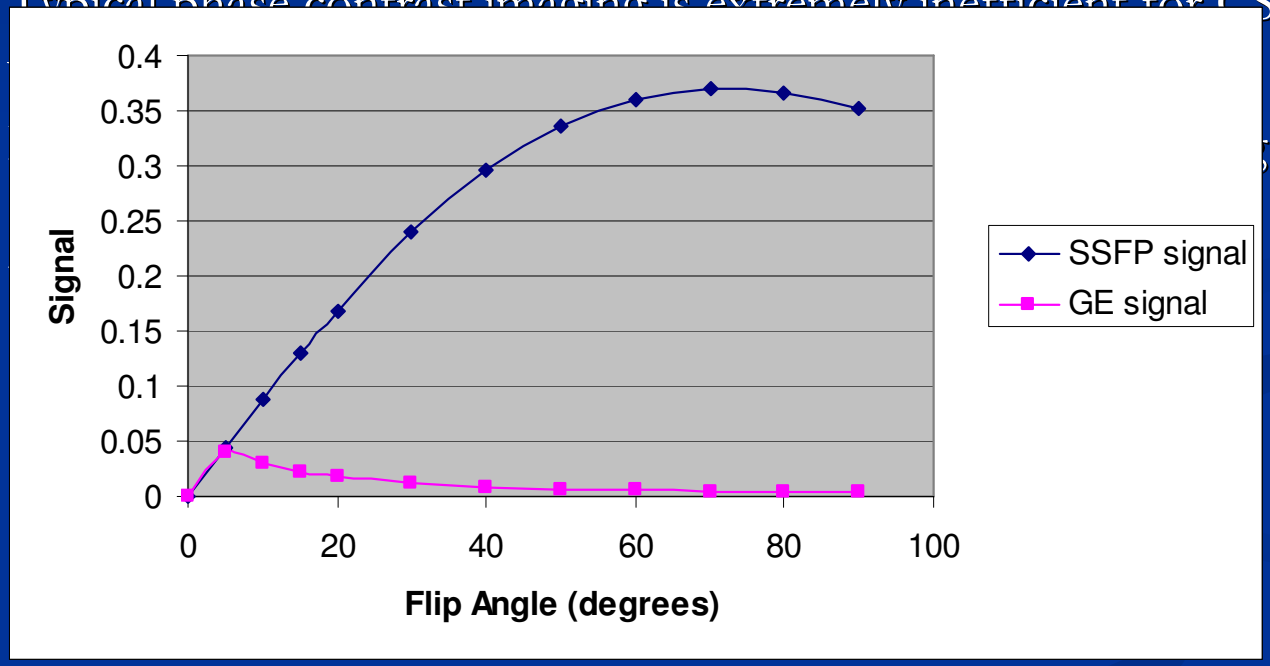
Image #	Flow Compartment	Purpose	Includes	Image plane	Encoding velocity
1	Cervical level vascular	Net vascular flow (arterial + venous)	extracranial carotids vertebrals jugulars	axial	80 cm/s
2	Intracranial vascular	Net intracranial (supratentorial) flow	intracranial carotids basilar sagittal & straight sinus	oblique axial	80 cm/s
3	Cortical venous	Measure of cortical flow	bridging cortical veins	off-midline sagittal	30 cm/s
4	Aqueduct	Ventricular CSF flow	aqueduct	oblique axial	10 cm/s
5	Prepontine cistern	Supratentorial SA CSF flow	PP cistern	axial	5 cm/s
6	Cervical SA space	Total SA CSF flow	cervical SAS	axial	5 cm/s
7	Other CSF	Convexity and 3rd vent. CSF pulsatility	3rd ventricle convexity SAS	coronal	5 cm/s

Flow studies
typical *CSF studies*



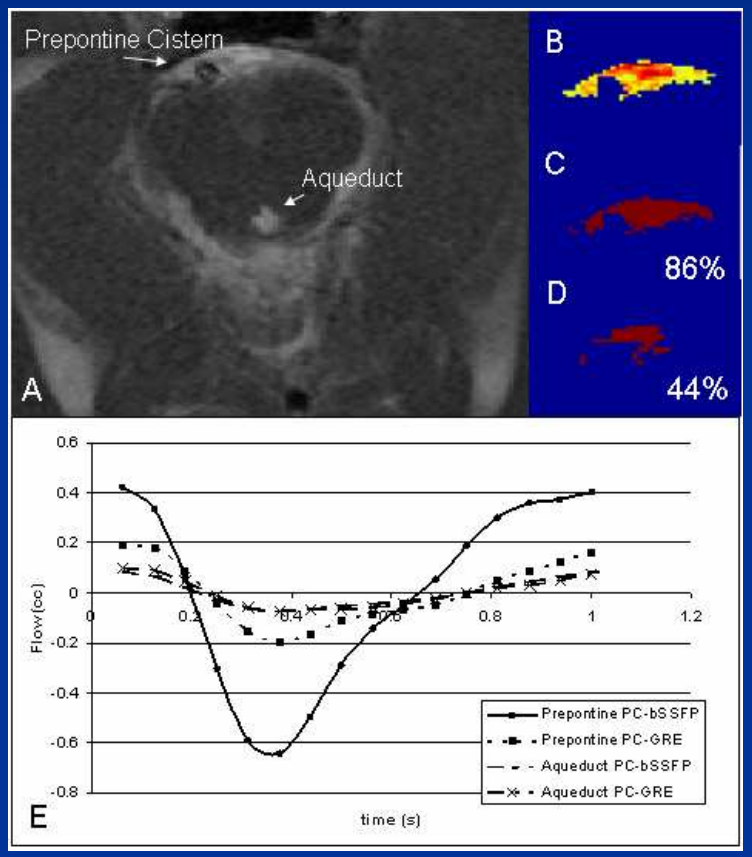
Improved Flow Imaging

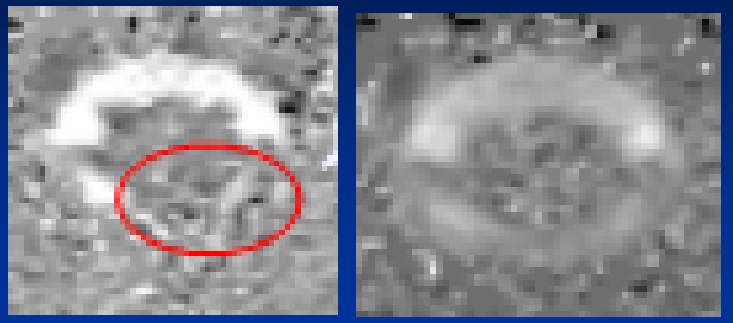
- Typical phase contrast imaging is extremely inefficient for CSF
-
-

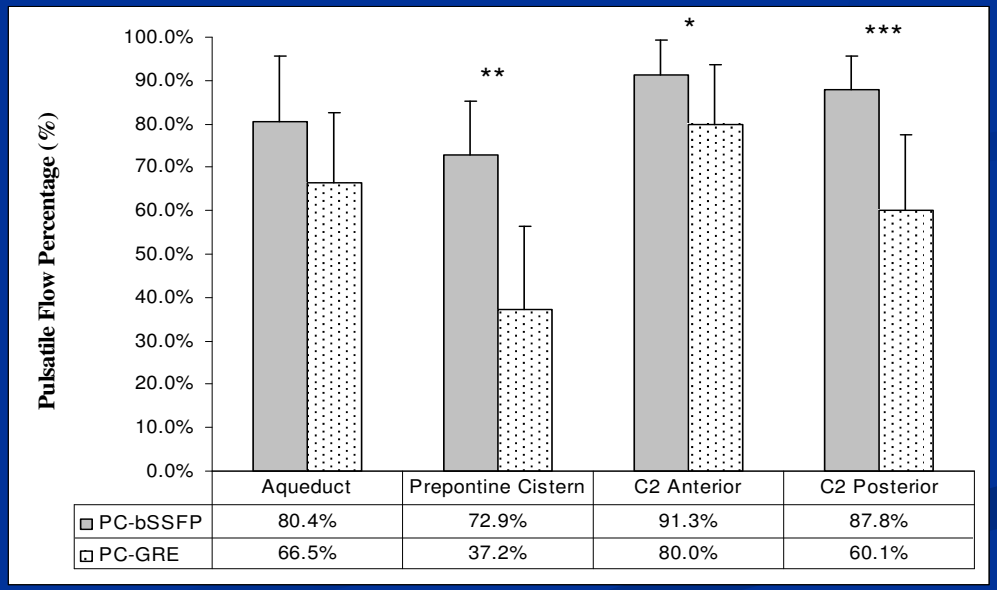


- Can generate images very rapidly

Improved CSF flow methods







Improved quantitation

Stroke volume measurements (mean ± standard deviation) for the four CSF regions measured with the two techniques in a healthy control population.

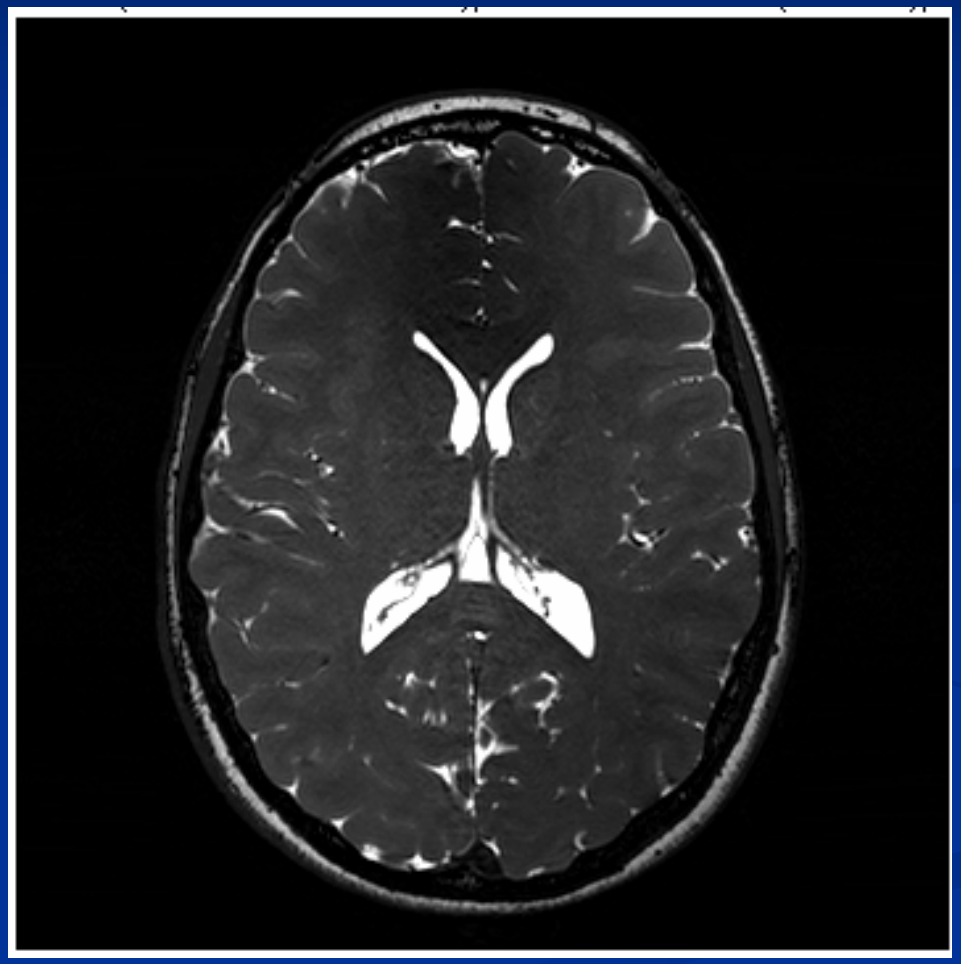
CSF Flow Region	Stroke Volume (μl)		p-value
	PC-bSSFP	PC-GRE	
Aqueduct	28.2 ± 16.0	25.44 ± 11.6	p = 0.2
Prepontine Cistern	217.1 ± 100.5	144.02 ± 107.8	*p < 0.05
Anterior SAS at C2	419.1 ± 150.3	357.03 ± 148.8	*p < 0.05
Posterior SAS at C2	273.3 ± 150.4	183.9 ± 116.3	*p < 0.0001

… and improved SNR

CSF Flow Region	Total CSF	
	SNR Gain	p-value
Aqueduct	2.02 ± 0.40	*p < 0.0005
Prepontine Cistern	2.92 ± 1.07	*p < 0.0005
Anterior SAS at C2	4.91 ± 2.91	*p < 0.005
Posterior SAS at C2	5.64 ± 3.38	*p < 0.0005

- Improved quantitation à accurate flow distributions
- Improved SNR à faster measurements/more flow planes

and structure …

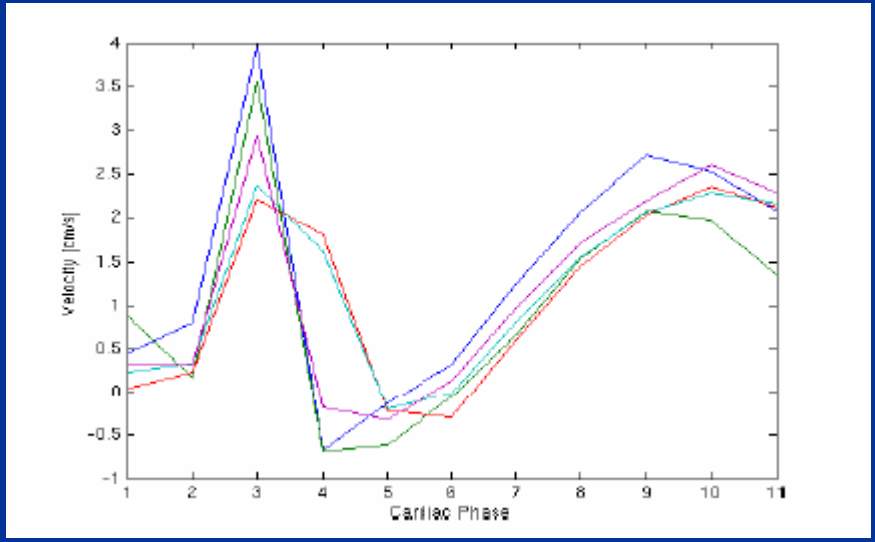


~ 0.6 mm isotropic resolution in < 4min

Pitfalls !!

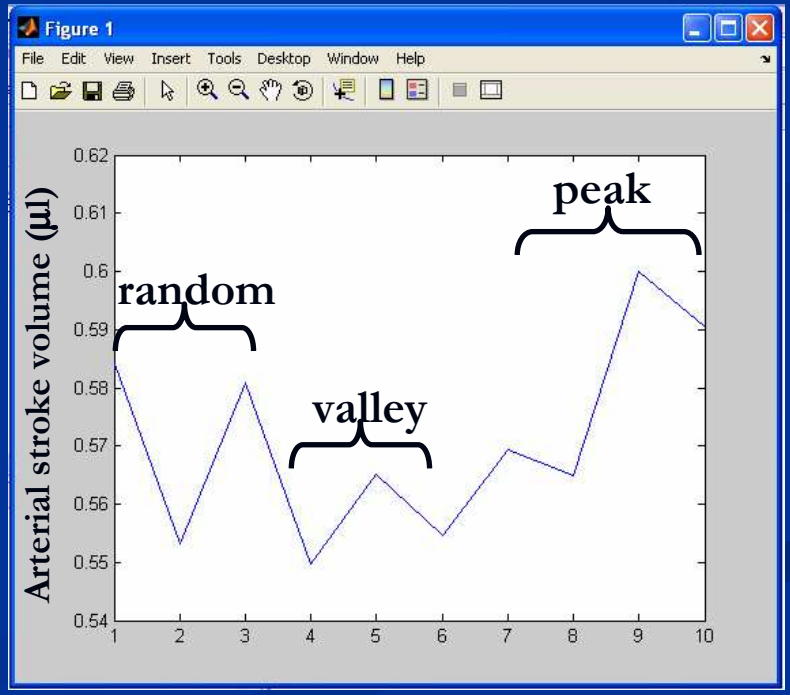
Respiratory effects

CSF flow variation over resp. cycle



From Santini et al, Proc ISMRM 2007, p. 3206.

Arterial flow variation with varying start point of scan



Quantitation issues

- **Make sure we are comparing apples with apples**

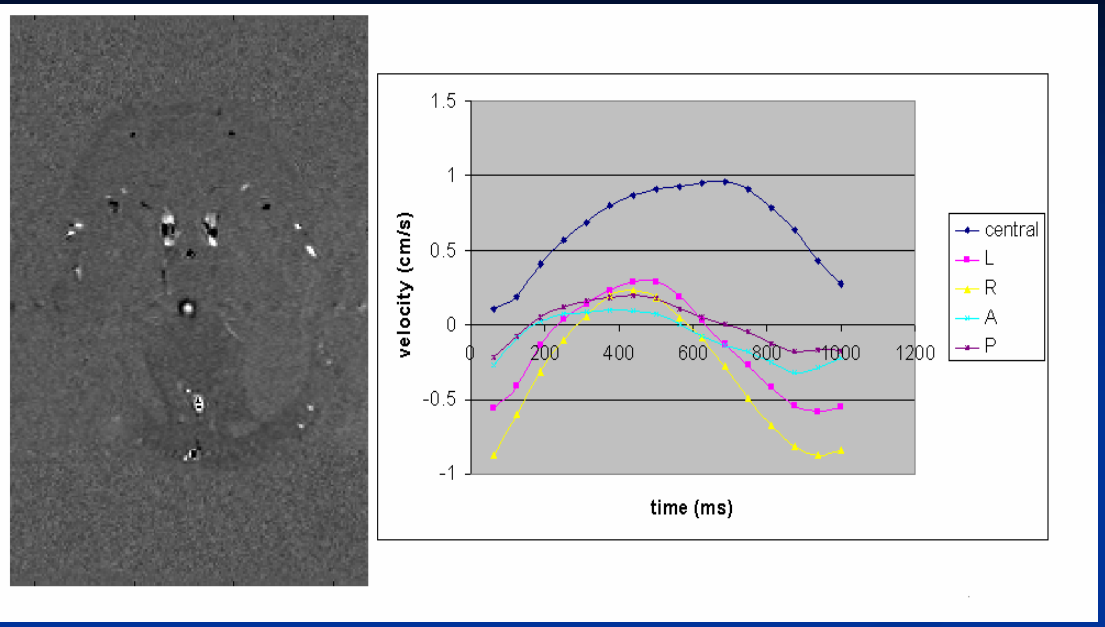
	Arterial stroke volume µL	Cervical CSF stroke volume µL	Venous compression µL
Controls	910	610	300
Hydrocephalus	970	470	500

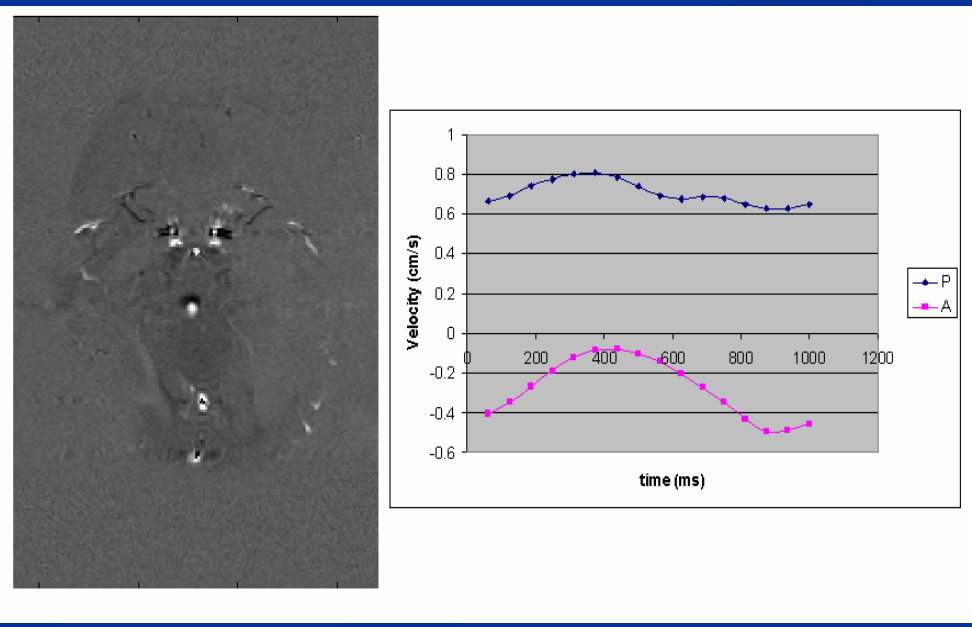
- **Accurate absolute quantitation**

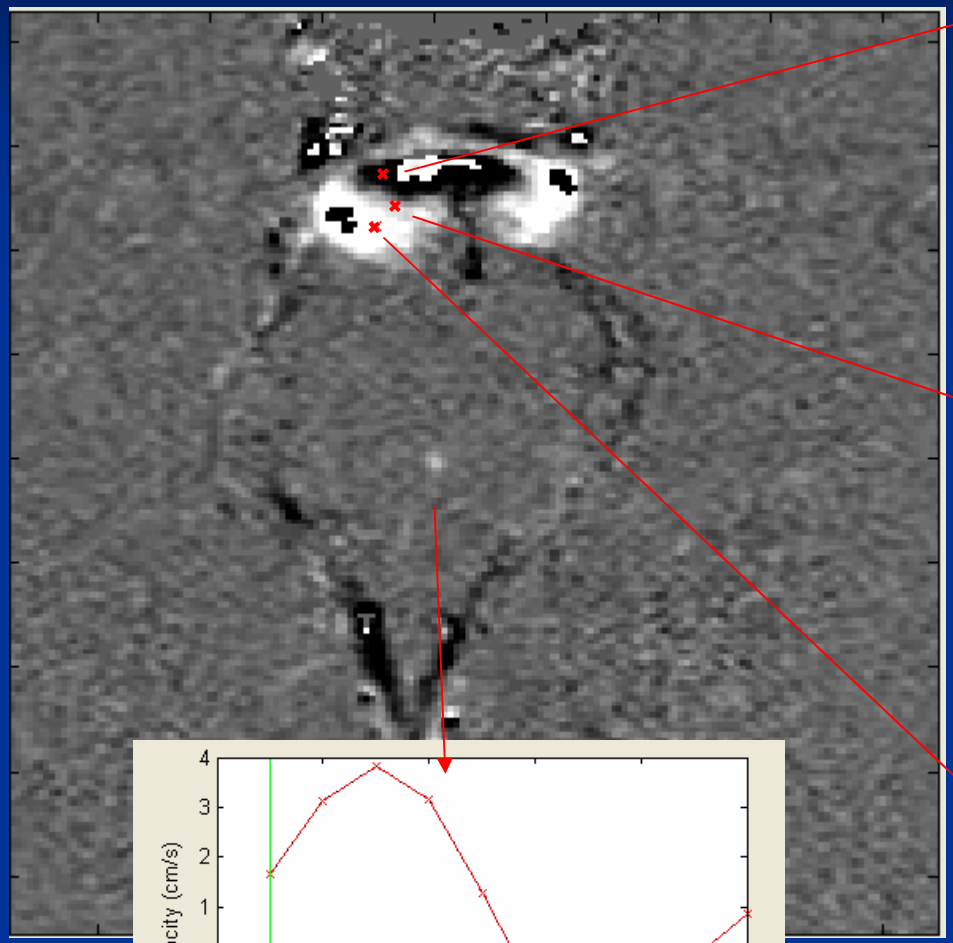
Results for CSF stroke volume @ CCJ

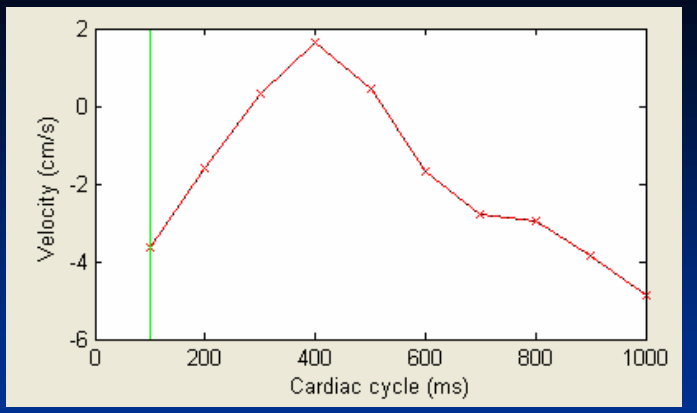
- **Greitz (1993) – 960 µL**
- **Baledent (2004) – 467 µL**
- **Wagshul (2006) – 610 µL**

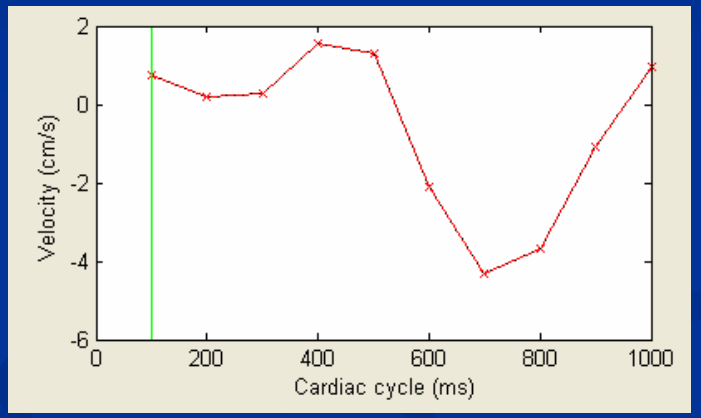
Modeling issues: Patient example



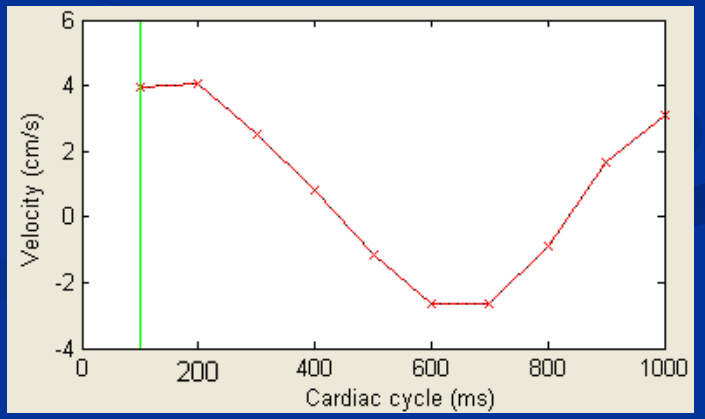





Velocity (cm/s)
2
0
-2
-4
-6
0
200
400
600
800
1000
Cardiac cycle (ms)


Velocity (cm/s)
2
0
-2
-4
-6
0
200
400
600
800
1000
Cardiac cycle (ms)

Velocity (cm/s)
4
3
2
1
0
-1
-2
0
200
400
600
800
1000
Cardiac cycle (ms)


Velocity (cm/s)
6
4
2
0
-2
-4
0
200
400
600
800
1000
Cardiac cycle (ms)

So, what can modeling do for us?

- Understanding the relationship between patient flow/pressure data and symptoms
- Predicting flow/pressure in portions of the intracranial space inaccessible to non-invasive measurements
- Predicting the effects of shunting and other therapeutic devices

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