Patient-Specific CFD Simulations of Vasospasm in 3 Different Cases

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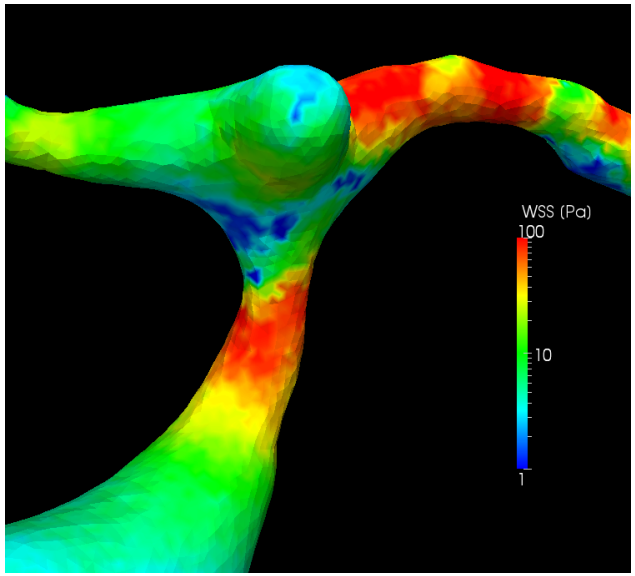
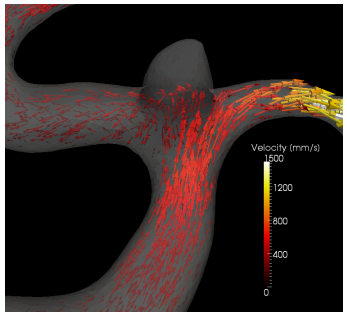
13th June 2013



We have done simulations using data from 3 subjects suffering from vasospasm

Introducing vasospasm
and our cases

Our method

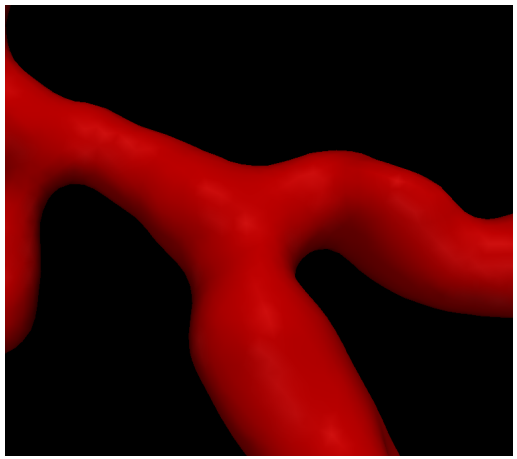


Results and
conclusions

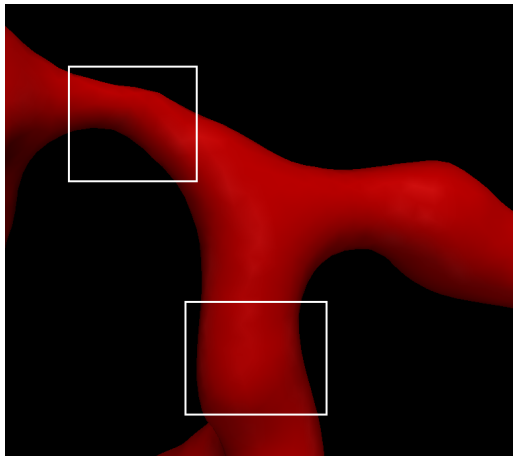
Vasospasm is a serious and common complication of cerebral aneurysm rutpure

- Causes constriction in surrounding arteries
- Onset usually 3-14 days after rupture
- A common cause of poor outcome of subarachnoid hemorrhage

Temporary, dramatic change in the blood vessels!



Normal state



Vasospasm

Treatment methods are available, but may be dramatic in themselves

Blood supply to the brain is vital: Doctors try to maintain cerebral perfusion!

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Blood supply to the brain is vital: Doctors try to maintain cerebral perfusion!

This can be done by several methods:

- Calcium antagonists
- Hemodilution (blood thinners)
- Hypervolemia (increase blood volume)
- Hypertensive drugs

Vasospasm has a highly complex pathophysiology

It is to a large degree unknown why vasospasms occur.

Possible factors include:

- Elevated endothelin 1 levels
- Decreased production of nitric oxide
- Changes in the electrical properties of smooth mucle cells
- Elevated CSF pressure
- and so on...

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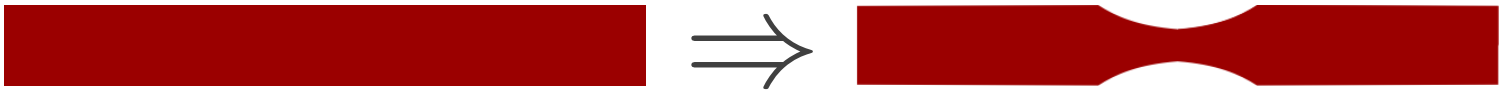
- and so on...

Therefore:
A very long way to use CFD as *the* tool for explaining vasospasm, but what about the hemodynamic effects of vasospasm?

Vasospasm as well as the treatment can cause great variations in the hemodynamics

”Idealized” situation: Straight vessel, vessel diameter constriction of 50% and no reduction in flow rate.



$$V \propto \frac{1}{r^2}$$ Peak velocity increase by a factor 4

$$\tau \propto \frac{1}{r^3}$$ WSS increase by a factor 8

$$\frac{dp}{dx} \propto \frac{1}{r^4}$$ Pressure drop increase by a factor 16

Major hemodynamic change during vasospasm - related to aneurysm development?

Hemodynamics is believed to can

- identify ruptured from unruptured aneurysms.
- predict growing aneurysms.
- alter arteries in days.

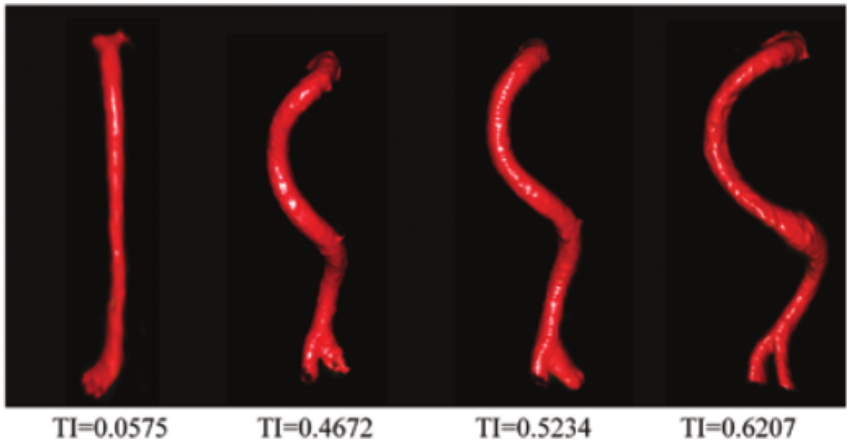


Figure from: Hoi et al., 2008

Vasospasm can cause dramatic changes in hemodynamics!

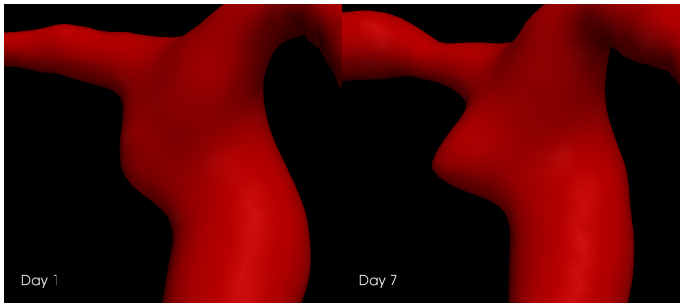
Three patients with vasospasm

Three patients with vasospasm

A

Rupture of right ICA sidewall aneurysm.

- Moderate vasospasm from day 3
- 3x velocity increase
- Significant aneurysm growth



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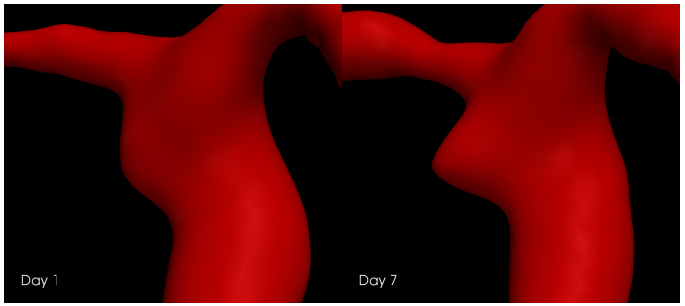
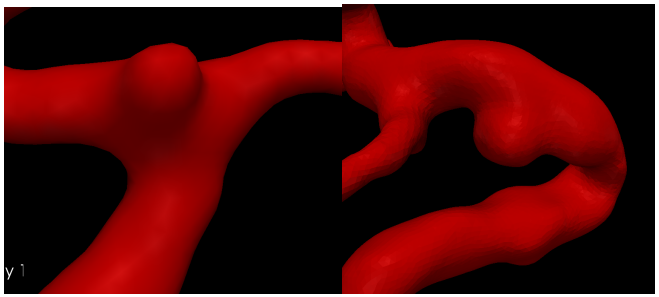


B

Rupture and surgery of large right ICA aneurysm

- Severe vasospasm in *left* part of CoW
- Unruptured aneurysms at left ICA and MCA
- Growth of MCA aneurysm, not of ICA aneurysm



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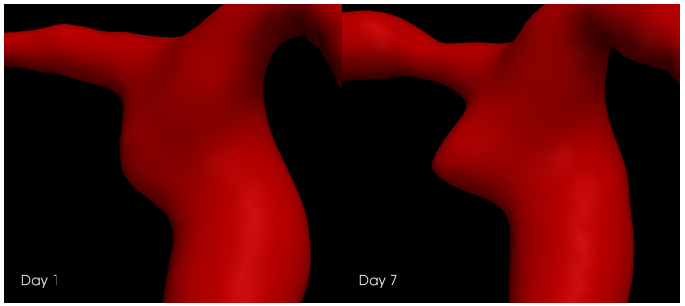

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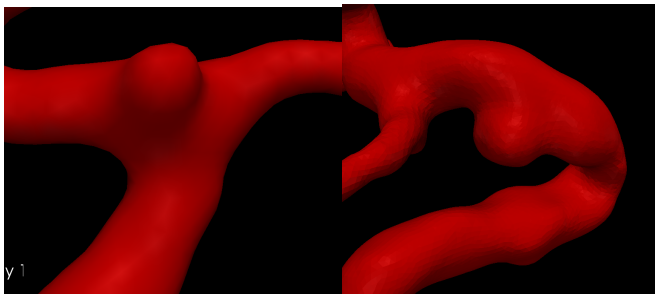
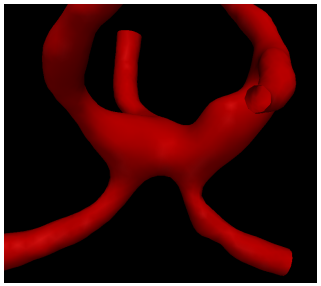


C

Rupture of ACA (A2) sidewall aneurysm

- CT images indicate growth
- ... but data disregarded because of high uncertainty



This motivates the question:

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Can hemodynamics be used to help explain aneurysm development during vasospasm?

The Navier-Stokes equations are solved by a pressure correction scheme implemented in FEniCS

- Incremental pressure correction scheme
- Semi-implicit convection handling
- Crank-Nicholson timestepping

$$\rho\left(\frac{\partial \mathbf{u}}{\partial t} + \mathbf{u}\cdot\nabla\mathbf{u}\right) = -\nabla p + \mu\nabla^2\mathbf{u} + \mathbf{f}$$

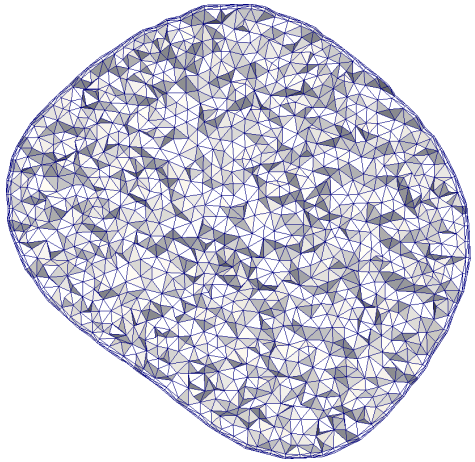
$$\nabla\cdot\mathbf{u} = 0$$

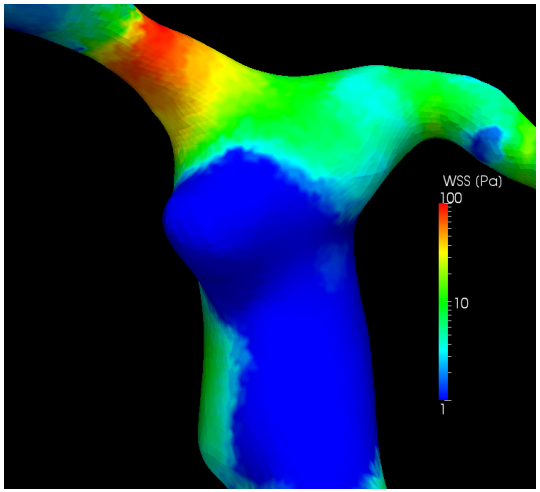
Scheme:

1. Solve tentative velocity using pressure from previous timestep
2. Solve Poisson equation to correct pressure
3. Use pressure correction to update velocity

We calculate common hemodynamic parameters on medium-sized meshes

- Piecewise linears
- 10000-20000 timesteps
- Approx. 2,000,000 tetrahedral cells
- Two boundary layers

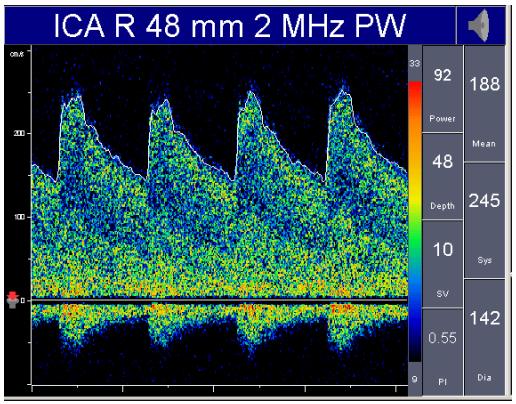




Hemodynamic parameters

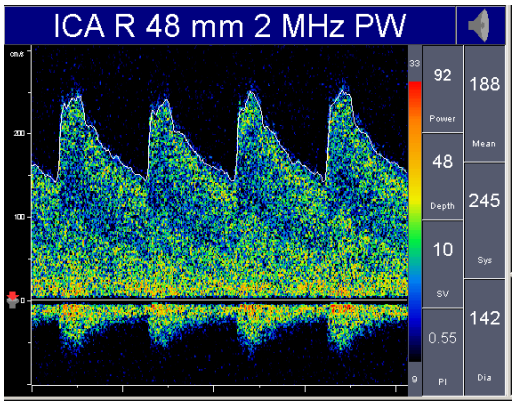
1. Time-averaged WSS
2. Pressure drop
3. Oscillatory shear index (OSI)

Inflow boundary conditions set to fit Doppler measurements



Pouiseille profiles at inlets to match Doppler velocities

Inflow boundary conditions set to fit Doppler measurements



Pouiseille profiles at inlets to match Doppler velocities

$$p = C\frac{Q}{A^{3/2}}$$

Outlet BC for similar shear at outlets

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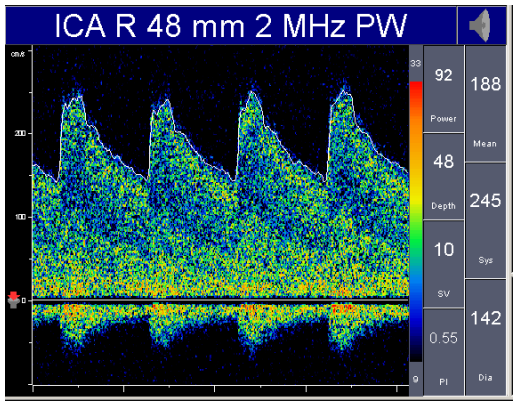


Pouiseille profiles at inlets to match Doppler velocities

$$p = C\frac{Q}{A^{3/2}}$$

Outlet BC for similar shear at outlets

$$\mathbf{u} = \mathbf{0}$$

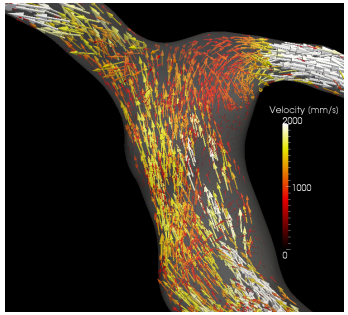
No-slip condition at walls

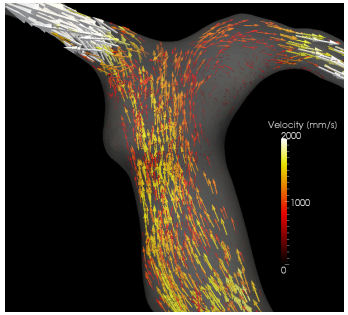
Case A

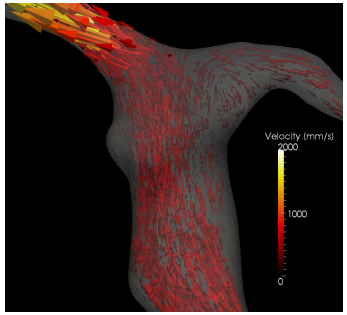
Case A

Case A

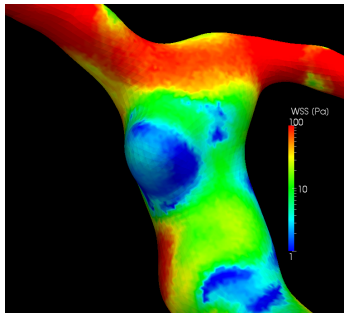
Velocity

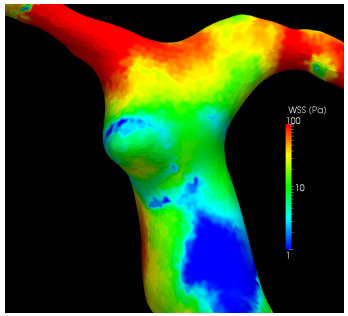


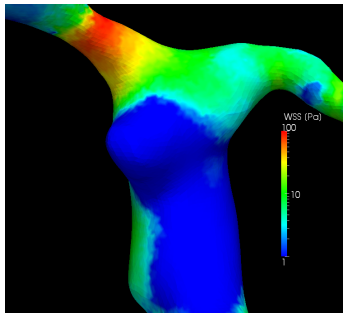




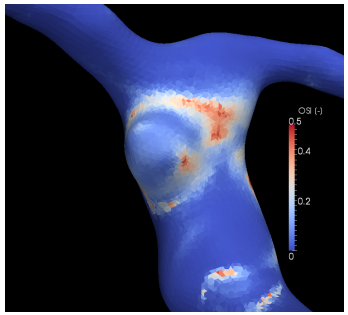
WSS

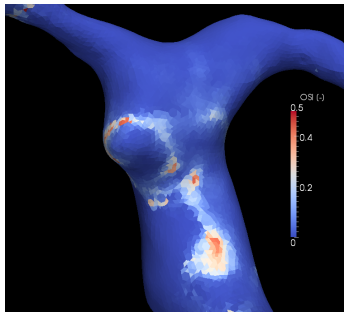


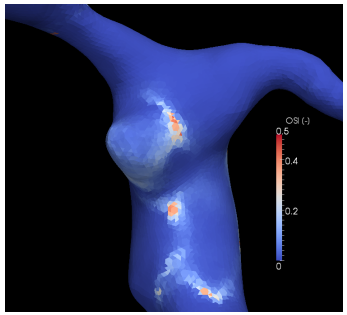




OSI



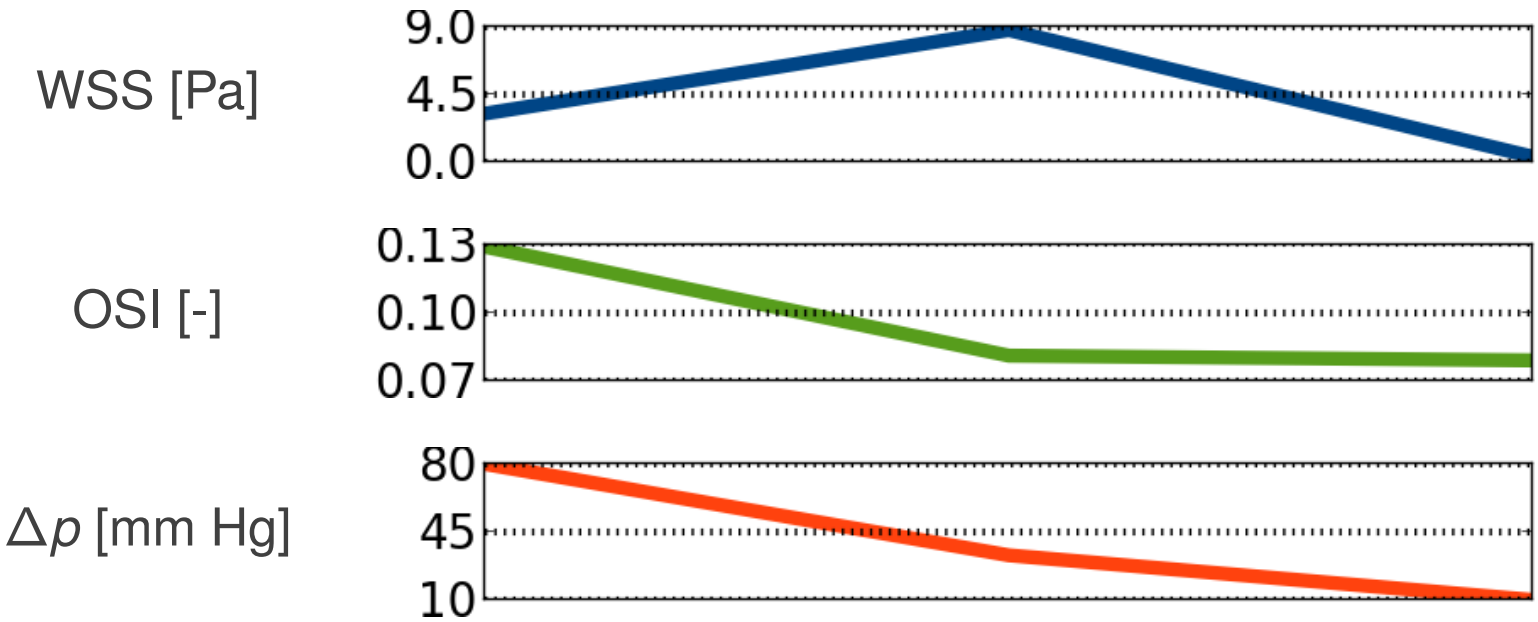




Day 3 Day 5 Day 7

Case A

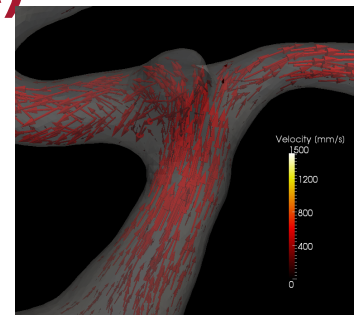
Day	3	5	7
Vasospasm	Moderate	Moderate	Small
Growth	52%	21%	3%

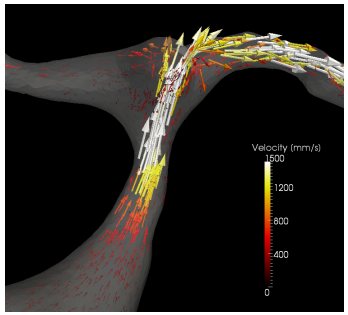


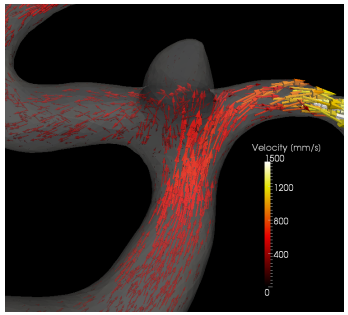
Case B (MCA)

Case B (MCA)

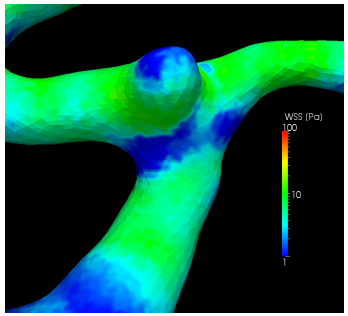
Velocity

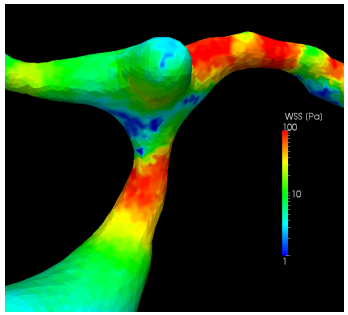


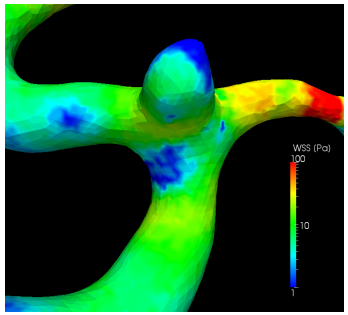




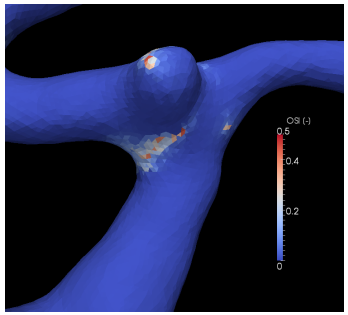
WSS

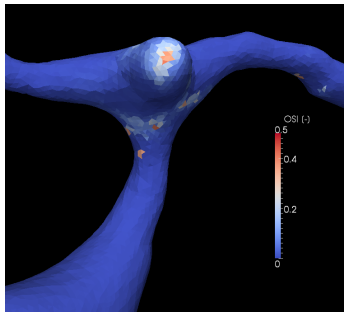


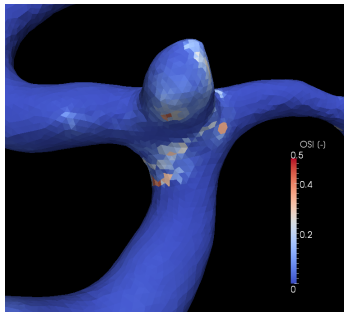




OSI



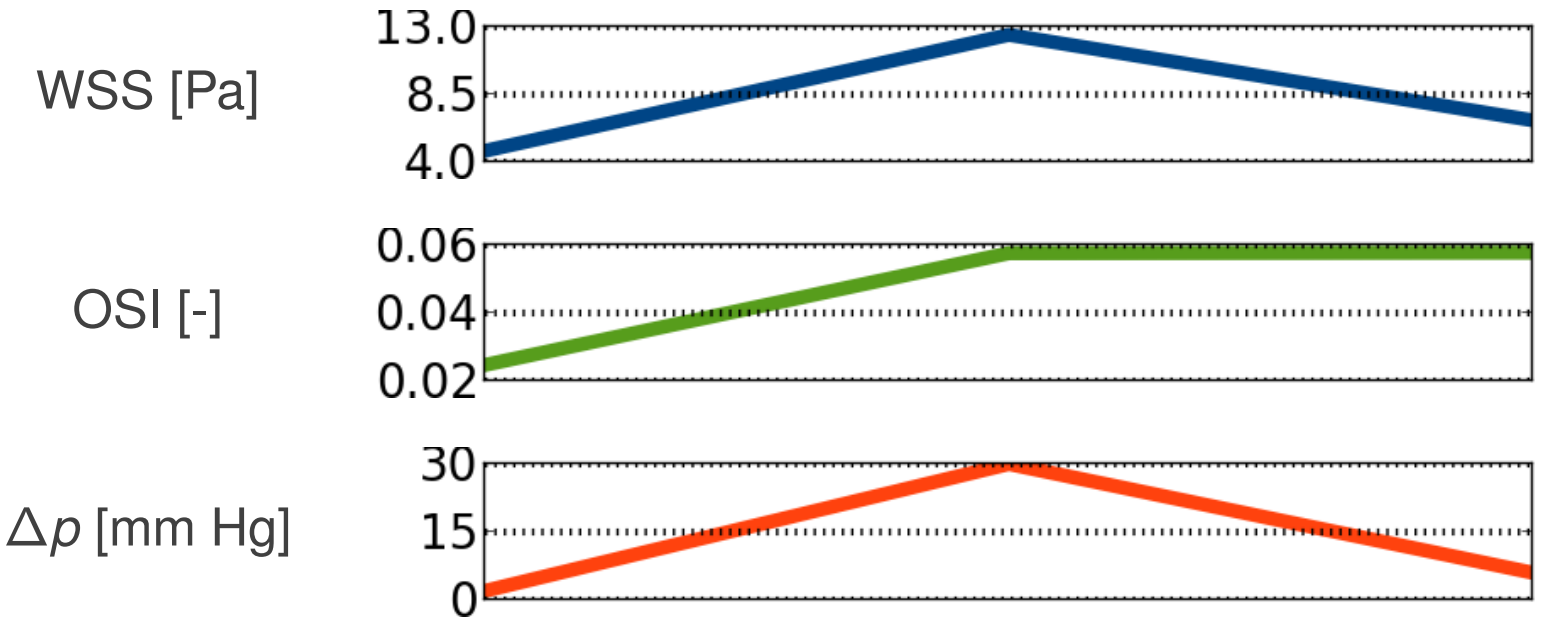




Day 1 Day 10 Day 16

Case B (MCA)

Day	1	10	16
Vasospasm	None	Severe	Small
Growth	-	15%	17%



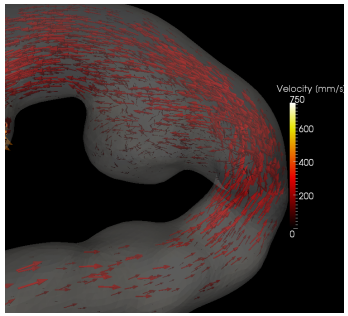
Case B (ICA)

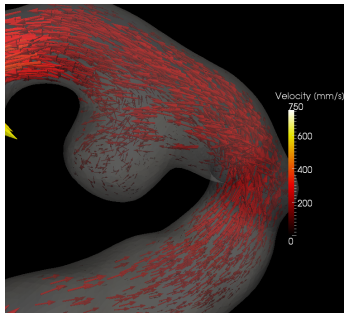
Case B (ICA)

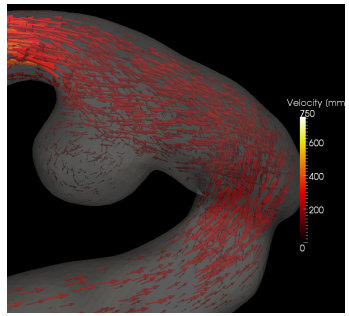
Velocity

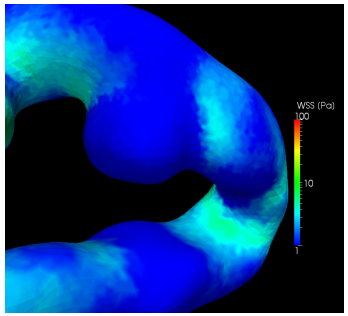
WSS

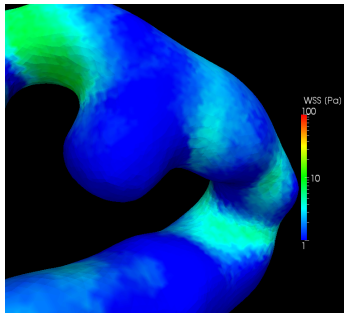
OSI

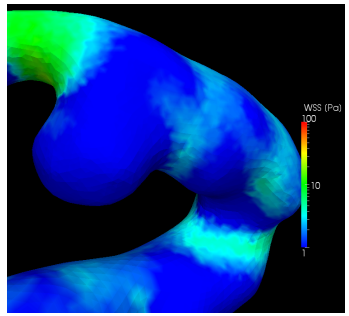


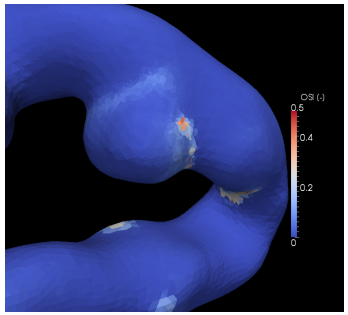


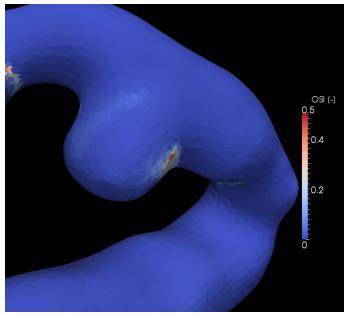


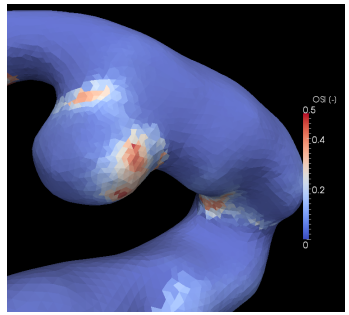








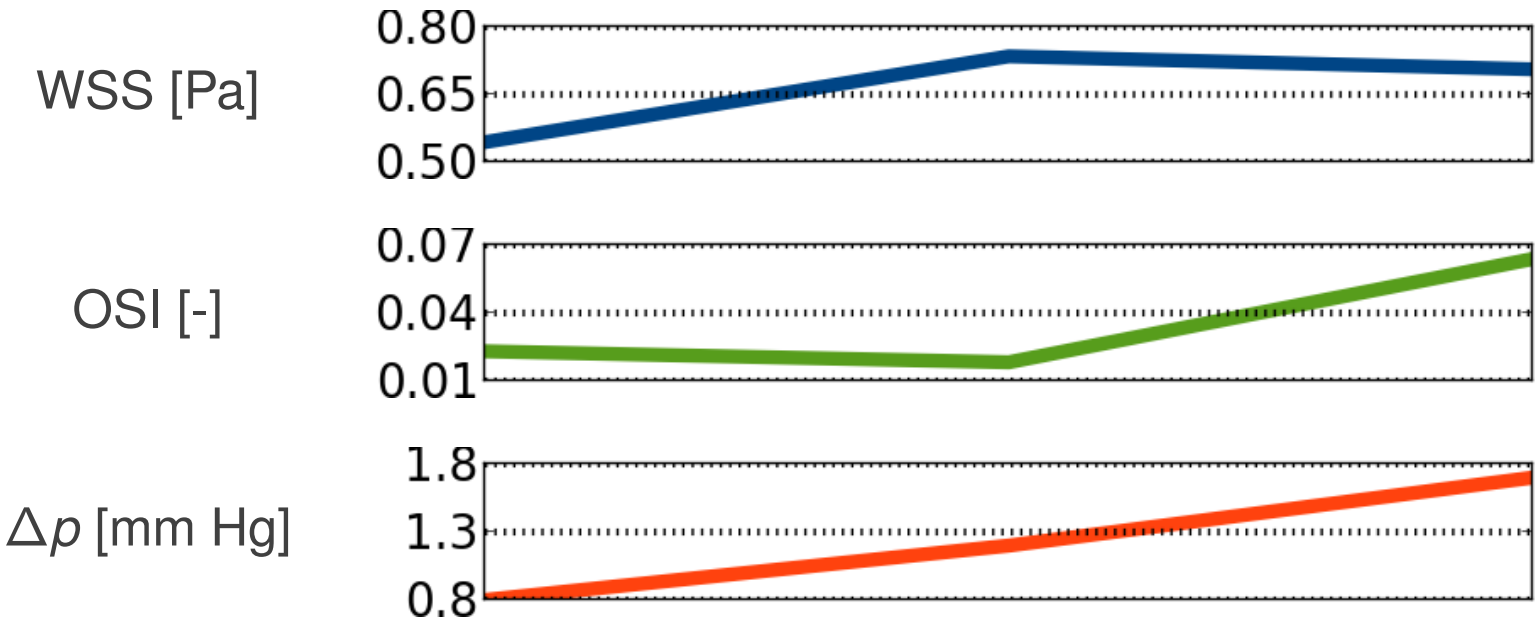




Day 1 Day 10 Day 16

Case B (ICA)

Day	1	10	16
Vasospasm	None	Moderate	Severe
Growth	-	8%	6%



Limitations

Several obvious limitations to this study

- Only two patients studied
- Uncertainties related to Doppler measurements, segmentation of narrow arterial segments and flow diversion
- Incomplete data sets

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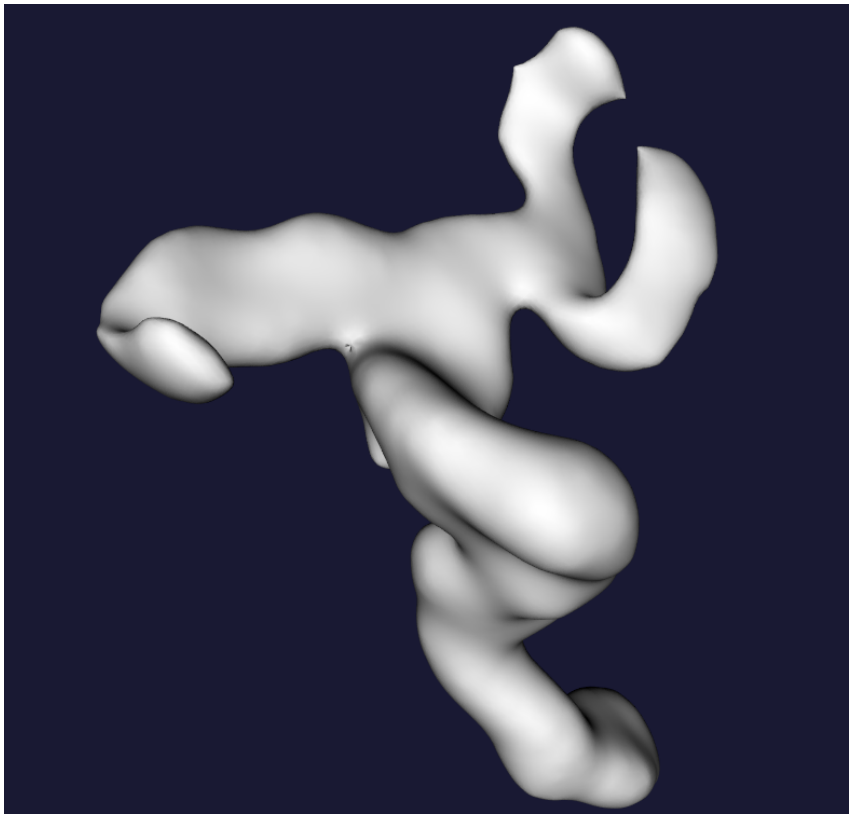

Positives and conclusions

Some points of interest:

- Hemodynamic indicators vary tremendously during vasospasm
- Difficult to correlate hemodynamics with aneurysm development
- A slight tendency of high OSI when growth is observed

CFD on vasospasm could

- improve understanding of short term effects of hemodynamic changes
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