Brain multimodal monitoring: neurosurgeon's perspective

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Hejčl Aleš

Neurosurgery Department, J. E. Purkinje University
Masaryk Hospital,
Ústí nad Labem, Czech Republic

What is the role of MMM in neurointensive care??

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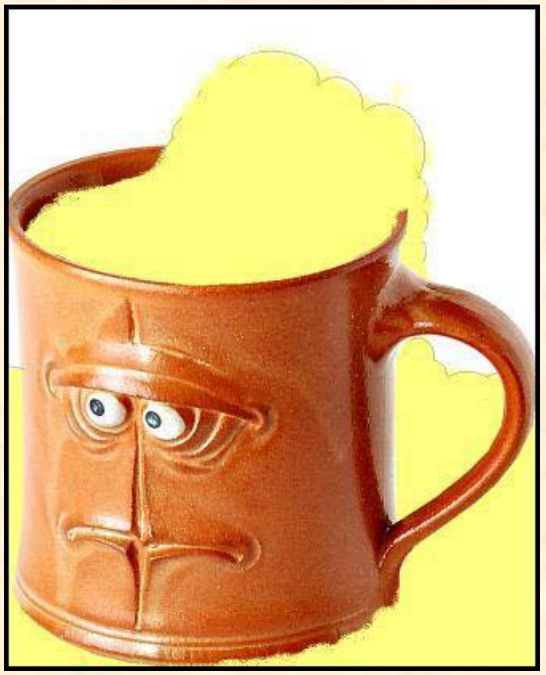
Keep the brain under physiological conditions



Prevent secondary damage:
edema, hemorrhage, isc hemia

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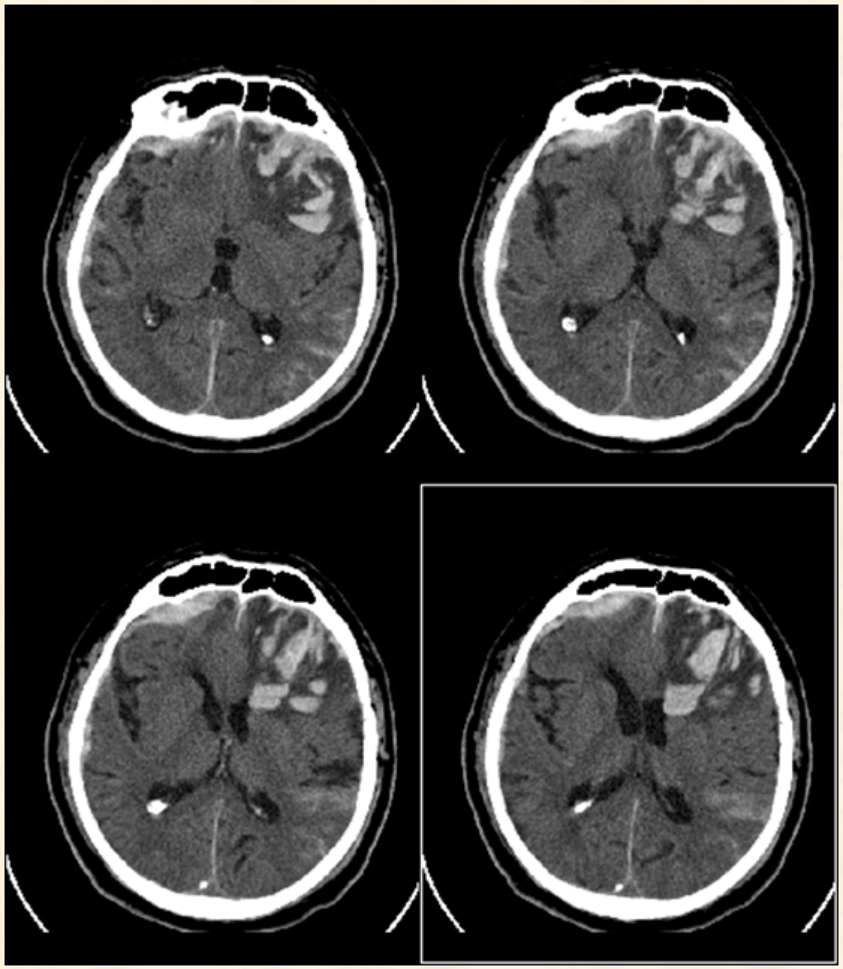
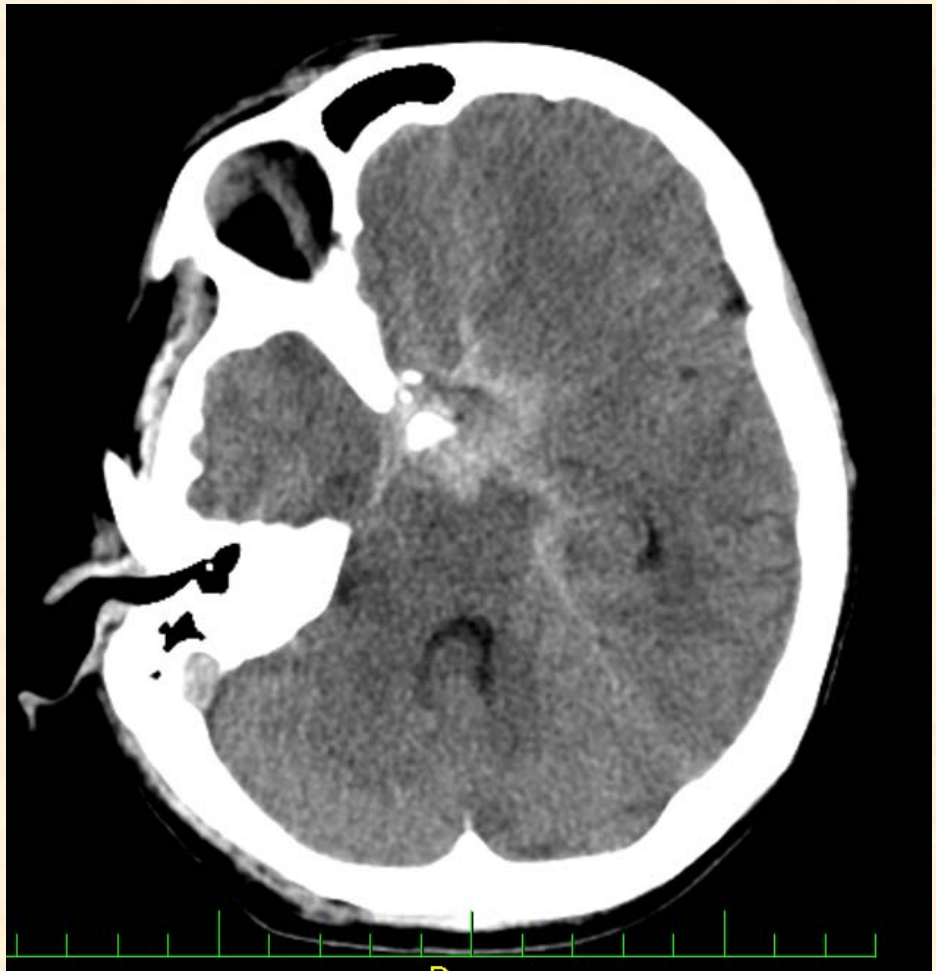
Which patients are „in danger“ of secondary brain damage??

Risk of secondary brain damage (edema, vasospasm, hemorrhage)

TBI



SAH after AN rupture



Standard monitoring techniques in SAH

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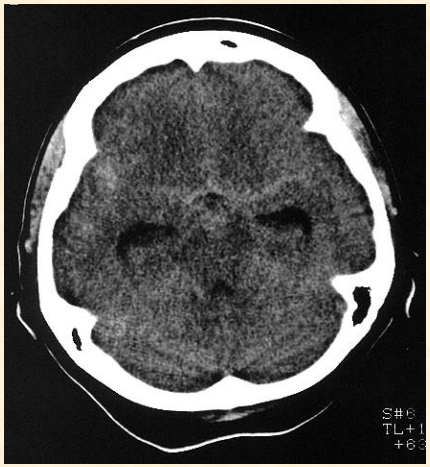
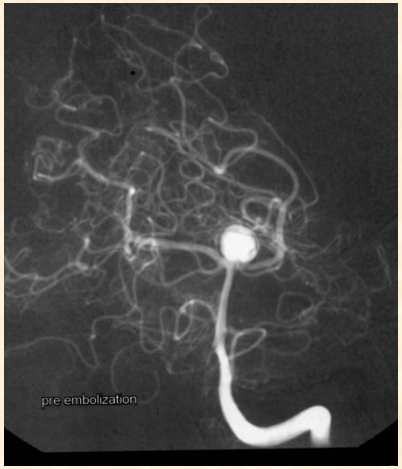
Clinical evaluation

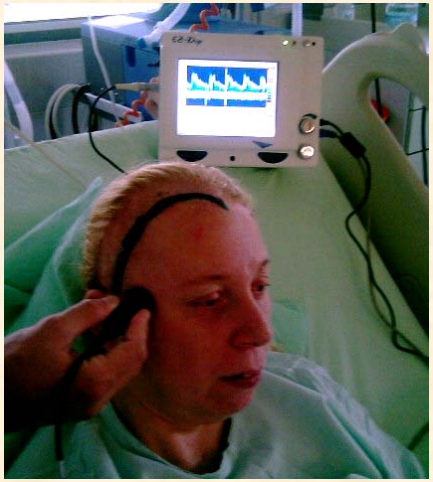
Brain CT



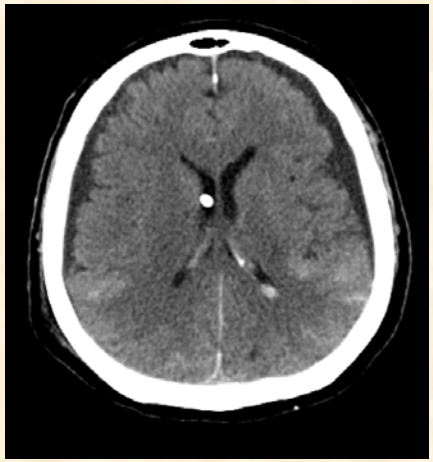
angiography



TCD



EVD



ICP

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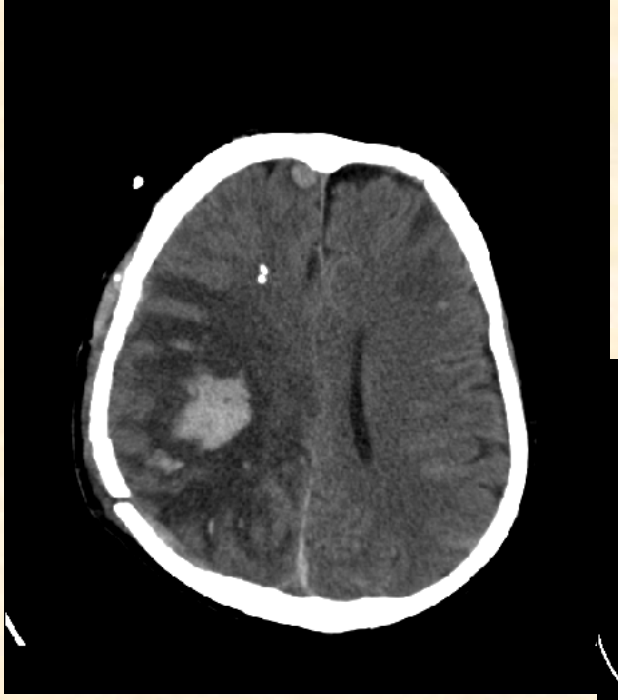
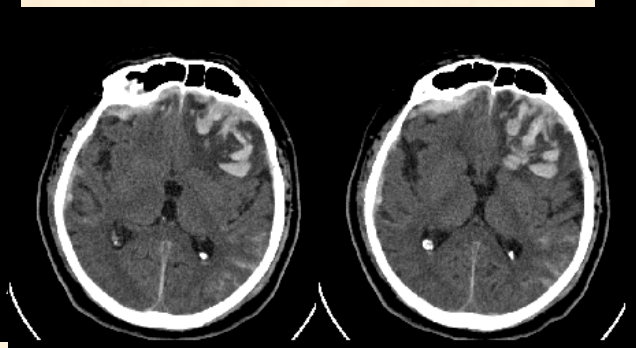
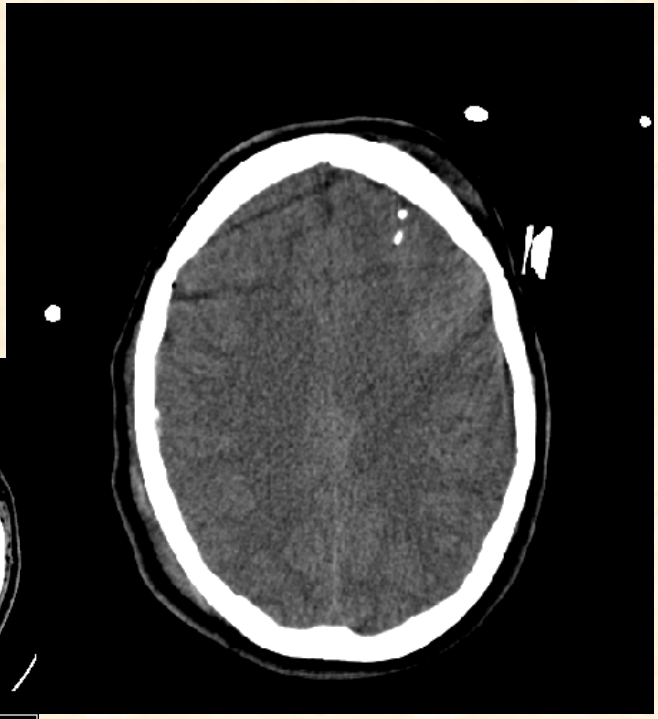
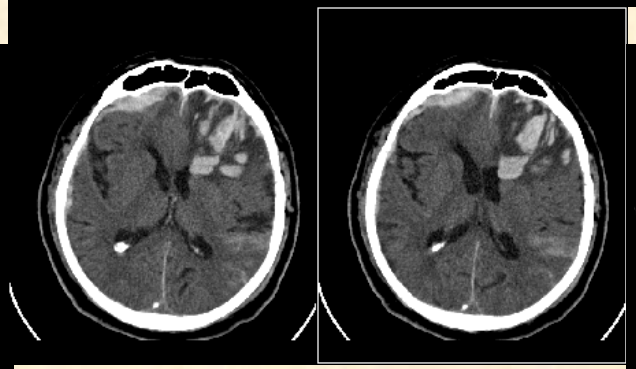
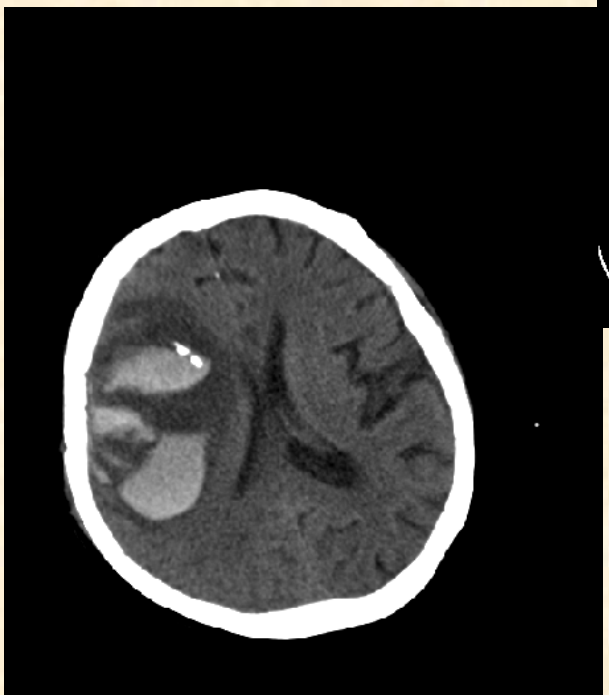
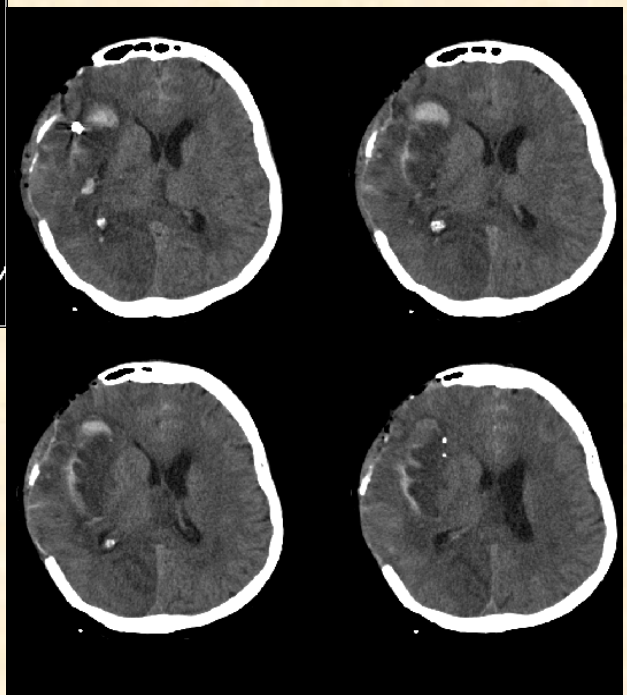
Indication for ICP monitoring

in patients with severe TBI (GCS score < 9) who have

- an abnormality on CT scan
- normal head CT scan if at least one of the following is present
  - age greater than 40 years, posturing on clinical examination
  - or a systolic blood pressure < 90 mmHg

- ICP > 20mmHg – treat „one size fits all“

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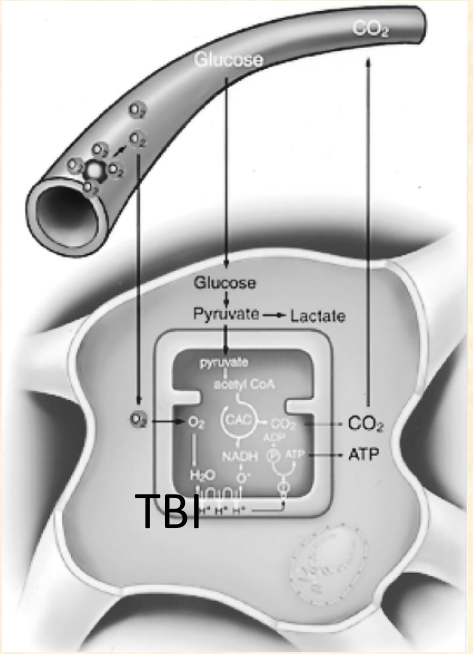
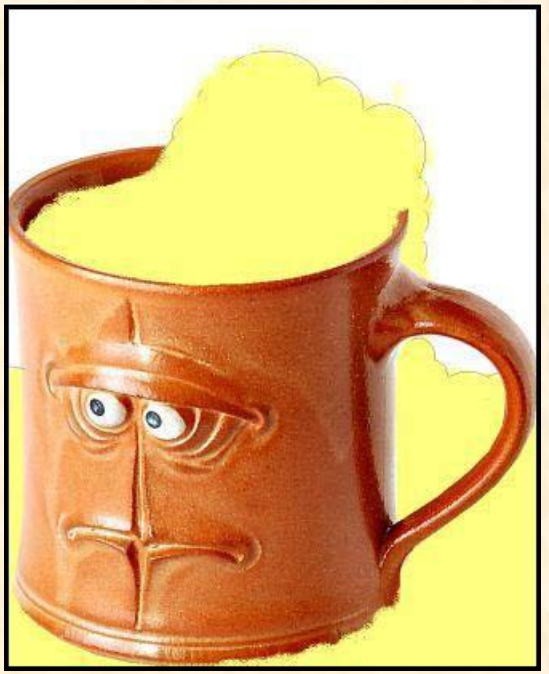
Why multimodal monitoring??

- **targetted therapy**
- **individualized therapy**
- elimination of therapeutic mistakes
- prediction of clinical results
- improve bad clinical results in patients with severe TBI and SAH

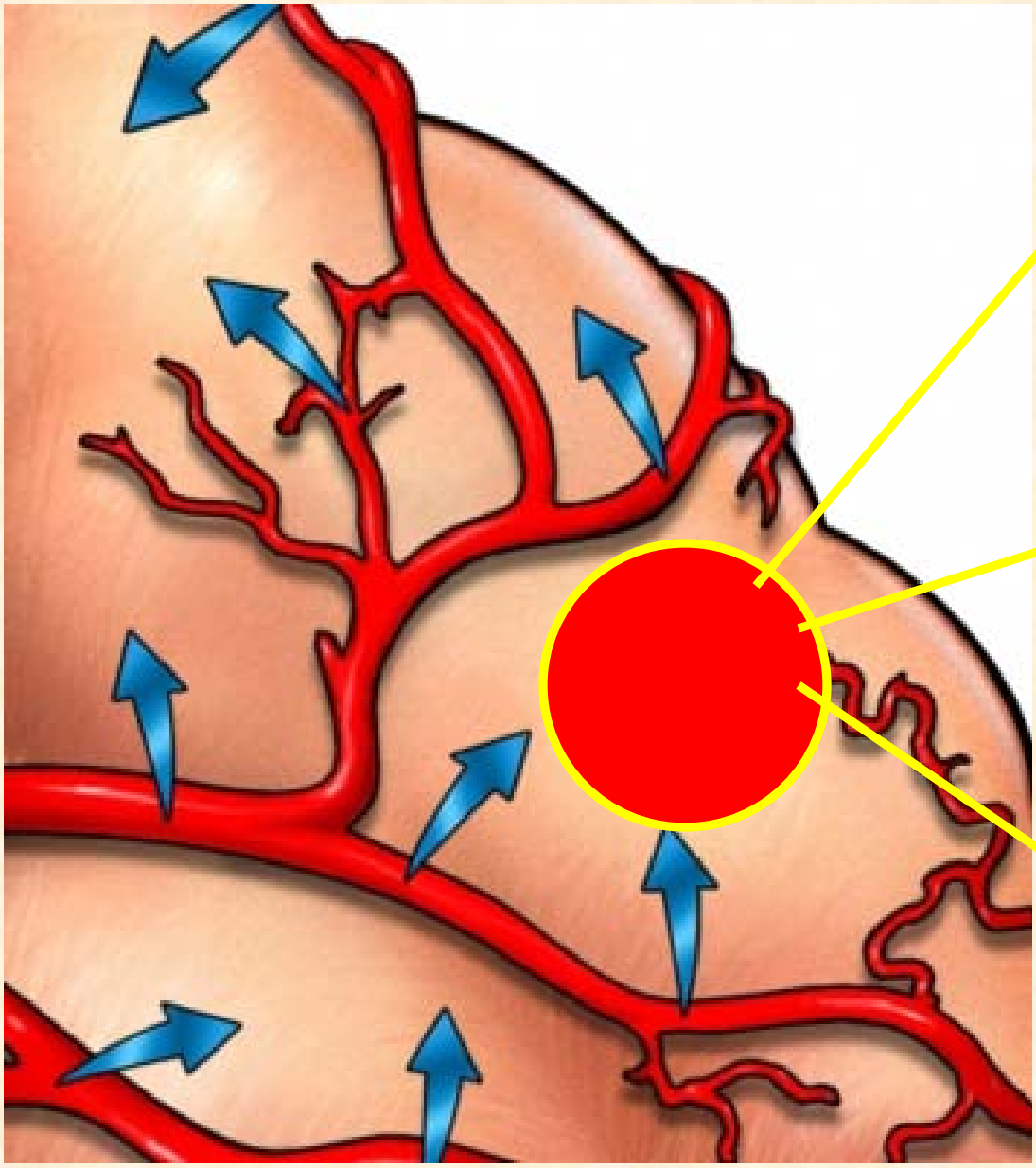


ICP monitoring in TBI questioned!!!

"…..**we were following far too simplistic an approach** and have individually developed methods of **using multiple monitors (multimodality monitoring) to fine tune and individualize treatment variables such as ICP** (rather than simply accepting a treatment threshold of 20 mm Hg)."



Chesnut et al. A trial of intracranial-pressure monitoring in traumatic brain injury. NEJM, 2012.



How much blood flows into the brain?

(Hemedex, CBF)

How much oxygen do we supply?

(Licox, ptiO$_2$)

How is brain metabolism doing?

(microdialysis)

Multimodal monitoring at our ICU

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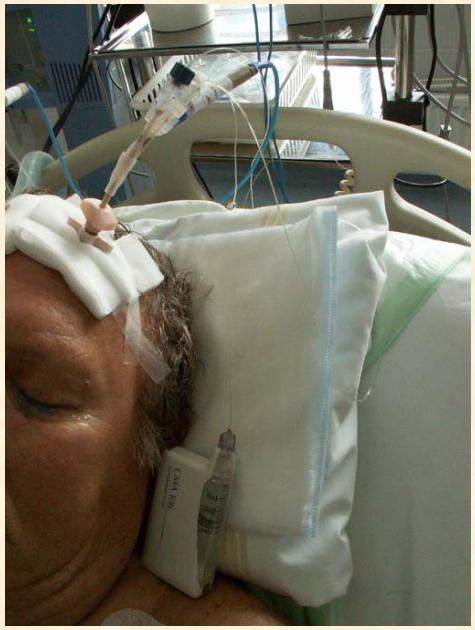
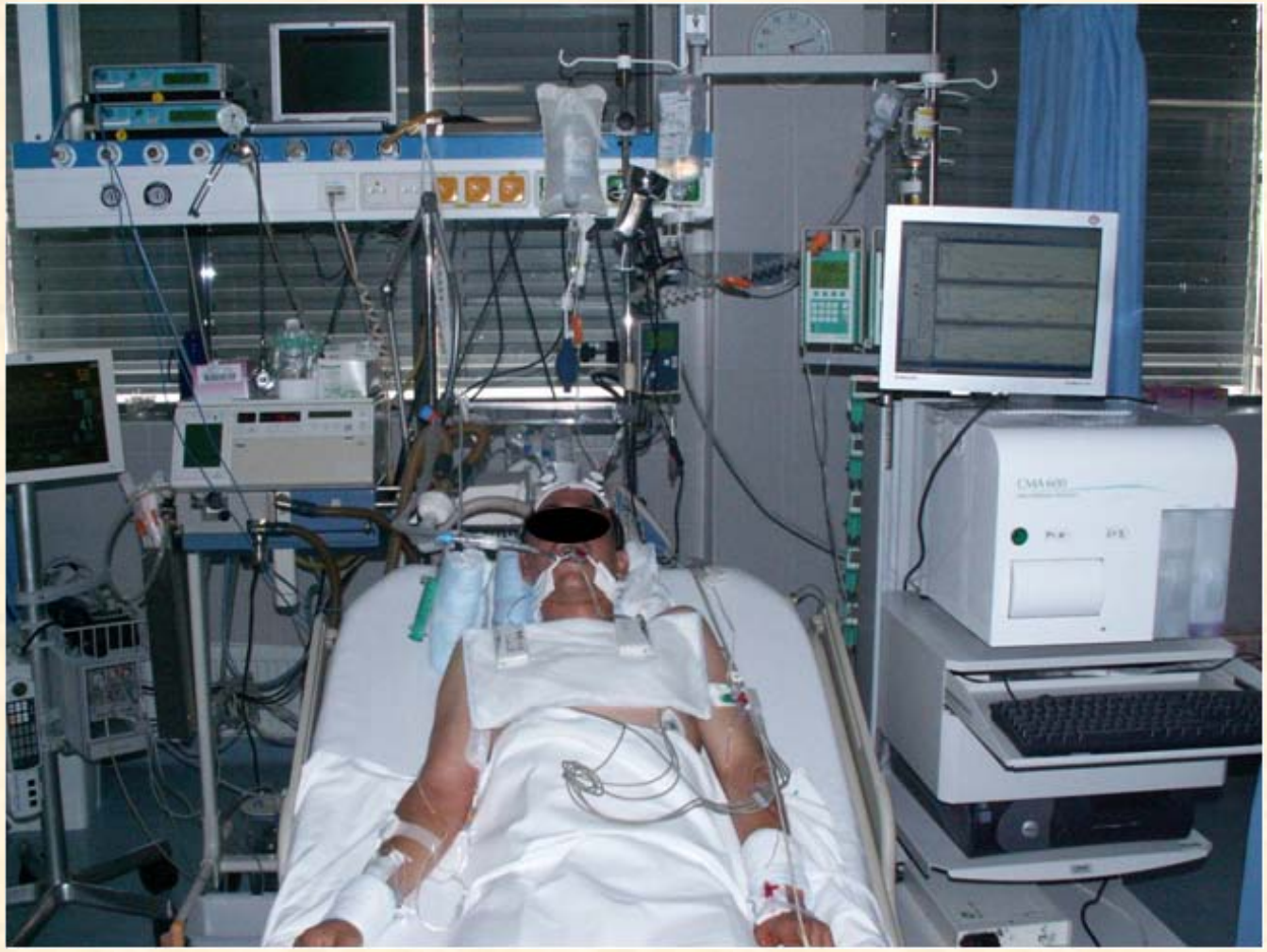
- bolt
- tunneling





Individualizing the CPP/ICP protocol based on tissue oxymetry in patients with TBI

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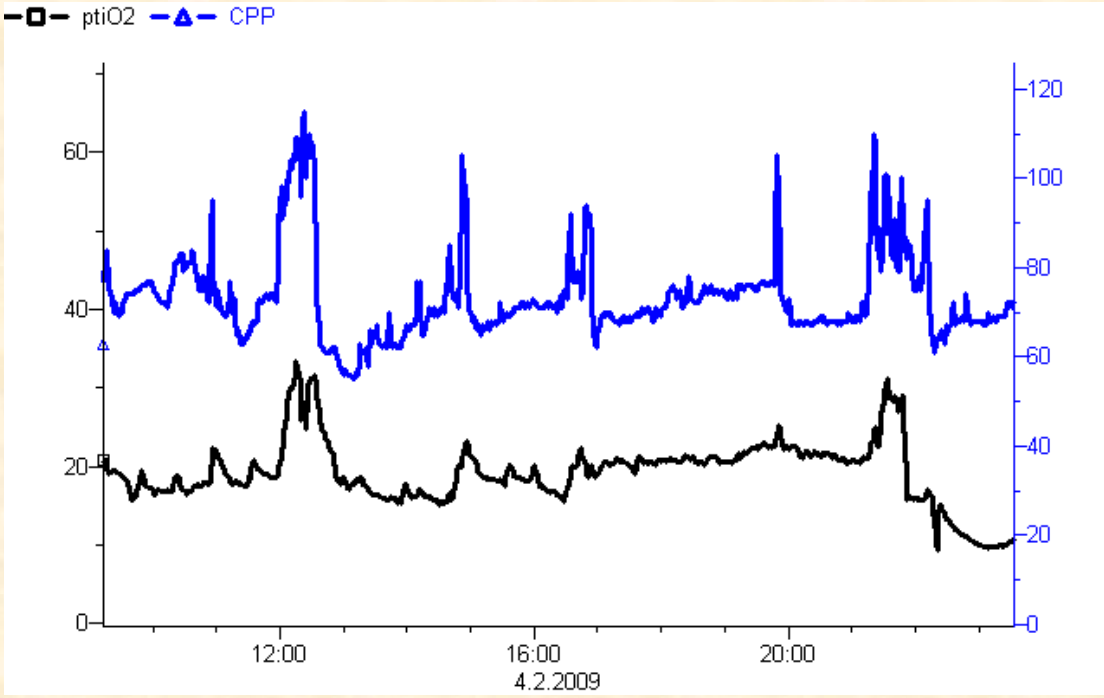
Does induced hypertension reduce cerebral ischaemia within the traumatized human brain? Coles, Brain 2004

Yes, in some patients it does. We need to identify the subset of patients.

„In pathological situations, the relationship between $PbtO_2$ and CPP may become linear, hence manipulating CPP to maintain $PbtO_2$ > 15–20 mmHg ($PbtO_2$-directed strategy) might optimize CBF and avoid secondary ischemia.“ *Bouzat 2013*



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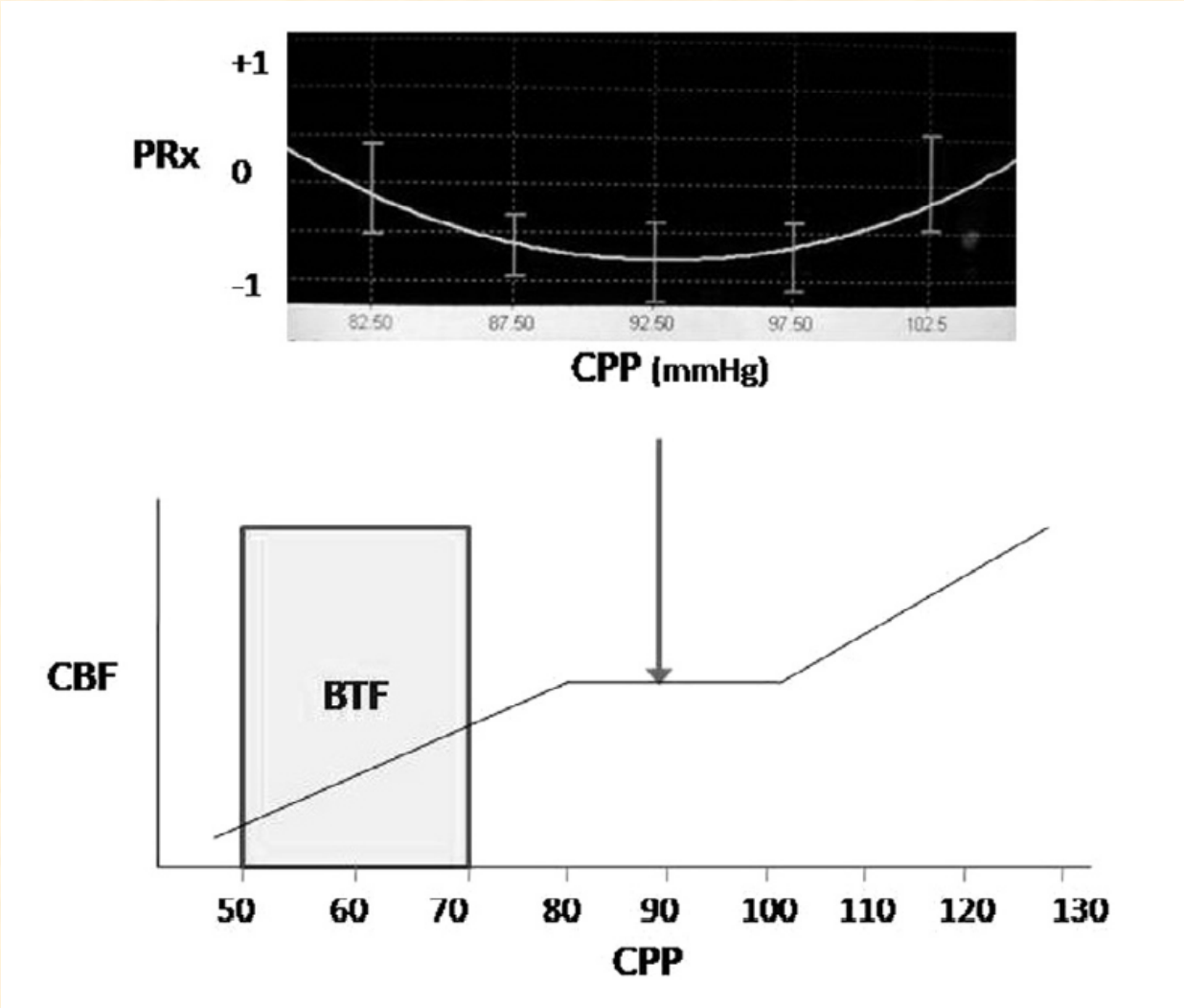
CPP based on PRx



Makarenko et al. 2015

Brain tissue oxymetry and metabolism can modify our therapeutic approach

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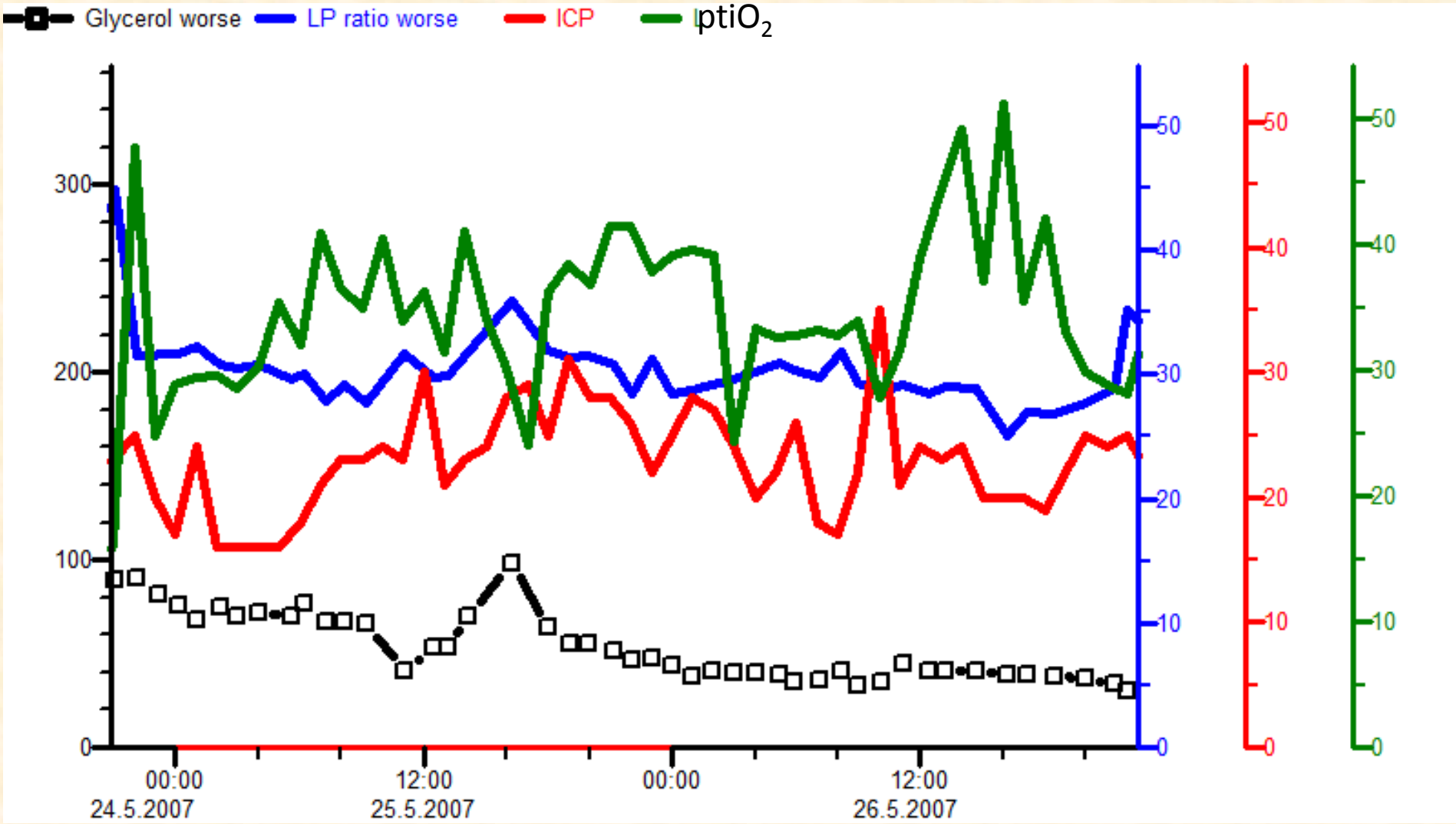

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Is decompressive craniectomy still worthwhile?

Unfavorable outcome:

51% in standard care vs.
70% in the DC group

- Selection of patients
- Indication criteria
- Timing

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 APRIL 21, 2011 VOL. 364 NO. 16

Decompressive Craniectomy in Diffuse Traumatic Brain Injury

D. James Cooper, M.D., Jeffrey V. Rosenfeld, M.D., Lynnette Murray, B.App.Sci., Yaseen M. Arabi, M.D., Andrew R. Davies, M.B., B.S., Paul D'Urso, Ph.D., Thomas Kossmann, M.D., Jennie Ponsford, Ph.D., Ian Seppelt, M.B., B.S., Peter Reilly, M.D., and Rory Wolfe, Ph.D., for the DECRA Trial Investigators and the Australian and New Zealand Intensive Care Society Clinical Trials Group*

Tissue metabolism and DC

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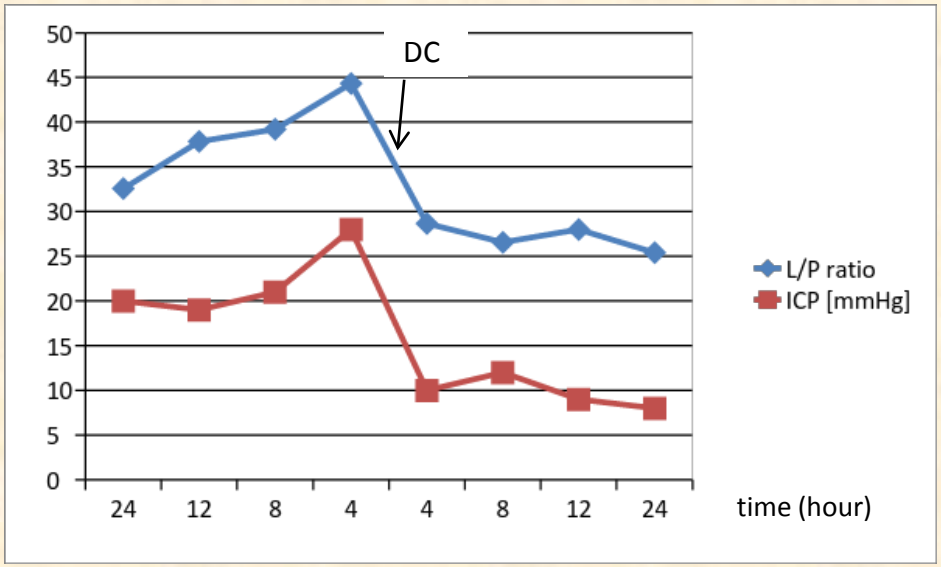
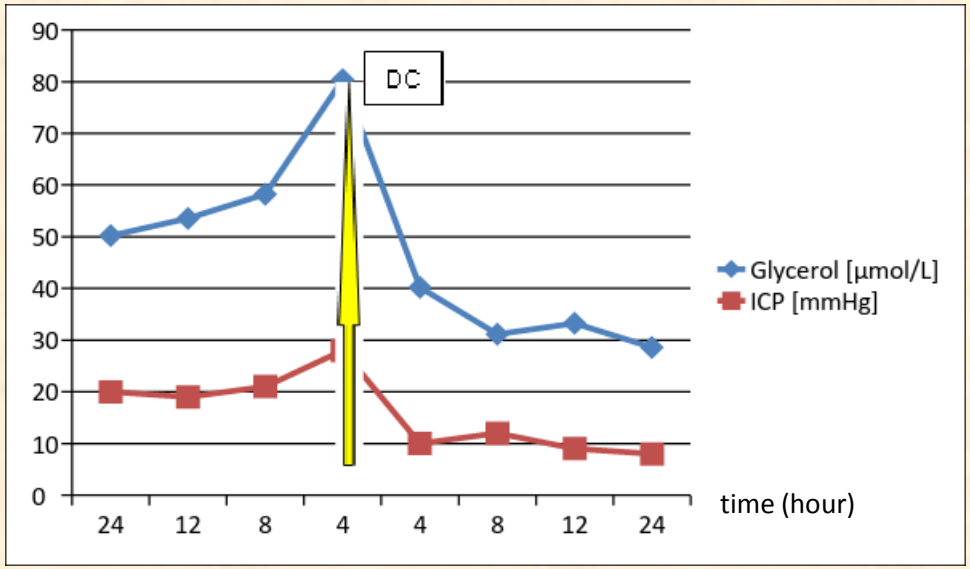
- Increased LP ratio and glycerol into pathological values may precede ICP elevation by 2-6 hours.
- Increased levels of the LP ratio and glycerol decrease back to normal values after decompressive craniectomy
- **Selective indication! Early timing!**





SAH due to aneurysm rupture

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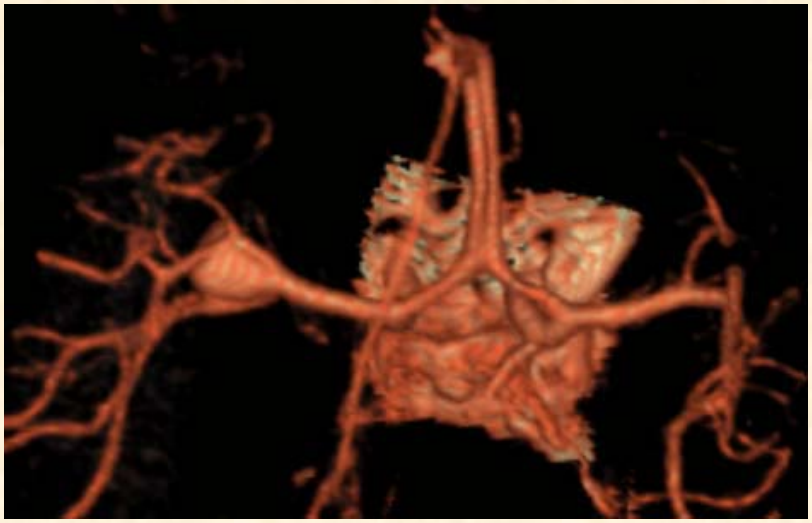
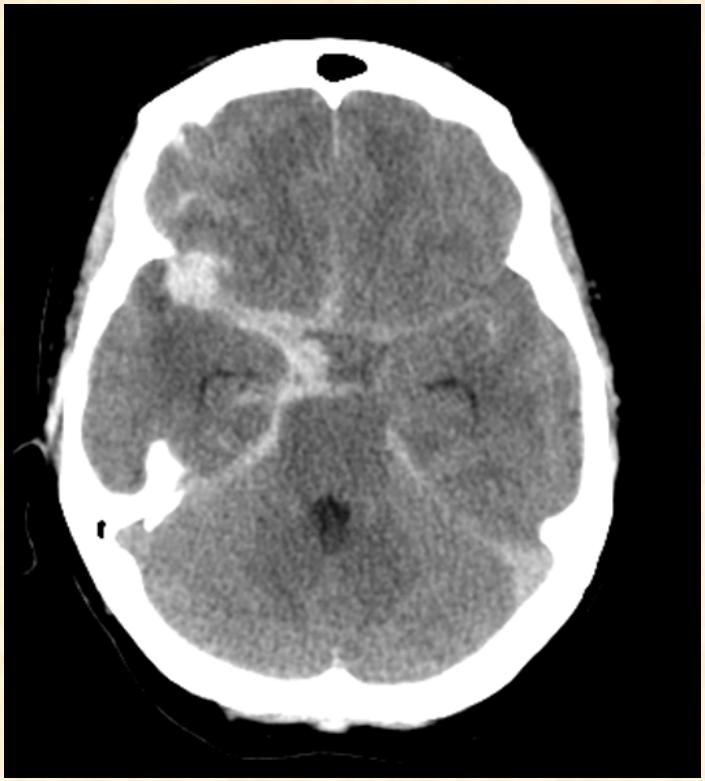
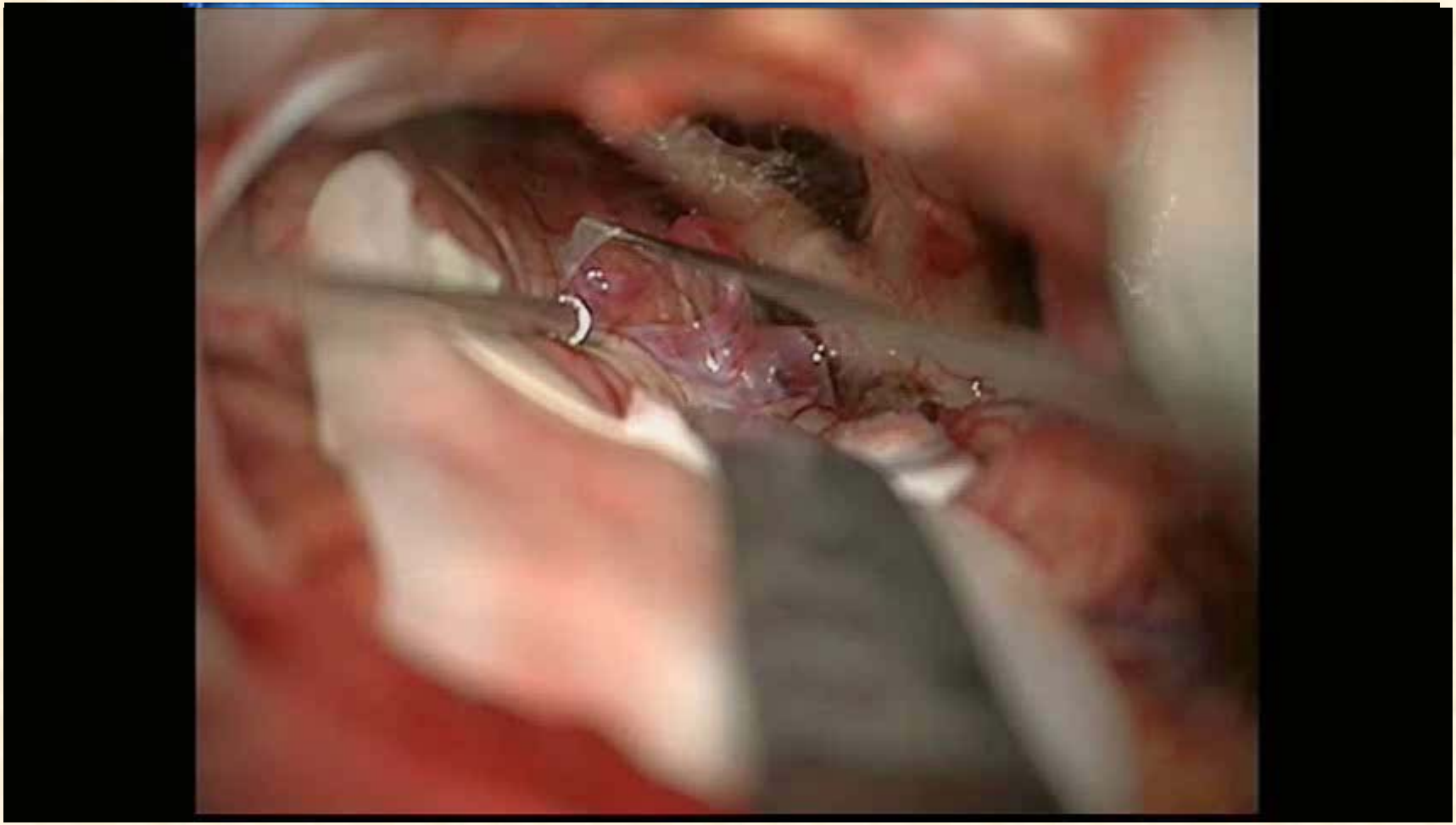




Clip or coil?.....*the trouble is someplace else*



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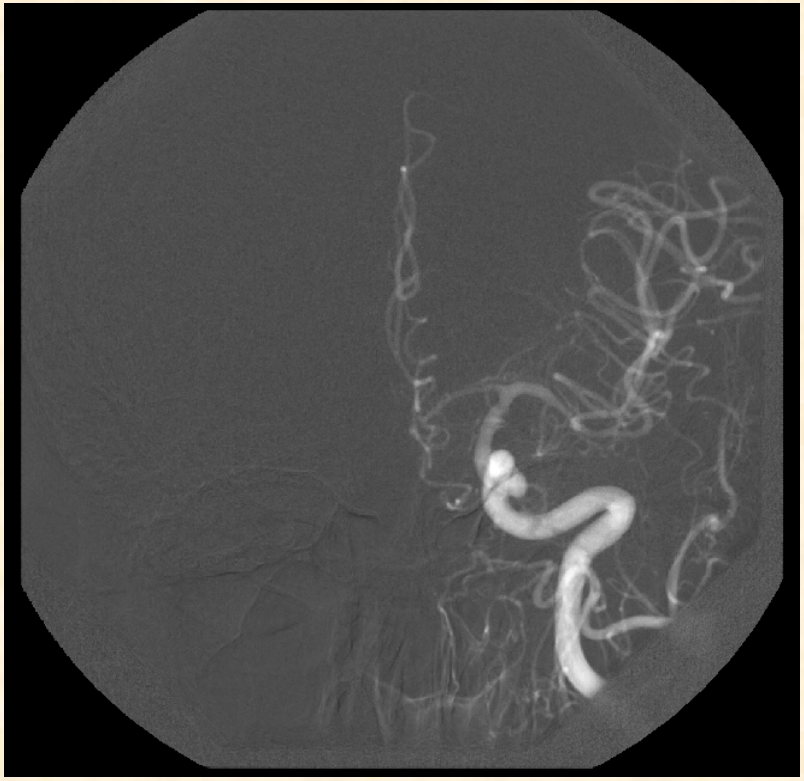
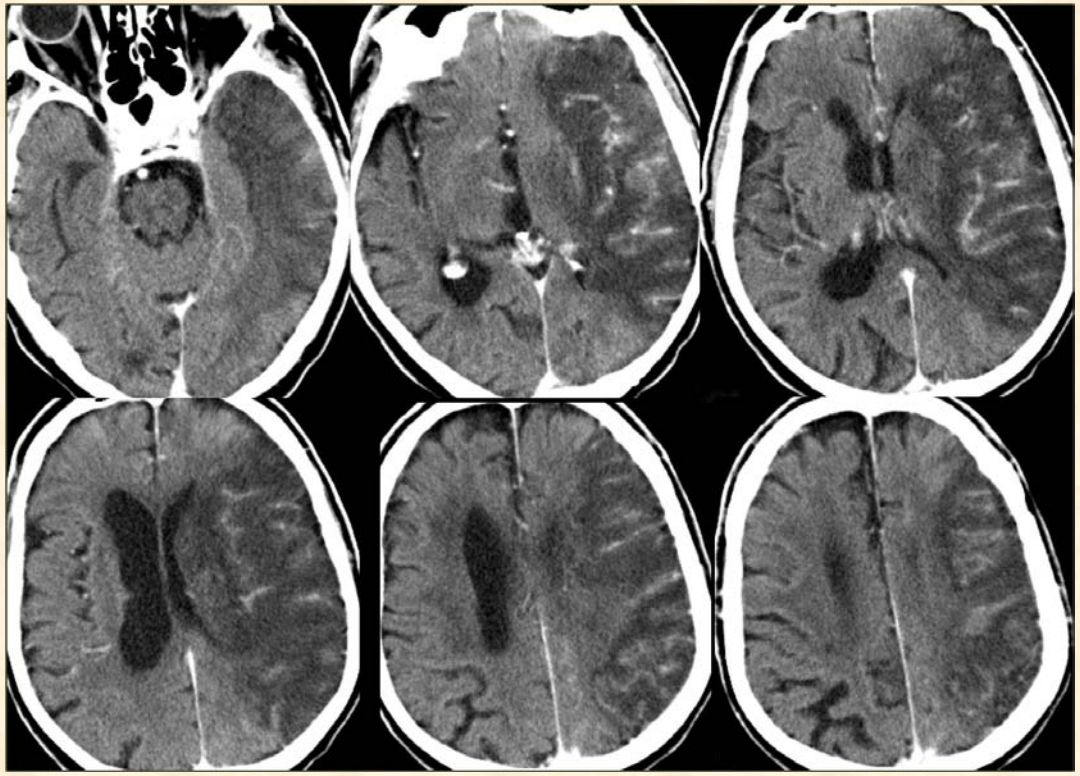
Delayed cerebral ischemia after SAH





vasospasm

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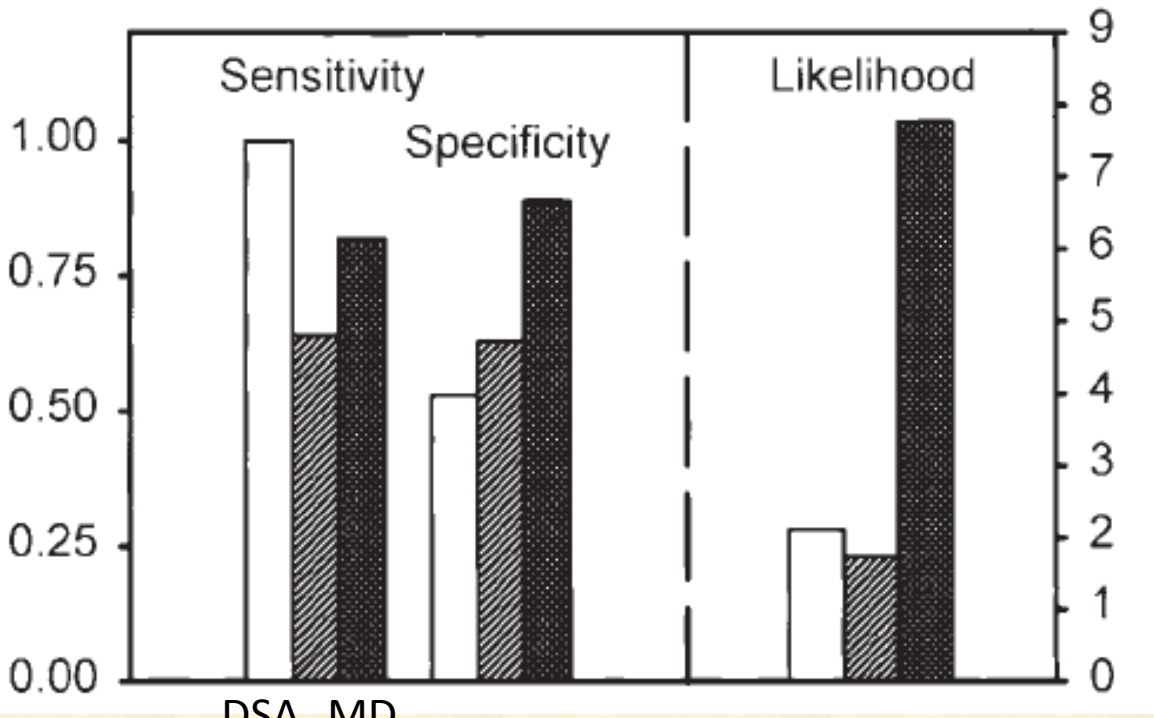
Brain metabolism monitoring and vasospasm after SAH



Unterberg et al. 2001

Increased LP a LG ratios preceed the development of DIND by more than 20 hours

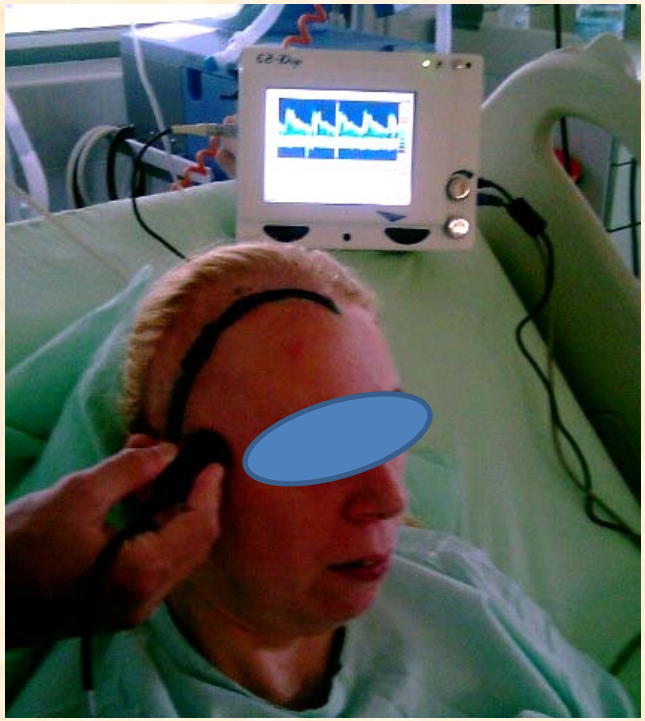
(Skjøth-Rasmussen et al. 2004)

2 attacks of aphasia and confusion due to cerebral vasospasm treated with spasmolytics

TCD (Type A, Class II)

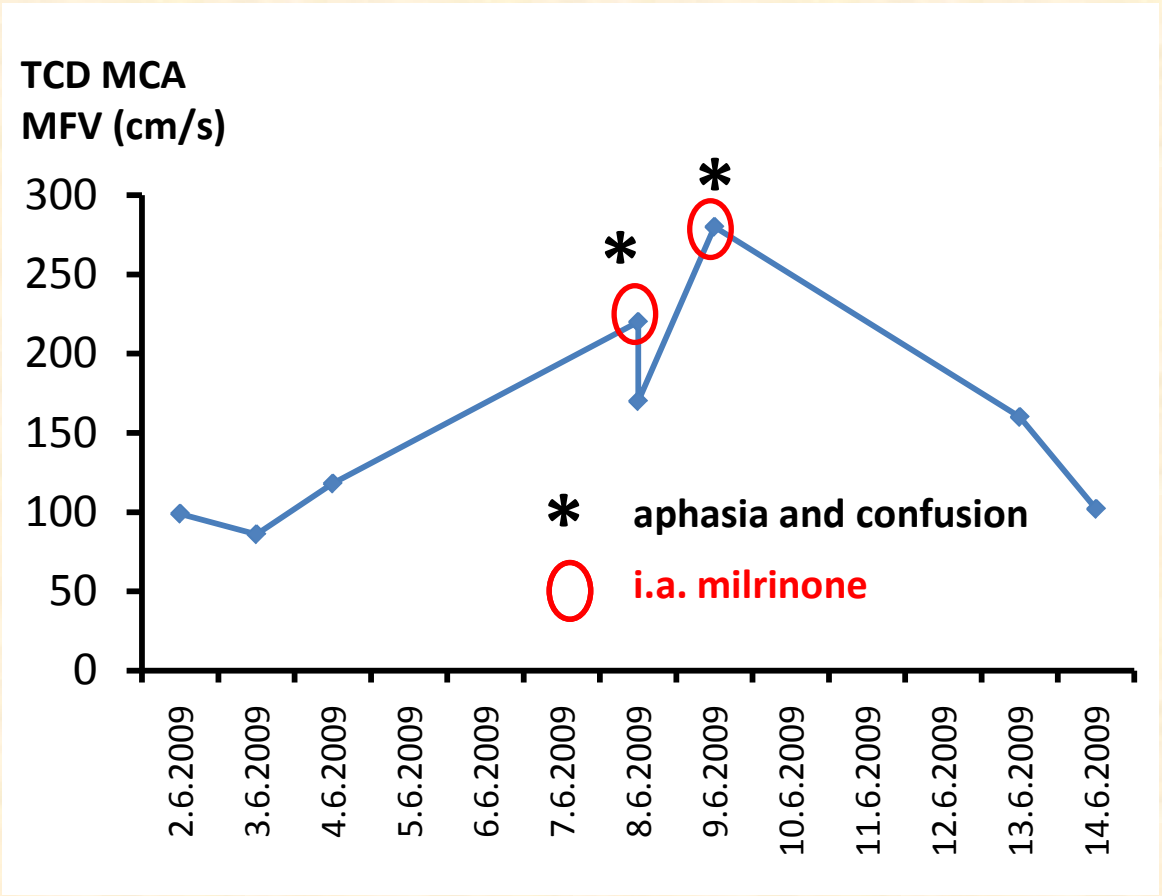
easy and noninvasive

bedside monitoring method



Mean difference (before-after i.a. spasmolytics) :

54cm/s



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Vasospasm treatment with spasmolytics

Clinical presentation	Number of patients	Positive response	No response	Worsening
Aphasia	7	7	0	0
Hemiparesis	5	3	1	1
Confusion	1	0	0	1
LOC	16	6	7(3)	0
TCD velocities	3	0	0	0
Admitted at VS	2	0	2	0

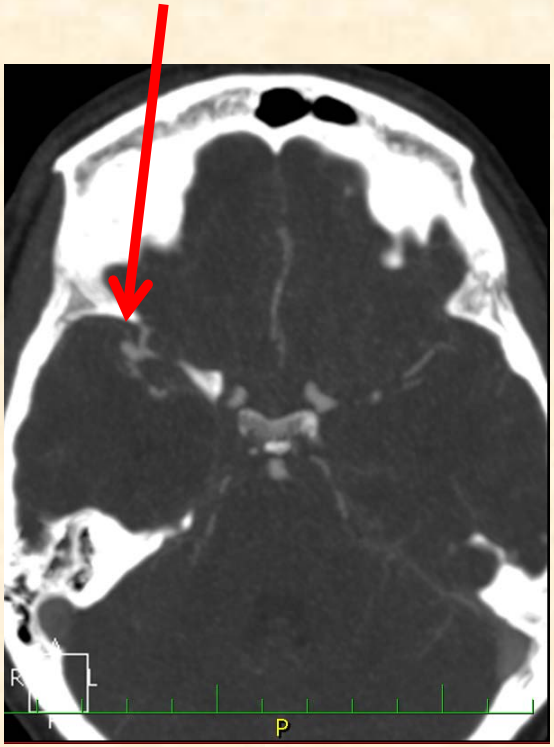
- Unsatisfactory outcome in patients with worsening of the level of consciousness

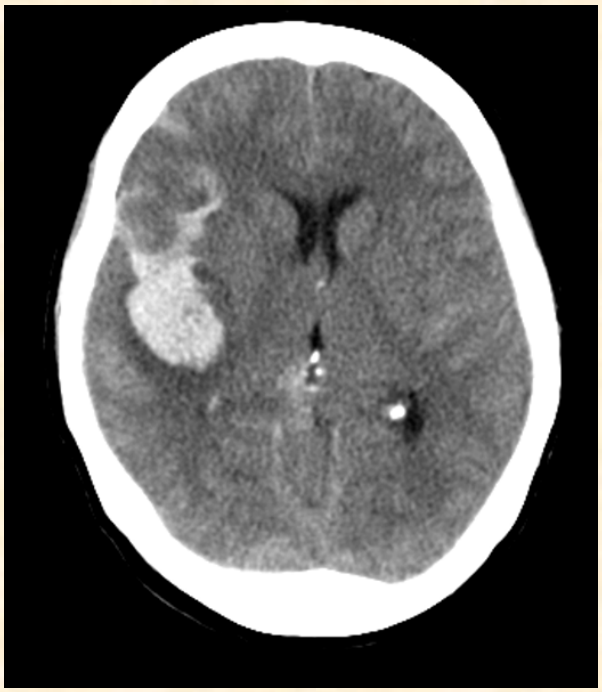
Brain metabolism monitoring in severe SAH patients

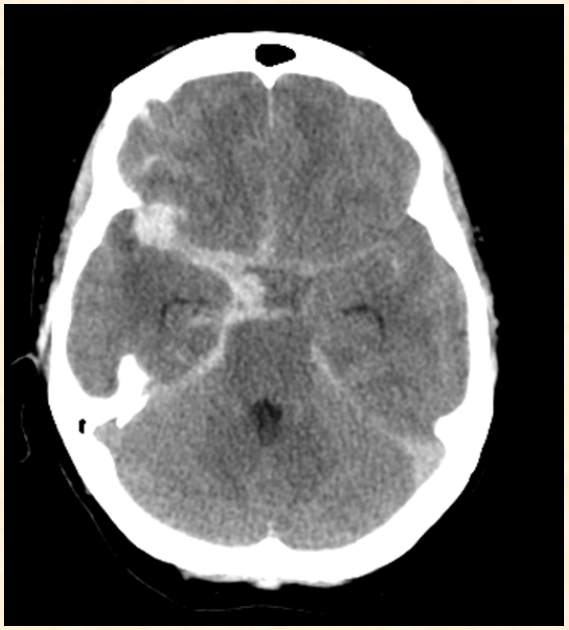
57 year old female

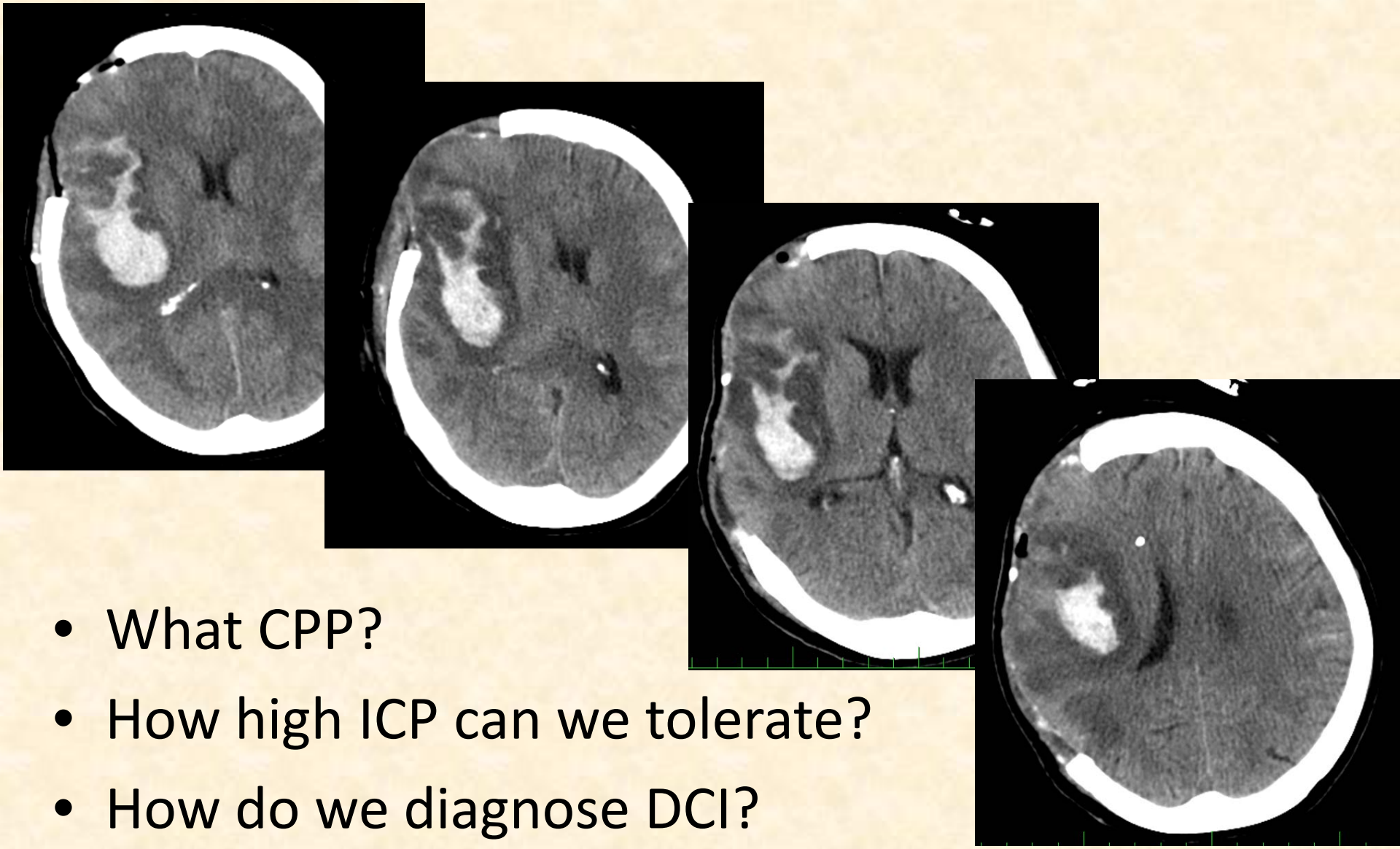
SAH, HH3, WFNS 3, Fisher 4

MCA aneurysm rupture









- What CPP?
- How high ICP can we tolerate?
- How do we diagnose DCI?

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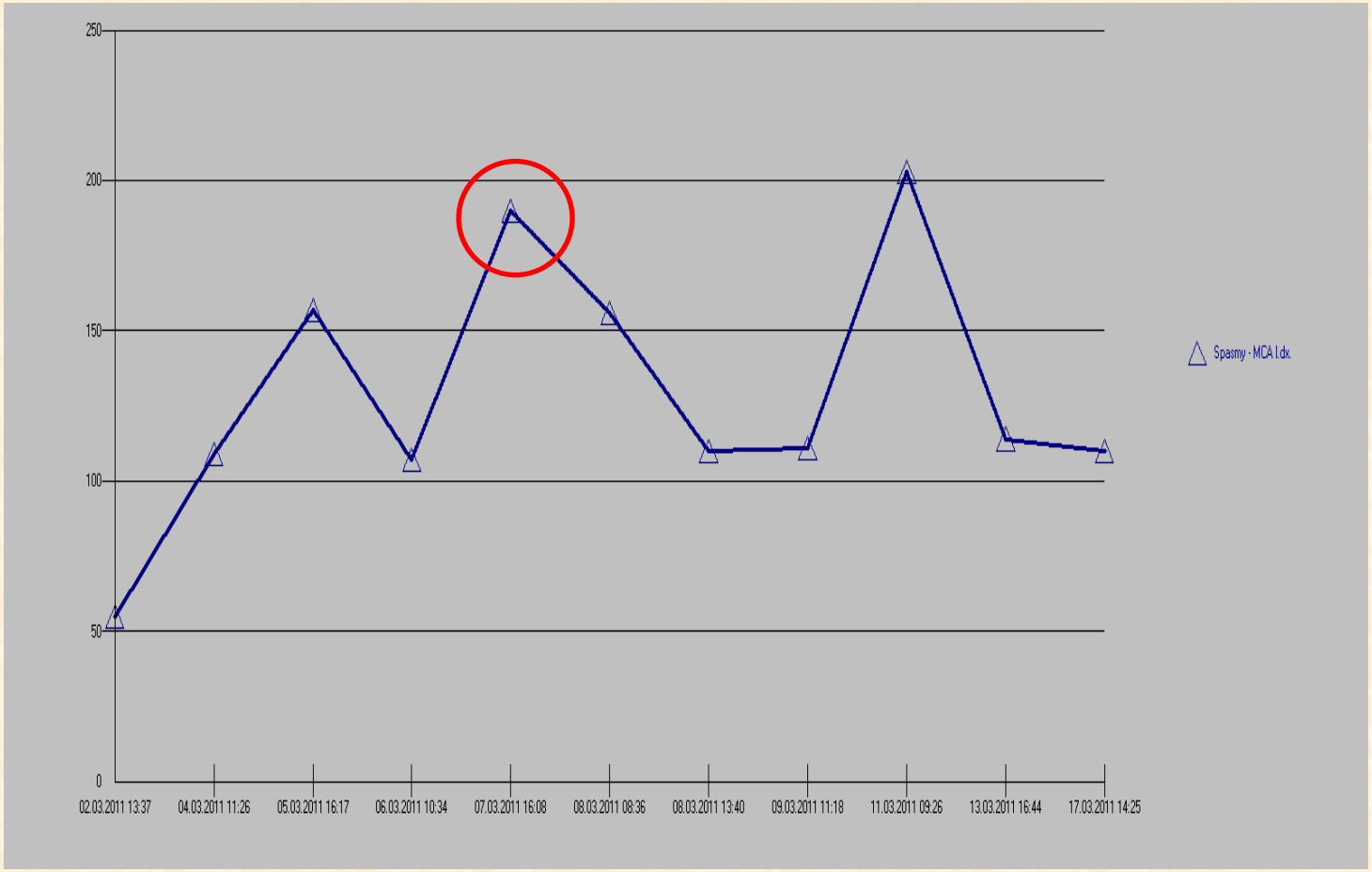
TCD monitoring for vasospasm



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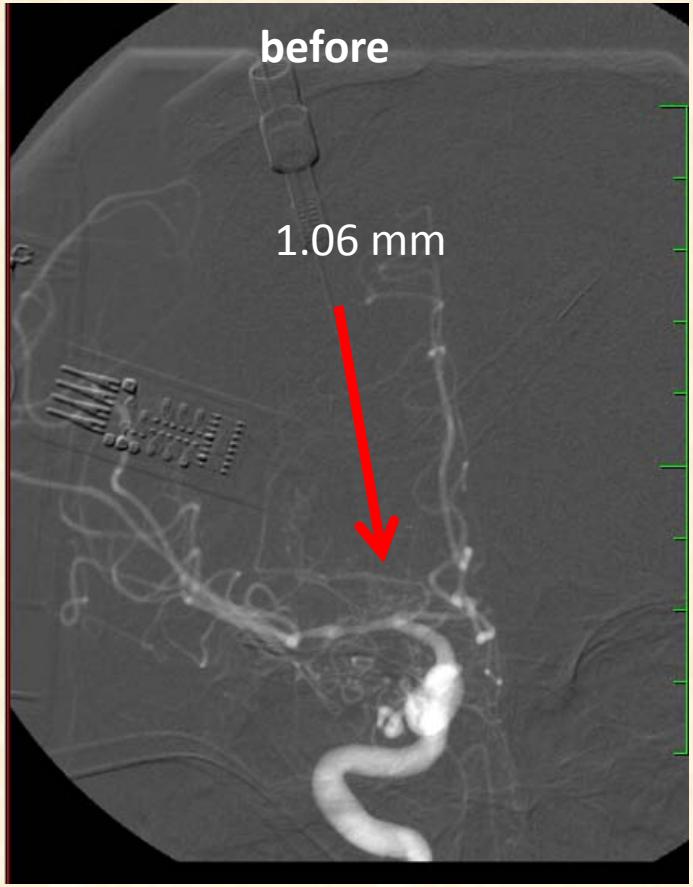
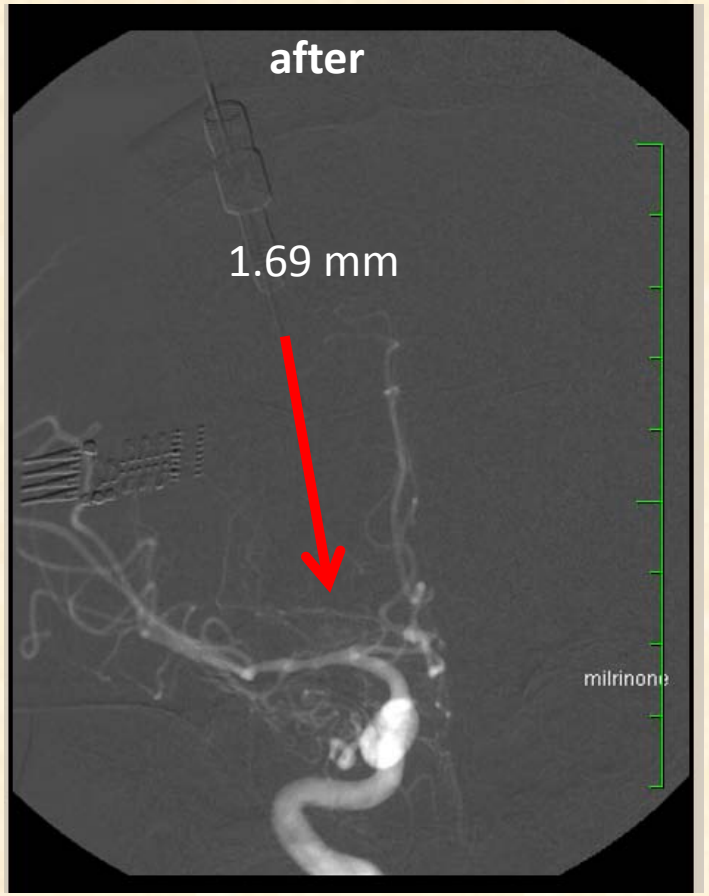
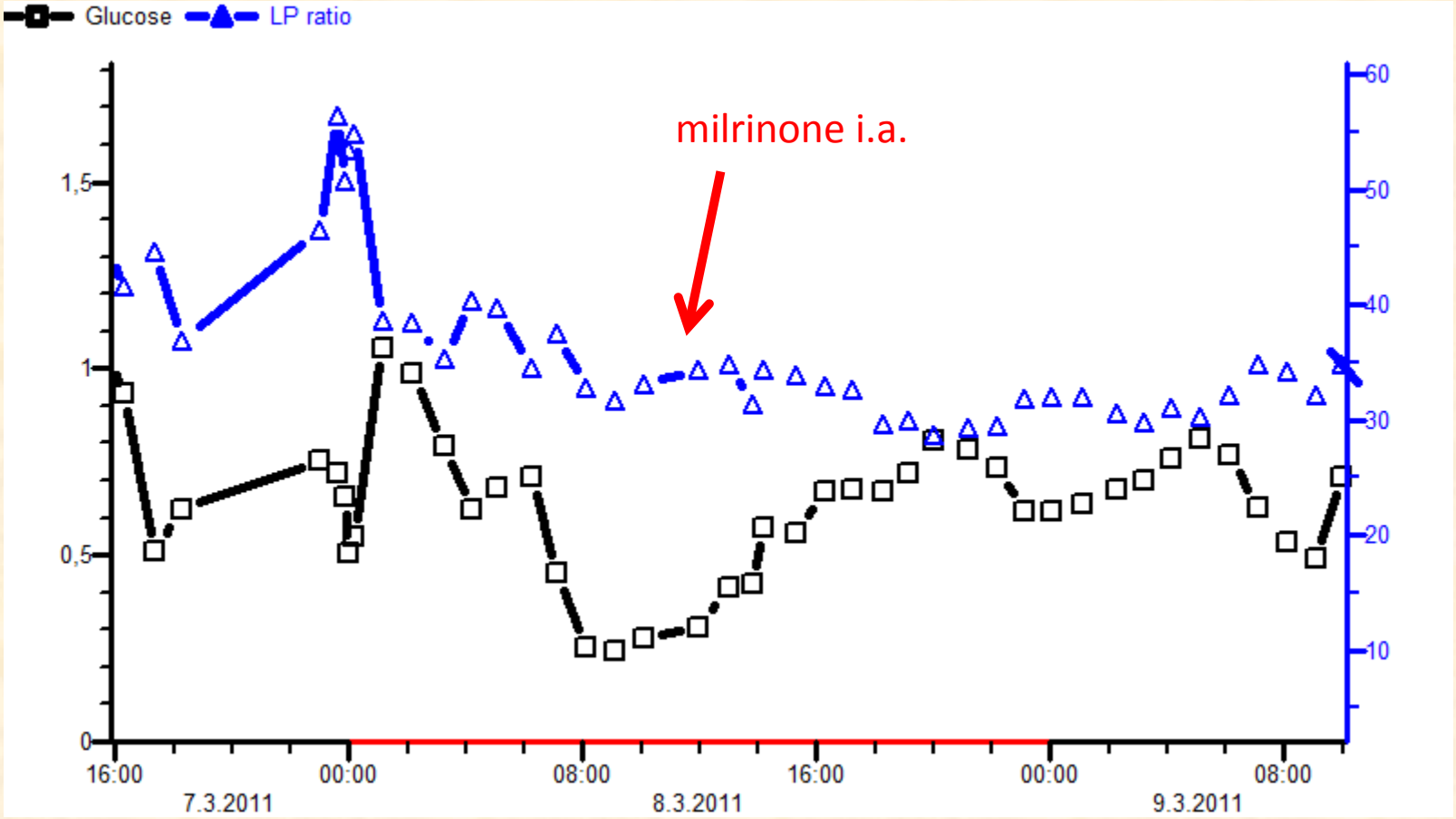
Intraarterial therapy of vasospasm using spasmolytics (8mg of milrinone)





Monitoring the therapy of vasospasm in sedated patients



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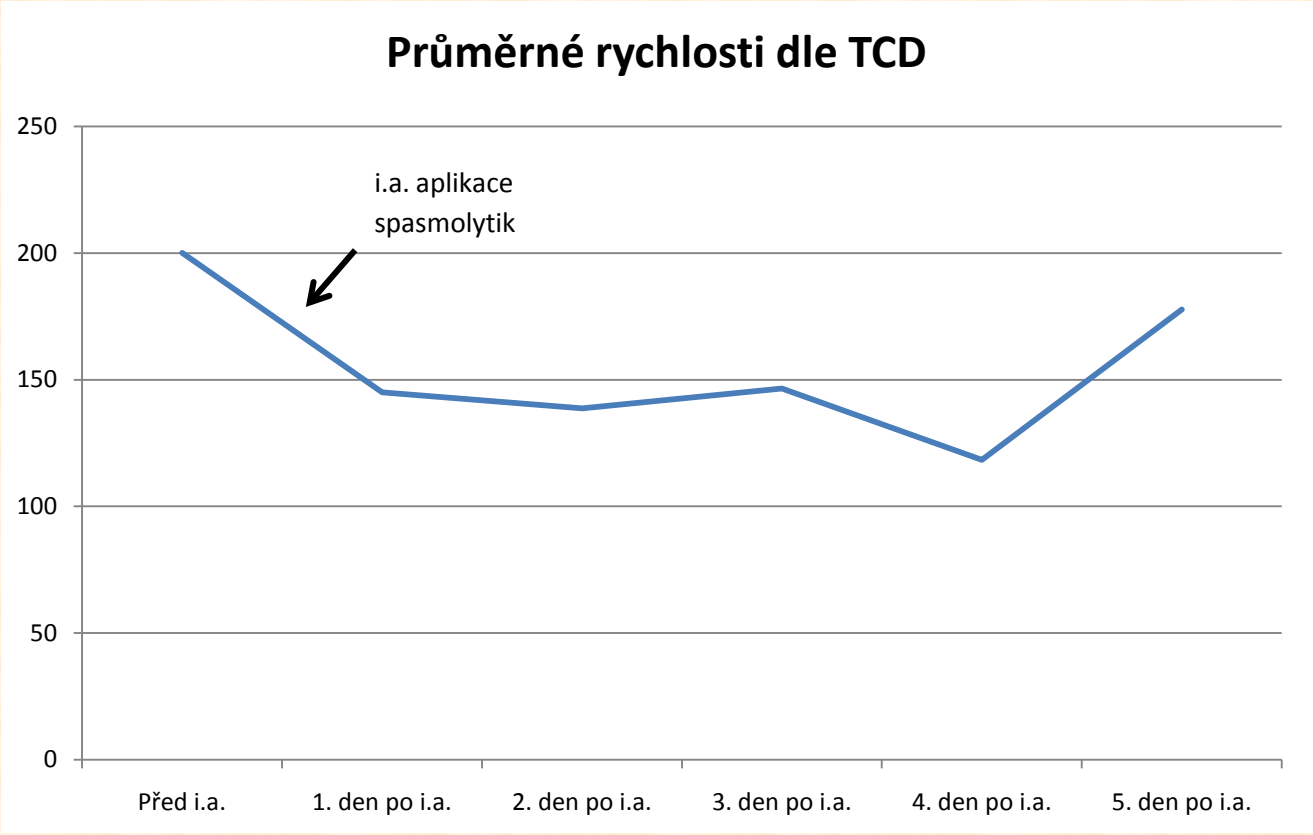
TCD monitoring for vasospasm



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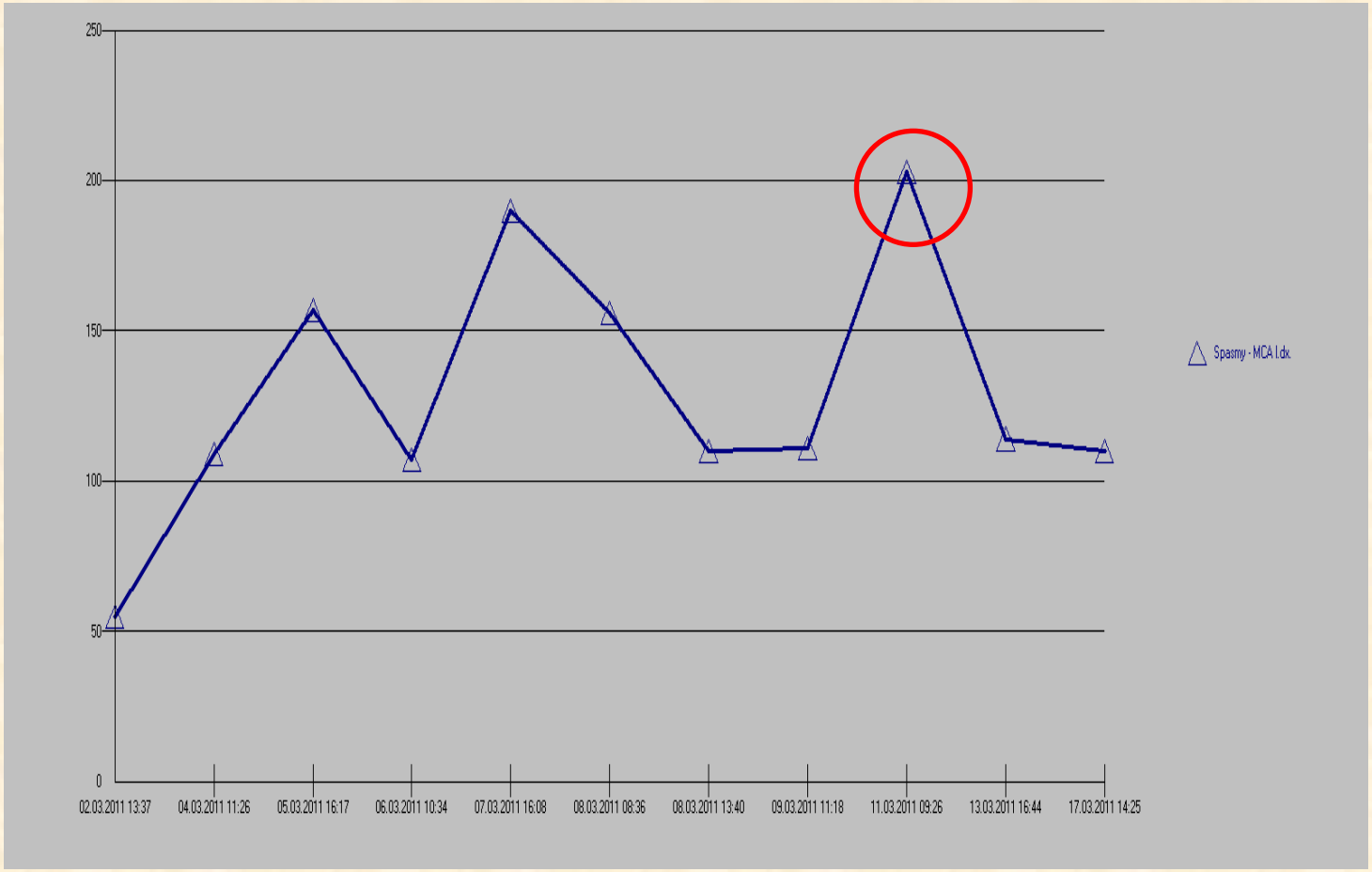

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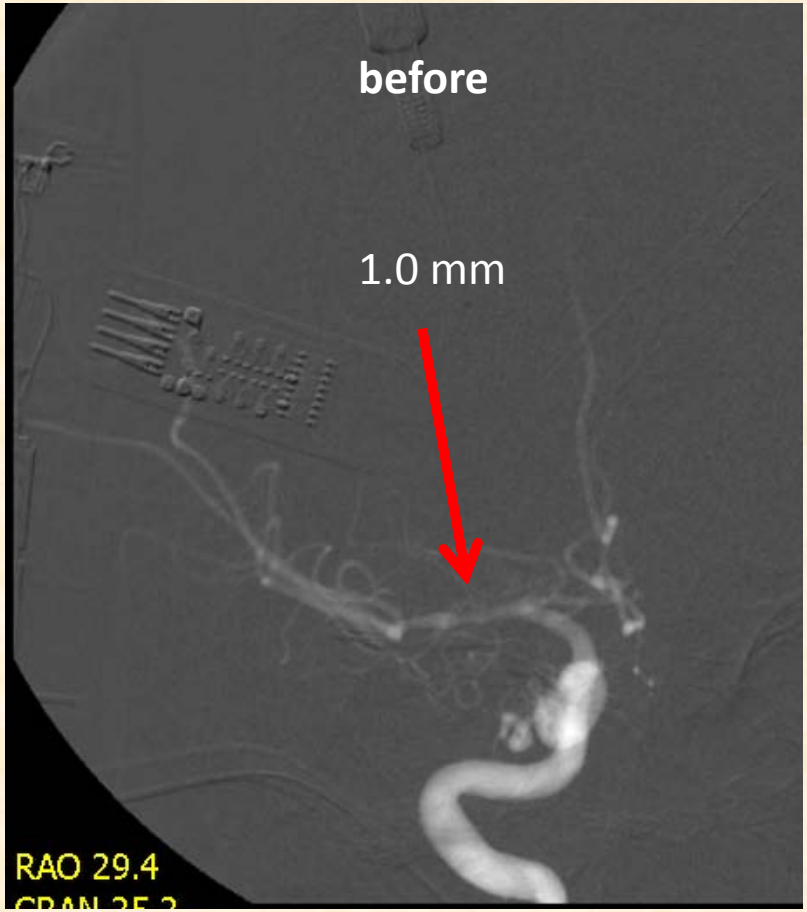
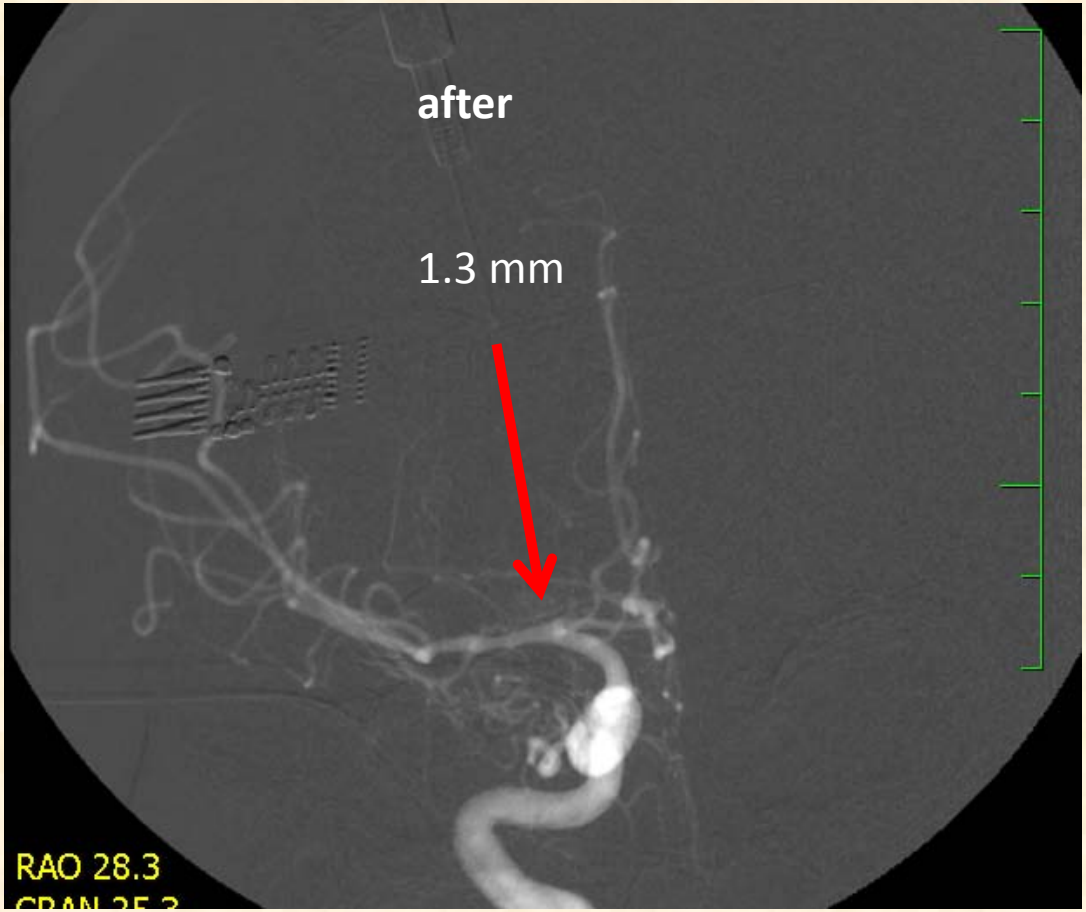
Repeated intraarterial therapy of vasospasm using spasmolytics (8mg of milrinone)





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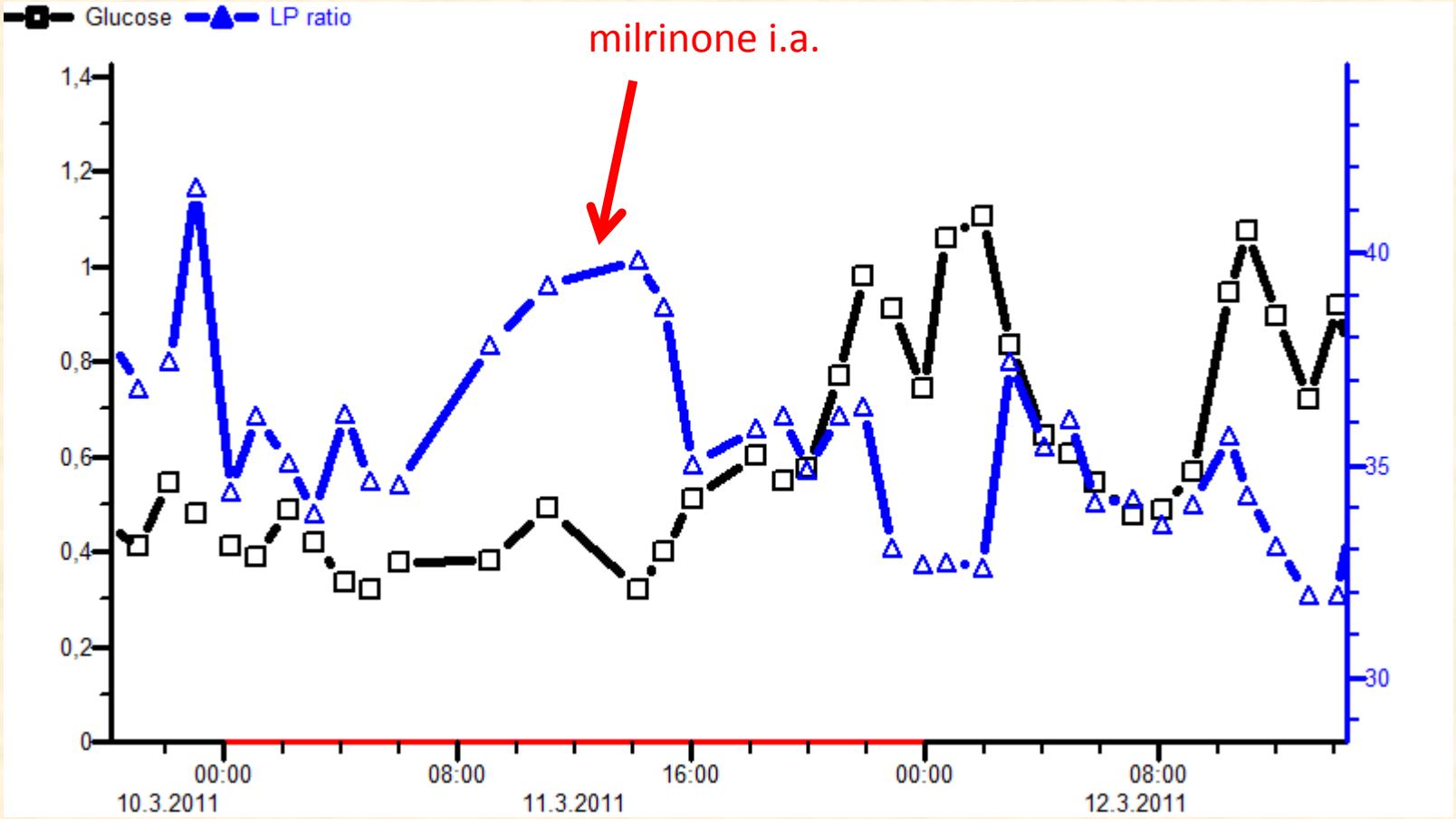

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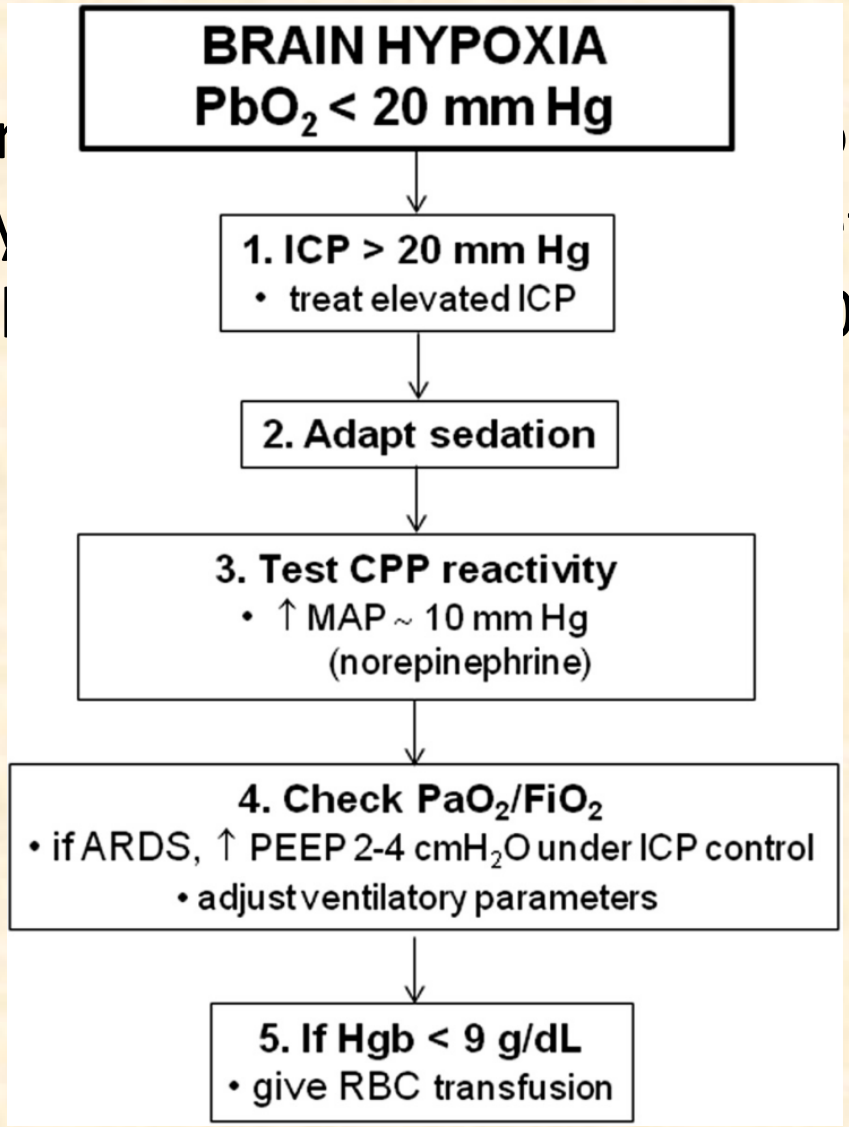
Practical management of low $PbtO_2$ in patients with severe TBI

Start inter … r $ptiO_2$ < 15 mmHg (hy … fore irreversibl … 07)



Bouzat, Ann Intensive Care, 2013

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Microdialysis “guidelines“

- Microdialysis consensus statement (Cambridge 2014):
  - Trends may be more important than absolute values
  - Low brain glucose – increase systemic glucose, in case of brain ischemia (high LP ratio) improve CPP
  - Level of importance: 1. glucose and LP ratio 2. glutamate 3. glycerol

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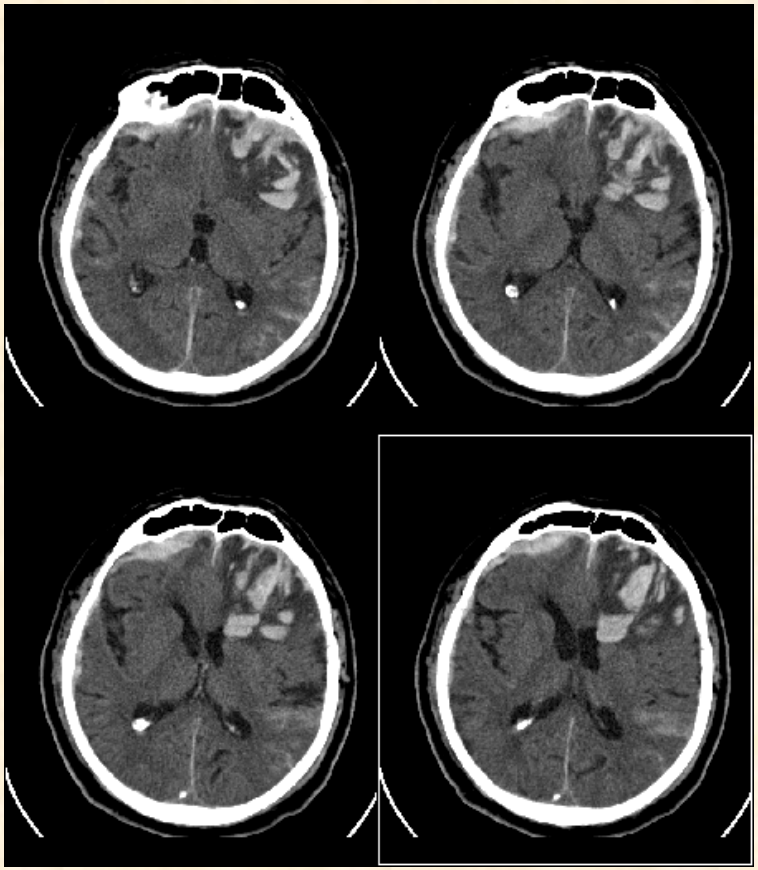
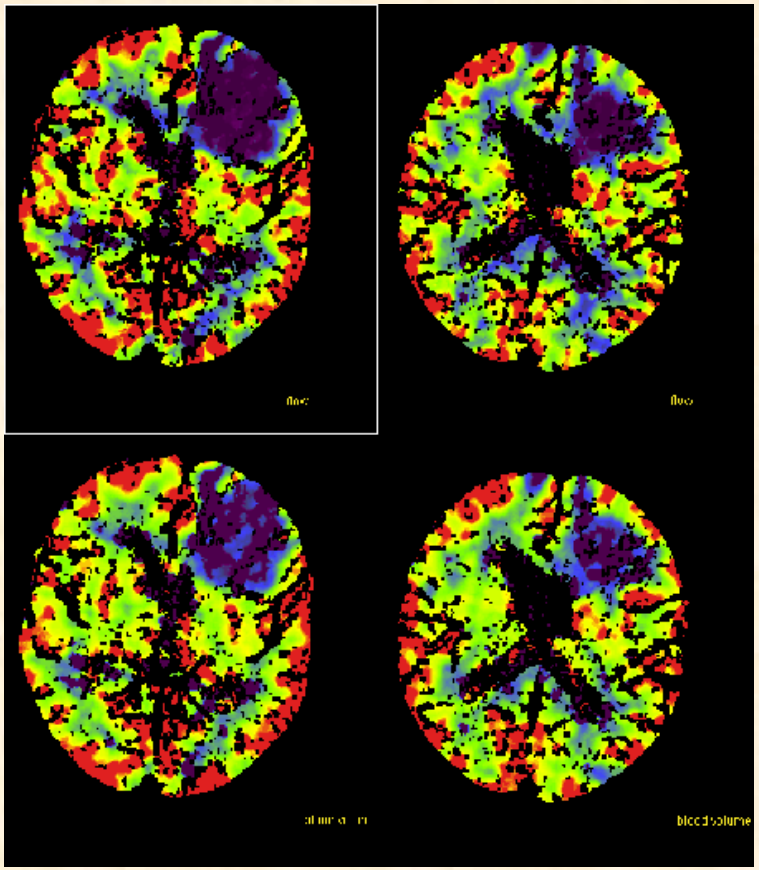
Bilateral brain monitoring

65 yo man, multiple trauma, GCS 8





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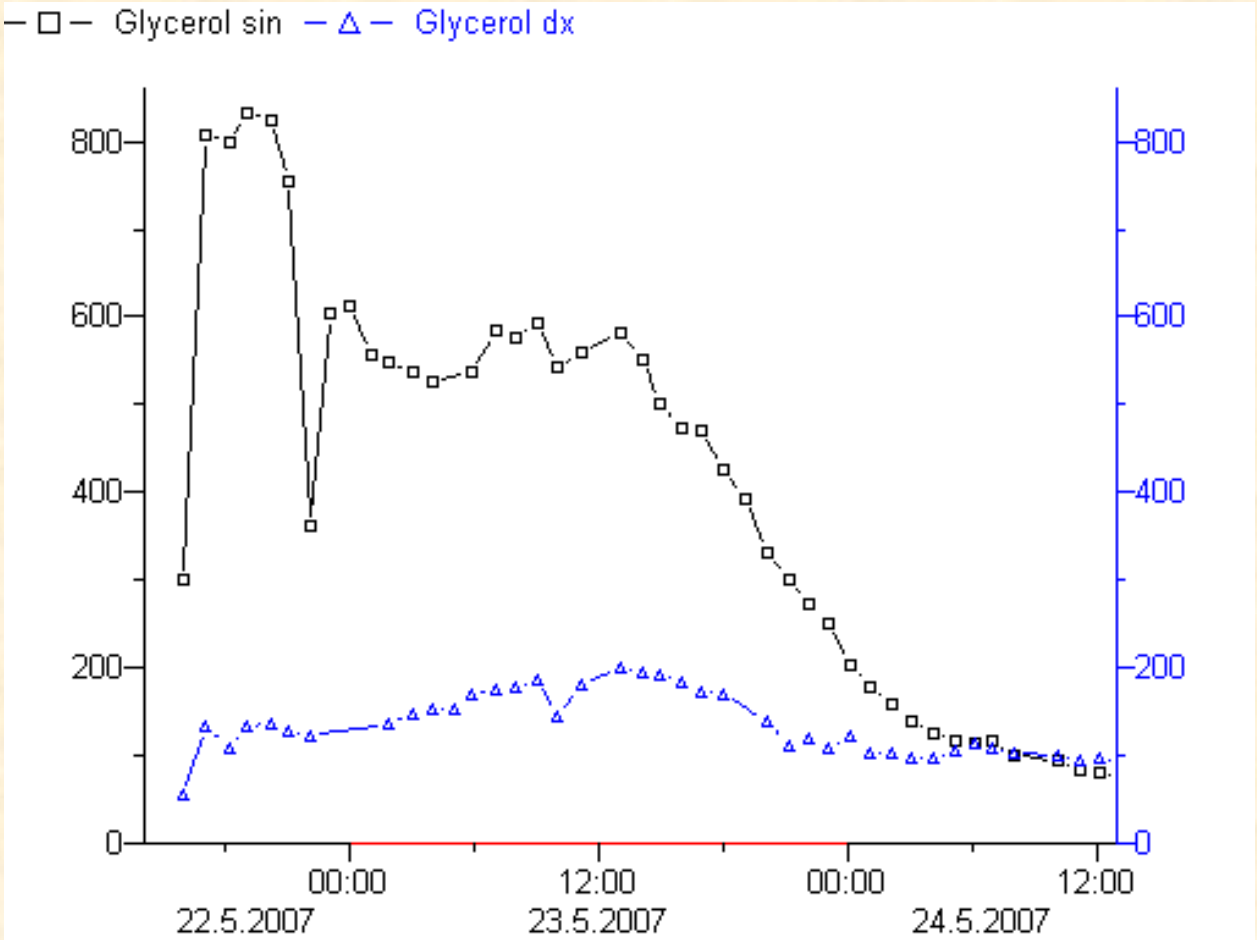
Glycerol during the initial days



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- Microdialysis consensus statement (Cambridge 2014):
  - Trends may be more important than absolute values
  - Low brain glucose – increase systemic glucose, in case of brain ischemia (high LP ratio) improve CPP
  - Level of importance: 1. glucose and LP ratio 2. glutamate 3. glycerol
  - Large molecules are studied with 100kDa catheters

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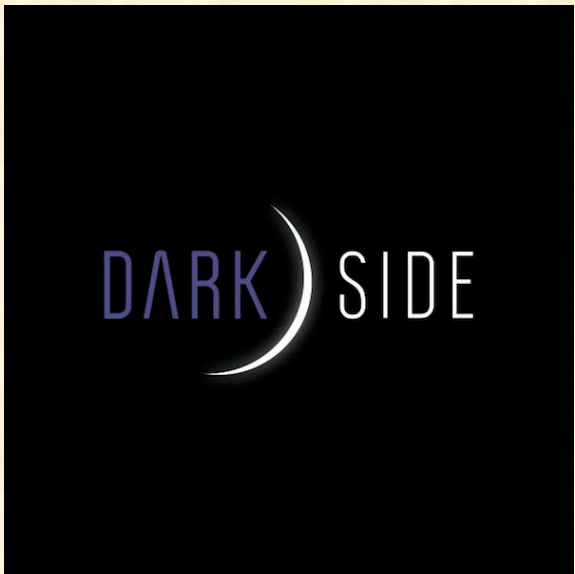
Limitations of MMM

- Data interpretation is sometimes difficult - a dedicated team is necessary (nurses)
- Application after worsening – often too late
- Technical limitations (microdialysis – „dry microvials“)



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Goal directed brain tissue oxygen monitoring versus conventional management in traumatic brain injury: an analysis of in hospital recovery.

Compared with ICP/CPP-directed therapy alone, the addition of $pBtO_2$ monitoring did not provide a survival or functional status improvement at discharge. The true clinical benefit of $pBtO_2$ monitoring will require further study.

Neurocrit Care. 2013 Feb;18(1):20-5.

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Conclusions

1. Multimodal monitoring provides evaluation of brain tissue physiology (blood flow, oxygenation, metabolism)

   in sedated/intubated patients, which may aid in the guidance of individualized monitored therapy in patients especially after TBI and SAH.

2. Despite the fact we are able to improve brain tissue oxygenation and metabolism with some current therapies, no proof of improved clinical outcome exists.

Spreading depolarization

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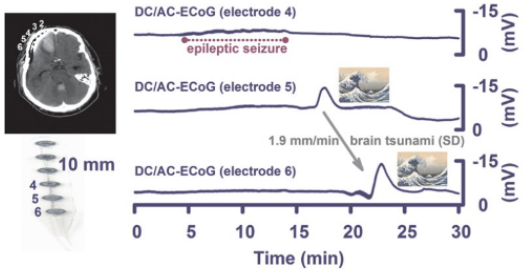
The Problem: Brain Tsunamis

In 1944, the Brazilian physiologist Aristides Leão made an unexpected discovery during his dissertation research at Harvard University. After intensely stimulating the brain's cerebral cortex, he observed a propagating wave of suppressed cortical function which he termed "spreading depression." Later work showed that these waves are massive short-circuits of the batteries that drive the brain's electrical activity. Since the normal polarity of these batteries is lost, these waves are called **spreading de-polarizations**. During a spreading depolarization, brain cells swell, cease electrical function, and enter a *twilight state of near-death*. Because of the profound perturbation, spreading depolarizations are nick-named **brain tsunamis**.



This figure illustrates the recording of spreading depolarizations in the human brain. Electrodes are placed on the surface of the brain after neurosurgical treatment of severe brain injury. Here, a large wave is recorded at electrode 5 and then spreads to electrode 6, moving through the brain at 2 mm/min. The waves are about 30 times the amplitude of normal brain activity and thus resemble 'tsunamis'. The relatively small scale of an epileptic seizure on electrode 4 can be seen for comparison.

Leão subsequently observed that similar changes occur after blocking blood flow to the brain, thus making the first connection between spreading depolarizations and brain injury. In the 1970's, researchers began to appreciate that these waves occur *spontaneously* in the brain after acute injury. By the 1990's, it was proven in animals that brain tsunamis **cause** the progressive death of brain tissue and expansion of injuries. Several studies showed that the number and duration of spreading depolarizations that occurred after stroke was a critical determinant of the size of the brain lesion that developed. However, convincing evidence that spreading depolarizations were relevant to human disease was still lacking. See History for how the story continues today.

Thank you for your attention!!

Indication criteria for MMM

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GCS < 9

trauma, SAH (ruptured AN)

CT pathology (contusion/multiple contusions, SAH with hematoma)

Trauma: conservative, after surgery (aSDH evacuation, DC)

SAH: usually HH3 or more, residual hematoma, edema, ischemia – prolonged ventilation