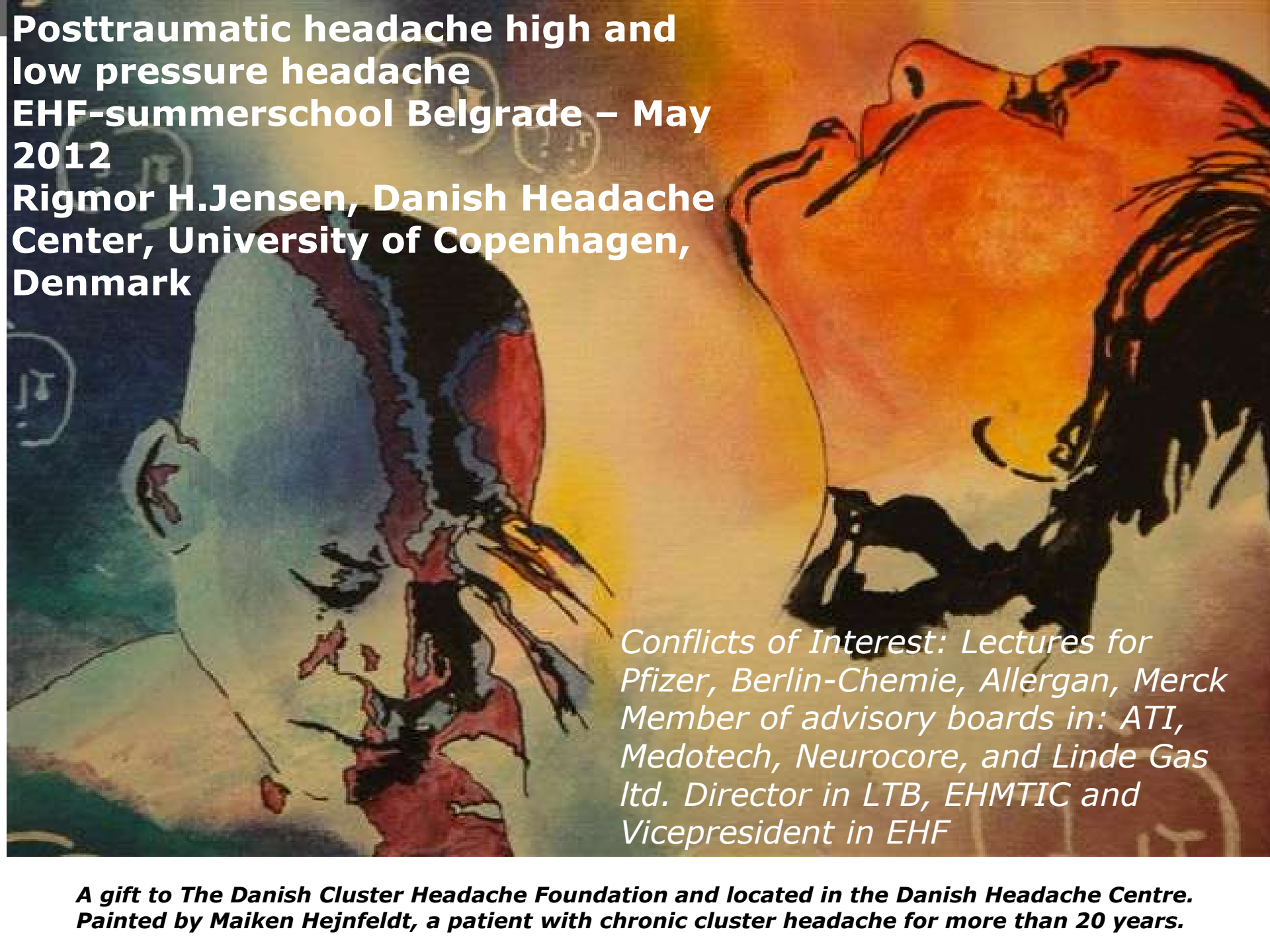


**Posttraumatic headache high and  
low pressure headache  
EHF-summer school Belgrade – May  
2012  
Rigmor H.Jensen, Danish Headache  
Center, University of Copenhagen,  
Denmark**



*Conflicts of Interest: Lectures for  
Pfizer, Berlin-Chemie, Allergan, Merck  
Member of advisory boards in: ATI,  
Medotech, Neurocore, and Linde Gas  
Ltd. Director in LTB, EHMTIC and  
Vicepresident in EHF*

***A gift to The Danish Cluster Headache Foundation and located in the Danish Headache Centre.  
Painted by Maiken Hejnfelddt, a patient with chronic cluster headache for more than 20 years.***



# Postramatic headache

## Chapter 5: Headache attributed to Head and/or Neck Trauma

- **5.1 Acute headache attributed to head injury**
- 5.1.1 Acute post-traumatic headache attributed to moderate or severe head injury
- 5.1.2 Acute post-traumatic headache attributed to mild head injury
- **5.2 Chronic headache attributed to head injury**
- 5.2.1 Chronic post-traumatic headache attributed to moderate or severe head injury
- 5.2.2 Chronic post-traumatic headache attributed to mild head injury
- **5.3 Acute headache attributed to whiplash injury**
- **5.4 Chronic headache attributed to whiplash injury**
- **5.5 Acute headache attributed to other head and/or neck trauma**
- **5.6 Chronic headache attributed to other head and/or neck trauma**
- **5.7 Acute headache attributed to craniotomy**
- **5.8 Chronic headache attributed to craniotomy**



## 5.1.2 Acute post-traumatic headache attributed to mild head injury

- Headache of any type, fulfilling criteria C and D
- Head trauma fulfilling criteria a and b:
  - None of the following:
    - Loss of consciousness >30 minutes duration
    - Glasgow Coma Scale (GCS) <13
    - Post-traumatic amnesia >24 hours in duration
    - Altered level of awareness >24 hours in duration
  - All of the following:
    - Symptoms and/or signs diagnostic of mild traumatic brain injury, manifest by ≥1 of the following immediately following the head injury:
      - > Transient confusion, disorientation, or impaired consciousness
    - Normal brain imaging (if performed)
      - > Loss of memory for events immediately before or after injury
      - > Other neurologic deficits such as focal weakness, numbness, ataxia, dysphasia
- Evidence of causation shown by:
  - Headache is reported to have developed within 7 days after:
    - head trauma
    - or after discontinuation of medications that impair the ability of the patient to report or sense headache following head trauma
- One or other of the following:
  - Headache resolves within 3 months after head trauma
  - Headache persists but 3 months have not yet passed since head trauma
- Headache is not better accounted for by another headache diagnosis



## 5.2.2 Chronic post-traumatic headache attributed to mild head injury

- Headache of any type, fulfilling criteria C and D
- Head trauma that meets criteria of a and b:
  - None of the following:
    - Loss of consciousness of >30 minutes duration
    - Glasgow Coma Scale (GCS) <13
    - Post-traumatic amnesia >24 hours in duration
    - Altered level of awareness >24 hours in duration
  - All of the following:
    - Normal brain imaging (if performed)
    - Symptoms and/or signs diagnostic of mild traumatic brain injury, manifest by  $\geq 1$  of the following immediately following the head injury:
      - > Transient confusion, disorientation, or impaired consciousness
      - > Loss of memory for events immediately before or after injury
      - > Other neurologic deficits such as focal weakness, numbness, ataxia, dysphasia<sup>[k1]</sup>
- Evidence of causation shown by:
  - Headache is reported to have developed within 7 days after:
    - head trauma
    - or after regaining consciousness following head trauma
    - or after discontinuation of medications that impair the ability of the patient to report or sense headache following head trauma
- Headache persists for >3 months after head trauma
- Headache is not better accounted for by another headache diagnosis



## Postramatic headache

- Prevalence and incidence?
- In US an incidence of 1.7 mio-3.8 mio TBI /Year
- Acute PTH affect 56-90% of all TBI, but time dependent and most pronounced in arly days after trauma
- Chronic PTH affect 10-44% with increased risk in females, young age, low education, and poor self rated health. Up to 20% of those may have CPTH after 4 years.
- Mechanisms: ?
- Treatment: ?



## Symptoms and Signs of PTH

- Diffuse dull headache, increase in intensity with physical activity
- Photophobia
- Phonophobia
- Concentrations problems
- Memory problems
- Tiredness
- Anxiety/Depression?
- Medication Overuse?



## Key points

- Traumatic axonal injury (TAI) after traumatic brain injury (TBI) is difficult to identify using conventional CT or MRI.
- Susceptibility-weighted imaging (SWI) is highly sensitive to the presence of microbleeds, which are a marker of TAI.
- Diffusion tensor imaging (DTI) provides quantitative information about white matter structure.
- DTI is abnormal after mild TBI (including blast injury) and changes dynamically in line with underlying pathological processes.
- DTI abnormalities correlate with the severity of cognitive and neuropsychiatric impairments, and are able to partly predict clinical outcome after mild TBI.

Investigating white matter injury after mild traumatic brain injury.

Sharp, David; Ham, Timothy

Current Opinion in Neurology. 24(6):558-563, December 2011.

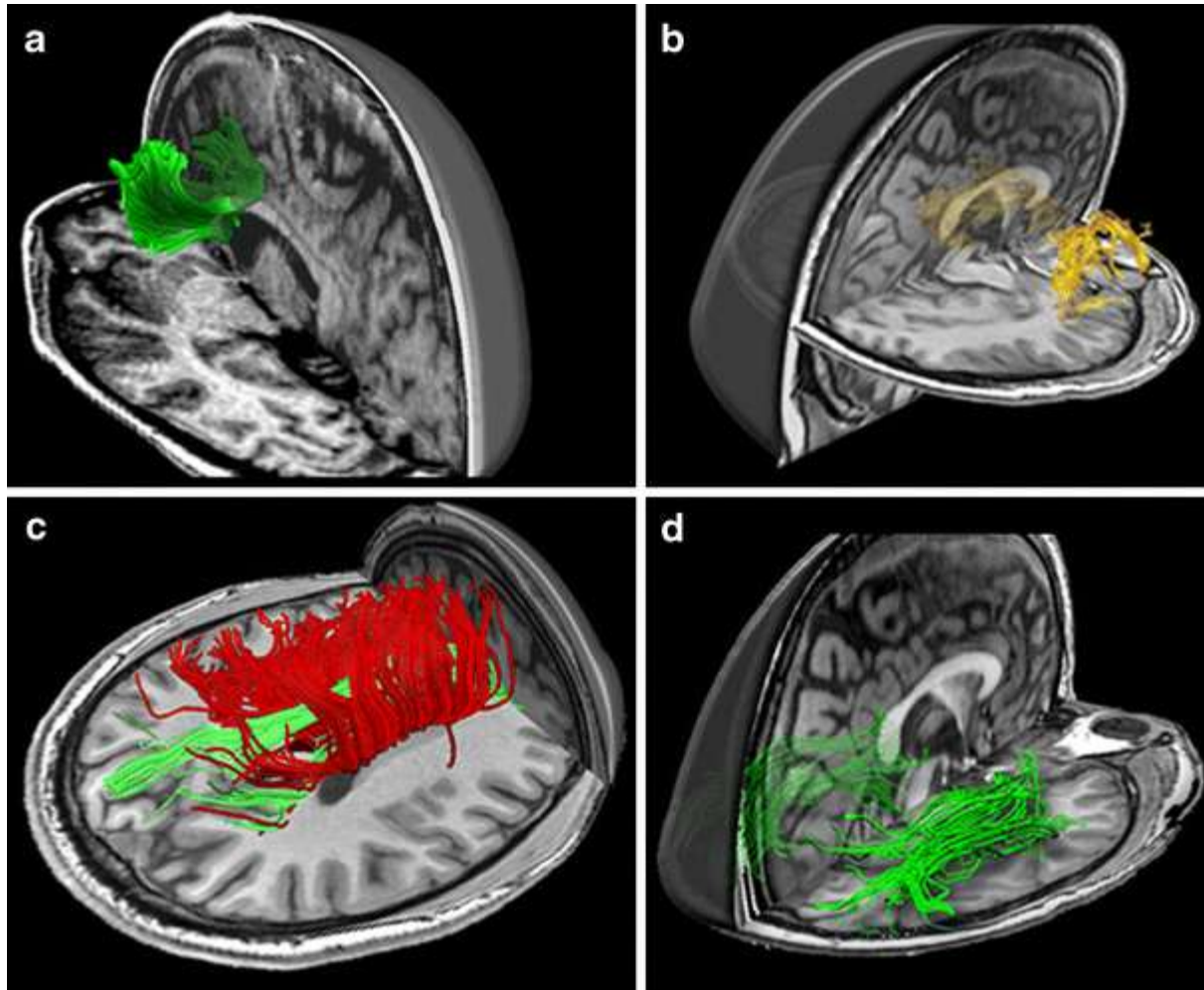
DOI: 10.1097/WCO.0b013e32834cd523

Box 1. no caption available





# Brain Imaging and Behavior Shenton et al 2012







## Treatment

- Very few evidence based studies
- Diary
- History
- Information
- Simple analgesics in restricted doses
- Detoxification in case of MOH
- Prophylaxis:
  - Amitriptyline, start low, go slow, very sensitive;
  - Betablockers? Topiramate?
- Psychological/social support

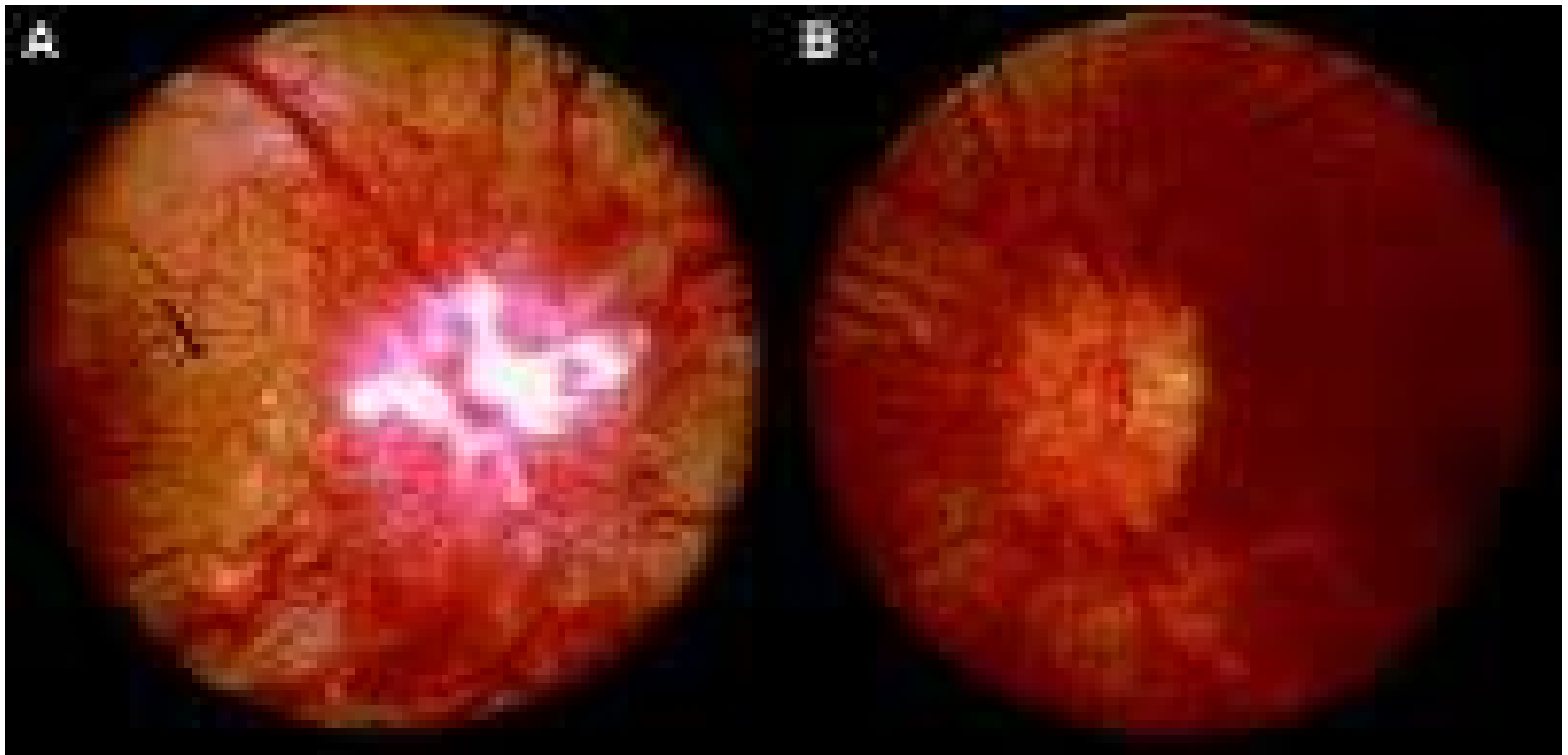


## Case

- 17 year old girl with mild episodic posttraumatic headaches for 6 months presents in ER with subacute, severe holocranial headache for 2 days. Double vision otherwise no visual symptoms
- No family history of headaches
- No reported exposition to hormonal therapy
- Questions: ?

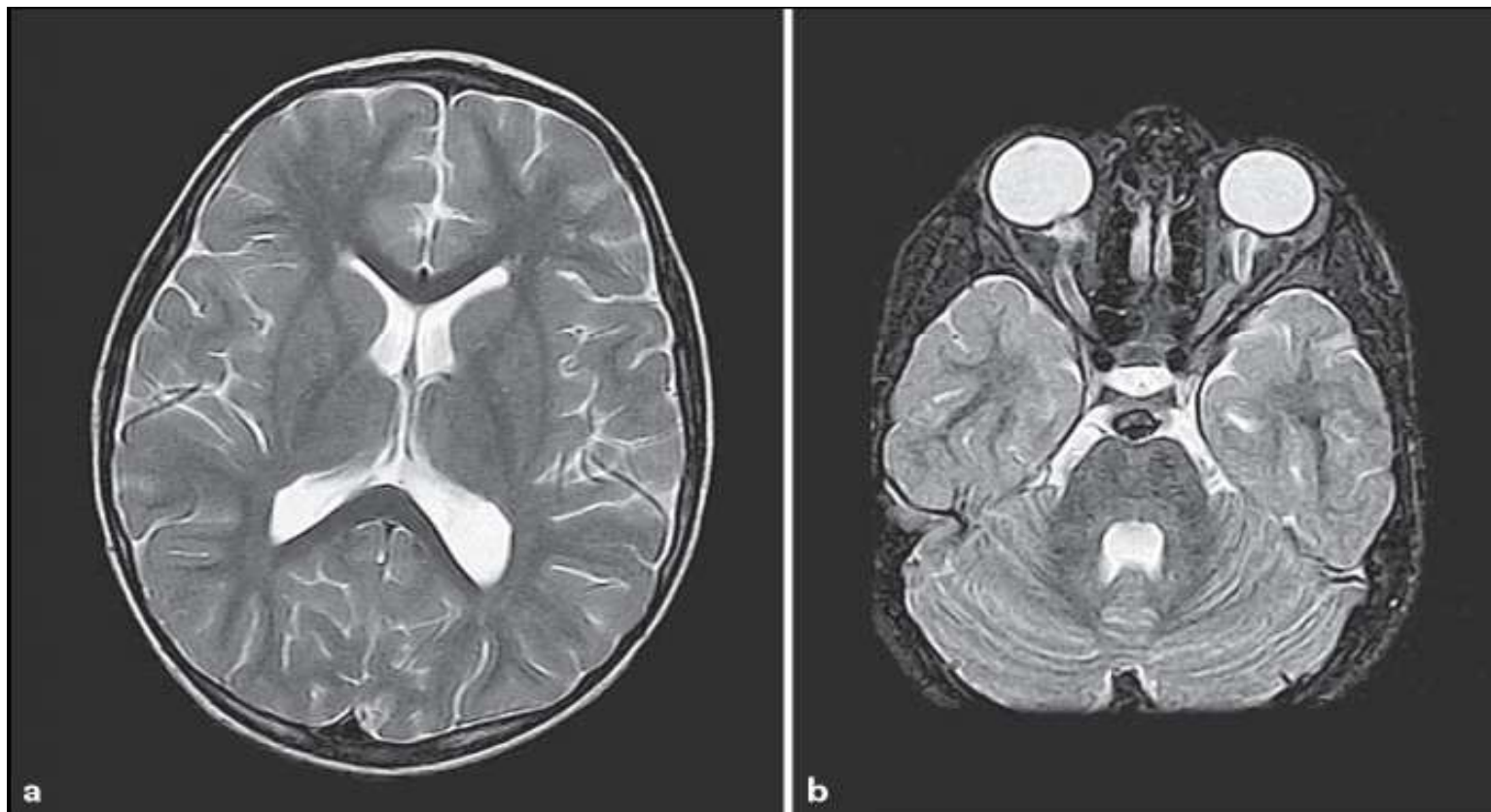


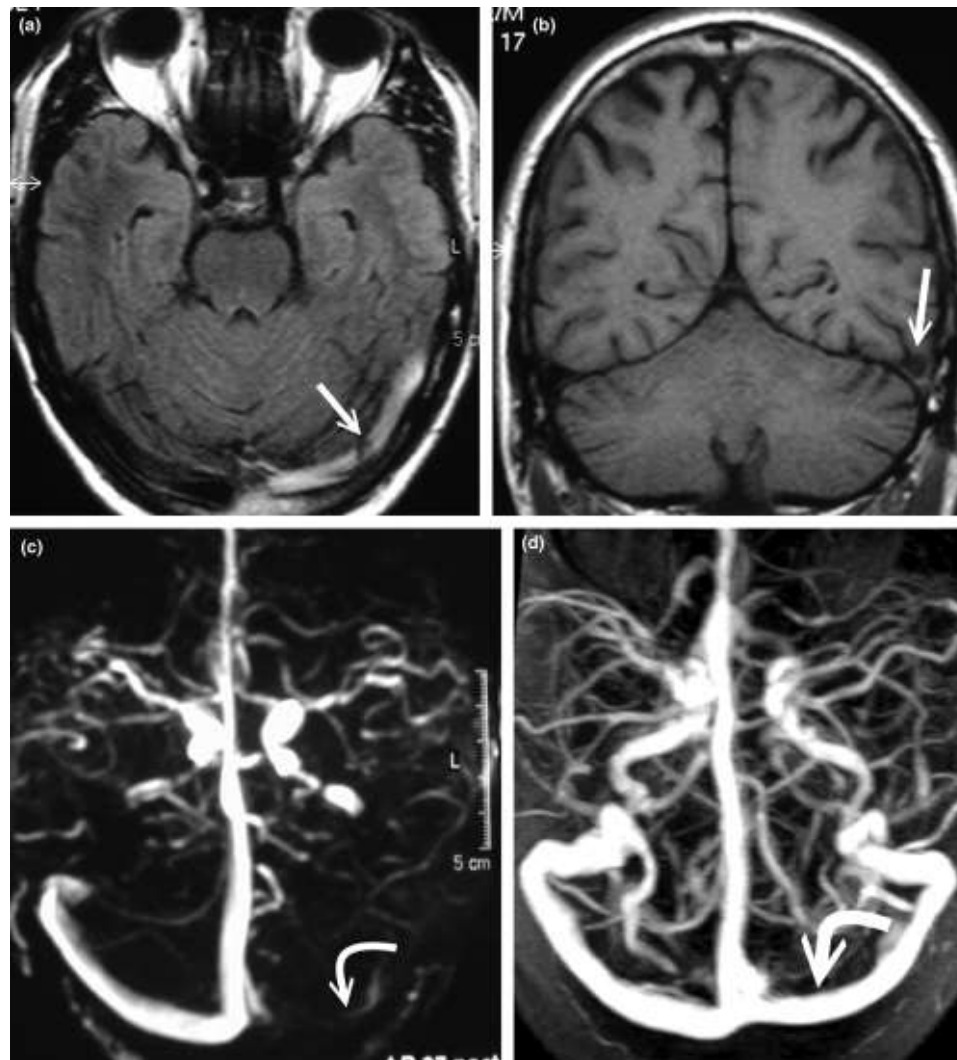
# Fundus apperance - Diagnosis?





## Case 9





Klingebl, R., Bauknecht, H.C., Bohner, G., Kirsch, R., Berger, J. & Masuhr, F.  
Comparative evaluation of 2D time-of-flight and 3D elliptic centric contrast-enhanced MR  
venography in patients with presumptive cerebral venous and sinus thrombosis.  
*Eur J Neurol* 2007; **14** (2), 139-143.



## **7. Headache attributed to non-vascular intracranial disorder**

**7.1 Headache attributed to high cerebrospinal fluid pressure**

**7.2 Headache attributed to low cerebrospinal fluid pressure**

**7.3 Headache attributed to non-infectious inflammatory disease**

**7.4 Headache attributed to intracranial neoplasm**

**7.5 Headache attributed to intrathecal injection**

**7.6 Headache attributed to epileptic seizure**

**7.7 Headache attributed to Chiari malformation type I**

**7.8 Syndrome of transient Headache and Neurological Deficits with cerebrospinal fluid Lymphocytosis (HaNDL)**

**7.9 Headache attributed to other non-vascular intracranial disorder**



## **7.1 Headache attributed to high cerebrospinal fluid pressure**

**7.1.1 Headache attributed to idiopathic intracranial hypertension (IIH)**

**7.1.2 Headache attributed to intracranial hypertension secondary to metabolic, toxic or hormonal causes**

**7.1.3 Headache attributed to intracranial hypertension secondary to hydrocephalus**





## 7.1.1 Headache attributed to IH

### **B. Intracranial hypertension fulfilling the following criteria:**

1. alert patient with neurological examination that either is normal or demonstrates any of the following abnormalities:
  - a) papilloedema
  - b) enlarged blind spot
  - c) visual field defect (progressive if untreated)
  - d) sixth nerve palsy
2. increased CSF pressure ( $>200$  mm H<sub>2</sub>O [non-obese],  $>250$  mm H<sub>2</sub>O [obese]) measured by lumbar puncture in the recumbent position or by epidural or intraventricular pressure monitoring
3. normal CSF chemistry (low CSF protein acceptable) and cellularity
4. intracranial diseases (including venous sinus thrombosis) ruled out by appropriate investigations
5. no metabolic, toxic or hormonal cause of intracranial hypertension



## IIH-epidemiology

- Incidence 1-2/100.000 in non-obese individuals
- Incidence 21/100.000 in obese women
- Prevalence?
- All ages (range 1 mth-?) but most frequent between 20-40 years
- Male/female ratio: 1/4-15



# IIH

## Ocular symptoms and signs

- Visual field defects
- VI nerve palsy
- Decreased visual function
- Enlarged blind spot
- Impaired contrast sensitivity
- Colour vision defects
- Afferent pupillary defect



# IDIOPATHIC INTRACRANIAL HYPERTENSION- symptoms

	Johnston	Rush	Corbett	Skau*
<b>Patients (no.)</b>	62	63	57	8
<b>Symptom (%)</b>				
• Headache	95	75	81	100
• Diplopia	31	35	33	0
• Visual blurring	65	68	-	75
• TVO	-	46	72	50
• Other	-	22	32	13
• Nausea and vomiting	24	21	?	75
• Dizziness	11	?	?	-
• Tinnitus	11	?	?	13
• Asymptomatic	0	5	?	-
• Other	15	19	?	-

• Median delay from onset to diagnosis 4 months (range 1-48 mths)\*



## IH- differential diagnosis

- Vascular diseases: Cerebral venous sinus thrombosis; AVM
- CSF hyperviscosity
- Haematological/ Endocrinological diseases
- Guillain-Barré
- Infections:  
Syphilis; Meningitis/encephalitis; Sinusitis, Otitis
- Pharmacological: Intoxications
- Circulatory: Hypertension; Congestive heart failure
- Neoplasms: Intracranial; lymphoma; Spinal cord tumour
- Others: Respiratory disease with CO<sub>2</sub> retention; Sleep apnoea syndrome; Bechet; SLE
- Ophthalmological diseases ?



## IH- prognosis

- Permanent visual loss in 40-87 %, severe in 10%
- Complete amplyopia in 5-10% !
- Recurrent episodes in 39%
- Optic atrophy in 9% (6 years follow up)
- High rate of relapse, and headache is a poor predictor

*(Wall 1991; Rowe 1998; Corbett et al 1982; Wall et al 1991; Kesler et al 2004, Yri 2011)*



# Idiopathic intracranial hypertension

- Obese woman of childbearing age
- Headache
- Papilloedema
- Pulsatile tinnitus
- Transitory visual obscurations
- Diplopia (VI palsy)
- Visual field defects
- Increased CSF pressure (>200 mm H<sub>2</sub>O in the non-obese, >250 mm H<sub>2</sub>O in the obese)



# Optical Coherence Tomography

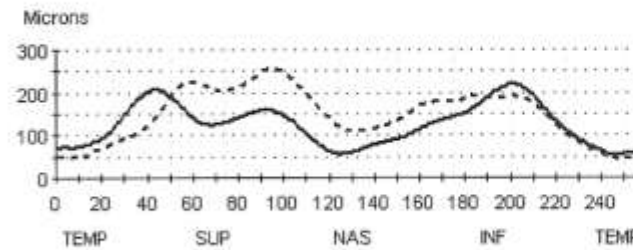
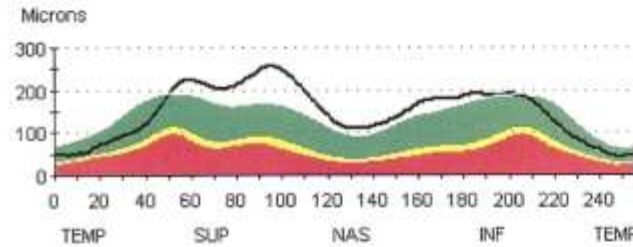
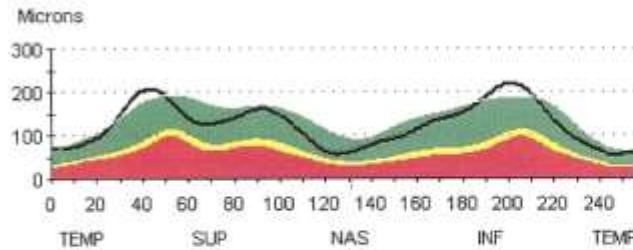
## Case 4

Day 0

BMI 31.4

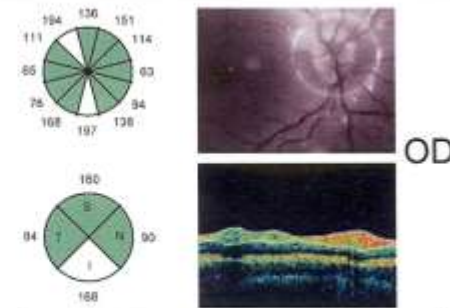
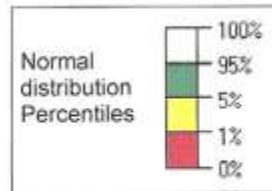
ICP = 31 cm H<sub>2</sub>O

Before Diamox

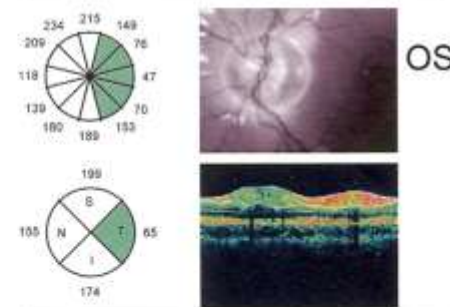


— OD - - - - OS

OD	Scans used	1, 2, 3
OS	Scans used	1, 2, 3



Signal Strength (Max: 10)	7
---------------------------	---



Signal Strength (Max: 10)	8
---------------------------	---

	OD (N=3)	OS (N=3)	OD-OS
Imax/Smax	1.06	0.74	0.32
Smax/Imax	0.94	1.35	-0.40
Smax/Tavg	2.46	3.97	-1.51
Imax/Tavg	2.60	2.94	-0.34
Smax/Navg	2.28	1.65	0.63
Max-Min	165.00	212.00	-47.00
Smax	206.00	256.00	-50.00
Imax	218.00	190.00	28.00
Savg	160.00	199.00	-39.00
Iavg	168.00	174.00	-6.00
Avg.Thick	125.57	148.32	-22.75

Signature:

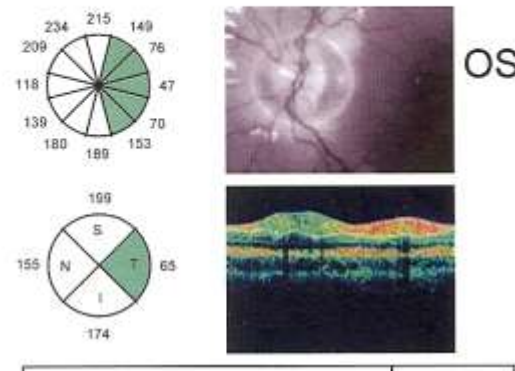
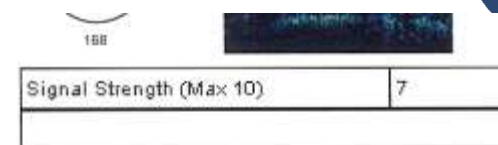
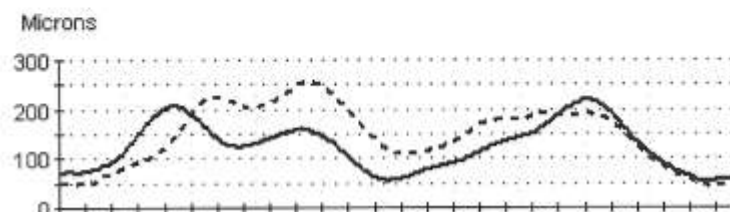
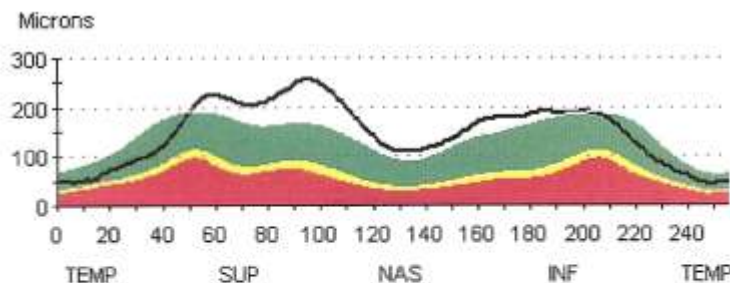
Dag 0 ICP 31 cmH<sub>2</sub>O BMI 31.4  
- diamox ordinere



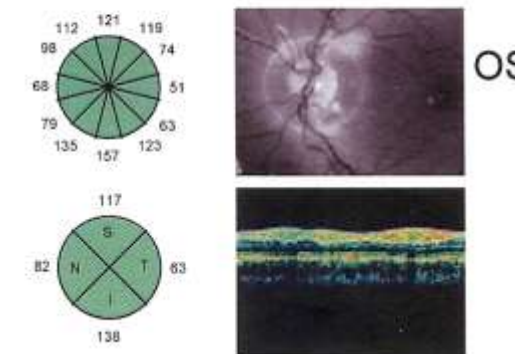
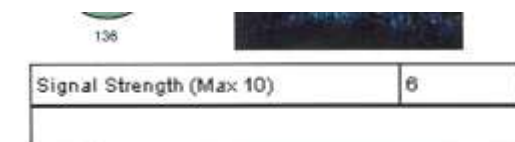
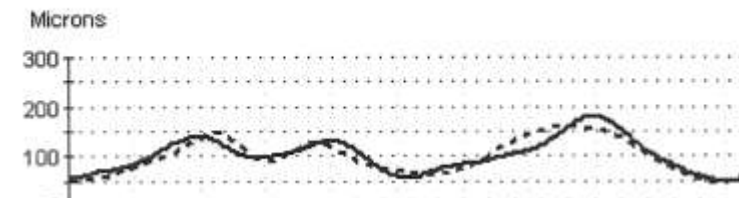
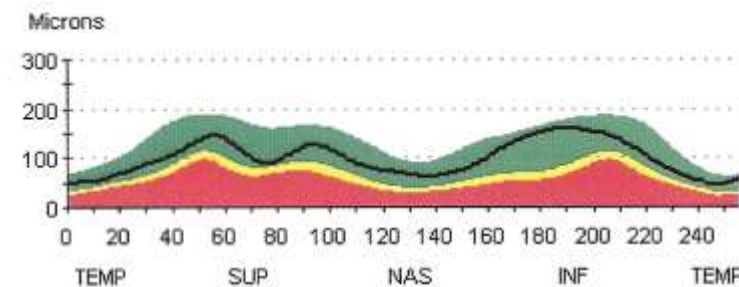


# Optical Coherence Tomography, Case 4, left eye

Day 0



Month 3





## IH- treatment

- Elimination of exogenic factors
- Weight loss
- Acetazolamide
- Furosemide
- LP-VP-Shunt
- N. opticus fenestration ?



## **IIH- conclusion I**

- Benign intracranial hypertension is not benign
- Idiopathic intracranial hypertension is not always idiopathic
- An early diagnosis is critical



## IIH- conclusion II

- Early and intensive treatment plan is critical
- Treatment is imperical and consist of Azetazolamide, diuretics, weight loss, and eventual shunting
- A systematic, multidisciplinary strategy and frequent, long term follow up are needed
- **Benign intracranial hypertension is not benign and early intervention is essential**



## Case 2

- 37-year old woman, previously infrequent migraine without aura, otherwise healthy
- On April 12th 2008 she experienced a buzzing sound in right ear. Five days later she developed subacute severe constant headache behind both eyes and in the parietal region. First days disappearance when lying down, worsening when she got up. Accompanying photophobia, nausea, stiffness in neck and dizziness
- Admitted to hospital April 19th
- Objectively: Decreased hearing on right side, tenderness in neck muscles, temperature 38.1
- What to do, differential diagnoses?



## Case 2

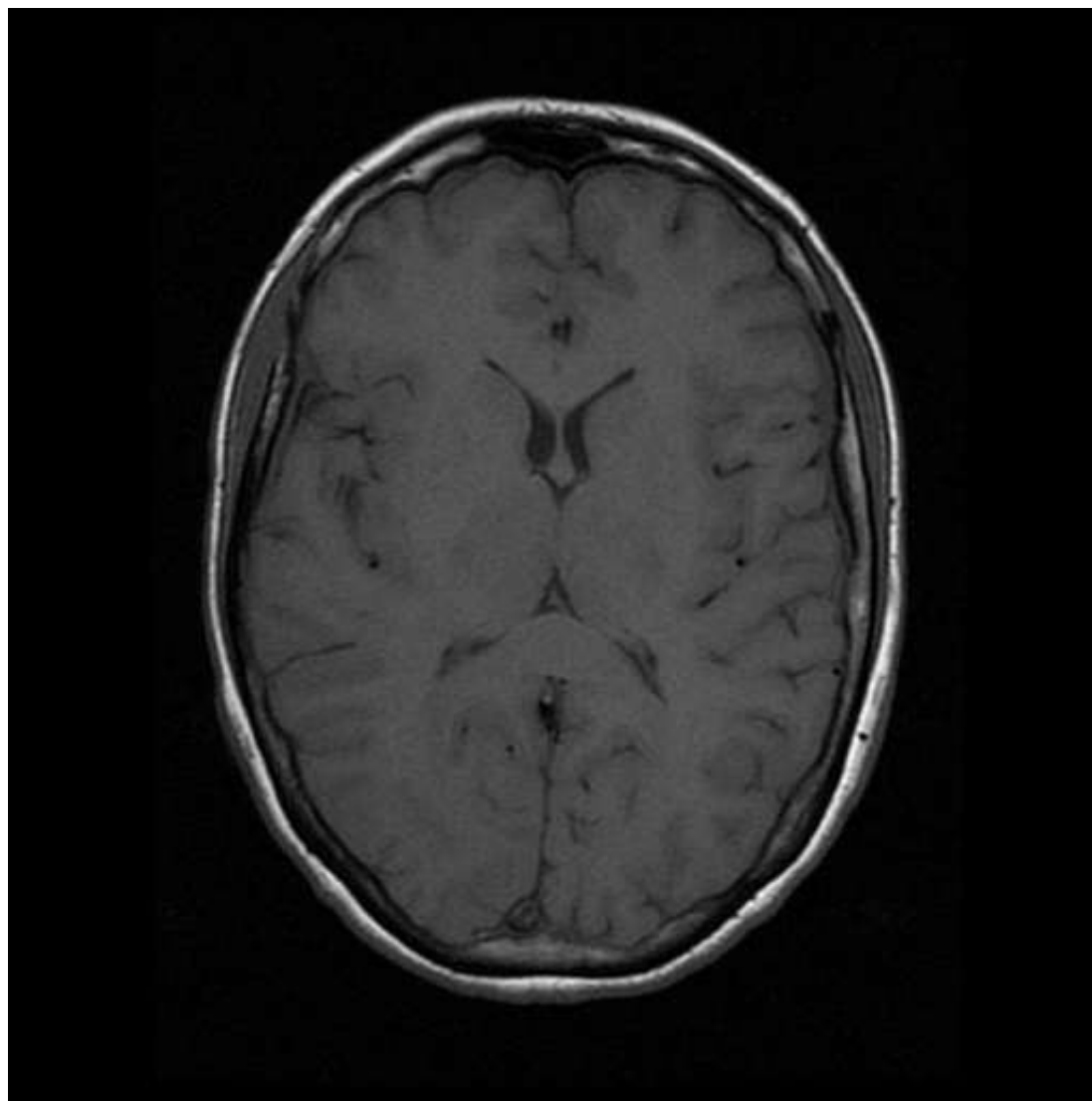
- Ct-scan with venous sequences normal
- Lumbar puncture, 3 leukocytes, protein 0.57, glucose 2.9, pressure 3.5 cm water
- Diagnosis? What to do?
- Ordering MR with contrast
- Plenty of cola and fluids, slight improvement
- Blood patch next day, staying in bed for 24 hours, slight improvement



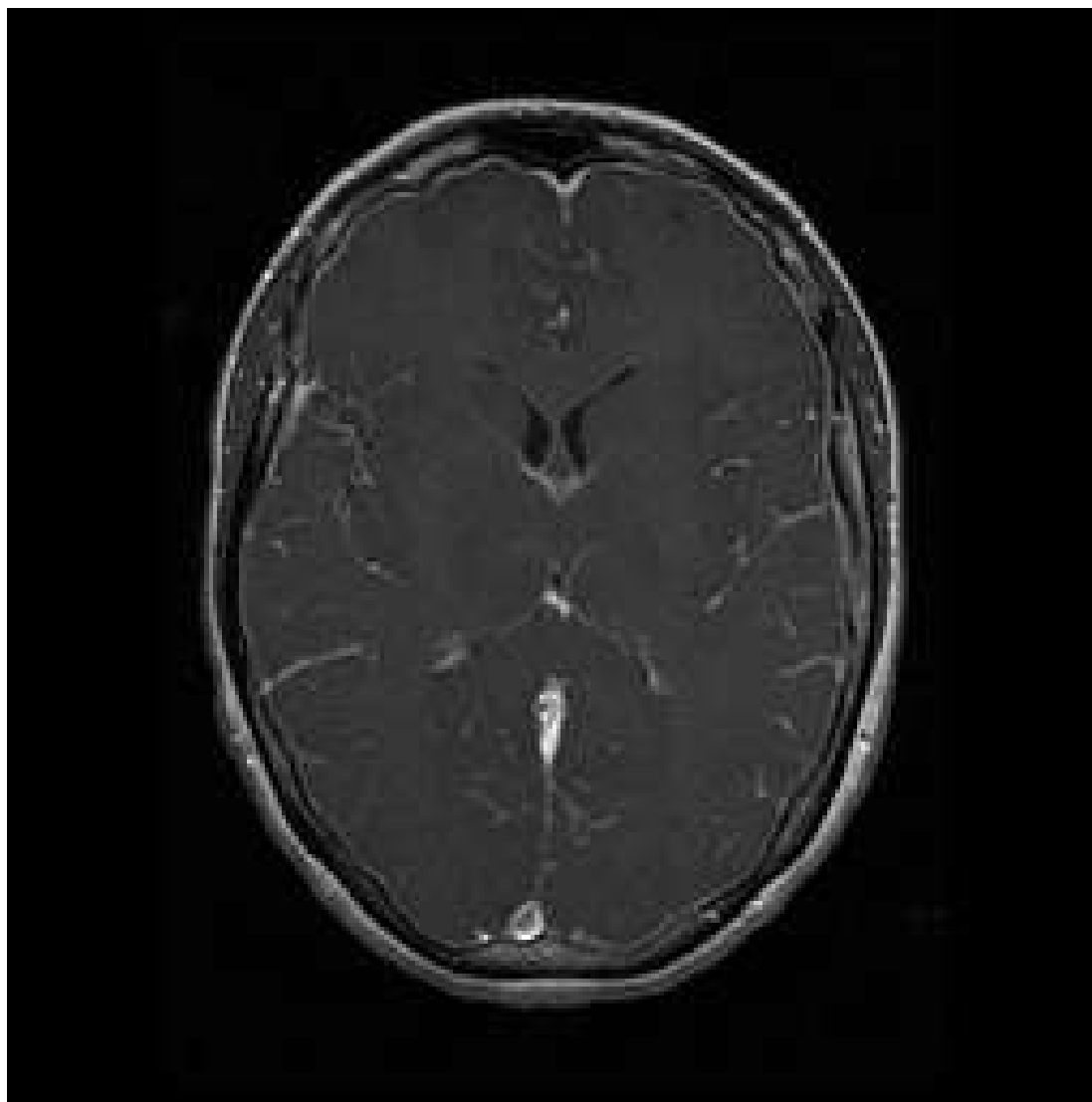
30.4.2008 – Sag, T2 without contrast



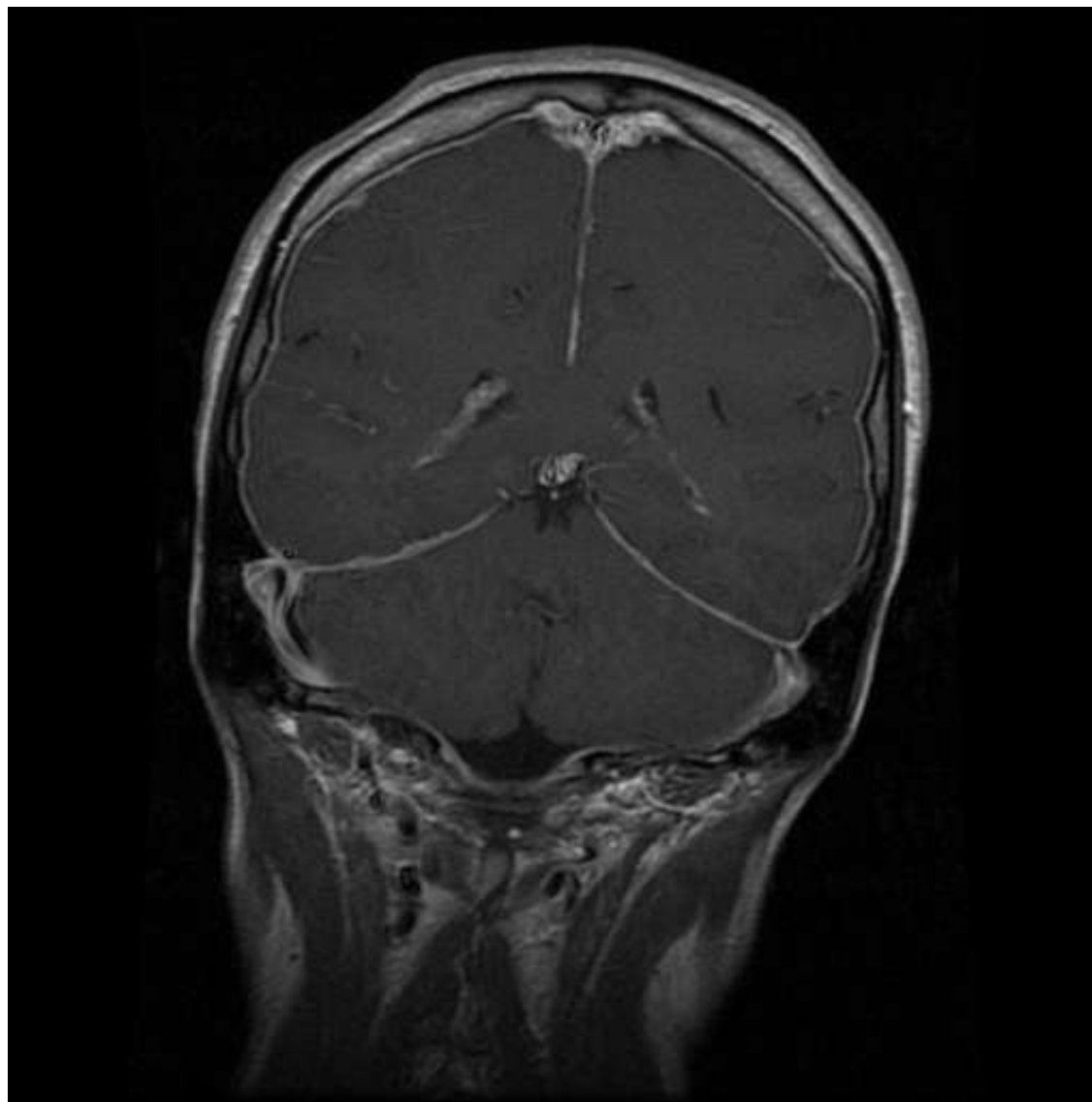
30.4.2008 – Ax, T1 without contrast



30.4.2008 – Ax, T1 with contrast



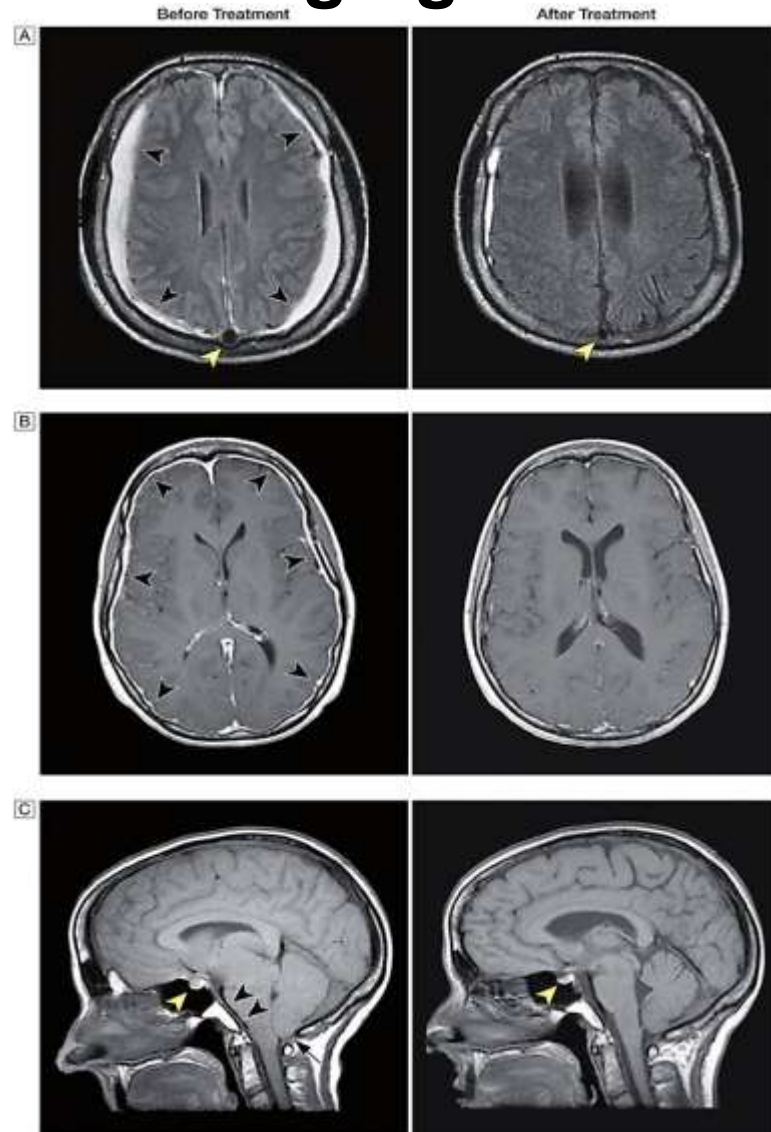
30.4.2008 – Cor, T1 with contrast



# Pretreatment and Posttreatment Magnetic Resonance Imaging



- S Subdural hygroma
- E Enhancement
- E Engorged veins
- P Pituitary hyperemia
- S Sagging of the brain



Schievink, W. I. JAMA 2006;295:2286-2296.

**JAMA**



## **7.2 Headache attributed to low cerebrospinal fluid pressure**

### **7.2.1 Post-dural puncture headache**

### **7.2.2 CSF fistula headache**

### **7.2.3 Headache attributed to spontaneous (or idiopathic) low CSF pressure**



## 7.2.1 Post-dural (post-lumbar) puncture headache

- A. Headache that worsens within 15 min after sitting or standing and improves within 15 min after lying, with  $\geq 1$  of the following and fulfilling criteria C and D:**
1. neck stiffness; 2. tinnitus; 3. hypacusia;
  4. photophobia; 5. nausea
- B. Dural puncture has been performed**
- C. Headache develops within 5 d after dural puncture**
- D. Headache resolves either:**
1. spontaneously within 1 wk
  2. within 48 h after effective treatment of the spinal fluid leak



## Case 2

- What to do now?
- Tablet coffee 100 mg x 3 (but tolerated only 1 + ½)
- Did not want another blood patch. Discharged May 5th
- Followed up in DHC 19.5. Still daily headache, now also sometimes when lying down. Also dizziness, nausea and neck stiffness. All together some improvement and some effect from coffee but still considerably affected, could not resume work
- Indometacin without effect. Offered new blood patch, but did not accept it





## Case 2

- 23.5: phone back, now interested in new blood patch. Performed 26.5, slight short-lasting effect
- What to do ?
- 28.5: third blood patch, immediately after blood patch major improvement, only vague symptoms, stayed in bed for 7 days
- 11.8: only very mild headache 2-3 times per week and intermittent decreased hearing for seconds after standing, well-being
- 23.2.2009: only very mild headache 1 time per week and intermittent decreased hearing for seconds after standing, well-being



# Spontaneous intracranial hypotension (SIH)

- Previously healthy young individuals
- Sudden severe headache/neck pain after strain, coughing, sneezing, lifting etc
- "An ice cube in an empty glass"
- Orthostatic at presentation, resemble post lbp h.
- A generalized connective tissue disorder?
- MRI: diffuse pachymeningeal Gd-enhancement

*In 10/10 SIH Miyazawa K et al Neurology 2003  
and in 59/59 SIH Mea E et al Neurol Sci 2007*