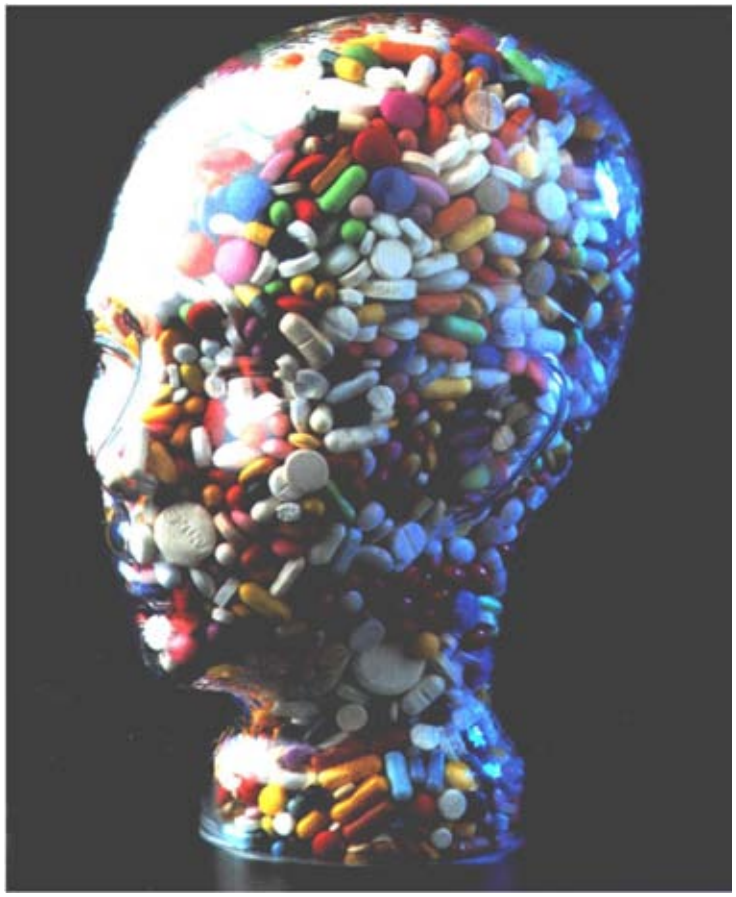
Management of secondary headaches WCN 2013



Rigmor Højland Jensen
Danish Headache Centre,
Department of Neurology,
Glostrup Hospital,
University of Copenhagen,
Denmark

Disclosures: Lectures for Pfizer, Berlin-Chemie, Allergan, Merck
Member of advisory boards in: ATI, Medotech, Neurocore, and Linde Gas ltd.
Director in LTB, EHMTIC and President in EHF

Headache Care: Organization of Headache Service

Level 3: Specialized headache centres
- Both inpatient and outpatient treatment
- Multidisciplinary treatment
- Education
- Research
- Organisation of networks with levels 1 and 2

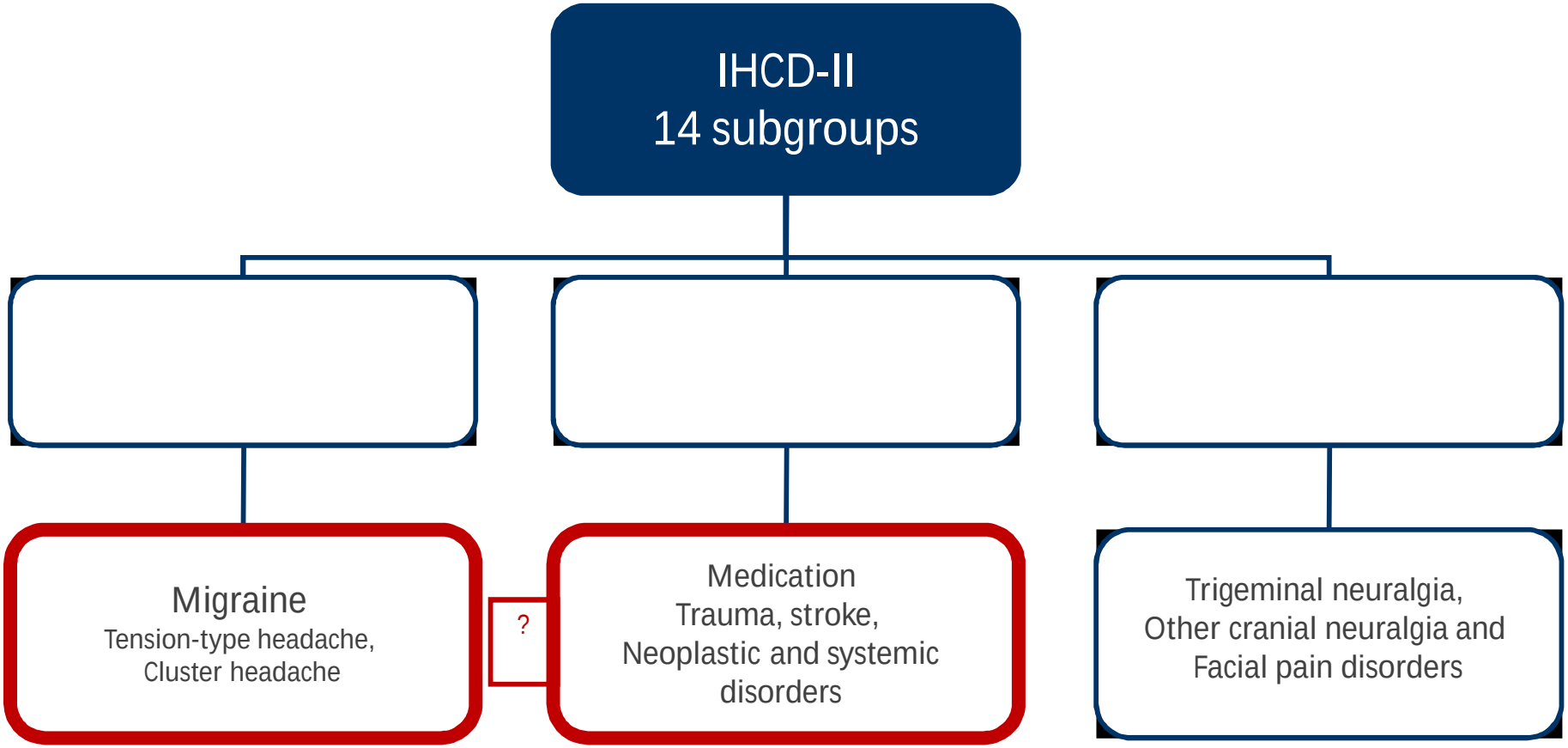
Level 2: Special Interest Headache Care

Secondary care or primary care with special interest in headache disorders
- Completion of special training
- Fulfills national guidelines and requirements for special headache/pain therapy

Level 1: General Primary Care

Primary care without special interest in headache disorders
-Following treatment guidelines
-Selecting patients for higher levels (gate-keeper function)
-Long-term care after discharge from levels 2 and 3

International Headache Classification, (ICHD- III beta 2013)



WHAT IS A SECONDARY HEADACHE?

"Etiology based, not symptom based as the primary headaches"

Standard Secondary Headache Diagnostic Criteria in ICHD III

A. Any headache fulfilling criterion C

B. Another disorder scientifically documented to be able to cause headache has been diagnosed

C. Evidence of causation demonstrated by at least two of the following:

1. headache has developed in temporal relation to the onset of the presumed causative disorder
2. one or both of the following:

a. headache has significantly worsened in parallel with worsening of the presumed causative disorder

b. headache has significantly improved in parallel with improvement of the presumed causative disorder

3. headache has characteristics typical for the causative disorder
4. other evidence exists of causation

D. Not better accounted for by another ICHD-III diagnosis.

Edition (ICHD III) – Basic Organization

Part 1: Primary headaches, chapters 1-4 (no other causative disorder)

1. Migraine
2. Tension-type Headache
3. Trigeminal Autonomic Cephalalgias
4. Other primary headaches

Part 2: Secondary headaches

5. Posttraumatic

6. Vascular disease

7. Other intracranial pathology

8. Substances

9. CNS infection
10. Homeostatic disorders
11. Cranium, Neck, Eyes, ENT, Sinuses, Mouth, Teeth, TMJ
12. Psychiatric

Part 3: Cranial Neuralgias and other facial pain

13. Neuralgias and neuropathy

Appendix

5. Headache attributed to head trauma

IHS code		ICD-10 code	
		Etiological code	Headache code
5.	Headache associated with head trauma		G44.88
5.1	Acute posttraumatic headache		G44.880
	5.1.1 With significant head trauma and/or confirmatory signs	S06	G44.880
	5.1.2 With minor head trauma and no confirmatory signs	S09.9	G44.880
5.2	Chronic posttraumatic headache		G44.3
	5.2.1 With significant head trauma and/or confirmatory signs	S06	G44.30
	5.2.2 With minor head trauma and no confirmatory signs	S09.9	G44.31

- **5.2.2 Persistent post-traumatic headache attributed to mild head injury**
- Headache of any type, fulfilling criteria C and D
- Head trauma with all of the following:
  - Either no loss of consciousness, or loss of consciousness of <30 minutes duration
  - Glasgow Coma Scale (GCS) ?13
  - Post-traumatic amnesia ?24 hours in duration
  - Alteration in consciousness ?24 hours in duration
    - 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
      - > Loss of memory for events immediately before or after injury
      - > Other neurologic deficits
- Evidence of causation shown by:
  - Headache develops within 7 days after head trauma
- Headache persists for >3 months after head trauma
- Headache is not better accounted for by another headache diagnosis

Management of Posttraumatic headache

- Mild – Moderate – Severe??
- Persistent TTH-like headache with photo-and phonophobia associated with cognitive symptoms
- No known mechanisms and pharmacological RCT ’s
- Clinical practise:
  - Acute attacks: Limited effect of analgesics and MOH-risk
  - Preventives most relevant:
  - Amitriptyline start low and go slow
  - Migraine Preventives (betablockers, antiepileptics)
  - Psychological counseling: Cognitive Behavioural Treatment, Relaxation and Biofeedback
  - Reassuring

Original Article

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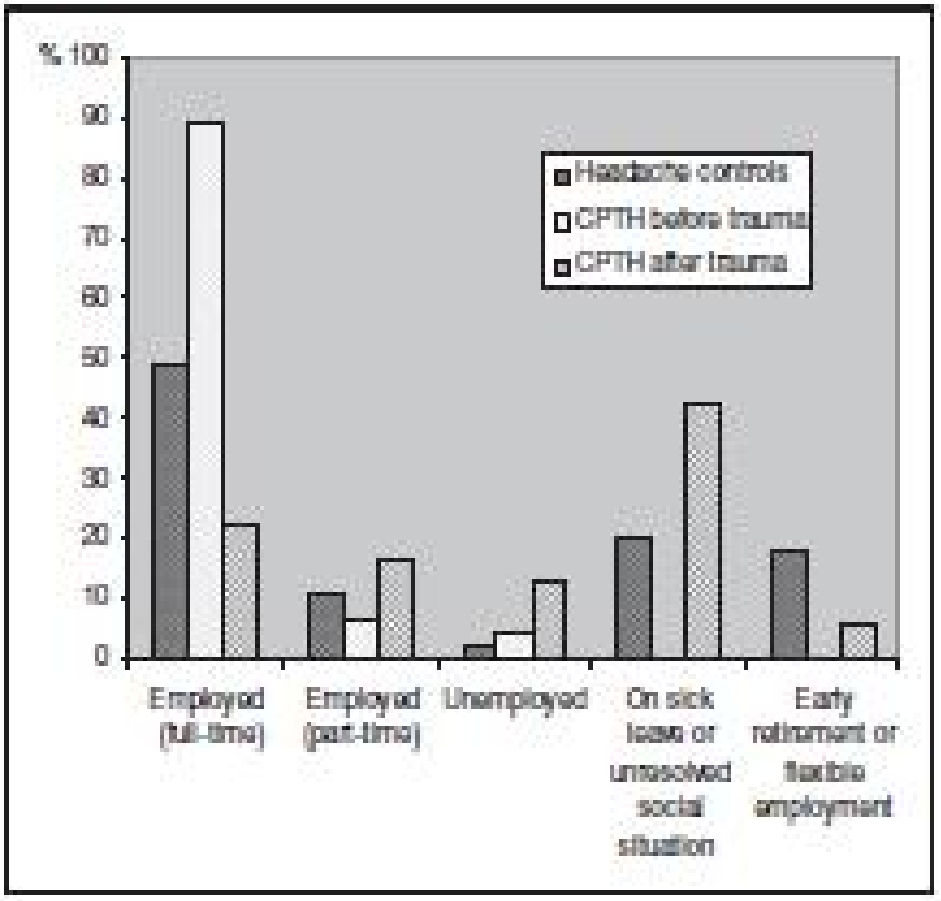
SAGE

Chronic post-traumatic headache after mild head injury: A descriptive study

Dorte Kjeldgard[1], Hysse Forchhammer[2], Tom Teasdale[3] and Rigmor H Jensen[1]



Quality of Life and Functioning in posttraumatic headache (*Kjeldgaard et al 2013*)

	CPTH ($N = 78–79$[a])	Control ($N = 44–45$[a])	Between groups		
	Mean (SD)	Mean (SD)	Mean difference	95% CI	p[b]
SF-36					
Physical Function	69.3 (20.0)	80.2 (17.5)	−10.9	(−18.0 to −3.86)	0.036
Physical Role	11.1 (22.6)	32.6 (37.3)	−21.9	(−34.2 to −9.55)	0.012
Bodily Pain	27.3 (17.9)	36.6 (18.7)	−9.30	(−16.0 to −2.56)	NS
General Health	51.5 (20.6)	49.3 (21.2)	2.16	(−5.52 to 9.84)	NS
Vitality	31.9 (18.8)	30.6 (17.9)	1.33	(−5.55 to 8.21)	NS
Social Function	42.3 (25.8)	58.1 (26.1)	−15.8	(−25.4 to −6.22)	0.012
Emotional Role	64.1 (41.3)	56.8 (45.8)	7.32	(−8.67 to 23.3)	NS
Mental Health	58.2 (18.2)	61.1 (21.1)	−3.01	(−10.1 to 4.13)	NS
Rivermead					
Sum total	31.4 (13.2)	22.7 (12.8)	8.67	(3.84 to 13.5)	0.012
Cognitive factor	2.5 (1.4)	1.5 (1.3)	1.01	(0.504 to 1.51)	<0.001
Emotional factor	1.7 (1.2)	1.2 (1.1)	0.437	(0.002 to 0.872)	NS
Somatic factor	1.9 (0.8)	1.5 (0.8)	0.433	(0.139 to 0.726)	0.048

Data are presented as mean (SD).

[a]The number of patients included in the calculations varies slightly because of missing values.

[b]p values are after Bonferroni correction.

Conclusion: Posttraumatic headache

- Symptomatic treatment with unspecific migraine preventives
- Low dose amitriptyline has a positive effect on headache and sleep but RCT's are lacking
- Psychological support, CBT and relaxation may be beneficial but evidence is scarce
- Mechanisms based treatment and new drugs targets are needed

Idiopathic Intracranial Hypertension ICHD-III Classification Criteria

1. Alert patient with neurological examination that is normal or demonstrates any of the following abnormalities:
    a) papilledema
    b) enlarged blind spot
    c) visual field defect (progressive if untreated)
    d) sixth nerve palsy

2. Increased CSF pressure (>250 mm H_2O) measured by lumbar puncture or by epidural or intraventricular pressure monitoring

3. Normal CSF chemistry (low CSF protein is acceptable) and cellularity

4. Intracranial diseases ruled out by appropriate investigations

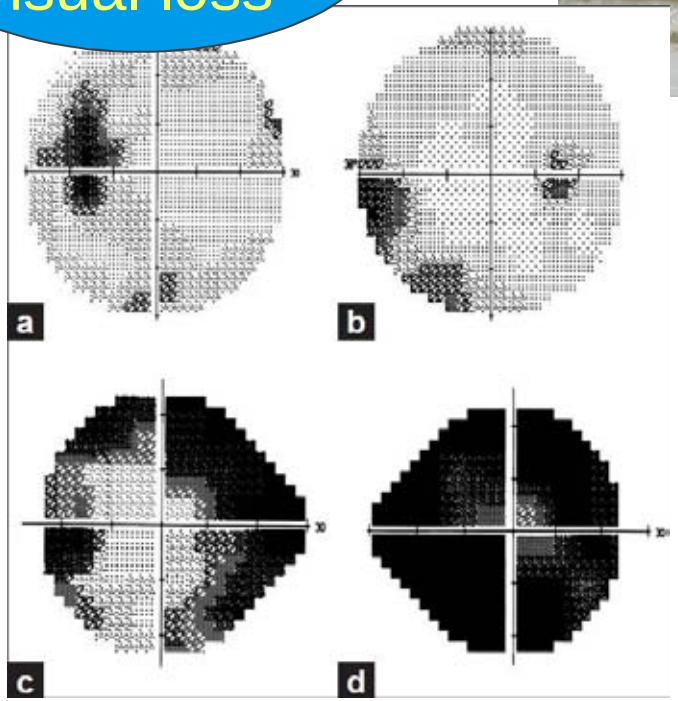
5. No metabolic, toxic or hormonal cause of intracranial hypertension

Background and signs of IIH

headache

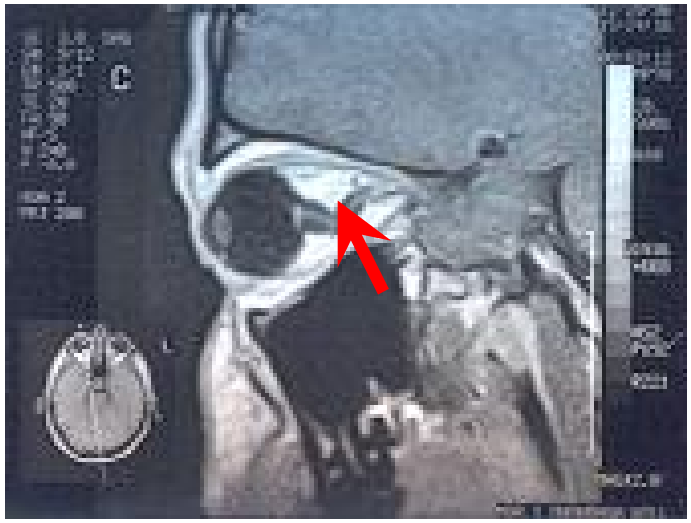
obese ? O

progressive
permanent
visual loss



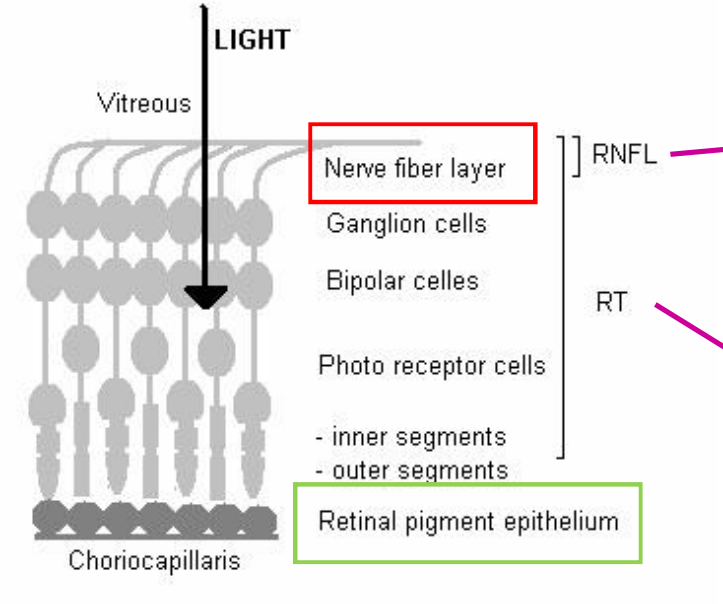
papilledema

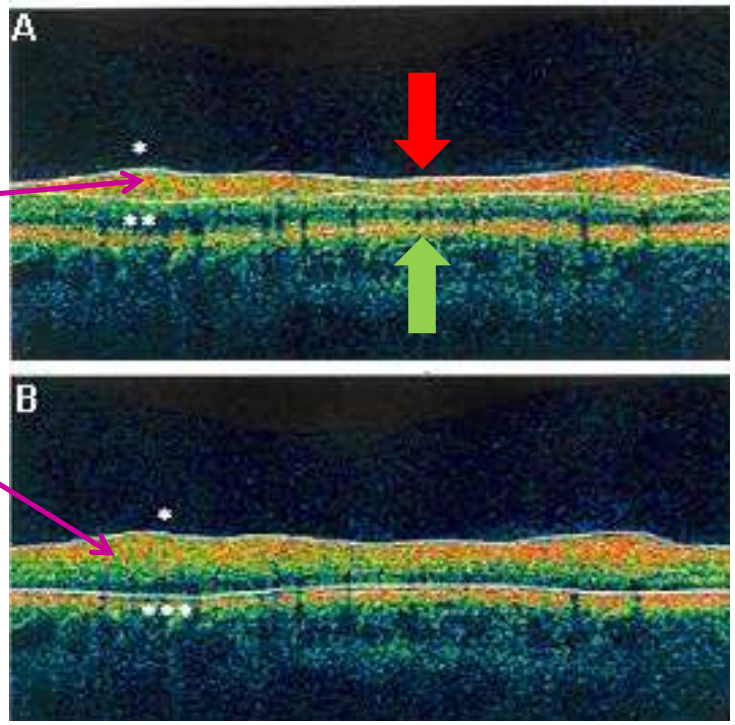
MR findings



25 IIH-patients and 25 controls
Most sensitive findings:
Empty sella and nerve sheat distension = most reliable signs.
Posterior globe flattening: specific but not sensitive. No changes in lateral ventricles (+VBM) and no relation to clinical presentations.
Jan Hoffmann et al Cephalalgia 2013

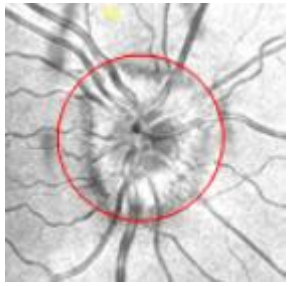
OPTICAL COHERENCE TOMOGRAPHY

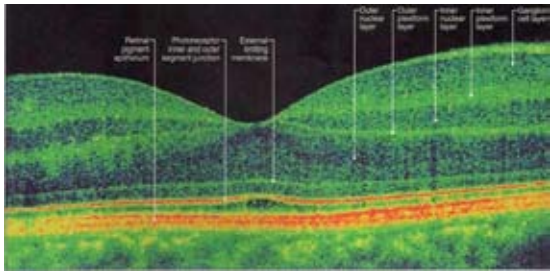


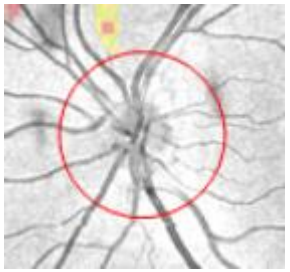


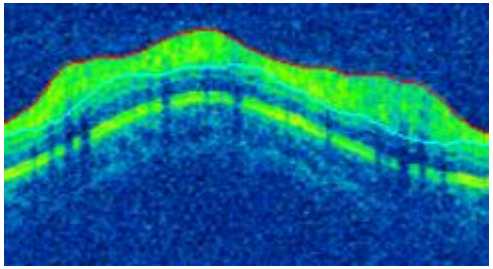
Onset

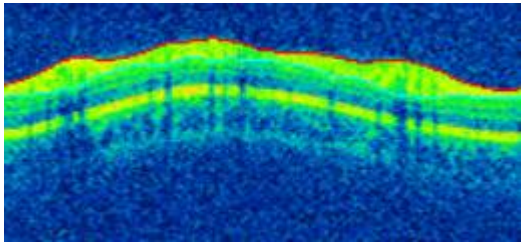
3 months

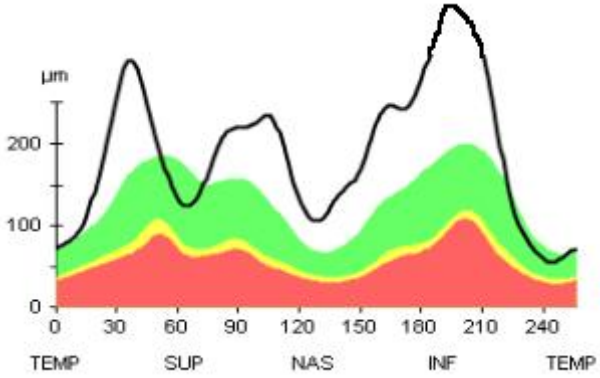


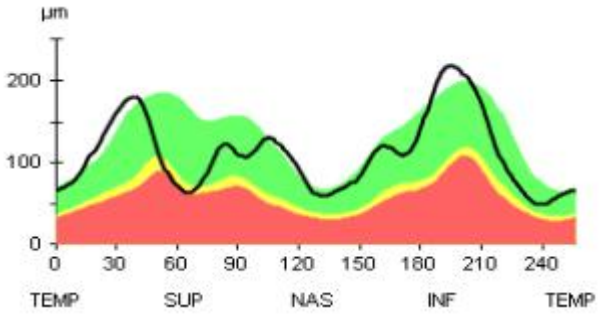


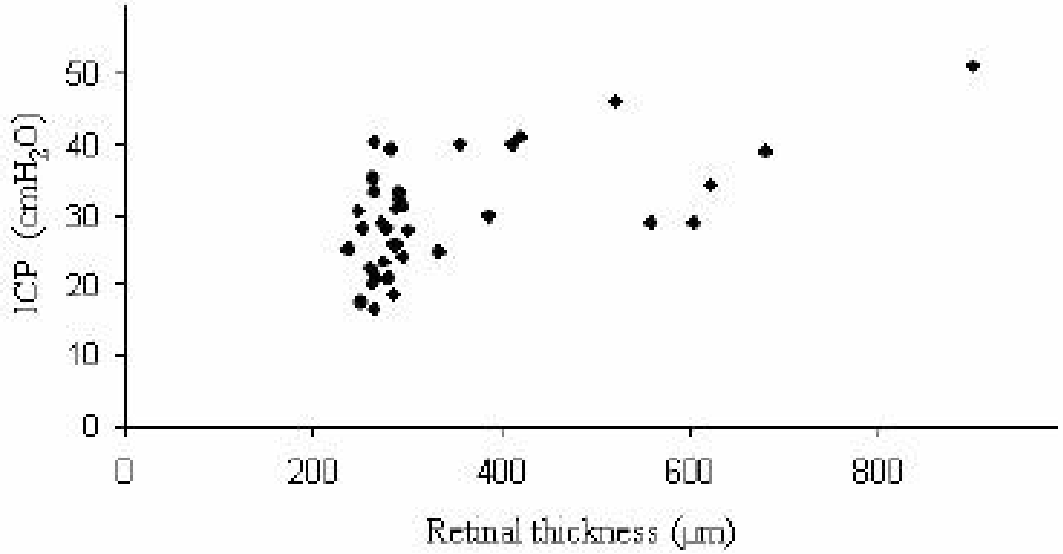


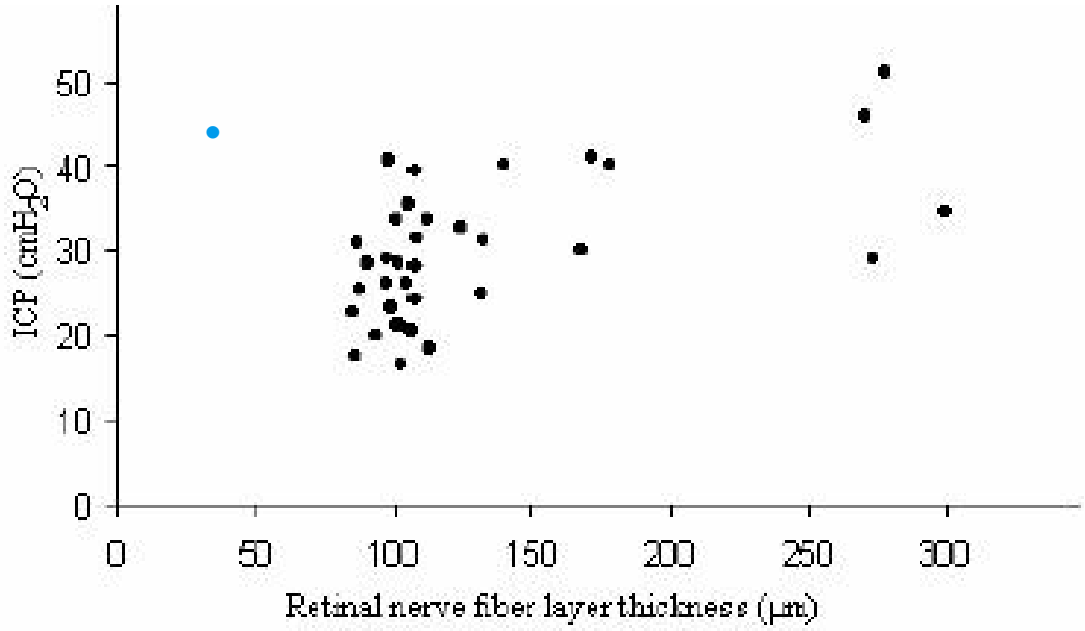












Scatter plots of ICP and RT (n=37) and RNFL (n=35) in all IIH subjects (N-/LH-/LL-IIH). Interocular means unadjusted for age and BMI.

Correlations:

N-IIH ICP vs RNFL
r = 0.5, p = 0.01

ICP vs RT
r = 0.5, p = 0.03

LH-IIH ICP vs RNFL
r = 0.7, p = 0.02

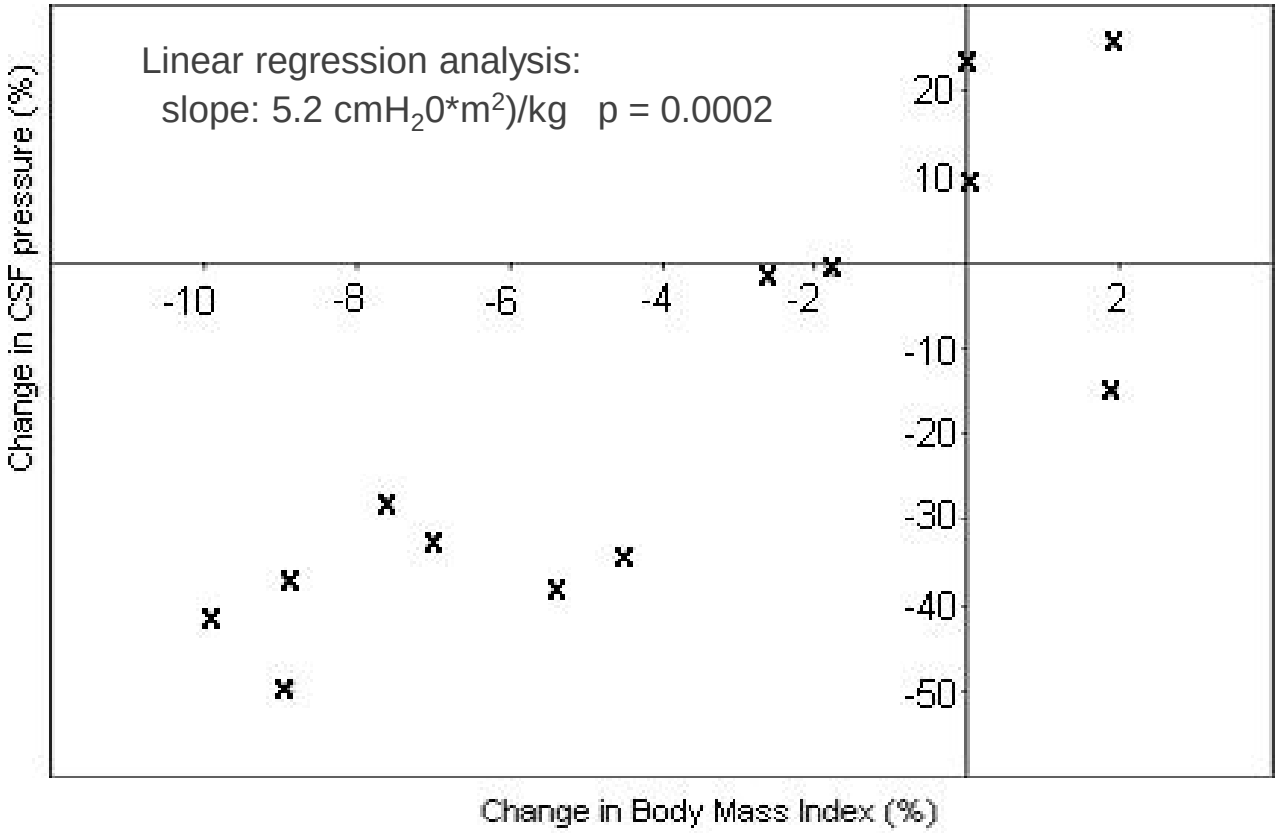
ICP vs RT
r = 0.6, p = 0.07

Skau et al 2010

ımark

GLOBAL EXCELLENCE IN HEALTH

Proportional change in BMI vs ICP



N = 13

Median ICP (cmH_2O)

baseline 31.0 (23.4->50)

final 24.0 (20.2-42.5)

$p < 0.02$

Mean BMI (kg/m^2)

baseline 33.4 (26.1-44.1)

final 31.1 (23.5-44.1)

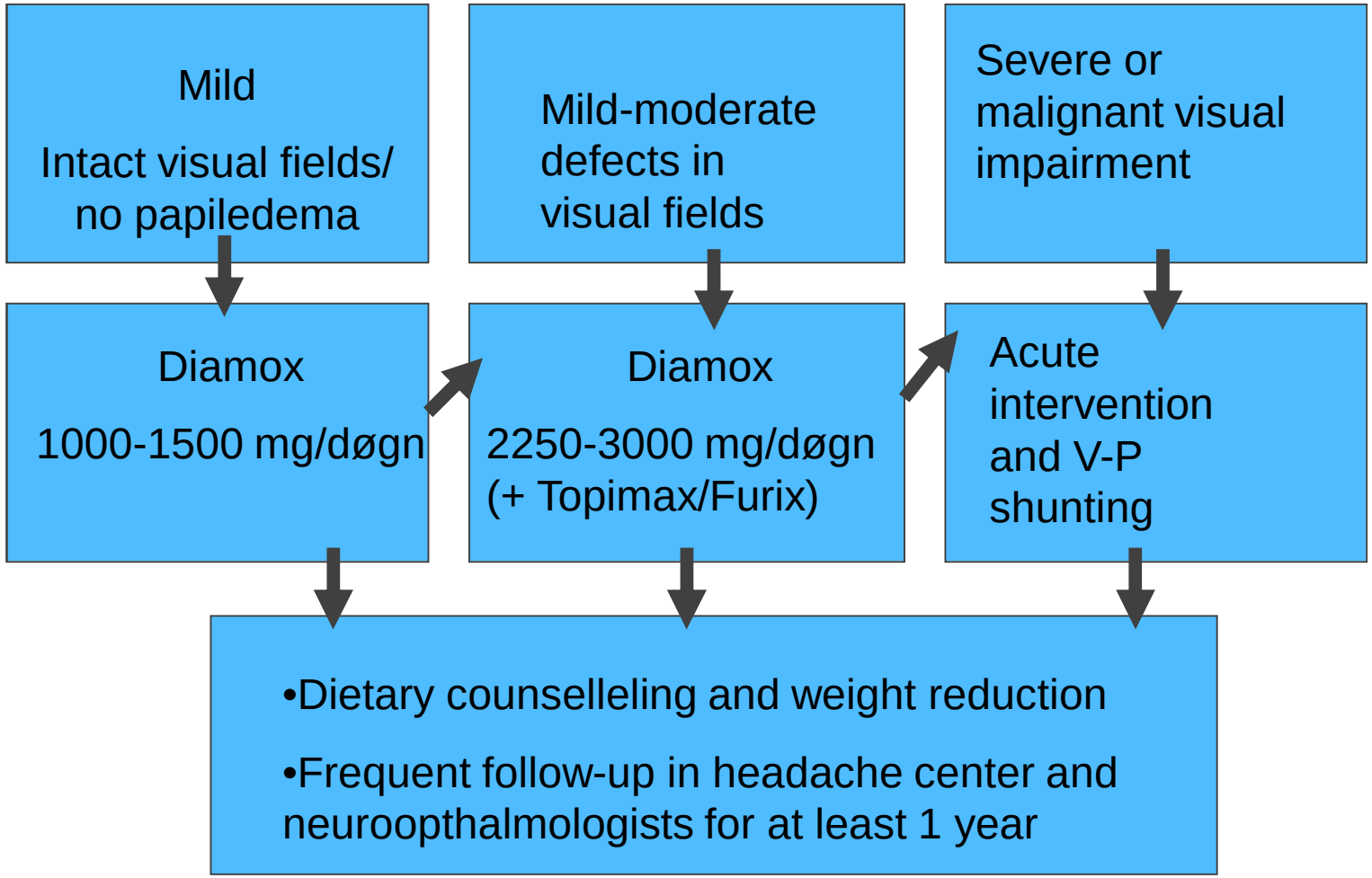
$p < 0.005$

Skau et al 2010

Idiopathic Intracranial Hypertension is not always Idiopathic

- Sinus venous trombosis
- Infections
- Inflammation
  - Sarcoidosis
- Endokrinological
  - parathyroid, thyroid, growth hormone, corticosteroid
- Neoplastic
- Uremia
- Toxic:
  - Tetracycline, steroids, vitamin A

Treatment strategy of IIH



DHC standard and Ball et al J Neurol 2011

Conclusions IIH

- The incidence of IIH is rapidly increasing in the wake of the obesity epidemics
- Exciting model of ICP regulation and headache?
- IIH (former benign intracranial hypertension) is not **benign** and not always **idiopathic**
- Active treatment with high doses of azetazolamid an weight loss are requiered
- Close follow up visits are needed to prevent relapse

"Medication-Overuse Headache" ICHD-III beta

... a system whereby medication overuse headache became a default diagnosis in all patients with medication overuse would encourage doctors all over the world to do the right thing, namely, to take patients off medication overuse as the first step in a treatment plan.

A. Headache present on ≥15 days/month

B. Regular overuse for >3 months of one or more acute/symptomatic treatment drugs as defined under sub form 8.2
   1. Ergotamine, triptans, opioids or combination analgesic medications on ≥10 days/month on a regular basis for >3 months

   2. Simple analgesics or any combination of ergotamine, triptans, analgesics, opioids on ≥10 days/ month on a regular basis for > 3 months without overuse of any single class alone

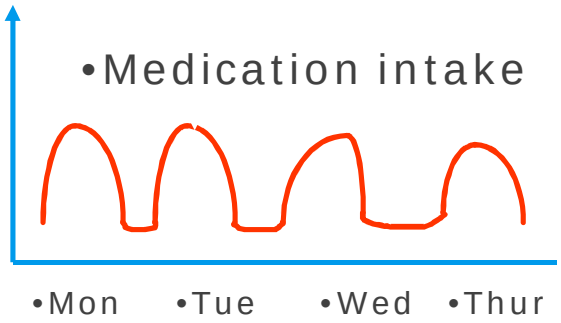
C. Not better accounted for by another ICHD-3 diagnosis
   (Headache has developed or markedly worsened during medication overuse)

Most important chronifying factor: medication overuse

- Chronic migraine
- Transformed migraine
- Chronic daily headache
- Chronic mixed headache
- Tension-type headache
- Post traumatic headache
- Post craniotomy headache

Clinical features (MOH)

Daily or almost daily headaches
Medication overuse



Dull diffuse headache

Mild to moderate

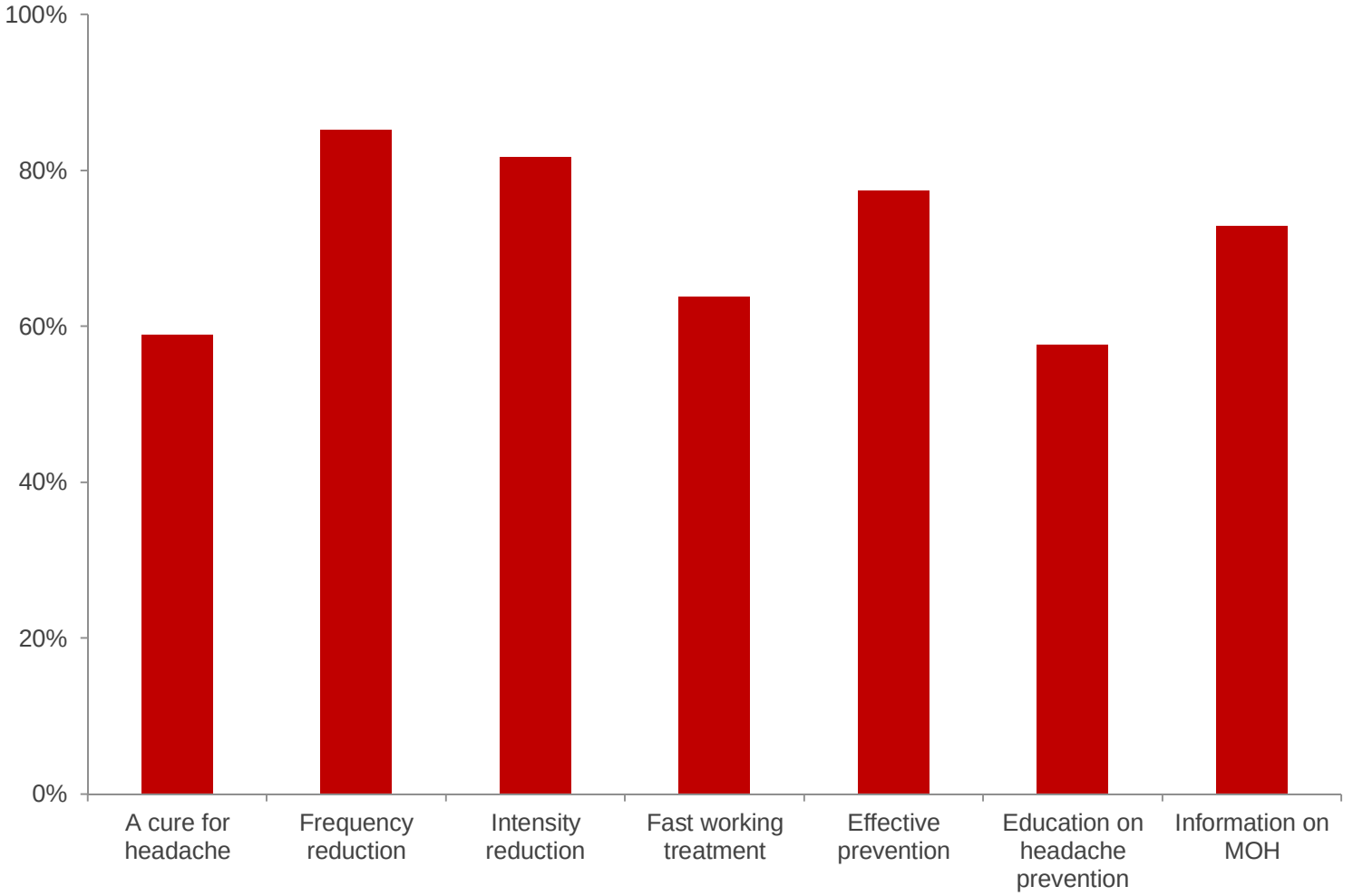
Holocranial

Without associated symptoms

Wake up with headache

Superimposed migraine like attacks

Headache Care: What does the patients want? MOH-Patient Expectations to treatment – A multicenter study.



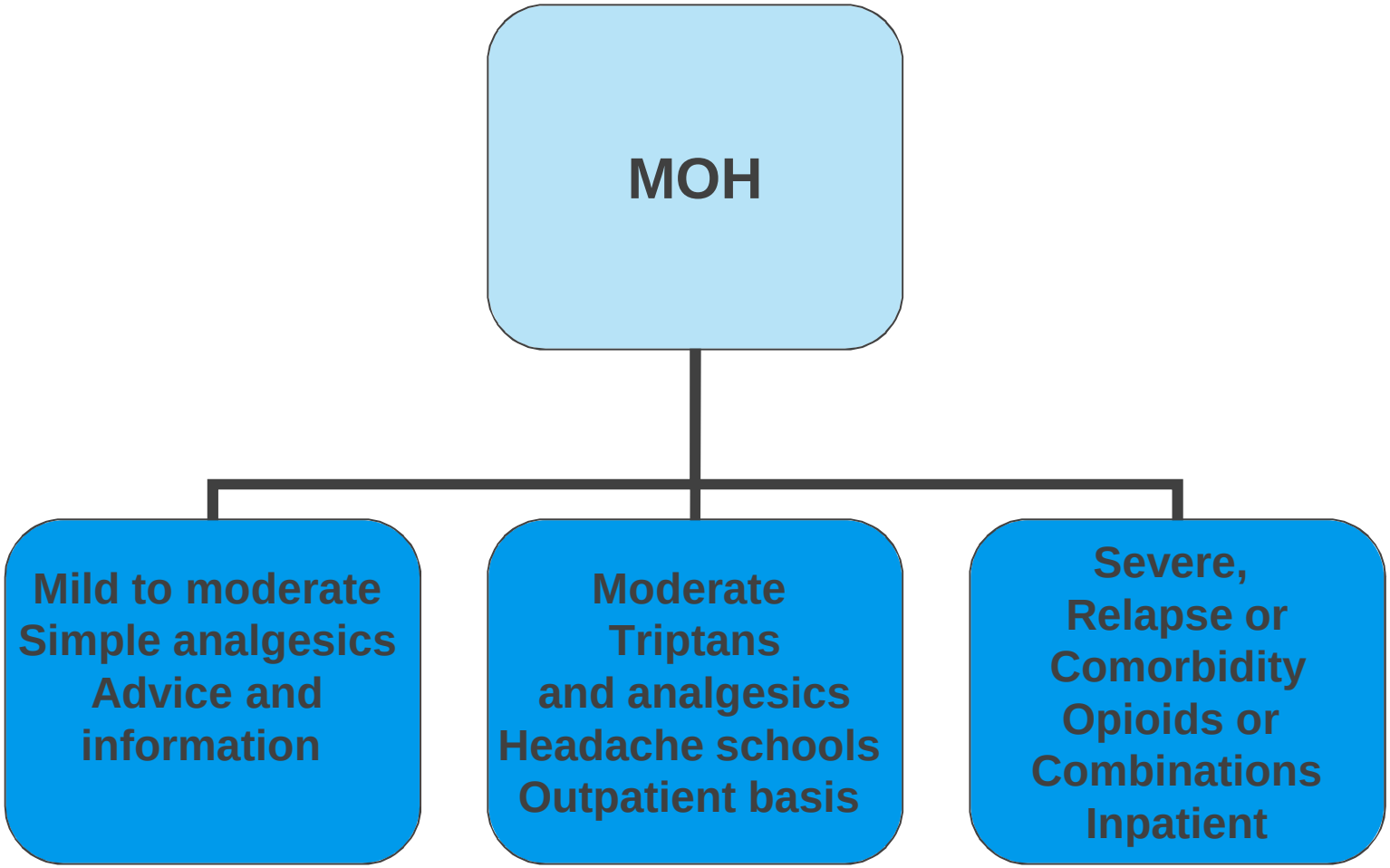
Munksgaard et al JHHP 2011

Medication overuse headache (MOH)

- 1 – 2% of the general population
- 20-30% in European Headache Centres
- 50-60 % in US Headache centers
- Favourable outcome by detoxifications in up to 60%
- ***BUT***
- To detox or not to detox?
- In-patient or out-patient basis?
- Little research on patients with treatment-resistant MOH
- Initial or delayed start of prophylaxis?

1) Stovner et al. 2008
2) Zeeberg et al 2006
3) Hagen et al. 2008
4) Evers, Jensen EFNS Guidelines 2011

Strategy for detoxification?



MOH in the general population

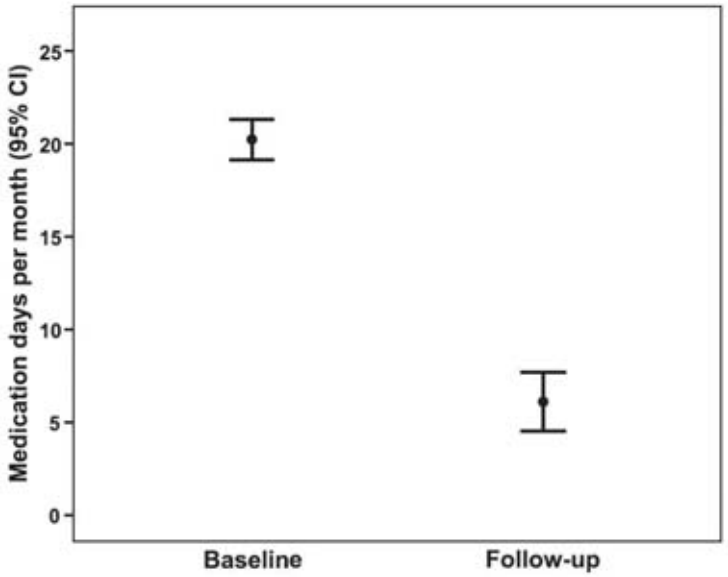
- Screening for MOH and chronic headache (1-2%)
- Simple verbal and written advice by neurologist
- N=109 patients
- Average duration of chronic headache 15.5 yrs
- Follow-up after 1.5 years:
- Headache frequency: 22 days/mth

- *Grande et al Eur J Neurol 2011*

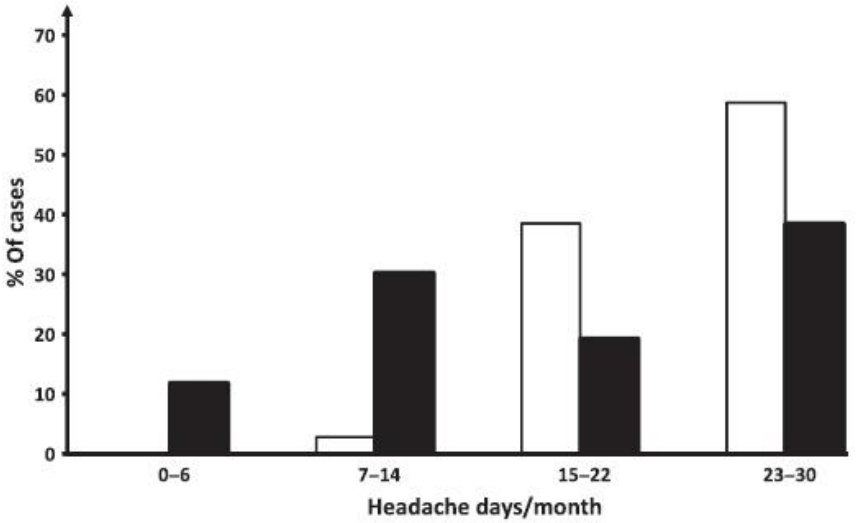
Medication and headache days

Mean follow-up-time was 1½ years

Mean medication days



Headache days per month



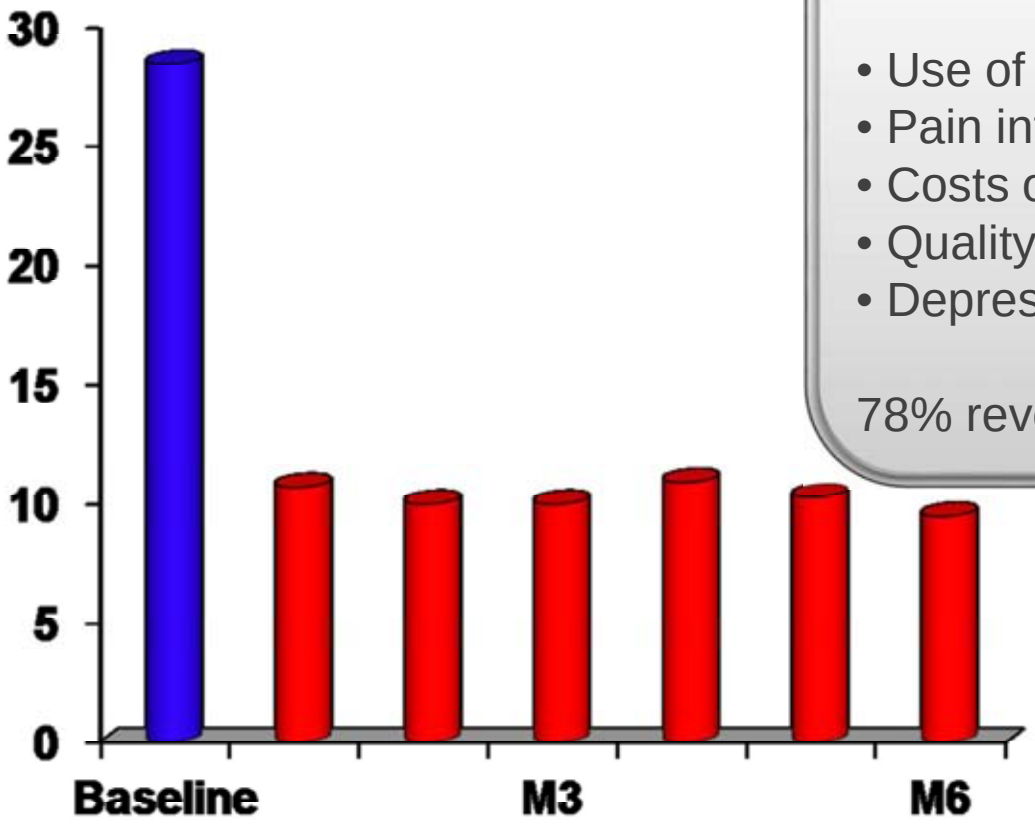
baseline (open bars) and follow-up (filled bars)

Cured from MO: 76%
Reversed to episodic headache: 42%
Frequency reduction: 22 to 6 days pr mth

Grande et al. Eur J Neurol 2011

Headache in the clinical population (frequency before and after detoxification and preventives in MOH (N= 651))

Headache days /month



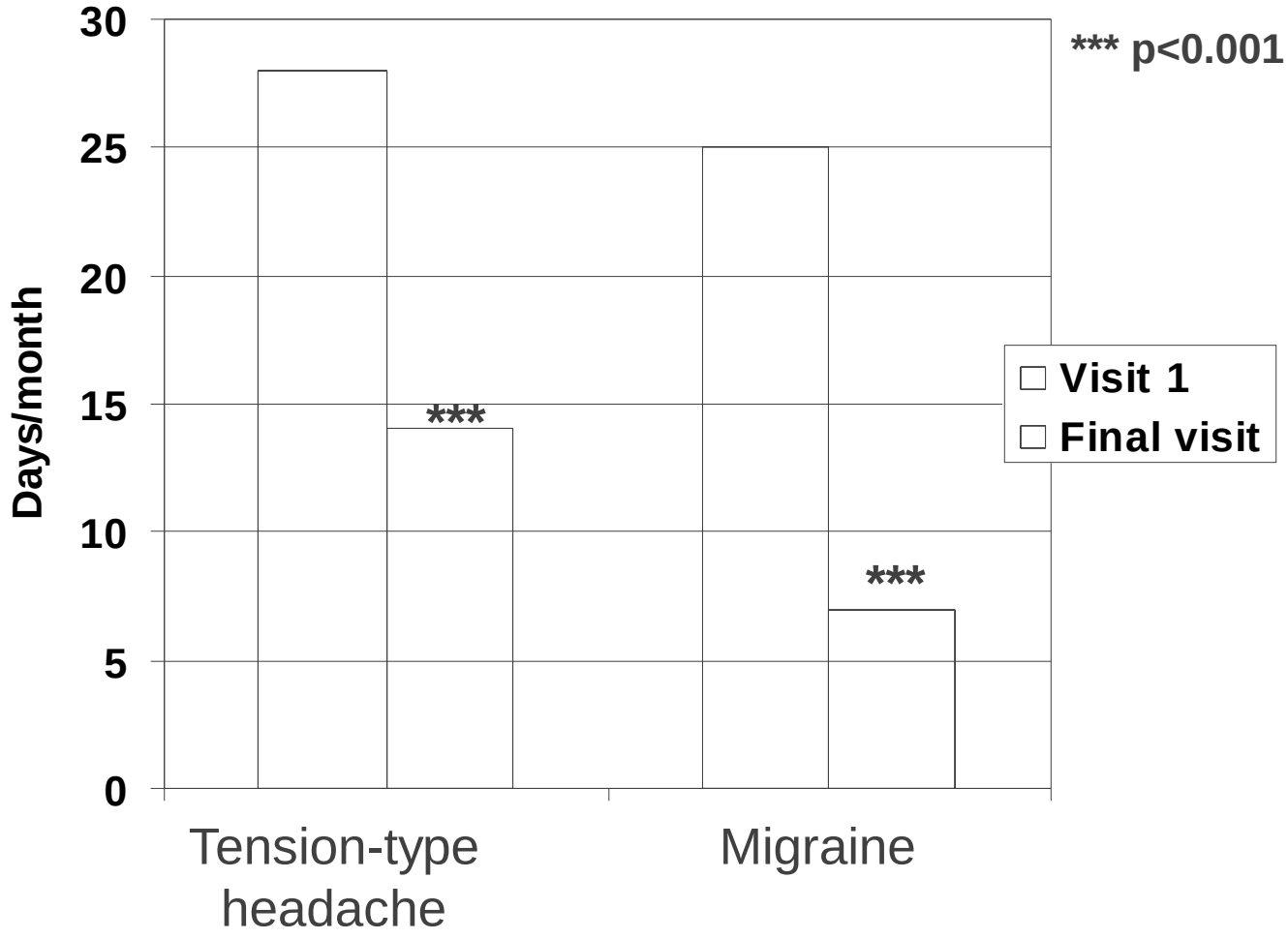
Highly significant effect on:

- Use of medication
- Pain intensity and duration
- Costs of health care
- Quality of life
- Depression and anxiety

78% reverted to episodic headache

Tassorelli et ComoEstas study group 2013

Effect of detoxification in a headache center

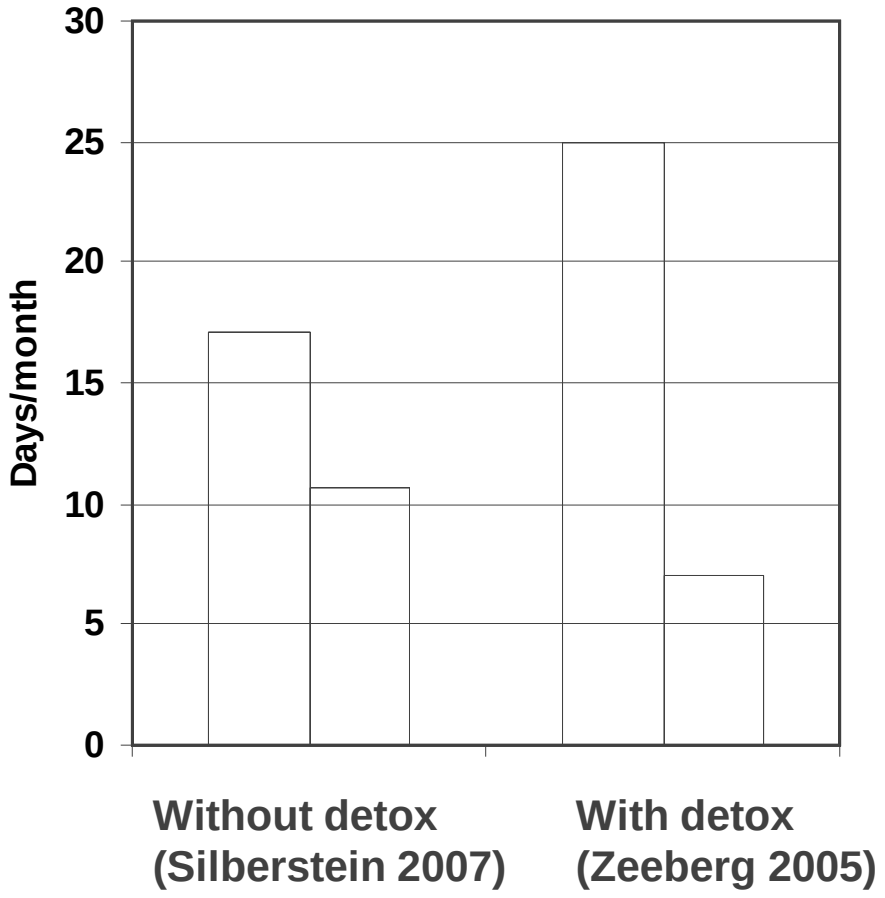


•***Zeeberg et al 2005***

Topiramate in ”chronic migraine” without detox

- Diener et al Cephalalgia 2007;27:814-823. 24 topiramate and 13 placebo completers, 78% MOH. Difference between active and placebo 20%
- Silberstein et al Headache 2007;47:170-180 and 2009;49:1153-1163. Same material. 92 topiramate and 90 placebo completers i.e. 55% completers. Effect size 10%. Medication use unchanged
- 3 months follow up

Effect of prophylaxis without and with detox



Treatment

- 98 patients
- MOH during 5 years
- Unsuccessfully treated by neurologists

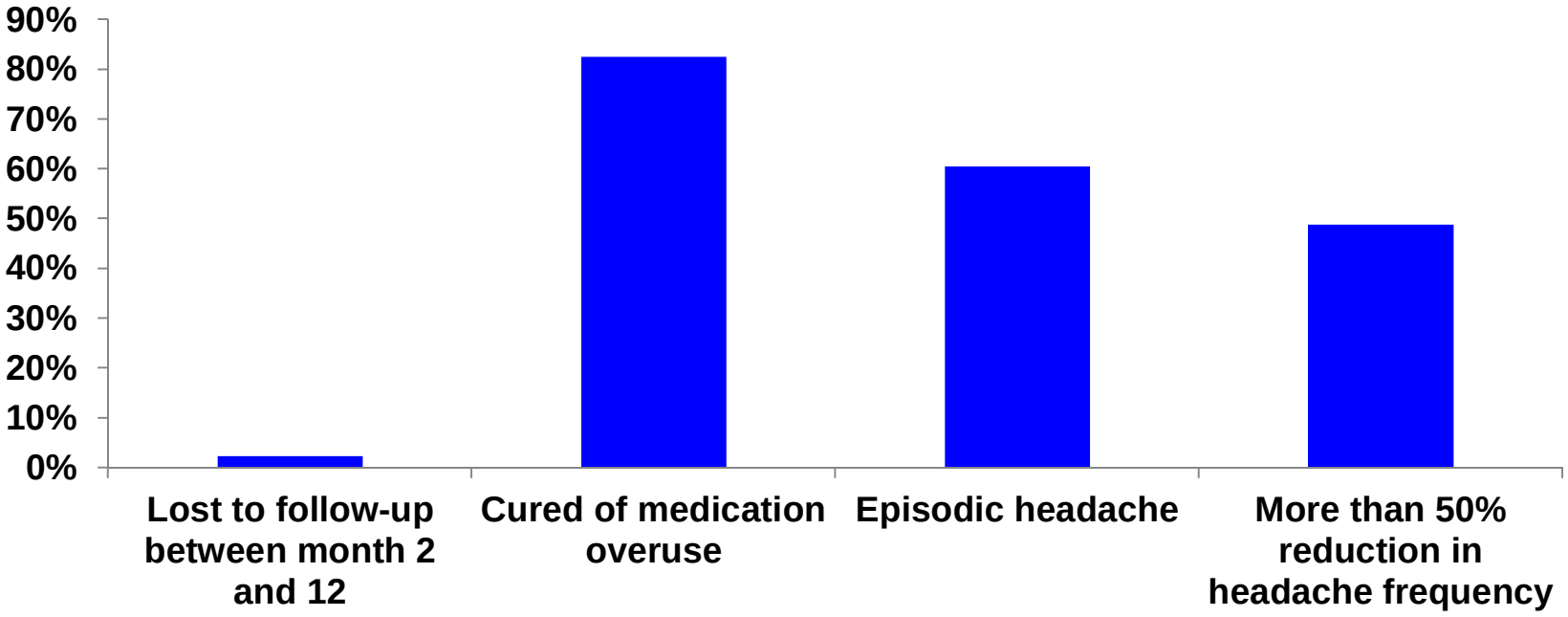
Aims

- Test 2 treatment regimens on:
- outcome
- use of medication,
- relapse,
- quality of life and
- cost-effectiveness

A	B
Individual based	Group-based, multidisciplinary
Prophylactics from Day 1	Prophylactics after 2 months if needed
Symptomatic medication 2 days/week	No symptomatic medication for 2 months
1-year follow-up	1-year follow-up

Results – in total

- 90% completed 2 months withdrawal
- 83% remained cured of MOH after 12 months

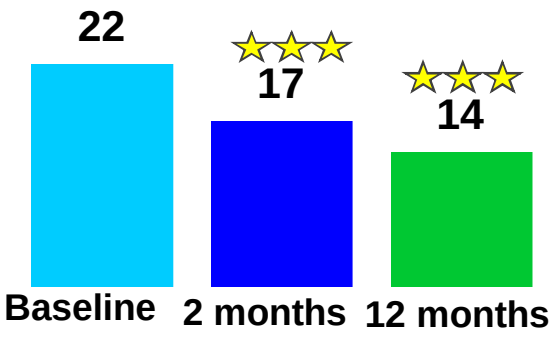


Munksgaard et al. Headache 2012

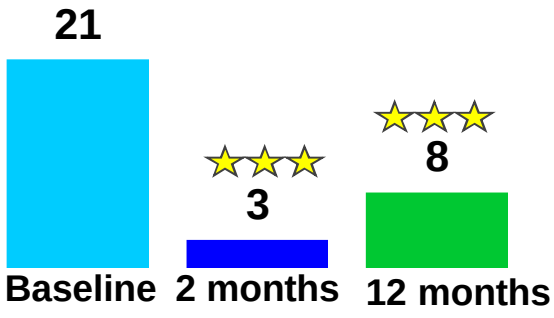
Results-in total

- 39% reduction in headache frequency (p<0.001)
- 63% reduction in medication use (p<0.001)

Headache frequency, days/4 weeks



Frequency of medication intake, days/4 weeks

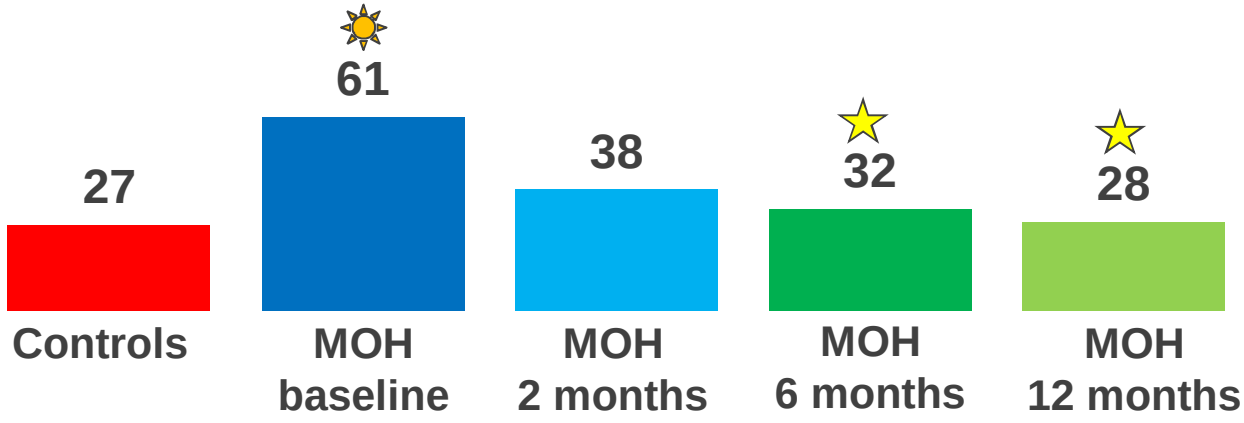


Munksgaard et al. Headache 2012

Results – pressure pain

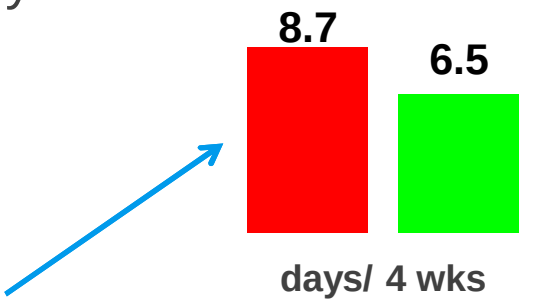
- pain thresholds: MOH < healthy (p<0.05)

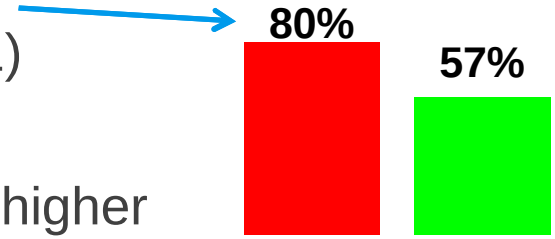
- suprathreshold pain (Pain scores 100 mm VAS):
- no difference extracephalic localisations
- cephalic locations:
  - ☀ MOH patients > healthy (p<0,05)
  - ★ baseline > 6 months and 12 months (p<0,05)



12 months follow-up after detoxification with or without prophylaxis from start
Munksgaard et al Cephalalgia 2012

- Similar headache frequency, duration, intensity and relapse rate in group A and B
- Group B: Higher quality of life (MIDAS)
- Group B: Reduced use of analgesics (p=0.02)
- Group B: Reduced use of prophylaxis (p=0.01)
- Group A: High use of health care service and higher number of visits in the headache centre (p<0.01)





Conclusions

- Very important to identify and treat secondary headaches
- IIH is not benign and the incidence is rapidly increasing
- Detoxification is rewarding and very effective in MOH
- MOH prevention is crucial
- Specific treatment for most sec. headaches and RCT's are lacking

the migraine trust

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