# Neurophysiology of insomnia

**A basis for neurofeedback application**

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# (Primary) Insomnia

Insomnia is a sleeping disorder characterised by (DSM-IV-TR, 2000):

- Sleep-onset or sleep maintenance complaints, and/or decreased sleep quality, presenting itself for at least one month
- Significant impairment in daytime functioning
- No other psychological, psychiatric or medical problems that can cause the insomnia complaint

# Behavioral perspective

- Trait and precipitating factors can result in acute insomnia
- Development of maladaptive coping strategies can give rise to subacute insomnia

↓

conditioned arousal and chronic insomnia

# Behavioral perspective

1. Physiological Arousal

   Physiological hyperarousal during the sleep onset period (SOP) and/or sleep (Vgontaz et al., 2001; Rodenbeck et al., 2002) and even daytime functioning (Bonnet & Arand, 1995)
   → body temperature, cortisol levels, metabolic rate,...etc.

2. Cognitive Arousal

   Intrusive (often negative) cognitions, which often lead to a disrupted sleeping pattern (Wicklow & Espie, 2000)

# Paradoxes of insomnia (1)

Perception of polysomnographic (PSG) sleep:

Objective PSG sleep ≠ subjective report of sleep

Sleep onset latency (SOL) is often overestimated in the subjective report compared to the PSG data

Total sleep time (TST) is often underestimated in comparison to the PSG data (Perlis et al., 2001; Edinger & Fins, 1995)

Interference mesograde amnesia? (Perlis et al., Phys & Behav, 2001)

# Cortical arousal

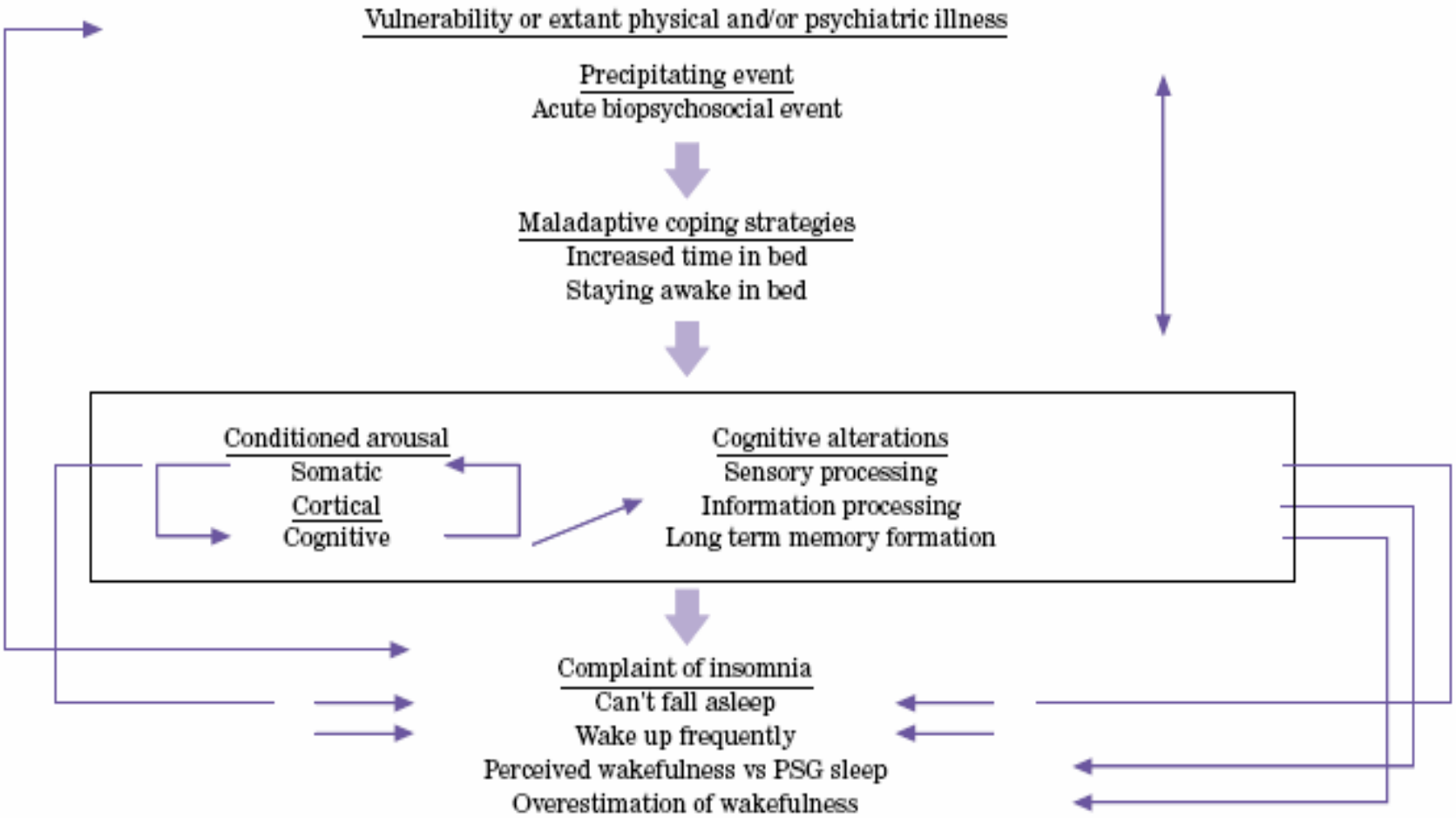
Cortical Arousal:

Heightened beta/gamma power during SOP and sleep
(Lamarche & Ogilvie, 1997; Merica et al., 1998; Perlis et al., 2001)

→ affects mesograde amnesia related to the sleep onset → sleep and wakefulness are more difficult to distinguish → explanation for paradoxes

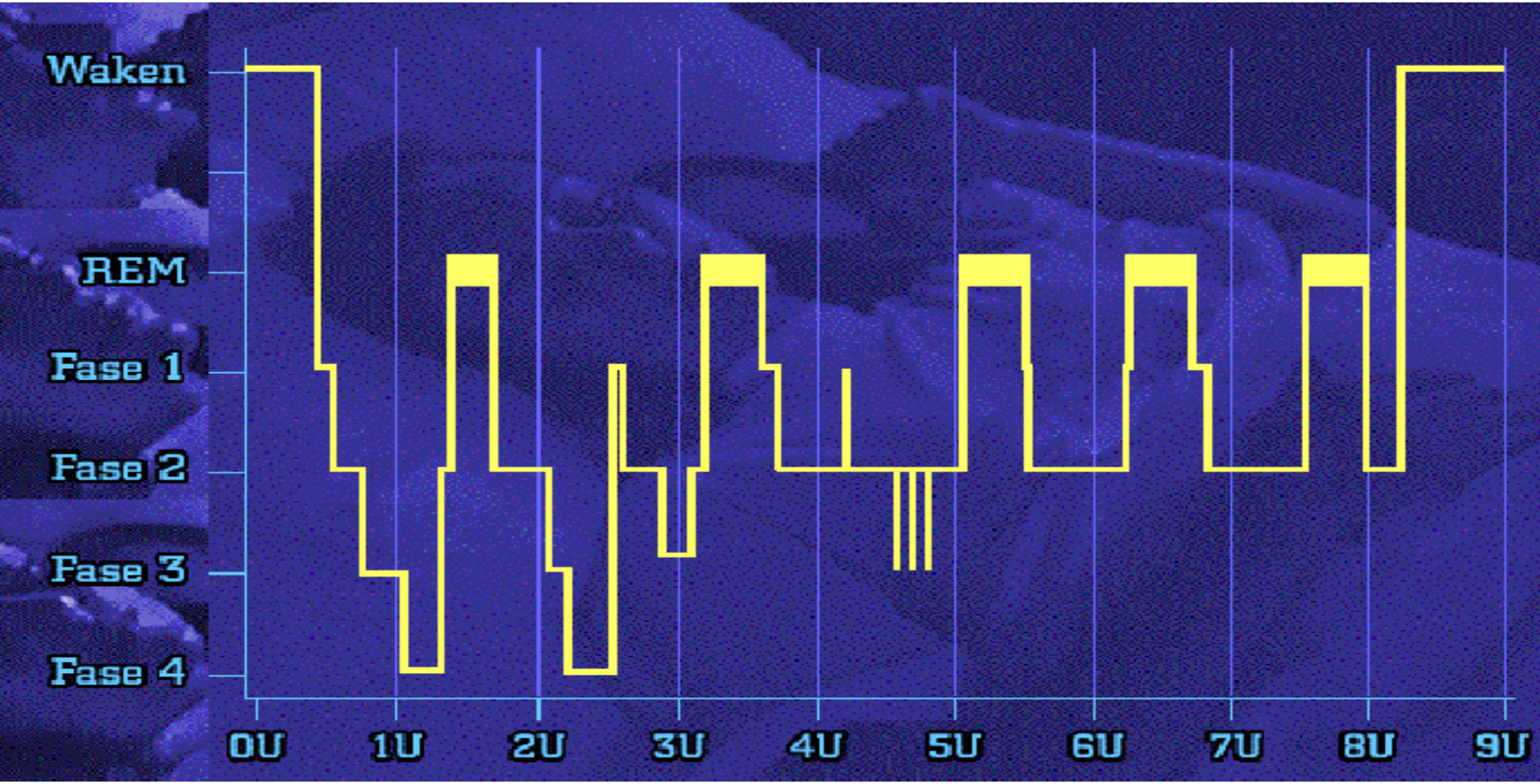
→ Neurocognitive perspective
(Perlis et al., J Sleep Res, 1997)

# Neurocognitive perspective




*(Perlis et al., J. Sleep Res., 1997)*

# Sleep EEG characteristics

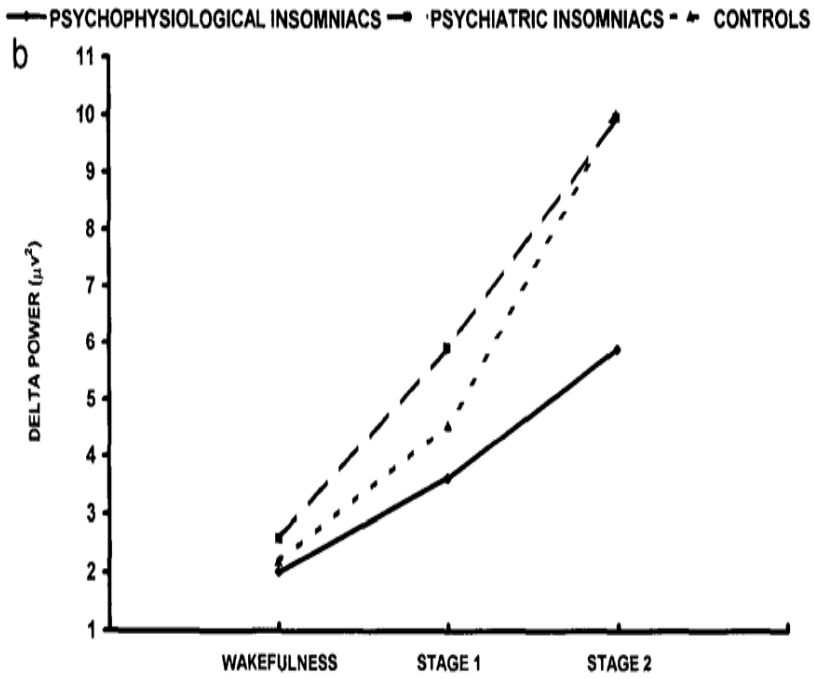


# Sleep EEG characteristics

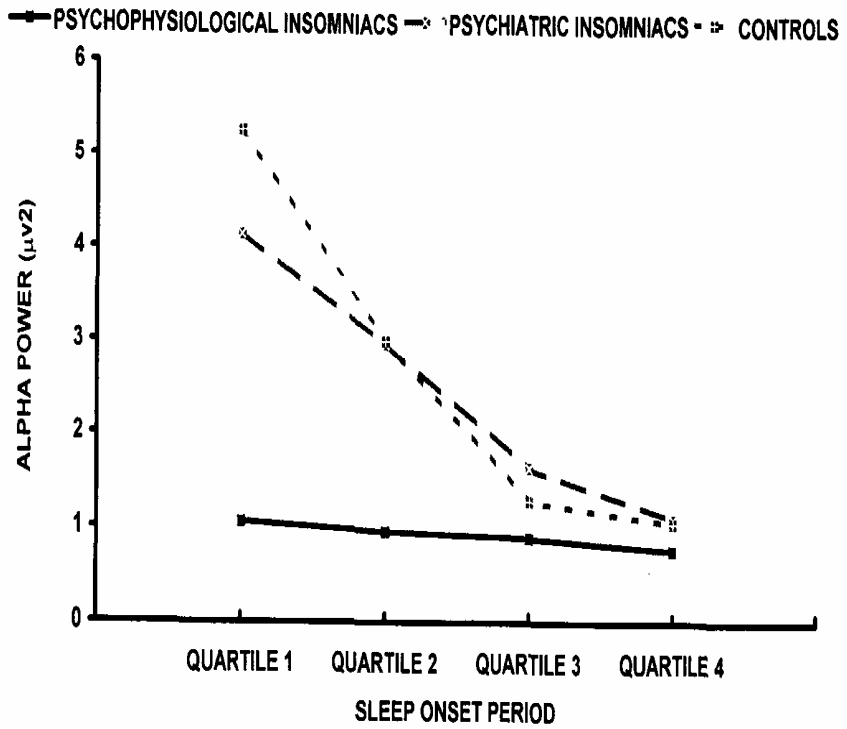
During the SOP (sleep onset period) and PSG sleep there is a significant difference in the EEG of insomnia patients in comparison to healthy sleepers or comorbid (secundary) insomnia

# Sleep EEG characteristics: Sleep onset period (SOP)

Delta power 1 – 4 Hz



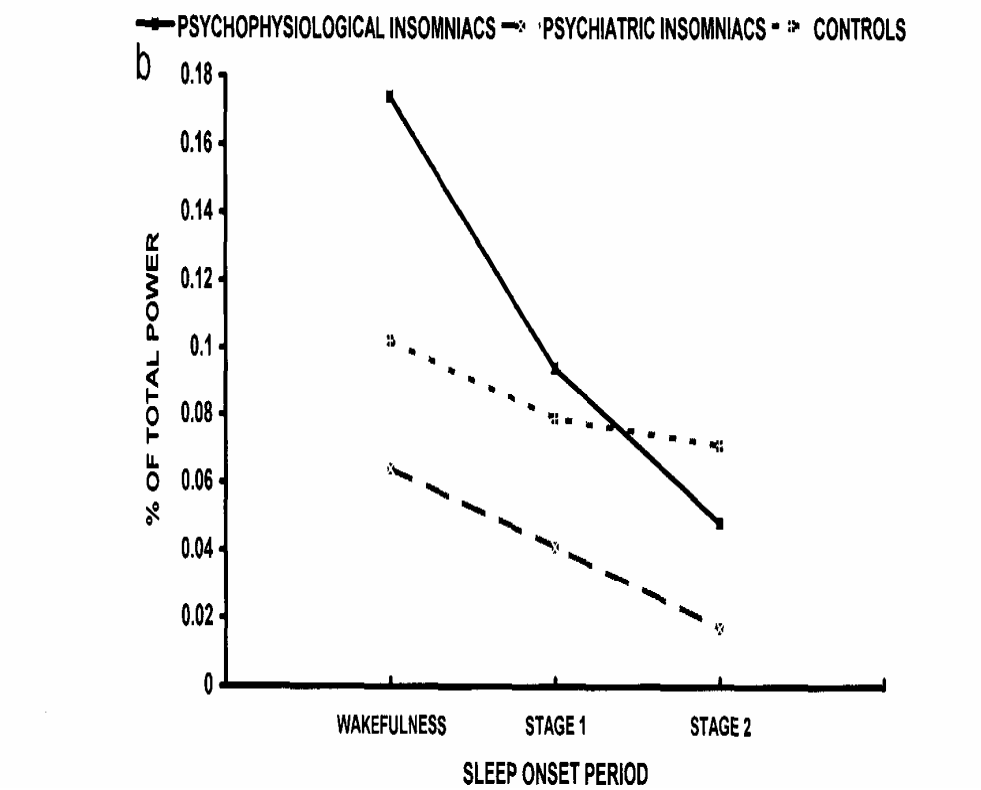

Alpha power 8 – 12 Hz




*(Lamarche & Ogilvie, Sleep, 1997)*

# Sleep EEG characteristics: Sleep onset period (SOP)

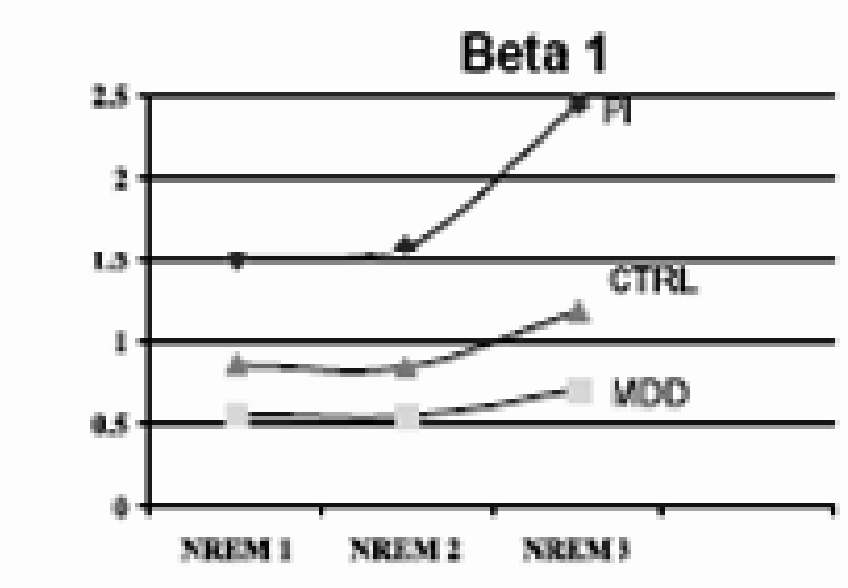
Beta power 15 – 25 Hz



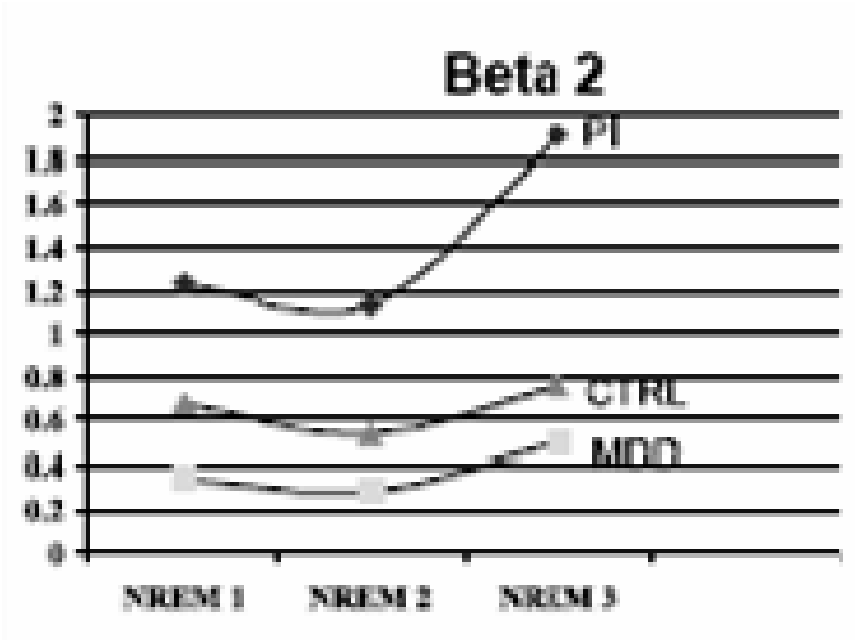

*(Lamarche & Ogilvie, Sleep, 1997)*

# Sleep EEG characteristics: NREM sleep

Beta 1: 14 – 20 Hz



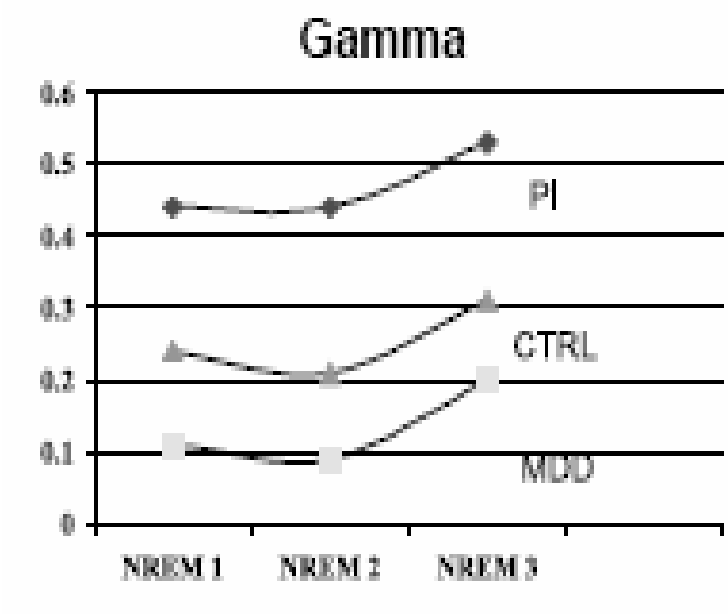

Beta 2: 20 – 35 Hz




*(Perlis et al., J. Sleep Res., 2001)*

# Sleep EEG characteristics: NREM sleep

Beta 3: 35 – 45 Hz




*(Perlis et al., J. Sleep Res., 2001)*

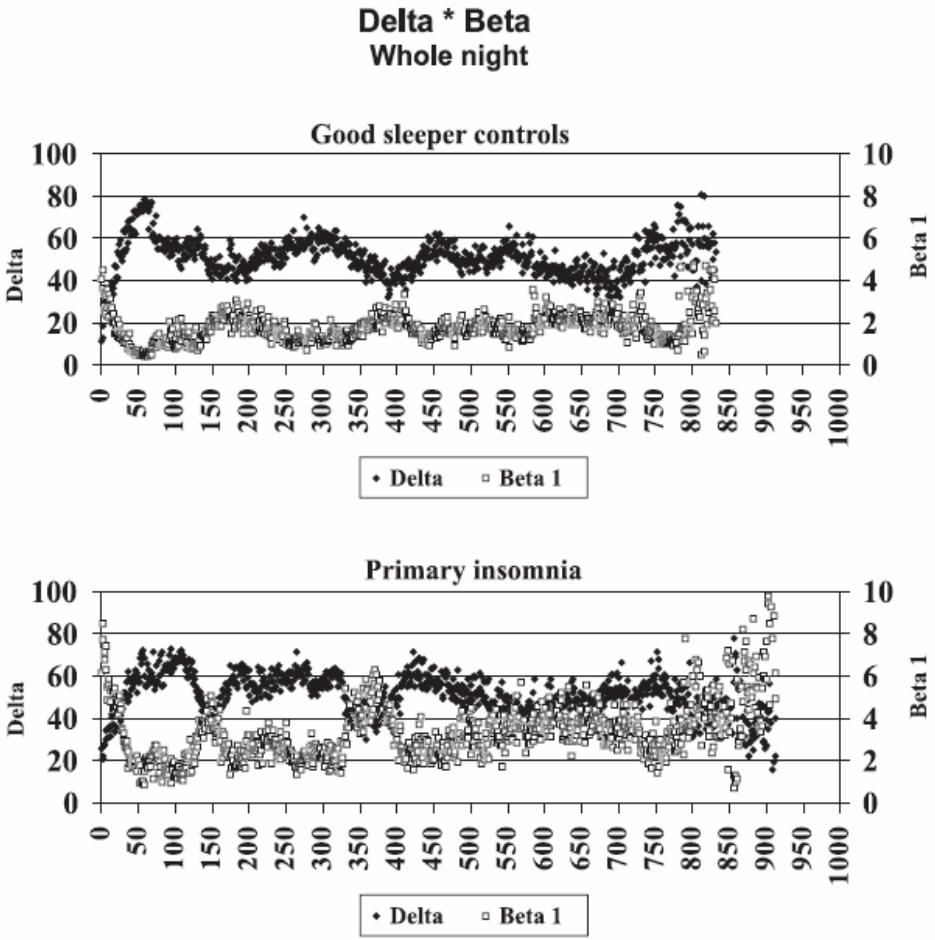
# Sleep EEG characteristics: REM sleep

- Deficit in delta and theta power
- Increased alpha power
- Increased sigma and beta power

➔ **SOP, NREM and REM: opposite EEG pattern in insomniacs**

(Merica et al., Eur J Neurosc, 1998)

# Sleep EEG characteristics: PSG sleep




- The inverted relation between delta and beta power observed in normal sleepers, disappears in the second part of the night in insomnia patients

*(Perlis et al., J. Sleep Res., 2001)*

# Treatment implications

- Pharmacotherapy: acute or short-term/transient insomnia. Not recommended for chronic insomnia (Holbrook et al., Can Med Assoc J, 2000; Smith et al., Am. J. Psychiatry, 2002)

- Cognitive-behavioural therapy (CBT): efficient for chronic insomnia (Morin et al., Sleep, 1999; Cervena et al., J. Sleep Res., 2004). Limitations:
  - Compared to the applications in other disorders, the effect sizes are moderate
  - +/- 20% of insomnia patients do not respond well enough to CBT

  → Focus on somatic or cognitive arousal not enough?!

# Neurofeedback

- SMR (12-14Hz) training in cats resulted in changes in sleep EEG: ↑ sleep spindle bursts, quiet sleep and ↓ motor excitability (Sterman et al., Science, 1970).
  ➔ intervention on wake EEG resulted in changes in sleep EEG.

- Thalamus plays an important role in the production of several EEG frequencies and information processing (Steriade et al., Science, 1993; Coenen, Neurosci Biobehav Rev, 1995). Regulates most neural input to cerebral cortex.

# Neurofeedback and insomnia

- Insomnia
  - Possible 24-hour CNS disorder → hyperarousal
  - Cortical hyperarousal during sleep: ↑ beta/gamma power
  - Disruption in information processes

- Neurofeedback
  - Intervention on the level of the CNS
  - More specific on the thalamocortical networks → important role in sleep and arousal (McCormick & Bal, Annu Rev Neurosc , 1997)
  - Resulting in improvement of cognitive functioning (Egner & Gruzelier, Neuroreport, 2001;Vernon et al., Int J Psychophys, 2003; Egner & Gruzelier, Clin Neurophys, 2004)

# Neurofeedback and insomnia (2)

- Hauri (1981): Frontal EMG / EMG-theta / SMR
  - Training on only 1 frequency band (4-8 Hz or 12-14 Hz)
  - No difference in PSG improvements between treatment groups
  - Correlation between baseline tension level and EMG-theta or SMR treatment outcome
- Hauri et al. (1982): replication theta versus SMR training
  - Same experimental conditions, except for EO in both protocols
  - Identical results (see supra)

# SMR protocol vs EMG training in Insomnia

## Objectives

- Application of neurofeedback protocol focussed on information processes and cognition
- Training at home
- Tele-neurofeedback: using internet connection

# Method

- 14 insomnia patients; 5 females, 9 men
- Extensive testing and psychiatric interview
- 2-week sleep diary and actigraphy
- 1 PSG pre- and posttreatment using Embla equipment and Somnologica Science® software

# Method

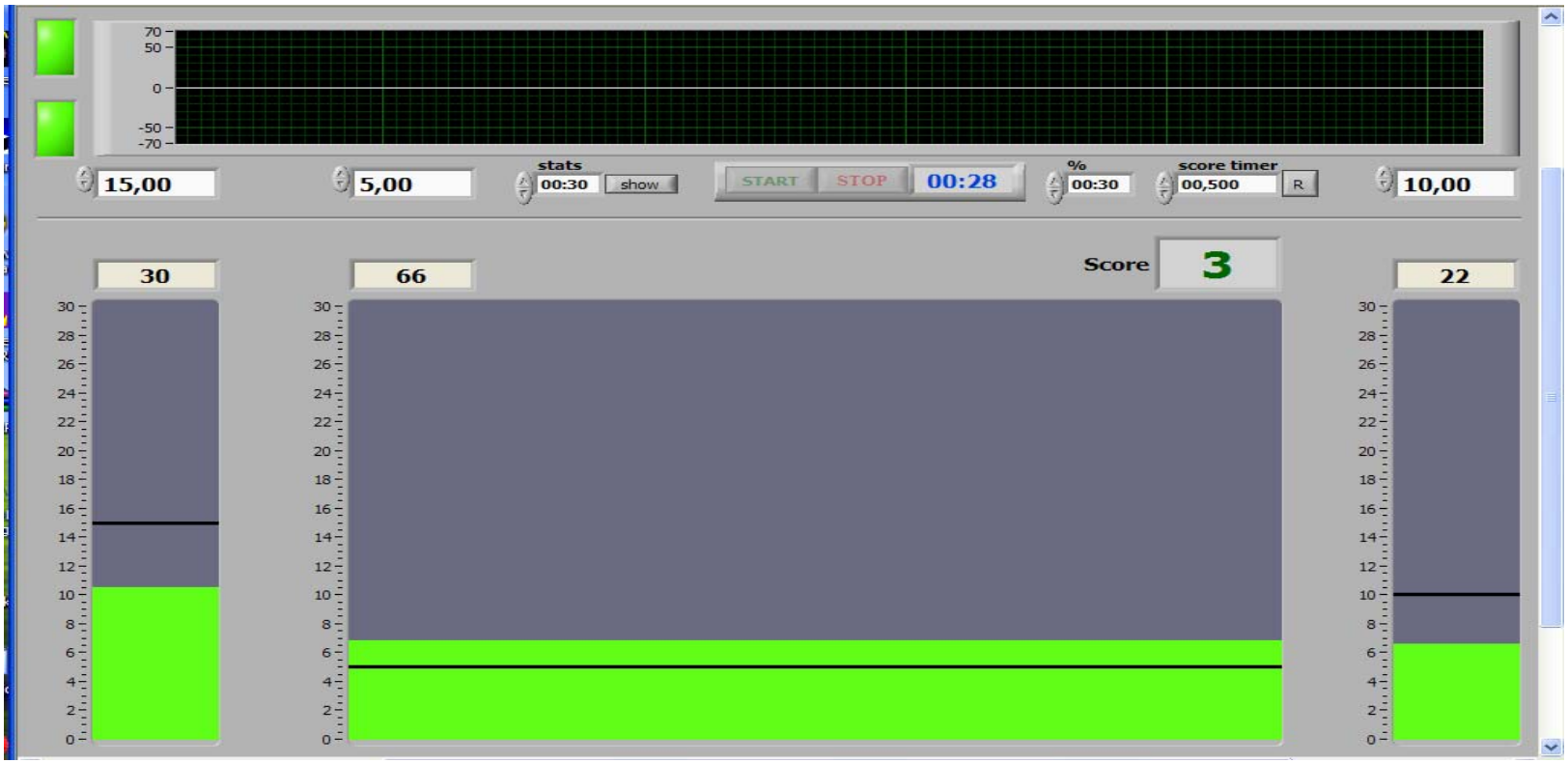
- A 19 lead EEG measurement using A.N.T. Equipment and Eemagine® software pre and posttreatment.
- EOG and EMG → visual artefact rejection
- Impedances were kept below 10 kOhm

# Method

Neurofeedback training:

- All participants received a training in electrode placement.
- Electrode placement: Cz-A2 (SMR), Fpz-A2 (Frontal EMG)
- 20 sessions over a period of 8 weeks. Alternating 2 or 3 sessions a week.
- Group 1 (n=8): inhibiting theta (4-8 Hz) and high beta (20-30 Hz), and reïnforcing SMR (12-15 Hz). ≈ Egner T., Zech T.F., Gruzelier J.H., Clin Neuroph, 2004
- Group 2 (n=6): Frontal EMG training ≈ relaxation training
- Randomised, single-blind, controlled study

# Method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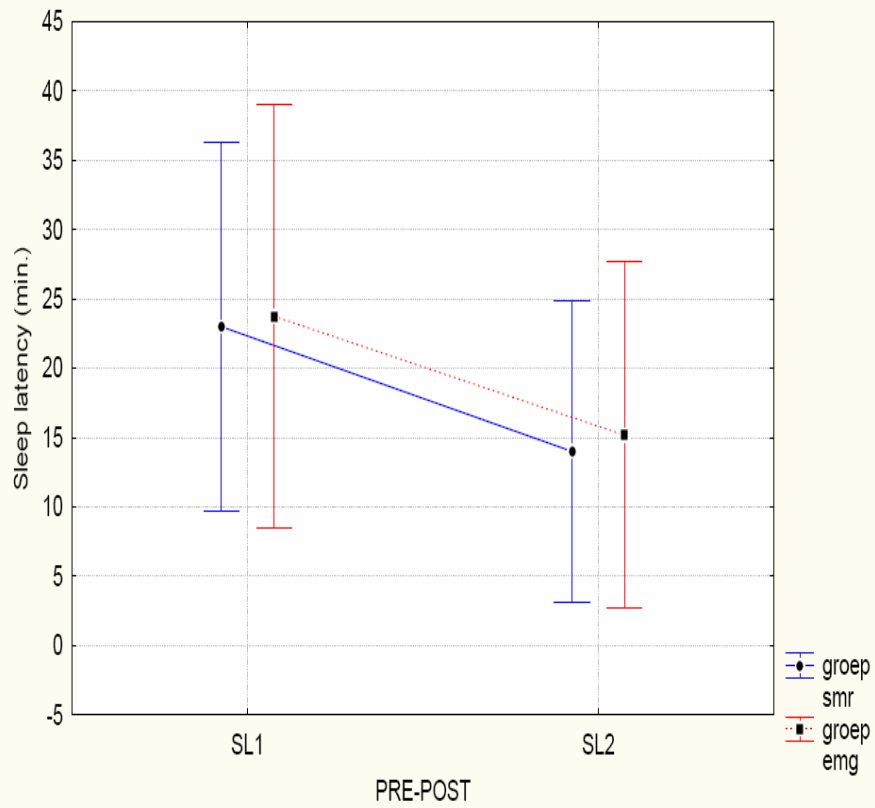
22

# Method

- Impedances were kept below 10 kOhm during neurofeedback training
- Neurofeedback sessions were performed at home, through a secured internet connection(vpn-connection and VNC software).
- Personal Efficiency Trainer (PET®) EEG equipment was used for neurofeedback sessions (Brainquiry n.v.)
- Software was developed by Brainquiry
- Statistica (Statsoft® v7.1): General linear model (GLM) repeated measures ANOVA

# Results (1)

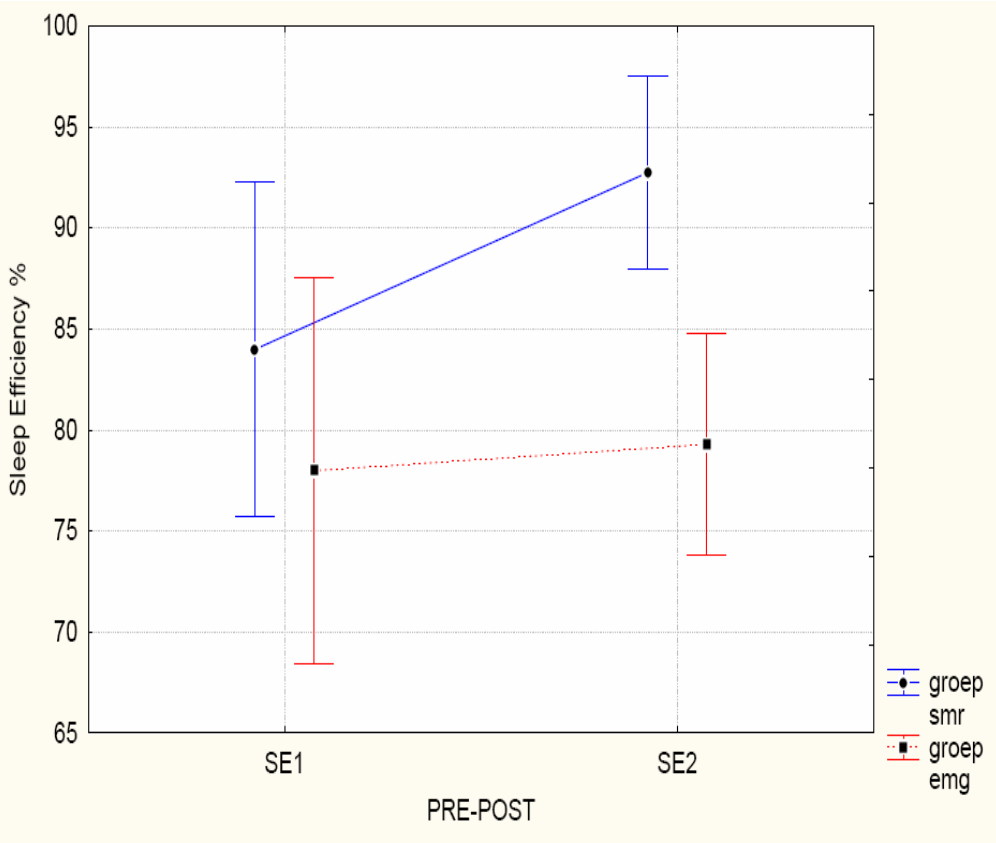
Sleep latency




- An overall improvement in sleep latency ($F(1,12)=6,46$; $p.<.05$)
- No significant difference in sleep latency between both treatment groups

# Results (2)

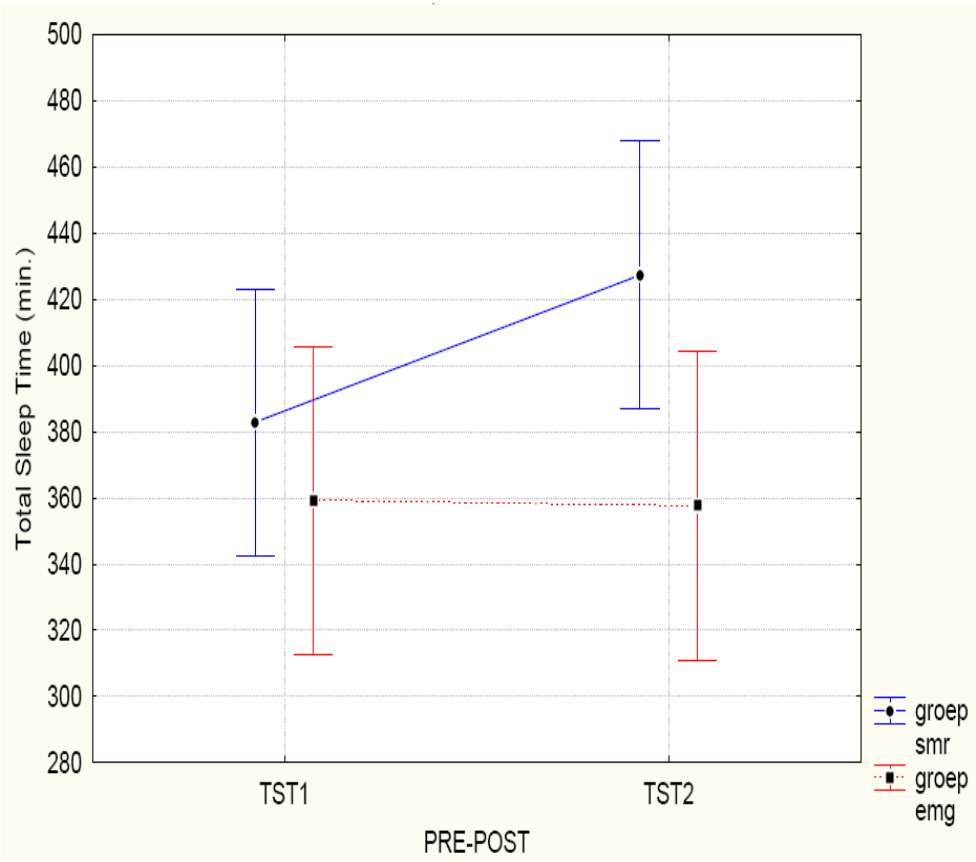
Sleep efficiency




- Significant main effect for treatment group ($F(1,12)=5,39$; $p.<.05$).
- No main effect pre post
- No interaction effect

# Results (3)

Total sleep time




- No significant main effect
- Significant interaction effect ($F(1,12)=4,94$; $p.<.05$)
- Post Hoc Tukey: Significant improvement in TST after SMR training ($p.<.05$)

# Results

- No drop outs occured during the 12 week study
- No significant side-effects were reported

# Conclusion

- Succesfull application of tele-neurofeedback training.
- The SMR protocol has significant effects on total sleep time in comparison to EMG biofeedback.
- Sleep latency is positively influenced by both trainingprotocols
- Limitations:
  - No habituation PSG night
  - Small sample size

# To be continued...

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