

PSYCHOLOGICAL PAIN TREATMENT IN FIBROMYALGIA: SYSTOLIC EXTINCTION TRAINING (SET) RESTORES BAROREFLEX SENSITIVITY, REDUCES PAIN SENSITIVITY AND CLINICAL PAIN REPORT

K. Thieme^{1,4}, PhD, H. Kraemer², MD, U. Koehler³, MD, T. Meller¹, MA, R. Malinowski¹, MA, W. Maixner⁴, DSS, PhD, R.H. Gracely⁴, PhD
¹Department of Medical Psychology, Phillips-University of Marburg, Germany, ²Hospital of Neurology, Univ. of Giessen, Giessen, Germany, ³Sleep Med., Philipps Univ. of Marburg, Marburg, Germany, ⁴Ctr. for Pain Res. and Innovation, Univ. of North Carolina, Chapel Hill, NC, USA

INTRODUCTION.

Important components of intrinsic pain regulatory systems are modulated by cardiovascular dynamics that influence baroreceptor sensitivity (BRS). The present study evaluated the effects of extinction training combined with electrical stimulation administered during either the systolic or diastolic phases of the cardiac cycle delivered in a randomized order ("systolic extinction training", SET) in patients with fibromyalgia syndrome (FMS). SET was compared to treatment with extinction training combined with electrical stimulation delivered independent of the cardiac cycle a "placebo" condition (PC).

METHOD.

Forty patients who fulfilled the American College of Rheumatology criteria for FMS and showed an elevated blood pressure response to a laboratory stressor termed hypertensive stress reactivity were randomly assigned to SET (n = 20), or PC (n = 20). Assessments of clinical pain, pain threshold, pain tolerance, baroreflex sensitivity (BRS), blood pressure and heart rate, cognitive and behavioural variables as well as sleep architecture and sympathetic outflow measured by microneurography were performed pretreatment and post-treatment as well as at 6 to 12 months posttreatment.

Design of Systolic Extinction Training - SET

10 Sessions with 2 hours in 5 weeks

Hour 1: Structured Extinction Training with Training of Perception and Increase of physical Activity
Hour 2: BRS modifying-Stimulation by delivering pain-free and two different pain stimuli adjusted by individual pain tolerance dependent on cardiac cycle

RESULTS.

Session	T1	T2	T3	T4
1	0	0	0	0
2	0	0	0	0
3	0	0	0	0
4	0	0	0	0
5	0	0	0	0
6	0	0	0	0
7	0	0	0	0
8	0	0	0	0
9	0	0	0	0
10	0	0	0	0
11	0	0	0	0
12	0	0	0	0
13	0	0	0	0
14	0	0	0	0
15	0	0	0	0
16	0	0	0	0
17	0	0	0	0
18	0	0	0	0
19	0	0	0	0
20	0	0	0	0

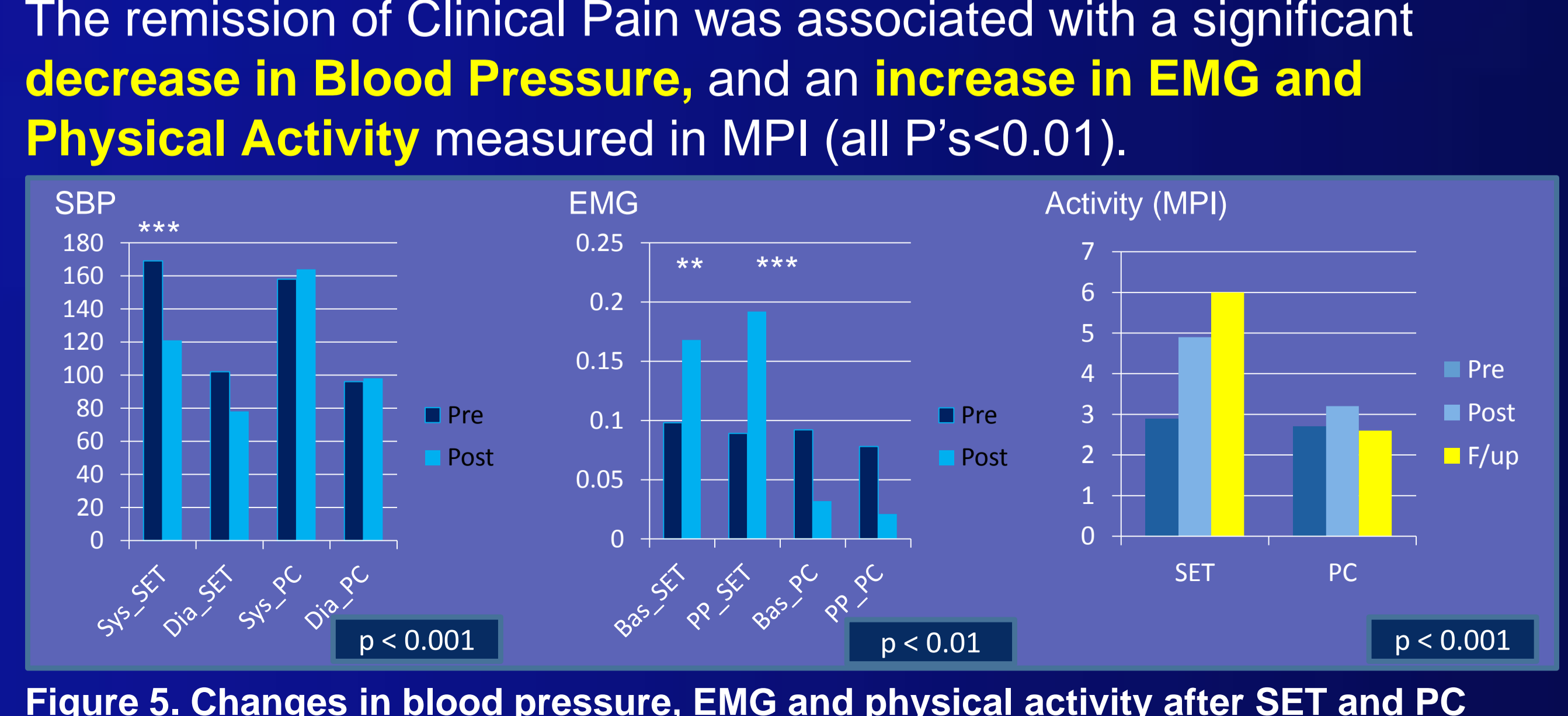
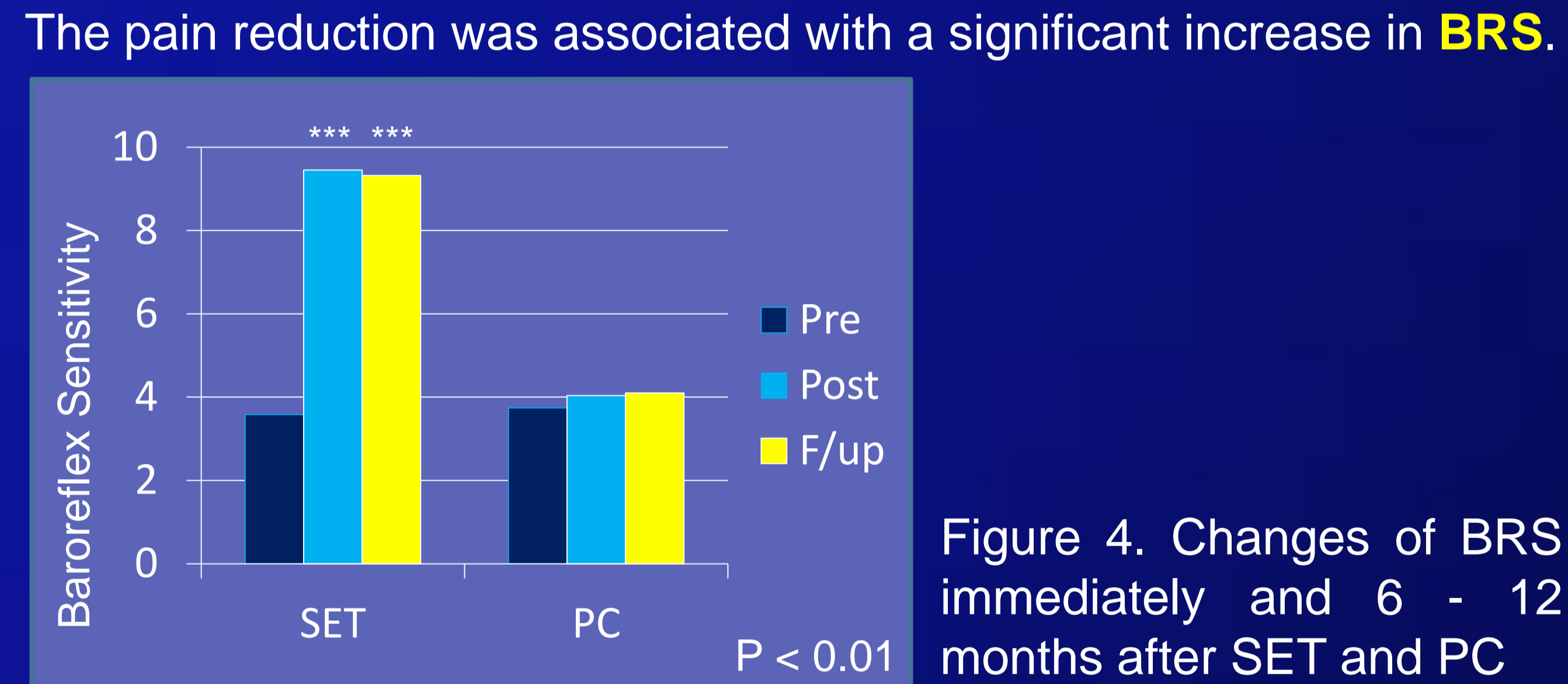
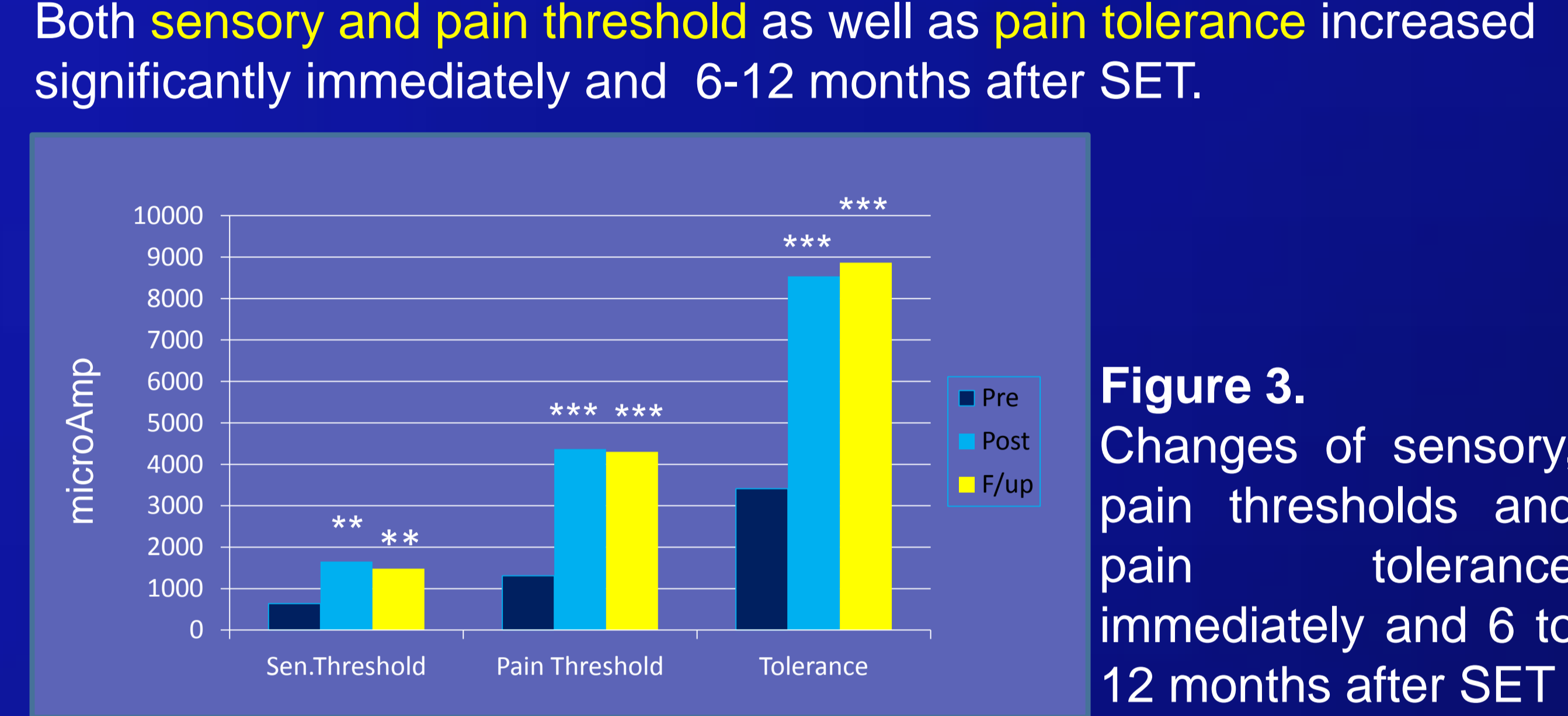
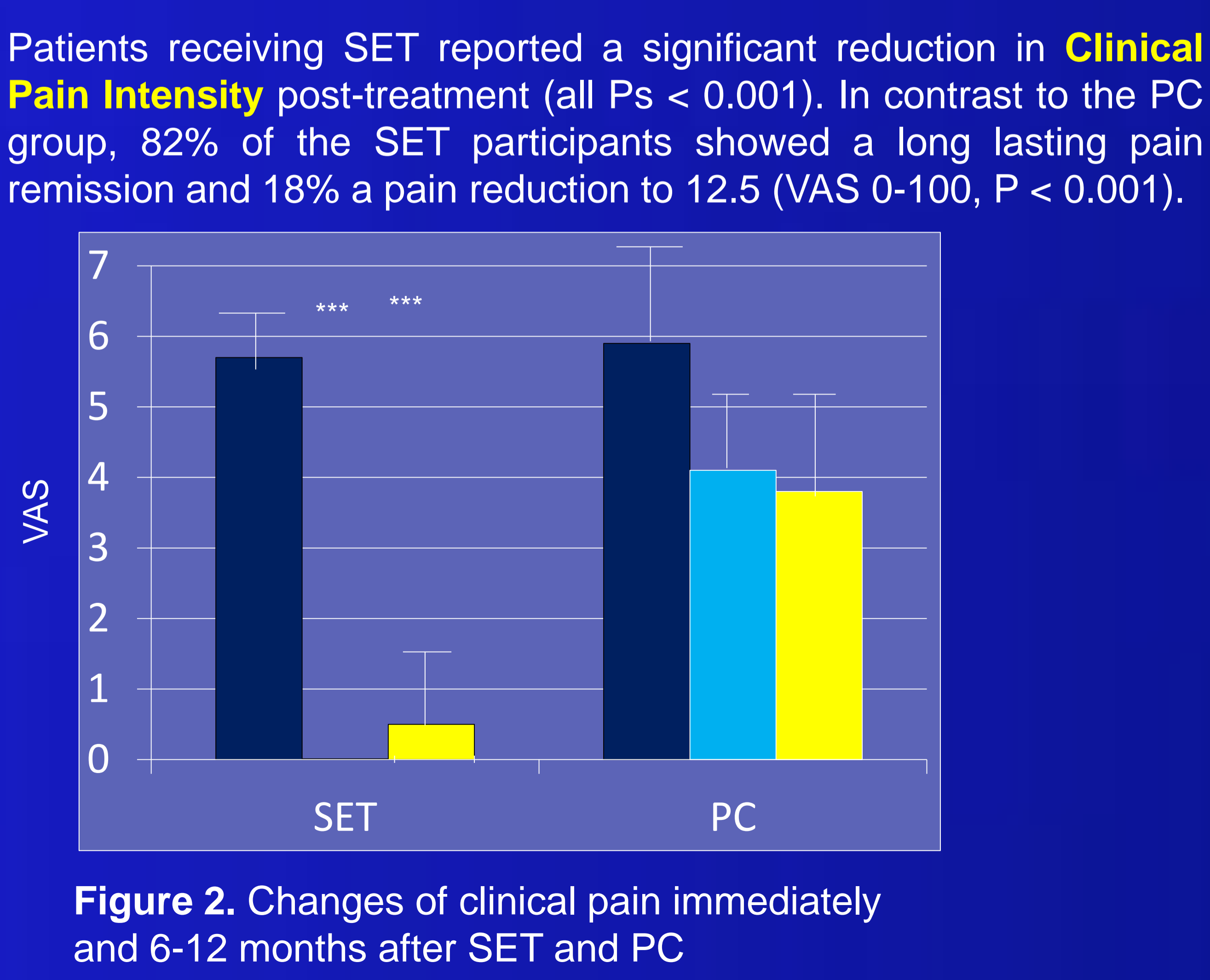
BRS-modifying Stimulation

- Assessment of ratings to define sensory and pain threshold as well as pain tolerance
- Calculation of 50% and 75% of pain tolerance
- First 8-minutes stimulation
- Assessment of ratings
- Renewed calculation of 50% and 75% of pain tolerance
- Second 8-minute stimulation
- Final assessment of ratings

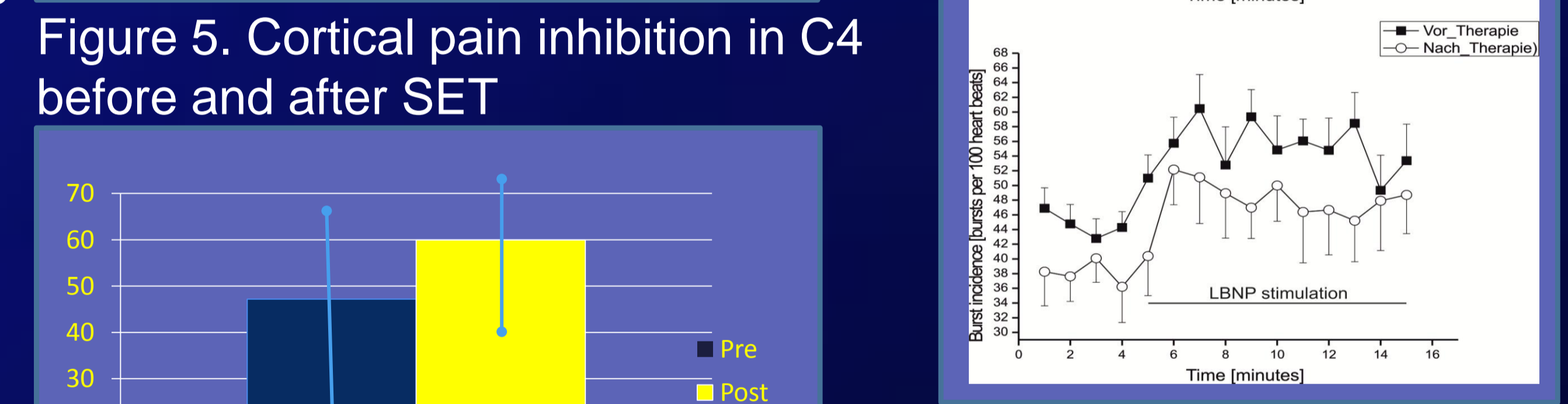
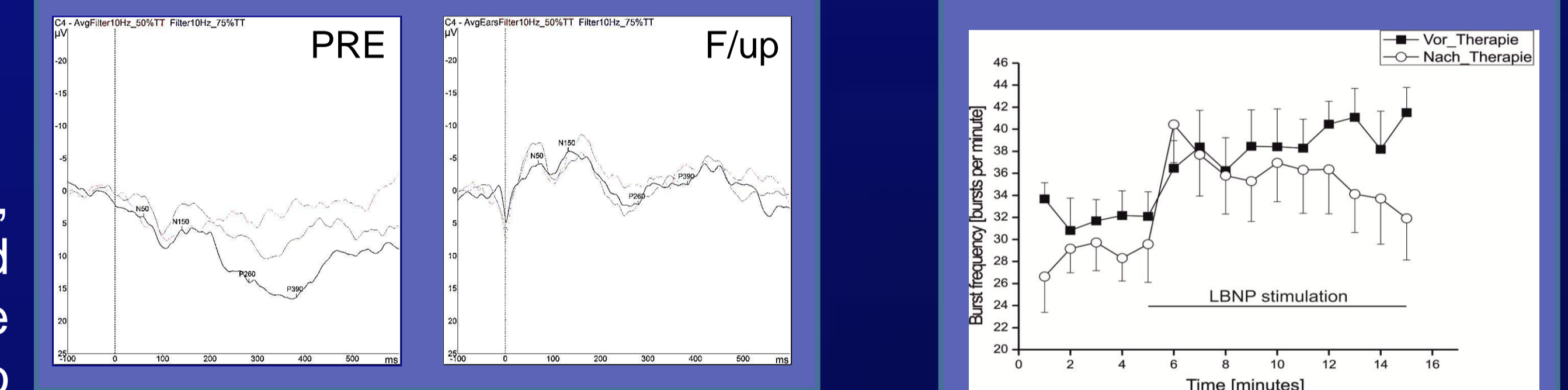
Session	T1	T2	T3	T4
1	0	0	0	0
2	0	0	0	0
3	0	0	0	0
4	0	0	0	0
5	0	0	0	0
6	0	0	0	0
7	0	0	0	0
8	0	0	0	0
9	0	0	0	0
10	0	0	0	0
11	0	0	0	0
12	0	0	0	0
13	0	0	0	0
14	0	0	0	0
15	0	0	0	0
16	0	0	0	0
17	0	0	0	0
18	0	0	0	0
19	0	0	0	0
20	0	0	0	0

Table 1. A single SP-protocol of Session 1 of SET

Table 2. A single SP-protocol of Session 10 of SET



In addition, the SET group demonstrated statistically significant higher improvements in central functions (all Ps < 0.001) as well as an improvement in sleep architecture (P < 0.01) and a reduction in sympathetic outflow (P < 0.01) compared the PC group.



CONCLUSION:

These results suggest that SET is effective in treating patients with FMS producing long lasting pain remission. Furthermore the findings show that the (1) greater blood pressure responses to stress predicts a greater reduction in clinical pain report and (2) increase in physical activity observed following SET is associated with a restoration of BRS, cortical pain inhibition, sympathetic outflow and sleep architecture. Even though baseline BRS is diminished in FMS patients, electrical stimulation delivered in a manner dependent on cardiac cycle phase when combined with extinction training is highly effective in reducing pain and restoring functions in a FMS subgroup characterized by hypertensive blood pressure stress reactivity.

Supported by German Research Foundation TH 899/7-1 and NIH R01AR054895-01A1