

Inhibition of Fibromyalgia Pain

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Background: Pain sensitivity is influenced by baroreflex sensitivity (BRS), involving nucleus tractus solitarius (NTS) mediated inhibition of pain, hypertension and unrefreshed sleep. In contrast to healthy individuals (HC), FM does not show the inverse relationship between BP and pain. Cardiac gated noxious and non-noxious electrical stimuli may normalize this FM dysfunction.

Methods: 30 pain-free normotensive HC and 30 FM experienced two 8-minutes-trials of randomly ordered non-painful and moderate and strong painful electrical stimuli to the fingers immediately *after* the systolic and diastolic peak (experimental protocol), and in a control condition with the same stimuli delivered independently of the cardiac cycle. Clinical pain, sensory, pain threshold, and pain tolerance were assessed before and after the trials. Blood pressure (BP), BRS, and evoked potentials were measured throughout the trials.

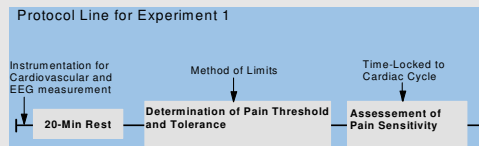


Figure 1: SP - Protocol

Event	Trigger	Delay	Duration mSec	Time on mSec	Time off mSec	Amp	Pause Sec
1	1	20%	250	7	20	DT	5
2	1	80%	250	7	20	DT	5
3	1	20%	250	7	20	HTT	5
4	1	80%	250	7	20	HTT	5
5	1	20%	250	7	20	TT	5
6	1	80%	250	7	20	TT	5
7							15

The table is performed 1 times with a pause of 15 sec. between each run and will be repeated 10 times.

$$30 \times 11 + 15 \times 10 = 330 + 150 = 480 \text{ seconds} = 8 \text{ minutes.}$$

The length of the stimuli is 250 millisecond and is given each 5 seconds. It characterizes a long-term-depression-design.

Analysis: Before, between and after the test trials, clinical pain and measures of sensory and pain threshold, as well a pain tolerance to electrical stimuli, were assessed. Blood pressure (BP), BRS, and evoked potentials were measured throughout the session.

Result1. Compared to the diastolic peak, N50, N150 and P260 evoked potentials were attenuated during the systolic phase in HC ($p < 0.005$) but not in FM.

Result 2. BRS was diminished in FM compared to HC ($p < 0.01$).

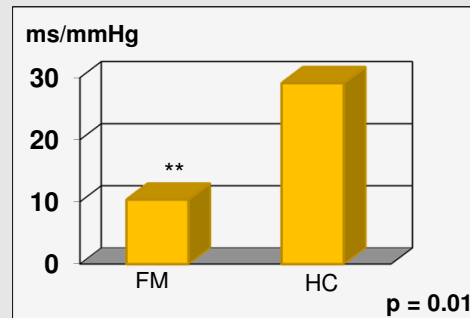
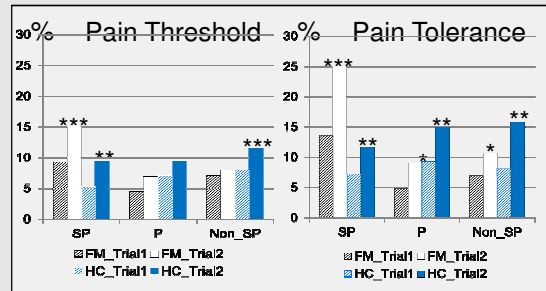


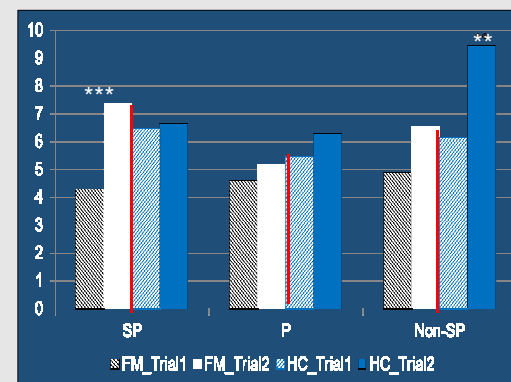
Figure 2: Differences in BRS between FM and HC

Result 3. Pain threshold and tolerance values increased by 14.4% and 24.6% in FM during the SP protocol and correlated with increases in BP and BRS and decreases in clinical pain report (all p 's < 0.01). In contrast, during the P protocol pain and tolerance thresholds were associated with greater N150 activity but not greater P260 and P390 activity or blood pressure.

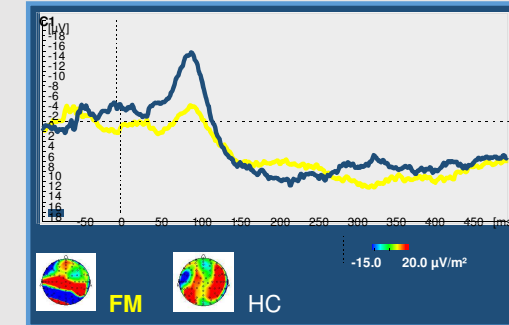


Result 4. The BRS was increased in FM compared to HC after the SP-protocol as a stimulation dependent on cardiac cycle. In contrast, HC showed an increase of BRS after the Non-SP-protocol (all p 's < 0.01).

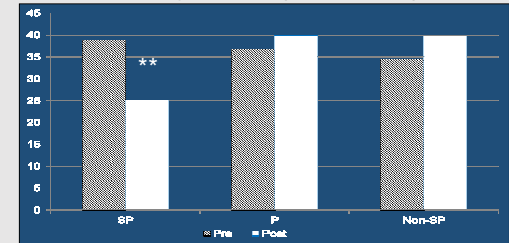
Figure 4: Differences in BRS between FM and HC after SP-, P- and Non-SP-Protocol



Result 5. The affective (P260) and cognitive (P390) components of pain, but not the attention (N50) and sensory (N150) components, showed greater responses in FM compared to HC during the SP protocol compared to the P protocol (all p 's < 0.005).



FM *Clinical Pain* significantly decreased to 35.79% during the SP - protocol (all p 's < 0.01).



Conclusion: Despite diminished BRS in FM, the combination of electrical pain and non-painful stimuli applied during the cardiac cycle diminished pain sensitivity and reduced fibromyalgia pain. Pain and stress reduction mediated by variations in BP serve as an instrumentally learned mechanism for stress inhibition in healthy people. In FM, this internal "coping" mechanism may be inactive or blocked. The SP protocol activated the internal "coping" mechanism that unblocked or facilitated pain inhibition in FM, possibly by increased activation of brain stem and basal forebrain regions involved in pain modulation.

