PS328

Cardiac Gating of Peripheral Afferent Stimulation restores Baroreflex Sensitivity, reduces Pain Sensitivity and Clinical Pain Report in Fibromyalgia Patients

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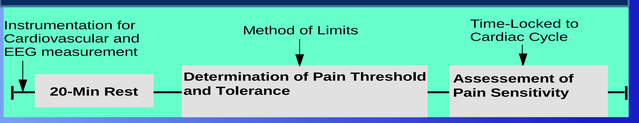
INTRODUCTION.

An important component of intrinsic pain regulatory systems is defined by cardiovascular dynamics that influence baroreceptor sensitivity (BRS). In healthy individuals, an elevation in resting arterial blood pressure is related to lower pain sensitivity.

This study tested: (1) whether this relationship is altered in fibromyalgia (FM) and (2) whether the introduction of noxious and non-noxious electrical stimuli introduced during systolic and diastolic phases of the cardiac cycle influence the perception of experimentally evoked and ongoing clinical pain.

METHODS.

Thirty pain-free normotensives (HC) and 32 FM received four 8-minutes-trials in which electrical stimuli were administered to the index finger during different phases of the cardiac cycle. In the test condition, non-painful electrical stimuli and painful electrical stimuli at 50% and 75% of the electrical pain tolerance were administered during both the systolic and diastolic phase in randomized order. In two control trials, one delivered only painful electrical stimuli and another delivered both non-painful and painful stimuli independent of the cardiac cycle phase.



Event#	Delay % or mS	Time On (mSec)	Time Off (mSec)	Duration (Count)	Amplitude	Pause (Sec)	Jump to	Repeat	Count	Group
1	20%	10	17	5	DT	5				A
2	80%	10	17	5	DT	5				A
3	20%	10	17	5	HTT	5				A
4	80%	10	17	5	HTT	5				A
5	20%	10	17	5	TT	5				A
6	80%	10	17	5	TT	5				A
7						15	1	10		

The events in this table occur 11 times with a pause of 15 sec. between each run.

Table 1: SP - Protocol

30 x 11 + 15 x 10 = 330 + 150 = 480 seconds = 8 minutes.
The length of the stimuli is 135 msec and is given each 5 seconds.

Analysis. The magnitude of clinical pain, sensory thresholds, as well as pain and tolerance thresholds to electrical stimuli were assessed before, between and after the test trials. BRS, blood pressure (BP), heart rate variability (HRV), surface electromyogram, (EMG) and respiration were measured throughout the session.

RESULTS.

Pain and tolerance thresholds were significantly different between FM and HC, increased by 15.1% and 25.2% in FM during the test protocol in contrast to 9.4% and 11.6% for HC.
In contrast during control trials,, the increases in thresholds in FM were significantly lower than the increases in HC (P<0.001).

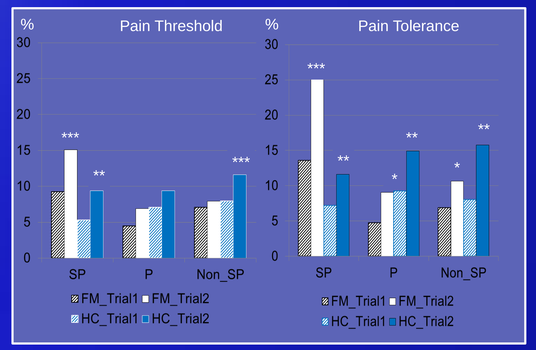


Figure 1: Changes of *Pain and Tolerance Thresholds* during the 1st and 2nd trial of SP-, P- and Non-SP in FM and HC

Blood Pressure in FM patients increased after the cardiac cycle related stimulations (SP- and P-protocol) but not after the Non-SP-protocol, a condition similar to the experience of real life. In contrast, blood pressure in HC increased also after the Non-SP-protocol (all p's<0.01).

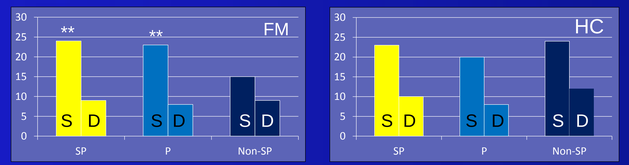


Figure 2: Changes of systolic and diastolic blood pressure between baseline and SP-, P- and Non-SP- protocols.

Prior to stimulation, **BRS** was diminished in FM compared to HC (p<0.01).

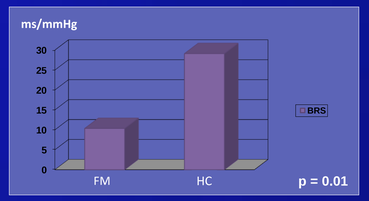


Figure 3: Differences in baseline *BRS* between FM and HC

BRS was increased in FM compared to HC after the SP-protocol, in which stimulation was dependent on the cardiac cycle. In contrast, HC showed increased BRS after the Non-SP-protocol, in which stimulation was independent of the cardiac cycle (all p's<0.01).

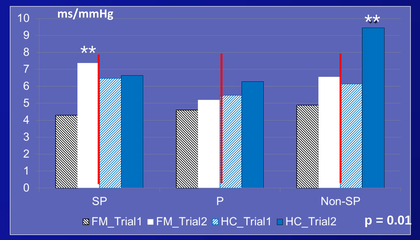


Figure 4: Differences in BRS between FM and HC after SP-, P- and Non-SP-Protocol

Vagal response (HF) increased in FM after the SP-protocol (p<0.01).

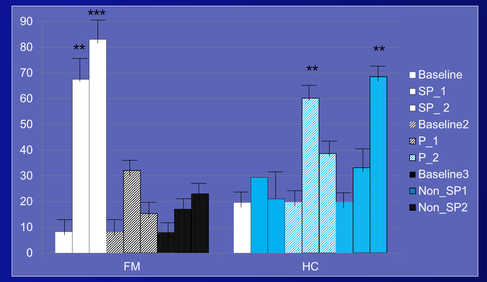
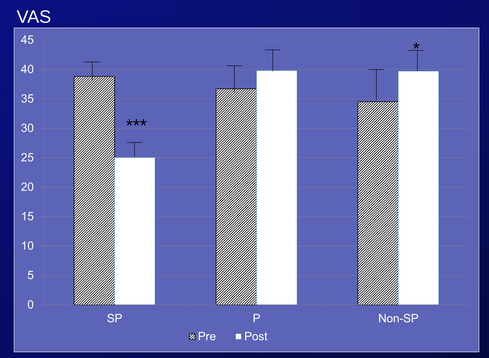


Figure 5: Differences in HF between FM and HC after SP-, P- and Non-SP-Protocols

FM ***Clinical Pain*** significantly decreased by 65.79% during the SP - protocol but not during the control protocols (all p's<0.01).



CONCLUSION:

Despite diminished BRS in FM, the combination of electrical painful and non-painful stimuli applied during specific phases of the cardiac cycle diminished pain sensitivity and reduced fibromyalgia pain. Pain and stress reduction mediated by variations in BP may serve as an instrumentally learned mechanism for stress inhibition in healthy persons.
In FM patients, 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.

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