A Review of Heart Rate Variability Biofeedback (HRVB) Treatment Outcome Studies with an Emphasis on Chronic Pain

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A Very Brief History of HRVB

- Paul Lehrer, Evgeny and Bronya Vaschillo
• We were getting mean HRs during exposure, but the data seemed inappropriate for that analysis.
• At the time, we knew little of HRV.
Measurement of HRV as Biomarker Grows Rapidly

- Biomarker for:
  - Cardiac Health
  - Psychological health
  - Emotional regulation
  - Other
Heart Rate Variability (RMSSD) predicting Amygdala Volume for Left and Right Hemispheres. Increasing Heart Rate Variability is associated with greater amygdala grey matter volume for the Lateral PreFrontal Cortex [LFPC] bilaterally ($F = 12.57$, R-Squared = 0.122, $p<0.0001$). Increasing Heart Rate Variability was associated with greater LPFC volume.
1983 with student Diane Herbs

- Our first attempt at HRV or RSA biofeedback
  - Compared to temp training for hypertension
    - Ss able to demonstrate learning quite readily
    - BP reductions comparable to other behavioral studies
  - Formulation of Mediational Model
Average RR-interval spectral power and RR intervals from 10 healthy supine subjects breathing to a nominal tidal volume of 1000 mL at seven breathing rates. From Eckberg DL, Circulation 1997;96:3224 –3232 (originally Brown et al., 1993)
The Afferent pathways

“The brain listening to the heart”
Figure 8. Afferent pathways. Diagram of the currently known afferent pathways by which information from the heart and cardiovascular system modulates brain activity. Note the direct connections from the NTS to the amygdala, hypothalamus, and thalamus. Although not shown, there is also evidence emerging of a pathway from the dorsal vagal complex that travels directly to the frontal cortex.
The vagus nerve: pathway to the limbic system

Brodmann 25
Extinction and the Treatment of Anxiety Disorders

• **Conclusions**

  • Extinction paired with VNS is more rapid than extinction paired with sham stimulation. As it is currently approved by the Federal Food and Drug Administration for depression and seizure prevention, VNS is a readily available and promising adjunct to exposure therapy for the treatment of severe anxiety disorders.
Heart Period Evoked Potential Across Conditions

Baseline vs Positive Emotion vs Negative Emotion vs Resonance Breathing

-1200 to 0
Training in HRVB vs EMG Biofeedback Assisted Progressive Muscle Relaxation (PMR) over 4 sessions

Assess HEP Pre and post training
SDNN between groups over time (p<.01)
HEP at 250 microseconds for both groups pre vs. post training (sign reversed)
Conclusions

- HRVB (probably as an adjunct to CBT or ACT) offers a promising treatment for depressive disorders
- It has the added advantage of improving autonomic homeostasis which has been shown to lead to improved cardiovascular function
- A possible mechanism is emerging
- It may eliminate some of the stigma associated with talk therapies and therefore increase compliance and reduce drop-outs (especially in minorities).
- Much more research is needed with active control groups.
<table>
<thead>
<tr>
<th>Disorder</th>
<th>Intervention</th>
<th>Design (Control)</th>
<th>Measures</th>
<th>Results</th>
<th>Reference(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asthma</td>
<td>HRVB + HT</td>
<td>vs. Sham EEG</td>
<td>Symptoms, lung function, medication</td>
<td>HRVB &gt; control</td>
<td>Lehrer et al., 2000; Lehrer et al., 2004</td>
</tr>
<tr>
<td>Chronic Obstructive Pulmonary Disease</td>
<td>HRVB + oximeter feedback</td>
<td>vs. TAU</td>
<td>6-minute walk</td>
<td>HRVB &gt; TAU</td>
<td>Giardino et al., 2004</td>
</tr>
<tr>
<td>Functional Gastrointestinal Disorders</td>
<td>Slow breathing + temp feedback</td>
<td>vs. TAU</td>
<td>Parent and child symptom ratings</td>
<td>Breathing &gt; control</td>
<td>Humphreys &amp; Gevirtz, 2000</td>
</tr>
<tr>
<td>Recurrent Abdominal Pain</td>
<td>HRVB</td>
<td>vs. Control</td>
<td>Symptom ratings and HRV measures</td>
<td>Symptom improvement associated with SDNN gains</td>
<td>Sowder et al., 2010</td>
</tr>
<tr>
<td>IBS</td>
<td>HRVB</td>
<td>vs. Hypnosis</td>
<td>IBS symptom severity scale, HADS</td>
<td>Both groups improved equally (HRVB slightly better)</td>
<td>Dobbin, Dobbin, Ross, Graham, &amp; Ford, 2013</td>
</tr>
<tr>
<td>Recurrent Abdominal Pain Integrated into other therapies</td>
<td>HRVB</td>
<td>Case study</td>
<td>Symptom log</td>
<td>Greatly improved</td>
<td>Masters, 2006</td>
</tr>
<tr>
<td>Cyclic Vomiting</td>
<td>HRVB</td>
<td>Case study</td>
<td>Vomiting frequency</td>
<td>Greatly improved</td>
<td>Slutsker, Konichezky, &amp; Goteiff, 2010</td>
</tr>
<tr>
<td>Recurrent Abdominal Pain</td>
<td>HRVB</td>
<td>TAU</td>
<td>IBS symptom measures</td>
<td>HRVB &gt; TAU</td>
<td>Ebert, 2013</td>
</tr>
<tr>
<td>Fibromyalgia</td>
<td>HRVB</td>
<td>vs. TAU</td>
<td>Standard FM scales</td>
<td>BFD &gt; TAU</td>
<td>Hassett et al., 2007</td>
</tr>
<tr>
<td>Cardiac Rehabilitation</td>
<td>HRVB</td>
<td>vs. Sham EEG</td>
<td>6-minute walk</td>
<td>HRVB &gt; sham EEG if LVEF &gt; 31</td>
<td>Swanson et al., 2009</td>
</tr>
<tr>
<td>Congestive Heart Failure</td>
<td>HRVB</td>
<td>vs. TAU</td>
<td>HRV measures (SDNN)</td>
<td>HRVB &gt; TAU</td>
<td>Del Pozo, Gevirtz, Scher, &amp; Guameri, 2004</td>
</tr>
<tr>
<td>Disorder</td>
<td>Intervention</td>
<td>Design Control</td>
<td>Measures</td>
<td>Results</td>
<td>Reference(s)</td>
</tr>
<tr>
<td>--------------------------</td>
<td>-----------------------</td>
<td>----------------</td>
<td>-----------------------------------</td>
<td>----------------------------------------</td>
<td>--------------</td>
</tr>
<tr>
<td>Coronary Artery Disease</td>
<td>HRVB + CBT</td>
<td></td>
<td>HRVB measures + adjustment scales</td>
<td>HRVB + CBT &gt; stress management</td>
<td>Nolan et al., 2005</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>vs. stress management</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Congestive Heart Failure</td>
<td>HRVB + stress</td>
<td></td>
<td>Case studies</td>
<td>Harvested heart tissue viability</td>
<td>Moravec, 2008; Moravec &amp; McKee, 2013</td>
</tr>
<tr>
<td></td>
<td>management</td>
<td></td>
<td></td>
<td>Training group equal to LVAD</td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>HRVB</td>
<td></td>
<td>Medication adjustment and BP</td>
<td>HRVB maintained BP with fewer meds</td>
<td>Reinke et al., 2007</td>
</tr>
<tr>
<td></td>
<td>vs. Sham EEG</td>
<td></td>
<td>BP, HRV, BRS</td>
<td>HRVB &gt; either control, improved on BP, HRV, and BRS measures</td>
<td>Lin et al., 2012</td>
</tr>
<tr>
<td>Prehypertensives</td>
<td>HRVB</td>
<td></td>
<td>Slow breathing and control, 3 month FU</td>
<td>Slow breathing = EMG feedback &gt; slow breathing alone</td>
<td>Wang et al., 2009</td>
</tr>
<tr>
<td>Prehypertensives</td>
<td>Slow abdominal</td>
<td></td>
<td>BP, HRV</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>breathing + EMG feedback</td>
<td></td>
<td>vs. Slow</td>
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<td></td>
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<tr>
<td></td>
<td>breathing alone</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chronic Muscle Pain</td>
<td>HRVB + myofascial</td>
<td></td>
<td>Four groups: pain and function measures</td>
<td>Combination superior to other interventions</td>
<td>Gordon &amp; Gevirtz, 2006; Vagades, 2011</td>
</tr>
<tr>
<td></td>
<td>release</td>
<td></td>
<td>stabilization exercises, HRVB alone, myofascial release alone, or combination</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>HRVB</td>
<td></td>
<td>Case studies</td>
<td>HRVB combined with physical release relieves pain</td>
<td>Gevirtz, 2006</td>
</tr>
<tr>
<td>Disorder</td>
<td>Intervention</td>
<td>Design (Control)</td>
<td>Measures</td>
<td>Results</td>
<td>Reference(s)</td>
</tr>
<tr>
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<td>--------------------------------------------------</td>
</tr>
<tr>
<td>OB/Gyn</td>
<td>HRVB</td>
<td>vs. TAU</td>
<td>Measures of pain, vitality and social</td>
<td>HRVB &gt; TAU</td>
<td>Hallman, Olsson, von Scheele, Melin, &amp; Lyskov, 2003</td>
</tr>
<tr>
<td>Preterm Labor</td>
<td>HRVB</td>
<td>vs. Control sessions</td>
<td>Preterm stress, preterm delivery</td>
<td>HRVB &gt; control for stress 13% vs. 33% preterm delivery (n.s.)</td>
<td>Siepmann et al., in press</td>
</tr>
<tr>
<td>PIH</td>
<td>HRVB (StressEraser)</td>
<td>vs. Matched case histories</td>
<td>BP, birth weight, gestation length</td>
<td>HRVB &gt; controls for birth weight and gestation length</td>
<td>Cullin et al., in press</td>
</tr>
<tr>
<td>PIH</td>
<td>Breathing and temperature</td>
<td>vs. activity management vs. TAU</td>
<td>BP levels logged daily</td>
<td>Biofeedback group halted; rising BPs vs. other groups</td>
<td>Sommers, Gevirtz, Jasin, &amp; Chin, 1989</td>
</tr>
<tr>
<td>Disorder</td>
<td>Intervention</td>
<td>Design (Control)</td>
<td>Measures</td>
<td>Results</td>
<td>References</td>
</tr>
<tr>
<td>---------------</td>
<td>--------------------</td>
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<td>----------------------------------------------</td>
<td>-----------------------------------</td>
</tr>
<tr>
<td>Depression</td>
<td>H RVB</td>
<td>No control, single group trial</td>
<td>BDI &amp; Hamilton</td>
<td>Depression reduced markedly</td>
<td>Karavidas et al., 2007</td>
</tr>
<tr>
<td></td>
<td>H RVB with StressEraser + DBT</td>
<td>vs. DBT + relaxation</td>
<td>BDI &amp; Hamilton</td>
<td>H RVB group superior</td>
<td>Zucker, Samuelson, Muench, Greenberg, &amp; Gevirtz, 2009</td>
</tr>
<tr>
<td></td>
<td>H RVB</td>
<td>Depressed vs. healthy control</td>
<td>BDI</td>
<td>Depressed patients reduced on BDI no changes in controls</td>
<td>Siepmann, Aykac, Unterdorfer, Petrowski, &amp; Mueck-Weymann, 2008</td>
</tr>
<tr>
<td></td>
<td>H RVB</td>
<td>vs. TAU after cardiac surgery</td>
<td>CES-D</td>
<td>H RVB &gt; TAU</td>
<td>Patron et al., 2013</td>
</tr>
<tr>
<td></td>
<td>H RVB</td>
<td>vs. Relaxation</td>
<td>BDI &amp; Hamilton</td>
<td>H RVB &gt; relaxation</td>
<td>Rene, Gevirtz, Muench, &amp; Birkhead, 2011</td>
</tr>
<tr>
<td></td>
<td>H RVB + DBT + Zoloft</td>
<td>vs. Zoloft</td>
<td>BDI &amp; Hamilton</td>
<td>H RVB + Zoloft alone</td>
<td>Rene et al., 2011</td>
</tr>
<tr>
<td>Anxiety Disorders</td>
<td>PTSD</td>
<td>HRVB</td>
<td>CAPS, trauma symptom checklist</td>
<td>H RVB &gt; TAU</td>
<td>Tan, Dao, Farmer, Sutherland, &amp; Gevirtz, 2011</td>
</tr>
<tr>
<td></td>
<td>HRVB</td>
<td>vs. Control</td>
<td>Information processing</td>
<td>H RVB &gt; information processing</td>
<td>Ginsberg, Berry, &amp; Powell, 2010</td>
</tr>
<tr>
<td></td>
<td>HRVB + DBT</td>
<td>vs. Relaxation</td>
<td>PCL</td>
<td>H RVB = relaxation</td>
<td>Zucker et al., 2009</td>
</tr>
<tr>
<td>Phobia</td>
<td>HRVB</td>
<td>Case example</td>
<td>approach phobic object</td>
<td>Improved phobic avoidance</td>
<td>Prigatano, 1972</td>
</tr>
<tr>
<td>Anxiety</td>
<td>HRVB</td>
<td>vs. Matched controls</td>
<td>Somatic symptoms</td>
<td>H RVB using HeartMath + control</td>
<td>Naca, 2009</td>
</tr>
<tr>
<td></td>
<td>HRVB</td>
<td>vs. Delayed treatment</td>
<td>Anxiety and mood</td>
<td>H RVB &gt; control</td>
<td>Henriques, Keffer, Abrahamson, &amp; Horst, 2011</td>
</tr>
<tr>
<td>Disorder</td>
<td>Intervention</td>
<td>Design (Control)</td>
<td>Measures</td>
<td>Results</td>
<td>References</td>
</tr>
<tr>
<td>---------------</td>
<td>-------------------------------</td>
<td>------------------</td>
<td>-----------------------------------------------</td>
<td>----------------------------------------------</td>
<td>------------------------------------------------</td>
</tr>
<tr>
<td>Stress</td>
<td>HRVB + stress management</td>
<td>vs. Control</td>
<td>Cholesterol, glucose, heart rate, blood pressure, positive outlook, and overall psychological distress.</td>
<td>HRVB + &gt; control on all measures, projected cost savings</td>
<td>McCraty, Atkinson, Lipsenthal, &amp; Arguelles, 2009</td>
</tr>
<tr>
<td>Sleep</td>
<td>HRVB + therapy</td>
<td>Single group study</td>
<td>Anxiety measures</td>
<td>Improvement</td>
<td>Reiner, 2003</td>
</tr>
<tr>
<td>Sleep Lab</td>
<td>HRVB (StressEraser)</td>
<td>Case report</td>
<td>Sleep log</td>
<td>Insomnia improvement long-term maintenance</td>
<td>Mc Lay &amp; Spira, 2009</td>
</tr>
<tr>
<td>Insomnia</td>
<td>HRVB</td>
<td>vs. Control</td>
<td>Sleep disturbance scale + actigraphy</td>
<td>HRVB &gt; controls</td>
<td>Eb ben et al, 2009</td>
</tr>
<tr>
<td>Performance</td>
<td>HRVB</td>
<td>vs. Sports psychology control</td>
<td>Hitting performance</td>
<td>HRVB &gt; controls</td>
<td>Strack &amp; Gevirtz, 2011</td>
</tr>
<tr>
<td>Golf</td>
<td>HRVB</td>
<td>vs. Neurofeedback vs. control</td>
<td>Refereed dance ratings</td>
<td>HRVB and neurofeedback &gt; control</td>
<td>Raymond, Sajid, Parkinson, Gruzeller, 2005</td>
</tr>
<tr>
<td>Dance</td>
<td>HRVB</td>
<td>vs. Neurofeedback vs. control</td>
<td>Refereed dance ratings</td>
<td>No effect on dance HRVB reduced anxiety</td>
<td>Gruzeller, Thompson, Brandt, &amp; Steffert, in press</td>
</tr>
<tr>
<td>Music</td>
<td>HRVB (emWave)</td>
<td>vs. Control</td>
<td>Performance anxiety measures</td>
<td>HRVB &gt; control</td>
<td>Thurber, 2006</td>
</tr>
<tr>
<td></td>
<td>HRVB or slow breathing</td>
<td>vs. Control</td>
<td>State anxiety</td>
<td>HRVB and slow breathing &gt; control</td>
<td>Wells, Outhred, Heathers, Quintana, &amp; Kemp, 2012</td>
</tr>
<tr>
<td>Disorder</td>
<td>Intervention</td>
<td>Design</td>
<td>Measure</td>
<td>Results</td>
<td>Reference</td>
</tr>
<tr>
<td>----------------------------</td>
<td>-----------------------</td>
<td>---------------------------</td>
<td>--------------------------------------</td>
<td>----------------------------------------------------------</td>
<td>-----------------------------------------------</td>
</tr>
<tr>
<td>Post-Partum Depression</td>
<td>HRVB (stress eraser)</td>
<td>Vs. TAU, (but random assignment)</td>
<td>Edinburgh Post Natal Depression Scale(EPND)</td>
<td>HRVB had less Anx., sleep disturbance at 1 month than controls</td>
<td>Kodama et al, 2014</td>
</tr>
<tr>
<td>Sleep</td>
<td>HRVB (Em Wave)</td>
<td>pilot</td>
<td>STAI, EPND</td>
<td>Dep reduced</td>
<td>Beckham&amp;Meltzer-Brody, 2013</td>
</tr>
<tr>
<td>Cardiac Rehab-CAD</td>
<td>HRVB</td>
<td>vs. Autogenic vs. control</td>
<td>HF amplitude during sleep</td>
<td>HRVB&gt; AT&gt; Control</td>
<td>Sakakibara et al, 2013</td>
</tr>
<tr>
<td>BP</td>
<td>Slow breathing @6/min</td>
<td>vs. music 10/min breathing</td>
<td>BP, BRS, HF</td>
<td>Slow breathing group &lt;BP, &gt;HF, &gt;SDNN, &gt;BRS</td>
<td>Pietro Amedeo Modesti • Antonella Ferrari • Cristina Bazzini • Maria Boddi, 2015</td>
</tr>
</tbody>
</table>
Pain Classification

A critical step in treatment
Pain

Acute Pain
  - No known pathology
    - Observe
  - Tissue Damage or Inflammation
    - RICE, Anti-Inflammatory, NSAIDS, Opioids, etc.

Chronic Pain
  - Peripheral Source
    - Stump, myofascial, burn, etc.
      - Tx fitted to source
  - Central
    - Phantom Limb, Thalamic, CRPS, FM
      - Systemic tx such as SABRE
Collaborators

• David Hubbard, M.D. & Myopoint
• Greg Berkoff, D.C.
• Sonja Banks, Ph.D.
• Carol Lewis, Ph.D.
• Walt McNulty, Ph.D.
• Toni Cafaro, Ph.D.
• Jeri Muse, Ph.D.
• Janeen Armm, Ph.D.
• Ali Oliviera, M.S., R.N.
• Jan Vagedes, M.D.
• Christopher Gordon, PT
• Frank Andrasik, Ph.D.
Comparative Costs of Musculoskeletal, Cardiovascular, & Cancer to the U.S. Economy
Chronic Muscle Pain-Epidemiology

• Second only to common cold for medical treatment
• 75-80 million people seeking treatment (Bonica, 1992)
• 550 million workdays lost
• $147 billion dollars lost in direct and indirect costs (Am. Assoc. Orthopedics, 1995)
• $245 billion 2001 dollars
• 70% of workers compensation claims
• 35% of work-disabling injuries (Calif. Work comp Bull, 1991)
Central vs. Peripheral Pain:

A key differential diagnostic distinction

• It is necessary to form a hypothesis on the source or sources of pain
  o Central
    • FM- Central allodynia or sensitization
    • CRPS- pain in a limb
    • Phantom limb pain
  o Peripheral
    • Myofascial Pain
    • IBS
    • Neuropathic Pain
  o Combination
Epidemiology-continued

- 45 million ER visits per year (Swiontkowski & Chapman, 1995)
- 70 million physician visits/year (Hollbrook, 1991)
- 425 million visits to chiropractors and “alternative” providers, $4.0 billion (Eisenberg, 1993)
- 20% of general population (Magni, 1993)
- 80% lifetime incidence (Bonica, 1990)
Oliveira, Gevirtz, & Hubbard (2005), *Spine*

- 126 Whiplash pts randomly assigned to video or normal ER tx
- Groups well matched
- Followed at 1, 3 & 6 months
- Video group (as compared to controls) showed good mastery of a content test on TPs \((F(1,124)=262.2, \text{Eta}^2=0.9)\)
- All ANOVAs and Chi^2 sig., \(p<.001\)
MUSCLE INJURY

Whiplash can cause injuries of neck muscles, ranging from minor strains and microhemorrhages to severe tears. Commonly affected muscles include the sternocleidomastoid muscle, scalene muscles, splenius capitis muscle, and longus colli muscle.
Short Form
Musculoskeletal Function Assessment

![Bar chart showing SMFA scores for different months and video conditions.]

- Month 1: No Video - Standard
- Month 3: No Video - Standard
- Month 6: No Video - Standard
- Month 1: Video - Standard
- Month 3: Video - Standard
- Month 6: Video - Standard
Verbal Rating Scale

<table>
<thead>
<tr>
<th>Month 1</th>
<th>Month 3</th>
<th>Month 6</th>
</tr>
</thead>
<tbody>
<tr>
<td>No Video</td>
<td>5</td>
<td>5</td>
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<tr>
<td>Video</td>
<td>1</td>
<td>1</td>
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</tbody>
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Verbal Rating Scale
Chiropractic Visits

- Month 1: No Video 35, Video 15
- Month 3: No Video 30, Video 10
- Month 6: No Video 25, Video 7
Physical Therapy Visits

![Bar chart showing the percentage of Physical Therapy Visits over three months (Month 1, Month 3, Month 6) with and without video. The chart indicates a decrease in visits over time for both groups.](image-url)
MRIs

Month 1
Month 3
Month 6
No Video
Video

%
ER Visits

![Bar chart showing ER visits over time with and without video assistance.](chart.png)
Urgent Care Visits

- No Video
- Video

Month 1
Month 3
Month 6
Taking Narcotics

[Bar graph showing the percentage of individuals taking narcotics over time, with categories for Month 1, Month 3, and Month 6. The graph compares 'No Video' and 'Video' conditions.]
Taking Muscle Relaxant

![Bar chart showing the percentage of taking muscle relaxants with and without video over the course of 6 months. The chart indicates a higher percentage of muscle relaxant usage in the absence of video compared to with video.]
Wearing Neck Brace

![Bar chart showing the percentage of patients wearing a neck brace at different time periods (Month 1, Month 3, Month 6) with and without video assistance. The chart indicates a higher percentage of patients wearing the brace without video assistance compared to those with video assistance at all time periods.]
Surgical Consultation

![Bar graph showing the percentage of no video and video consultations over months 1, 3, and 6.](image)
Cut Back Activities
Bed Rest

![Bar Chart]

- **Month 1**: 35% (No Video)
- **Month 3**: 5% (No Video, Video)
- **Month 6**: 5% (No Video, Video)

**Legend**:
- Orange: No Video
- Yellow: Video
Bed Rest

![Bar chart showing Bed Rest data for different months with and without video support. The chart indicates a significant increase in Bed Rest occurrence from Month 1 to Month 6, with a notable drop in the subsequent months.]
Number Missed Workdays Due to Injury
Primary Care Doctor Office Visits

Month 1  | Month 3  | Month 6
-------: | -------: | -------: |
No Video | Video  | No Video
---      | ---     | ---
70%      | 50%     | 60%
Relationship between attribution of pain etiology and pain improvement

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<thead>
<tr>
<th>Month</th>
<th>SMFA Std Pain scores</th>
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<tr>
<td>1</td>
<td>25</td>
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<tr>
<td>3</td>
<td>20</td>
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<tr>
<td>6</td>
<td>15</td>
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</tbody>
</table>

- **Orange bar**: Attribute pain to muscle tension
- **Yellow bar**: Does not attribute pain to muscle tension
Relationship between attribution of pain etiology and pain improvement

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<tr>
<th>SMFA Std Pain scores</th>
<th>Month 1</th>
<th>Month 3</th>
<th>Month 6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Attribute pain to muscle tension</td>
<td>7</td>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td>Does not attribute pain to muscle tension</td>
<td>15</td>
<td>20</td>
<td>22</td>
</tr>
</tbody>
</table>
Why was a 12 minute video so effective in preventing chronic neck/head pain?

- Answer is the core of this talk
Where Does the Pain Come From?

- Key question-seldom asked
- Is there a strong central component?
  - Is the pain a neurological construction absent peripheral mechanisms?
  - Evidence of central factors
    - Serotonergic systems
    - Dopaminergic systems
    - Antidepressant medication
    - Imaging studies
    - Small % of the variance
- Muscle Fatigue
  - No evidence for byproducts which would indicate fatigue
- Peripheral Mechanisms- Trigger Points
Typical Diagnosis

- Cervical Strain
- Lumbar Strain
- Repetitive Strain Injury
- Tension Headache
- TMJ or TMD
- Myofascial Pain Syndrome
Wait a minute here, Mr. Crumbley, maybe it's not kidney stones after all!
Typical Misdiagnoses

- Ruptured or bulged disc
- Pinched nerve
- Carpal tunnel syndrome
- Tennis elbow
- Bursitis
- Thoracic Outlet Syndrome
- Depression (or other Psychiatric diagnosis)
- Fibromyalgia
Etiological Theories

- Fatigue/Posture models
- Inflammation models
- Micro-lesion
- Subtle metabolic abnormality
- Trigger Points
Travell & Simons’
Myofascial Pain and Dysfunction:
The Trigger Point Manual
VOLUME 1. Upper Half of Body

Second Edition

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	Emory University School of Medicine
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JANET G. TRAVELL, M.D.*
LOIS S. SIMONS, M.S., P.T.
Consultant, Myofascial Pain and Dysfunction
Illustrations by Barbara D. Cummings
with contributions by Diane Abeloff and Jason Lee

* Dr. Janet Travell’s genius and medical insight identified in the first edition the clinical picture of individual myofascial pain syndromes and many perpetuating factors. In addition, we were most fortunate to have had the benefit of her advice in preparing some of this edition. She emphasized the importance of including a new chapter that covers the respiratory muscles and supplied unique pearls of clinical wisdom that sprinkled this revision.
Trigger Points (TrPs) I

- Trigger point is the sine qua non of Myofascial Pain Syndrome (MPS)
- Associated stiffness
- Localized point tenderness in muscle
- Stimulation produces local and referred pain
- Often with a palpable taut band
TrPs II

- Twitch
- Trigger because like a gun trigger is initiated with pressure
- Produces pain in another place-(target)
Figure 5.2. Overlapping pain referral patterns (red) from myofascial trigger points (Xs) in various masticatory and cervical muscles produce typical unilateral or bilateral migraine or tension-type headache pictures.
toid attachment of the sternocleidomasto-
doid. They reported inducing referred pain by applying digital pressure to these
tender muscles and by injecting hypertonic salt solution into them, location un-
specified.

Sternal Division
(Fig. 7.1A)

An active TP in the lower end of the
sternal division refers pain downward
over the upper portion of the sternum (Fig.
7.1A). This is the only downward refer-
ence of pain from this muscle.\textsuperscript{49,53} True
trigeminal facial neuralgia is not accom-
pained by sternal pain, which, when also
present, suggests the sternocleidomasto-
doid myofascial syndrome.

When an unusual TP is activated in the
lowest part of the sternal division, where
that division may merge with a slip of the
inconstant sternalis muscle, the TP is as-
associated with a paroxysmal dry cough that
can be precipitated by mechanical stimu-
lation of the TP.

At the mid-level of the sternal division,
TPs refer pain homolaterally, arching
across the cheek (often in finger-like pro-
jections) and into the maxilla, over the
supraorbital ridge and deep within the or-
bital.\textsuperscript{51} Pain may be referred on the same
to the external auditory canal.\textsuperscript{\textbf{46,47}} The
quality of the pain is described by patients
to be aching as in the deep pain defined
by Kellgren.\textsuperscript{22} The TP along the inner
margin at the mid-level of this division
refer pain to the pharynx and to the back
of the tongue during swallowing,\textsuperscript{5} which
causes "sore throat," and to a small round
area at the tip of the chin.\textsuperscript{53} Marbach\textsuperscript{39}
shows a similar pattern that includes the
cheek, temporomandibular joint and mas-
toid areas.

In the upper end of the sternal division,
TPs refer pain to the occipital ridge behind,
but not close to the ear, and to the vertex
of the head like a skull cap, with scalp
tenderness in the pain reference zone.

Autonomic concomitants of TP in the
sternal division relate to the homolateral
eye and nose.\textsuperscript{49,53} Eye symptoms include
excessive lacrimation, redness (vascular
engorgement) of the conjunctiva, apparent
"ptosis" (narrowing of the palpebral fissa-
ure) with normal pupillary size and re-
actions, and visual disturbances. The
"ptosis" is due to spasm of the orbicularis
oculi muscle, rather than to weakness of
the levator palpebrae muscle. The spasm
is caused by increased excitability of mo-
tor units within the reference zone of ster-
nal division TPs. The patient may have to
tilt the head backward to look up, because
of inability to raise the upper eyelid. Visual
disturbances include not only blurring of
vision,\textsuperscript{46} but also dimming of perceived

![Figure 7.1. Referred pain patterns (solid red shows essential zones and strik-](Image)

Table 8-2  Interrater Reliability of Examinations for Trigger Point Characteristics, Kappa Values

<table>
<thead>
<tr>
<th>Examination</th>
<th>Wolfe et al.\textsuperscript{300}</th>
<th>Nice et al.\textsuperscript{207}</th>
<th>Njoo et al.\textsuperscript{210}</th>
<th>Gerwin et al.\textsuperscript{87}</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spot tenderness</td>
<td>0.61</td>
<td>0.66</td>
<td>0.84</td>
<td>0.70</td>
<td></td>
</tr>
<tr>
<td>Jump sign</td>
<td></td>
<td>0.70</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain recognition</td>
<td>0.30</td>
<td>0.58</td>
<td>0.88</td>
<td>0.70</td>
<td></td>
</tr>
<tr>
<td>Palpable band</td>
<td>0.29</td>
<td>0.49</td>
<td>0.85</td>
<td>0.59</td>
<td></td>
</tr>
<tr>
<td>Referred pain</td>
<td>0.40</td>
<td>0.38</td>
<td>0.41</td>
<td>0.54</td>
<td></td>
</tr>
<tr>
<td>Twitch response</td>
<td>0.16</td>
<td>0.41</td>
<td>0.69</td>
<td>0.47</td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>0.35</td>
<td>0.38</td>
<td>0.49</td>
<td>0.74</td>
<td></td>
</tr>
</tbody>
</table>
Needle EMG Activity

Trigger Point

Adjacent Non-Tender Muscle Fibers

Active Needle Electrode

Surface Reference Electrode

Active Needle Electrode
Myofascial Trigger Points Show Spontaneous Needle EMG Activity (Hubbard & Berkoff, 1993)

Mean nEMG amplitude in Microvolts

- Normal Ss
- Muscle Pain Ss

- Trigger Point
- Adjacent Non-tender

Bar chart showing the comparison of mean nEMG amplitude between normal individuals and those experiencing muscle pain.
Effects of Curare on nEMG in TPs and Adjacent, (Non-tender) Sites

Pre Injection Post

TP nEMG

Adjacent nEMG
The Effect of Phentolamine Injection on TP & Adjacent nEMG

Phentolamine 2.5mg injected directly into trigger point in patient with myofascial pain
David Hubbard, MD, Dept Neurology, University of California, San Diego
Phentolamine effect on the spontaneous electrical activity of active loci in a myofascial trigger spot of rabbit skeletal muscle*1.

Archives of Physical Medicine and Rehabilitation, Volume 79, Issue 7, Pages 790 - 794

J. Chen, S. Chen, T. Kuan, K. Chung, C. Hong
Clinical Efficacy of Phenoxybenzamine (Myotech)
Italian Spindle Studies

- Passatore, Deriu, Grassi, & Raotta (1996), J. Auton N.S.
- Grassi, Deriu, & Passatore (1993) J. Physiology
- Grassi & Passatore (1990) Functional Neurology
  - Found strong response in spindle could be elicited by sympathetic cervical nerve stimulation, abolished by alpha-adrenergic blockade, unaffected by sympathetically induced vasomotor changes.
  - “These data suggest that, when the sympathetic nervous system is activated under physiological conditions, there is a marked depression of the stretch reflex which is independent of vasomotor changes and is probably due to decrease in sensitivity of muscle spindle afferents” (Grassi, Deriu, & Passatore, 1993, p.163)
Needle EMG-Guided Biopsy of a Trigger Point

- Extramuscular muscle fibers
- Intramuscular muscle fibers
- Spindle capsule
Figure 15.11 A muscle spindle and its innervation.
Muscle Spindle
“...we have observed alpha-EPS (end plate spikes) coactivation and even independent EPS activation, not connected to muscle contraction.”
Spindles are sympathetically preparing for motor action
muscle pain

spindle capsule

pain & pressure receptors

intrafusal muscle fibers

adrenalin receptor

sympathetic tension
Myofascial Factors in Low Back Pain

Deconditioning of lumbar extensors, particularly longissimus and multifidus muscles

Injury to muscle attachments may result in pain and delayed healing

Muscle spindles provide feedback mechanism for muscle tension. Sensitivity of spindles modulated by gamma efferent system and by sympathetic innervation of spindles. Sympathetic hyperactivity can result in painful spasm of spindles.
Trigger Point vs. Adjacent nEMG vs. Frontal EMG

autogenic relaxation training on EMG activity in myofascial trigger points. *Journal of Musculoskeletal Pain, 6, #4.*
Conditions


---

**Psychophysiology and Biofeedback, 22, 137 (abstract)**
nEMG Across Ergonomic & Stress Conditions

Personality Traits and Tp Worsening

- In 86 1st year graduate students, the Penn State Worry Questionnaire (among a number of other predictors) predicted trigger point worsening 2-3 months later over the course of increasing stress in an academic semester, $r = .35$, $r^2 = .123$ (Armm, Gevirtz, Hubbard, & Harpin, 1999)
Mediational Model of Muscle Pain

- Lack of Assertiveness
- Situations requiring self-assertion
- Worry or FNE ongoing
- Prolonged Sympathetic Activity/Vagal Withdrawal
- Overexertion
- TP or Spindle Spasm Pressure on Capsule
- Muscle Pain- Myofascial Pain- TP Pain
Insert Myopoint Video Here
Testing whether laughter is the best medicine
PAIN DRAWING

SHADE IN WITH A PENCIL ALL AREAS YOU HAVE PAIN.
(Don't forget to include the head or areas of lesser pain).
Use small x's to show any areas of numbness or tingling.
<table>
<thead>
<tr>
<th>Source of Pain</th>
<th>Diagnosis</th>
<th>Physiology</th>
<th>Symptoms</th>
<th>Exam</th>
<th>Tests</th>
<th>Treatment</th>
<th>Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Muscle</td>
<td>Strain Injury</td>
<td>Muscle stretch receptor overstretch, and adrenaline overactivity</td>
<td>Widespread pain, stiffness</td>
<td>Trigger Points</td>
<td>Trigger point EMG shows spasm localized to the nidus of the trigger point</td>
<td>Gentle stretch</td>
<td>Resolvable but tension must be addressed</td>
</tr>
<tr>
<td></td>
<td>Low Back Pain</td>
<td></td>
<td>Increased with exertion, immobilization and tension</td>
<td>Isometric contraction increases pain</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Repetitive Trauma</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>‘TMJ’</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Tension Headache</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Myofascial Pain</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nerve</td>
<td>Radiculopathy</td>
<td>Compression and ischemia of nerve</td>
<td>Ribbons of pain, tingling and numbness</td>
<td>Stretching nerve increases pain, decreased sensation in nerve distribution</td>
<td>MRI, CT or myelogram</td>
<td>Surgical decompression if nerve compression persists</td>
<td>After surgery, risk of residual nerve damage</td>
</tr>
<tr>
<td>Compression</td>
<td>Carpal Tunnel</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Disc Herniation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nerve Damage</td>
<td>Neuropathy</td>
<td>Demyelination or disruption of nerve</td>
<td>Same as nerve compression</td>
<td>Decreased sensation in nerve distribution, loss of reflexes</td>
<td>EMG-HVC</td>
<td><em>Anti-depressants</em></td>
<td>Permanent</td>
</tr>
<tr>
<td></td>
<td>Radiculopathy</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nerve Traction</td>
<td>Thoracic outlet</td>
<td>Stretching or crowding of nerve bundles</td>
<td>Tingling of entire arm or leg</td>
<td>Palpation and contraction of muscles increases tingling.</td>
<td>EMG-HVC</td>
<td>IS NORMAL</td>
<td>Same as for muscle pain</td>
</tr>
<tr>
<td>Syndrome</td>
<td>Sympathectomy</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>RESOLVABLE ONCE MUSCLE PAIN RESOLVED</td>
</tr>
<tr>
<td></td>
<td>Piriformis Syndrome</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Joint</td>
<td>Osteoarthritis</td>
<td>Thinning and roughening of joint space</td>
<td>Pain localized to joint</td>
<td>Passive movement of joint increases pain</td>
<td>X-ray</td>
<td>Non-steroidal anti-inflammatories</td>
<td>Slowly progressive</td>
</tr>
<tr>
<td></td>
<td>Degenerative Spine Disease</td>
<td></td>
<td></td>
<td>Thinning of joint degenerative changes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Rheumatoid Arthritis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Migraine</td>
<td>Migraine</td>
<td>Shunting of blood through the meninges</td>
<td>Attacks of pain, lasting 1-3 days, with nausea</td>
<td>Normal exam</td>
<td>None</td>
<td>Imitrex</td>
<td>Variable</td>
</tr>
<tr>
<td>Headache</td>
<td>Complex Regional Pain Syndrome</td>
<td>Constriction of blood flow in the hand or foot</td>
<td>Pain in the hand or foot that does not fit a nerve pattern</td>
<td>Skin texture and temperature changes</td>
<td>Skin temperature measurement</td>
<td>Biofeedback</td>
<td>Variable</td>
</tr>
<tr>
<td></td>
<td>Fibromyalgia</td>
<td>Unknown</td>
<td>Diffuse pain, fatigue, insomnia, depression</td>
<td>Diffuse tenderness in all four quadrants of body</td>
<td>None</td>
<td>Anti-depressants</td>
<td>Slowly progressive</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Non-steroidal anti-inflammatories</td>
<td>Variable</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Chronic Fatigue Syndrome</td>
<td></td>
</tr>
</tbody>
</table>
Differential Diagnosis of FM and MPS
(adapted from Schneider, 1995)

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Fibromyalgia</th>
<th>Myofascial Pain Syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain pattern</td>
<td>Bilateral &amp; Widespread</td>
<td>Regional: Specific referred pain patterns</td>
</tr>
<tr>
<td>Morning fatigue</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Sleep disorder</td>
<td>Yes: strong correlation with FM</td>
<td>Sometimes: secondary to pain &amp; discomfort of MPS</td>
</tr>
<tr>
<td>Soft tissue findings</td>
<td>Tender point</td>
<td>Trigger point</td>
</tr>
<tr>
<td>Palpable changes</td>
<td>None</td>
<td>Distinct “nodularity” over TP; Palpable, taut “ropy” bands with associated features</td>
</tr>
<tr>
<td>Female/Male ratio</td>
<td>10-20:1</td>
<td>1:1</td>
</tr>
</tbody>
</table>
### Symptoms

<table>
<thead>
<tr>
<th>Fibromyalgia</th>
<th>Myofascial Pain Syndrome</th>
</tr>
</thead>
</table>

#### History / presentation
- **Fibromyalgia**: Chronic, widespread pain; morning fatigue, stiffness and pain of unknown cause.
- **Myofascial Pain Syndrome**: History of acute or chronic muscle strain or injury; regionalized pain.

#### Treatment approach
- **Fibromyalgia**: Treatment is systemic: Low dose anti-depressants, Aerobic Exercise, Psychotherapy, Chiropractic manipulation.
- **Myofascial Pain Syndrome**: Treatment is specific and local: Accupressure, Therapeutic stretch, Injection.
Treatment Considerations

I

• Education/Attribution Shift
• Use grid, video, articles, persuasion, diagrams, etc. to achieve shift in causal attribution
• Physical management
  o Acupressure, acupuncture, theracane, tennis balls, passive stretches, moist heat, spray and stretch
  o Injections (Phenoxybenzamine, botulinum toxin type A, dry needling)
Sharp Hospital Treatment Model I

- 1992 to present
- Average weekly census = 225 patients (one of largest in world)
- 61% managed care, 33% workers’ compensation
- 67% reduction in following year health care costs
- Despite average of 3.8 years disability, 67% returned to work
Parasympathetic

“Accentuated Antagonism”

• “Vagal ‘tone’ predominates over sympathetic tone at rest. Under normal physiological conditions, abrupt parasympathetic stimulation will inhibit tonic sympathetic activation and its effects at rest and during exercise. This response is known as ‘accentuated antagonism’ “(Olshansky et al., 2011, p.863; Yang and Levy, 1984; Schwegler and Jacob, 1975; Levy, and Zieske, 1969)
common levator
Muscle for the nose
and the upper lip.

levator muscle
Specifically for the
upper lip.

Santorini's cartilage

cheek muscle

depressor muscle
which lowers the angle of the
mouth or the lips
(depressor angulae oris or triangularis).

zygomaticus minor
muscle

zygomaticus major
muscle

orbicular muscle of
the lips

depressor muscle of the
lower lip or chin

chin (mental) muscle
Sharp Hospital Treatment Model II

- Muscle pain education/differential diagnosis
- Cognitive coping
- Physical coping/gentle stretching
- Medication management
- Use of interns
- De-emphasize traditional psychological models
Treatment II: sEMG
Biofeedback Techniques

• Muscle awareness
  o Biofeedback, bilateral, symmetry, traps, frontal

• Frontal EMG
  o Cultivated Low Arousal
  o Facial Muscle Feedback

• Bi-lateral Trapezius Tx
  o For Bracing or Splinting
  o For Symmetry
  o For Breathing

• Specific Muscle Placement
VLF (.001 - .07Hz)
LF (.08 - .14Hz)
HF (.15 - .4Hz)
Figure 8. Afferent pathways. Diagram of the currently known afferent pathways by which information from the heart and cardiovascular system modulates brain activity. Note the direct connections from the NTS to the amygdala, hypothalamus, and thalamus. Although not shown, there is also evidence emerging of a pathway from the dorsal vagal complex that travels directly to the frontal cortex.
The Trigger Point Therapy Workbook
YOUR SELF-TREATMENT GUIDE FOR PAIN RELIEF
Clair Davies, N.C.T.M.B.

Foreword by David G. Simons, M.D., coauthor of Travell & Simons’ Myofascial Pain and Dysfunction: The Trigger Point Manual

"I believe this book will help end a great deal of needless suffering and prevent a great deal of unnecessary surgery."

—Devin Starbuck, coauthor of Fibromyalgia & Chronic Myofascial Pain

The proven method for overcoming soft-tissue pain now available in a practical step-by-step format.

Fibromyalgia • Chronic Myofascial Pain Syndrome • Low Back Pain • Carpal Tunnel • Tennis Elbow • Neck and Jaw Pain • Frozen Shoulder • Arthritis • Headaches • Sore Knees and Feet • Accident Trauma • Joint Pain and Muscle Aches • Sports and Repetitive Strain Injuries
Treatment Considerations

III

• Breathing/relaxation
  o Cultivated low arousal, breathing retraining, mindfulness techniques, etc.
  o Resonant Frequency Training

• Problem solving
  o Using awareness of muscle tension, try to remedy causal situation (seeing the big picture)
Cognitive interventions

Since the model hypothesizes that persistent sympathetic activity (even if low level) stimulates activity in the TrPs, we now look for the “smoking cognitive gun”. This is likely to center around distorted self-schema such as: “I’m only a valid person if I am pleasing others”, or “If I am not perfect I am worthless”.

Pain ratings across time

(Vagades, Gordon, Gevirtz, Andrasik (2013)  N=124)
Composite pain measure
Wrap-Up

• Summary of the arguments presented today
  o Epidemiology
  o Pathophysiology
  o Treatment

• Implications: Mind/Body techniques should be the first line treatment for muscle pain disorders