Of Veterans Affairs Health Benefits Burials & Memorials About VA Resources N VA » Health Care » HSR&D » Cyberseminars » Upcoming Cyberseminars — Catalog Health Services Resources N	A SITE MAP [A-Z] News Room Loca	ations Contact Us				
Health Benefits Burials & Memorials About VA Resources I VA » Health Care » HSR&D » Cyberseminars » Upcoming Cyberseminars — Catalog Health Services Resource 8 Development	News Room Loca	ations Contact Us				
VA » Health Care » HSR&D » Cyberseminars » Upcoming Cyberseminars — Catalog						
Health Services Research & Development		VA » Health Care » HSR&D » Cyberseminars » Upcoming Cyberseminars — Catalog				
Health Services Research & Development						
HSR&D Home						
About Us Upcoming Cyberseminars — Catalog	Upcoming Cyberseminars — Catalog					
Research Topics						
Career Development Program Upcoming Archive Searce	ch Cyber Seminar Catalog »	go				
▶ Centers						
← Cyberseminars Date Time (EST) Title	Series F	Presenters Register				

Applied HRV Data Interpretation for the Clinician Ginsberg, JP (Jack)

Heart Rate Variability Biofeedback (HRVB) professionals regularly use quantitative and graphic methods of analysis of heart rate to understand the physiological and emotional status of pain patients. This presentation will use actual pre-post HRVB data from Veterans with PTSD and chronic pain to illustrate how acquisition of the skill of self-regulation through HRVB affects heart rate patterns and screen displays and is related to reduced symptoms and improved level of function.

JP (Jack) Ginsberg, PhD

Licensed Clinical Psychologist/Neuropsychologist and Principal Investigator, **Dorn VA Medical Center Basic Science Research Assistant Professor** University of South Carolina, School of Medicine, **Dept of Pharmacology, Physiology & Neuroscience** Columbia, SC jay.ginsberg@va.gov 803.776.4000 x6644

Disclaimer and Disclosure

- Neuropsychologist, interest in PTSD and cognitive psychophysiology
 - Not expert in cardiology, physiology, medication, or pain
- No conflict of interest, affiliations, or product endorsement
- Slides are original, available on internet, or acknowledged
 - Not copyrighted but please acknowledge
- Some portions presented previously
- Models are didactic and heuristic
 - Correct only as far as they go
 - Referenced and consistent with current scientific literature
 - Corrections, revisions, expansions, updates are appreciated
 - Sketchy, incomplete, simplified, and not elaborated in context
 - Not writing medical cardiovascular physiology textbook or research proposal

Materials that are included in this presentation may include interventions and modalities that are beyond the authorized practice of mental health professionals. As a licensed professional, you are responsible for reviewing the scope of practice, including activities that are defined in law as beyond the boundaries of practice in accordance with and in compliance with your professions standards. Actual Sample of Interbeat Intervals (IBI)

Msecs

Velocity Curve (IBI vs sequential beat)

Sequential beat

12 13 14 15



705	716	754	787	787	762	748	723	754
779	790	800	785	772	764	749	726	721

Tachygram (ibi in msecs vs time in secs)







Tachygram

A tachygram is a graph of heart rate (HR), either as inter-beat interval (IBI) or as instantaneous HR (BPM), over a continuous time period. The illustrations above are IBI in msecs measured as RR.

Sine Wave







72 BPM, Max-Min 20 1 cycle/10 sec=6 cycles/min =12 beats/cycle Vs 60 BPM, Max-Min 30 1 cycle/10 sec=6 cycles/min =10 beats/cycle







mathematically derived from a sine/cosine function.

In the illustration above there are 10 beats in 10 seconds (avg BPM=60); note however that the beats would not be not evenly spaced 1 second apart





The Fourier Transform will transform any sinusoidal wave form into a frequency spectrum. The transformed frequency spectrum is quantified and analyzed in terms of 'power' or area under the curve, across a range of frequencies. Power is directly related to variance of the untransformed time series.

HRV Analysis Results

rmibitot - xe/xe/xex - xe xet xet Page 1/1



Total

LEAF

3340

8.973

3301

5.996

LFAF

Kubios HRV report



0

0

0.1

0.2

Frequency (Hz)

0.3

0.4

0.5

0 0.1

0.2

Frequency (Hz)

0.3

0.5

0.4

VLF: 0.0033-0.04Hz=(300-25) secs/cycle=(0.2-2.4) cycles/min

- Minimum 5 minute recording (24 hour is best)
- More strongly predictive of all-cause mortality and inflammation than the other bands
- Origins of VLF power unclear
 - Intrinsic (pacemaker)
 - Predominantly SNS activity however low PNS power also present

LF: 0.04–0.15 Hz=(25-6.7) secs/cycle=(2.4-9) cycles/min

- Minimum 2 min recording
- 'Baroreceptor range' during rest
- Power from SNS, PNS, and baroreceptors
 - SNS not above ~0.1 Hz
 - PNS dominant >0.07 Hz

HF: 0.15–0.40 Hz=(6.7-2.5) secs/cycle=(9-24) cycles/min

- Minimum 1 minute recording
- 'Respiratory band'
- PNS power



Idealized HRV parameters - 1 min recording		
Mean HR	72.0	
Max-Min	20.0	
SDNN (ms)	83.9	
RMSSD (ms)	47.0	
VLF power (ms ²)	0	
LF power (ms ²)	6959.0	
LF peak (Hz)	0.1	
HF power (ms²)	4	

Kubios v. 2.0

Physiological Bases of HRV

- I. Respiratory Sinus Arrhythmia (RSA)
 - Abdominothoracic Pump, Respiratory Pump, Cardio-respiratory Coupling, Cardiopulmonary Reflex, Lung-heart Pump
 - Results from interactions of
 - Respiration (e.g. rate, depth, volume, effort)
 - Vagal parasympathetic tone
 - Oscillations in cardiac output
 - Occurs at normal respiration rate
 - 0.15-0.4 Hz=9-14 BrPM
 - May be measured as either instantaneous HR or IBI
 - Max-min ('peak to trough') over a single respiratory cycle or averaged over many respiratory cycles
 - HRV indices
 - A higher resting vagal tone may be adaptive
 - Energy reserve capacity for active states
- II. Resonant Frequency Breathing (RFB)
 - Occurs in LF, around 0.1 Hz=~6 BrPM
 - Baroreceptor reflex (BR)
 - Interrelations of respiration, HR, BR, BP, and vasomotor tone (VT)
 - 'Coherence'

RSA: Respiration and Vagal Parasympathetic Tone-I

- Normal Respiration produces rhythm of cardiac acceleration and deceleration
 - Cardiac acceleration on inhalation
 - Cardiac deceleration on exhalation
- Synchronization of respiratory and cardiovascular processes and regulation of energy exchange
- Vagal tone is measured by HR responses to cholinergic and adrenergic stimulation and blockade



RSA: Respiration and Vagal Tone-II

- RSA magnitude is affected by respiration and vagal tone separately
- Vagal tone reflects oscillations of cardiac vagally modulated parasympathetic (cholinergic) effects upon the sinoatrial node and, therefore, HR
- Respiratory parameters (e.g. rate, volume or depth) affect or confound the function linking RSA and vagal tone
 - RSA due to respiration rate and cardiac vagal tone can dissociate
- RSA magnitude more closely related to changes in respiratory parameters than to changes in cardiac vagal tone
 - Greater tidal volume, lower breath rate increase RSA with constant vagal tone
- HR max-min is sensitive to respiration rate independent of vagal tone
- HF power changes may not be accompanied by changes in HR
- HF power and RSA do not represent vagal tone
- RSA magnitude is affected by vagal tone and beta-adrenergic status
 - Vagolytic agents (e.g. atropine, muscarinic cholinergic antagonists) block Ach and increase beta-2 adrenergic activity
 - Decrease RSA, increase HR, no BP change
 - Abolish LF and HF power
 - Blocks SA Ach released by vagus
 - Does not alter HR in absence of vagal nerve activity
 - Beta-adrenergic blockade (e.g. propranolol, atenolol)
 - Increase RSA, decrease HR and BP

RSA: Respiration and Oscillation in Cardiac Output (Intra-thoracic Pressure, Venous Return)

- RSA is also elicited by cardiovascular reflexes due to changes in venous filling of the heart during normal respiration
- Respiratory activity influences venous return to the heart. Inspiration expands the right atrium, lowers intra-pleural pressure, enhances venous return, increases HR
- Increasing the depth of inhalation promotes venous return (like pulling out on a bellows or syringe), enhances cardiac output, and increases HR
 - Cardiac output = stroke volume x HR

• Low pressure arterial baroreceptors at veno-atrial junctions of the heart ('cardio-pulomonary receptors') are innervated by myelinated vagal afferents and respond to atrial filling and contraction. During inhalation, right atrial expansion and reduced pressure increase venous return, blood volume stretch recptors, and HR via withdrawal of vagal parasympathetic activity to the SA node (Bainbridge Reflex). During exhalation, right atrial contraction and increased pressure decrease stretch receptors which decreases venous return and HR via activation of vagal reflex.



RSA is affected by inhalation vs exhalation changes in venous return

and cardiac output



Increasing the depth of respiration promotes venous return through changes in right atrial (chest cavity) pressure. During inspiration, the chest wall expands as the diaphragm descends, causing right atrial pressure to fall which facilitates venous return.. As pressure falls and venous return flow rises, cardiac rate accelerates... During expiration, the opposite occurs.. Increasing right atrial pressure impedes venous return and slows HR... Increasing the depth of ventilation increases the range of HR over the respiratory cycle. **Resonance Frequency Breathing (RFB)**

- HRV is related to frequency of respiratory cycle
- At ~ 6 breaths/minute =10 seconds per breath=0.1 Hz = 0.1 cycles/second
 - = 1 cycle/10 seconds
 - = 6 cycles/60 seconds
 - = 6 cycles/minute
- @RFB, respiration and HRV synchronize
 - 'Resonance'
 - Indicator of 'Coherence'

0° phase relationship between oscillations in respiratory and HR cycles





When HRV and respiration are synchronized, a spectral peak occurs at the RFB, ~0.1 Hz, due to resonance of HRV amplitude.

Resonance Frequency Breathing: Effect of Various Respiration Frequencies on HRV Averaged RSA Amplitude and Phase Frequency Characteristics



- Measured continuous HR in 5min paced breathing periods of 0.5, 0.25, 0.143, 0.11, 0.077, 0.055, 0.04, 0.029, and 0.02 Hz
- Paced breathing (imposed) sinewave oscillations in HR
- Calculated avg HR_{max}- Hr_{min}
- 6 per respiratory cycle
 - <u>The highest amplitude of max-</u> <u>min and 0° phase occurred at</u> <u>same frequency of breathing,</u> <u>~0.1 Hz</u>



Phase Relationships of Sine-Wave Oscillations

RSA (HR max-min) Differs between People but all have max ~0.1 Hz



- Breathing ~0.1Hz caused highest HR oscillation amplitude for all Ss
- HR always synchronized with breathing at this frequency (phase=0°).
- Peak HR amplitudes at ~0.1 Hz (resonance in CVS) at this frequency.
- Peak HR amplitude found in range of <u>0.075–0.108 Hz (=4.5-6.5 BrPM)</u>

Resonance is the tendency of a system to oscillate with greater amplitude at some frequencies than at others. Relative maximum frequency of oscillation is the system's **resonance frequency**. At these resonance frequency, even small periodic driving forces can produce large amplitude oscillations



Pushing a person in a swing is an example of resonance. Pushing a swing in time with its resonant frequency will make the swing go higher and higher (maximum amplitude), while attempts to push it at a faster or slower tempo results in smaller arcs.

Coherence of Cardiac Rhythm: Effect of RFB on HRV coherence.com (Richard Brown, MD and Stephen Elliott, Ph.D.)



The difference between the max and min HR for each cycle is shown along the center; averaging across consecutive cycles yields HRV(avg), one of the many measures of HRV.

30 BrPM (0.5 Hz) , HRV(avg) = 2

7.5 BrPM (0.125 Hz), HRV(avg) = 11

5.5 BrPM (0.092 Hz), HRV(avg) = 34



Baroreflex activates resonance ('Coherence')





Respiration, HR, and BP are interrelated BP: Sys/Dias, Pulse, Mean Arterial

- 1. The Valsalva Maneuver
- 2. Traube-Hering and Mayer Waves
- 3. Baroreceptor Reflex (BR)
 - a. HR goes down when blood pressure goes up, and HR goes up when blood pressure goes down
 - b. The baroreflex amplifies HR oscillations at its resonance frequencies
 c. HRV Resonance @RFB→Coherence

The Valsalva Maneuver

- Real-time blood pressure and heart rate responses to deep breathing provide autonomic information
- RSA and Valsalva maneuver share a BP and HR linking mechanism
- In a normal Valsalva maneuver, BP and HR both normalize. In autonomic failure, HR remains high and BP stays low
- Quantified by the Expiratory/inspiratory (E/I) or Valsalva ratio =

Longest R-R interval (phase IV) /

Shortest R-R interval (phase II)

- E/I ratio reflects PNS afferent (baro-receptor) and PNS and SNS efferent (parasympathetic and sympathetic) relation
- E/I > 1.20 is normal, <= 1.20 is abnormal

Procedure for the Valsalva Maneuver

- 1. While continuously recording BP and HR:
- 2. Take a deep breath, pinch your nose, and keep your mouth closed.
- 3. Try to exhale moderately forcefully for 10 to 15 seconds, as if inflating a balloon.
- 4. Gently release after about 15 seconds and breathe normall
- 5. Produces 4 stages of HR and BP changes



HR and BP: Traube-Hering and Mayer Waves

- Named for Ludwig Traube, Karl Konstantin Hering, and Siegmund Mayer
 <u>Taube-Hering</u>
- Rhythmical variations in blood pressure with a frequency varying from 6 to 10 cycles per minute (0.10 to 0.16 Hz)
- Related to variations in vasomotor tone
- Discovered in 1865 by Traube
- Confirmed in 1869 by Hering

<u>Mayer</u>

- Oscillations similar to Taube-Hering waves observed in 1876 by Mayer
 - Thought they might be a separate entity than Traube-Hering
- Frequency ~0.1 Hz (10-second waves), correlated with HRV
- Due to oscillating sympathetic vasomotor tone (VT) of arterial blood vessels
- Cyclic waves in arterial blood pressure
- Waves in arterial blood pressure brought about by <u>oscillations</u> <u>in baroreceptor and chemoreceptor reflex</u> control systems.
- Vasodilation due to action on alpha adrenergic receptors
- Abolished or attenuated by blockade of alpha-adrenergic receptors Seen both in ECG and continuous blood pressure
- Arterial blood pressure linear frequency coupling with SNS
- Low frequency and non-synchronous with ventilatory pattern
- Frequency does not depend on gender, age or posture
- Shift to lower frequency may be associated with risk of hypertension

Baroreceptor Reflex (BR)



How does it work? BR mediates BP changes in response to HR changes via pressure receptors in the aortic arch and carotid arties. These baroreceptors increase discharge rate when stretched by BP elevation caused by increased HR. This signal (through CNIX from carotid and CNX from aorta) goes to the cardiovascular control center in medulla which then decreases vasomotor SNS and dilates vessels and increases cardiac vagal PNS, so that HR decreases. When BP falls, BR lower discharge leads to vasomotor SNS increase and vessel constriction, cardiac SNS increase and PNS withdrawal, and results in HR acceleration.

Resonance of HRV due to BR at RFB

At RFB (0.07-0.11 Hz, and corresponding to Mayer waves), the BRmediated BP oscillatory period is 180° out of phase with HR oscillations. HR oscillations are thus amplified at resonant frequency. Maximal BP is reached after ~5 second delay from the previous cycle of BR-mediated BP increase and therefore occurs at the same time that HR reaches a minimum, which lowers HR even further; conversely, minimal BP occurs as HR reaches maximum and increases HR even more.

~ 5 sec time delay in BR produces arterial pressure oscillation resonance with HRV						
@RFB	 HR and respiration periods synchronize; → BR + 5 second delay → BP 180° out of phase with HR; resonance of HRV; 	→ Coherence				




Ginsberg, J. P., & Nagpal, M. (2016). Disruption of Bradycardia During Vigilance: Autonomic Cardiac Dysregulation is Prelude to Disinhibition, Hyperarousal, and Attention Bias in Combat Veterans with PTSD. In *A Multidimensional Approach to Post-Traumatic Stress Disorder-from Theory to Practice*. InTech. HR Amplitude Resonance due to BP Phase Frequency Shift from BR 5-second delay creates the sharp 0.1 Hz peak characteristic of Coherence



Baroreceptor reflex (BR)

- HR changes in response to Respiration
- BR changes in response to HR to maintain homeostasis
- BP changes in response to BR
- HR amplitude resonates with BP at RFB
 - Fixed time delay ~5 sec in BR produces 0.1 Hz arterial pressure oscillation that resonates HR amplitude at 0.1 Hz
- RFB synchronizes respiration, HR, BR, BP
- BR also important in 0.03 Hz VT oscillations
 - Complex relationship between HR and 0.03 Hz VT oscillations

'Coherence'

- Uncertainty whether Mayer waves are independent of BR
 - ?Central oscillator?
 - ?Additive to resonance?



Triggered by respiration @`0.1 Hz, BR links HR and BP via CNS, produces HRV resonance, and maintains BP homeostasis. 0.03 Hz oscillations of VT also influence HR and BR.

Properties of HRV Coherence

- · Produced by resonant frequency breathing (RFB)
- RFB occurs ~0.1 Hz (=6 cycles/minute=10 sec/cycle=10 sec/ period)

Produced by HRV Biofeedback

• Also produced by other stimuli at 0.1 Hz frequency (e.g. rhythmical muscle tension, chanting, picture presentation, etc.)

• Due to interactions between cycles of respiratory sinus arrhythmia (RSA) and baroreflex feedback control of vasomotor tone

- 0° phase between respiratory and sinusoidal HR $\,$ cycles

- 180° phase between baroreflex and sinusoical HR cycles
- Associated with maximum RSA (max-min HR over respiratory cycle)
- Discrete sharp peak in power spectrum at resonant frequency
- Associated with improved adaptive behaviors
 - alertness, responsiveness
 - emotional self-regulation
 - cognitive function
- distinct from "relaxation"
- Healthy people do not have Coherence during non RFB periods
- Extended high coherence may be a sign of inflexibility of cardiac adjustment

Summary: RSA and BR work together to produce HRV

- 1. RSA
 - Relatively small amplitude HR oscillations
 - Frequency range of normal respiration
 - ~10-24 breaths/minute= 0.17Hz-0.40Hz
 - HR accelerates on inspiration (SNS)
 - Returns to resting HR on exhalation (PNS)
 - Oscillations of cardiac output
 - Affects intra-thoracic pressure and venous return \rightarrow HR
 - Smaller BP oscillations (Traube-Hering waves?)
 - Hypertension reduces RSA and vagal tone
- 2. BR
 - BP changes in response to HR for homeostasis (?Mayer waves?)
 - RFB synchronizes respiration, HR, BR, BP→'Coherence'
 - Larger amplitude HR oscillations than RSA
 - Can be very large
 - Frequency range of ~4.2-7.5 breaths/minute (= 0.07-0.12 Hz),
 - Increases max and min of HR compared to slow respiration cycle
 - Average HR may not change
 - Important in 0.03 Hz VT oscillations
 - HRVB reduces BP

Attaining Coherence: HRV Biofeedback



"I THINK YOU SHOULD BE MORE EXPLICIT HERE IN STEP TWO,"



The heart rhythm pattern shown in the top graph is characterized by its erratic, irregular pattern (incoherence), and associated with negative emotions such as anger or frustration. The bottom graph shows a coherent heart rhythm pattern, observed when an individual is experiencing sustained, modulated positive emotions such as gratitude or happiness.

Three components of Autonomic Self-Regulation a

- 1. HRV Biofeedback = resonant frequency breathing
- 2. Mindful attention
- 3. Positive emotional state

ASR coaching essential elements

- Paced breathing at resonant frequency and the production of HRV Coherence through HRV Biofeedback
- Mindfulness or imagery focused on breathing and the heart. Focused attention on air entering and exiting the chest and passing thorough the heart
- Positive emotional state (PES). Occupy the mind during the HRVB session with thoughts of compassion, gratitude, apreciation, etc.











Time-Domain Results

Variable	Units	Value
Mean RR*	(ms)	946.4
STD RR (SDNN)	(ms)	30.1
Mean HR*	(1/min)	63.46
STD HR	(1/min)	2.02
RMSSD	(ms)	17.1
NN50	(count)	5
pNN50	(%)	1.9
RR triangular index		6.816
TINN	(ms)	115.0

Frequency–Domain Results

FFT spectrum (Welch's periodogram: 256 s window with 50% overlap)





Time-Domain Results

Variable	Units	Value
Mean RR*	(ms)	928.4
STD RR (SDNN)	(ms)	17.0
Mean HR*	(1/min)	64.65
STD HR	(1/min)	1.18
RMSSD	(ms)	8.7
NN50	(count)	0
pNN50	(%)	0.0
RR triangular index		4.534
TINN	(ms)	75.0

Frequency–Domain Results

FFT spectrum (Welch's periodogram: 256 s window with 50% overlap)



Figure 1 (a – d) depicts the Pre-Post HRVB Training the R-R Interval Tachogram and Power Spectra Density of one PTSD+ subject.

Pre-Training





HRV Power Spectrum

Peak Power at 0.099 Hz = 960.4 ms²; Total LF Power = 2344.4 ms²/Hz



Calculation of McCraty's Coherence Ratio (CR) from the HRV **Power Spectrum**





Figure 3 A typical heart rate variability (HRV) recording over a 15-minute period during resting conditions in a healthy individual

McCraty, R., & Shaffer, F. (2015). Heart rate variability: new perspectives on physiological mechanisms, assessment of self-regulatory capacity, and health risk. *Global Advances in Health and Medicine*, *4*(1), 46-61.

Beyond Coherence?

Calculation of the Parasympathetic Ratio (PR) from the HRV Power Spectrum



HRV(B) Case Data

PTSD 2 Pre-



Frequency-Domain Result FFT spectrum) **Time-Domain Results** RR Spectrum 0.013 Variable Units VLF LF HF Variable Units Value 0.00-0.04 0.04-0.15 Frequency band (Hz) 0.15-0.40 1005 Mean RR* (ms) PSD (s²/Hz) (Hz) 0.040 0.070 0.257 Peak frequency Mean HR* 60 (bpm) 33 264 359 Power 54 (ms²) Min HR (bpm) Power (log) 3.507 5.575 5.885 Max HR 70 (bpm) 54.74 Power (%) 5.08 40.16 SDNN 26.3 (ms) 42.31 57.66 28.7 RMSSD Power (n.u.) (ms) NN50 72 (beats) pNN50 (%) 9.54 657 Total power (ms²) 0.3 0.4 0.5 0.1 0.2 0 RR triangular index 8.89 Total Power 6.487 (log) Frequency (Hz) TINN 135.0 (ms) LF/HF ratio 0.734 Stress Index (SI) 13.7 EDR (Hz)

Peak (0.04-0.26 Hz)	Peak Power	Total Power	CR
0.26	153.2	656.5	0.23
Peak (0.07-0.40 Hz)	Peak Power	Total Power	PR
0.26	153.2	656.5	0.23





Time-Doma	in Results		Frequency-Do	main	Result	T spectri	im)	
			Trequency-Domain Results in opecation				RR Spectrum	
Variable	Units	Value	Variable	Units	VLF	LF	HF	0.114
Mean RR*	(ms)	841	Frequency band	I (Hz)	0.00-0.04	0.04-0.15	0.15-0.40	
Mean HR*	(bpm)	71	Peak frequency	(Hz)	0.040	0.087	0.167	2
Min HR	(bpm)	64	Power	(ms ²)	9	2076	145	王
Max HR	(bpm)	83	Power	`(log)	2.207	7.638	4.974	(s ²
SDNN	(ms)	43.6	Power	(%)	0.41	93.11	6.49	8
RMSSD	(ms)	24.5	Power	(n.u.)		93.49	6.51	2 I
NN50	(beats)	8						
pNN50	(%)	3.24	Total power	(ms ²)	2229			
RR triangular	index	10.33	Total Power	(log)	7.709			4 NIVAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAA
TINN	(ms)	201.0	LF/HF ratio		14.354			0 0.1 0.2 0.3 0.4 0.5
Stress Index	(SI)	10.4	EDR	(Hz)	-			Frequency (Hz)

Peak (0.04-0.26 Hz)	Peak Power	Total Power	CR
0.09	1769.8	2229.3	0.86
Peak (0.07-0.40 Hz)	Peak Power	Total Power	PR
0.09	1769.8	2229.3	0.86

PTSD 2 Pre-



PTSD 2 Post-



PTSD 2 Pre-



Time-Domair	n Results		Frequency-D	omain	Result≰FF	T spectru	ım)	RR Spectrum
Variable	Units	Value	Variable	Units	VLF	LF	HF	0.013
Mean RR* Mean HR* Min HR Max HR SDNN RMSSD NN50 pNN50 RR triangular i TINN Stress Index (S	(ms) (bpm) (bpm) (bpm) (ms) (ms) (beats) (%) ndex (ms) SI)	1005 60 54 70 26.3 28.7 72 9.54 8.89 135.0 13.7	Frequency ban Peak frequency Power Power Power Power Total power Total Power LF/HF ratio FDR	d (Hz) (Hz) (ms ²) (log) (%) (n.u.) (ms ²) (log) (Hz)	0.00-0.04 0.040 33 3.507 5.08 657 6.487 0.734	0.04-0.15 0.070 264 5.575 40.16 42.31	0.15-0.40 0.257 359 5.885 54.74 57.66	(H ₂) 00 0 0.1 0.2 0.3 0.4 0.5 Frequency (Hz)
	-			(, .=)				

Peak (0.04-0.26 Hz)	Peak Power	Total Power	CR
0.26	153.2	656.5	0.23
Peak (0.07-0.40 Hz)	Peak Power	Total Power	PR
0.26	153.2	656.5	0.23

eHFpp>LFpp RMSSD>SDNN





PTSD 5 Pre-



Peak (0.04-0.26 Hz)	Peak Power	Total Power	CR
0.26	45.7	428.6	0.12
Peak (0.07-0.40 Hz)	Peak Power	Total Power	PR
0.29	91.7	428.6	0.27

PTSD 5 Post-



Time-Domain Results

Frequency-Domain Result FFT spectrum)

Variable	Units	Value
Mean RR*	(ms)	854
Mean HR*	(bpm)	70
Min HR	(bpm)	59
Max HR	(bpm)	84
SDNN	(ms)	36.4
RMSSD	(ms)	23.5
NN50	(beats)	25
pNN50	(%)	4.15
RR triangular ir	ndex	10.79
TINN	(ms)	186.0
Stress Index (S	i)	10.7

					_	
Variable	Units	VLF	LF	HF	RR Spectrum	_
Frequency band	d (Hz)	0.00-0.04	0.04-0.15	0.15-0.40	0.060	
Peak frequency	(Hz)	0.033	0.120	0.153		
Power	(ms^2)	48	1228	153	2	
Power	(log)	3.875	7.113	5.032	£	
Power	(%)	3.37	85.90	10.72	(8 ⁻	
Power	(n.u.)		88.90	11.10		
Total power	(ms ²)	1430				
Total Power	(log)	7.265				
LF/HF ratio		8.011			a ver home	
EDR	(Hz)	-			0 0.1 0.2 0.3 0.4	0.5
					Frequency (Hz)	

Peak (0.04-0.26 Hz)	Peak Power	Total Power	CR
0.12	739.2	1429.7	1.07
Peak (0.07-0.40 Hz)	Peak Power	Total Power	PR
0.12	739.2	1429.7	1.07

PTSD 5 Pre-



PTSD 5 Post-



Time-Domain Results

Frequency-Domain Result FFT spectrum)



Pain 17 Pre-



Time-Domain Results

Frequency-Domain Result FFT spectrum)

Variable	Units	Value	Variable
Mean RR*	(ms)	833	Frequenc
Mean HR*	(bpm)	72	Peak freq
Min HR	(bpm)	64	Power
Max HR	(bpm)	96	Power
SDNN	(ms)	27.5	Power
RMSSD	(ms)	19.4	
NN50	(beats)	13	Total pow
pNN50	(%)	1.80	Total Pow
RR triangular	index	6.94	LF/HF rati
TINN	(ms)	154.0	EDR
Stress Index (SI)	13.1	

Variable	Units	VLF	LF	HF	RR Spectrum
Frequency band	(Hz)	0.00-0.04	0.04-0.15	0.15-0.40	0. <u>013</u>
Peak frequency	(Hz)	0.037	0.047	0.203	
Power	(ms^2)	83	489	119	
Power	(log)	4.417	6.193	4.775	운
Power	(%)	11.99	70.80	17.15	°4s
Power	(n.u.)		80.44	19.49	
Total power	(ms ²)	691			
Total Power	(log)	6.538			l h a h
LF/HF ratio		4.128			0 M MMMMMMMMMMMMMMMMMMMMMMMMMMMMMMMMMM
EDR	(Hz)	-			0 0.1 0.2 0.3 0.4 0.5
					Frequency (Hz)

Peak (0.04-0.26 Hz)	Peak Power	Total Power	CR
.05	181.9	690.5	0.36
Peak (0.07-0.40 Hz)	Peak Power	Total Power	PR
.08	69.2	690.5	0.10

Pain 17 Post-



Peak (0.04-0.26 Hz)	Peak Power	Total Power	CR
0.10	202.5	631.7	0.47
Peak (0.07-0.40 Hz)	Peak Power	Total Power	PR
0.10	202.5	631.7	0.47

Pain 17 Pre-



Time-Domain Results

Frequency-Domain Result FFT spectrum)

Variable	Units	Value	Variable	Units	VIE		HE	RR Spectrum
Mean RR*	(ms)	833	Frequency	band (Hz)	0.00-0.04	0.04-0.15	0.15-0.40	0.013 05
Mean HR*	(bpm)	72	Peak freque	ency (Hz)	0.037	0.047	0.203	
Min HR	(bpm)	64	Power	(ms ²)	83	489	119	
Max HR	(bpm)	96	Power	(log)	4.417	6.193	4.775	H
SDNN	(ms)	27.5	Power	(%) (nu)	11.99	70.80	17.15	
RMSSD	(ms)	19.4		(1.4.)		00.44	13.45	정 .10
NN50	(beats)	13	Total power	(ms ²)	691			
pNN50	(%)	1.80	Total Power	r `(log)	6.538			A land
RR triangular	index	6.94	LF/HF ratio		4.128			4 WWWWWWWWW
TINN	(ms)	154.0	EDR	(Hz)	-			0 0.1 0.2 0.3 0.4 0.5
Stress Index	(SI) `́	13.1						Frequency (Hz)

Peak (0.04-0.26 Hz)	Peak Power	Total Power	CR	
.05	181.9	690.5	0.36	
Peak (0.07-0.40 Hz)	Peak Power	Total Power	PR	
.08	69.2	690.5	0.10	

LFpp>eHFpp SDNN>RMSSD

Pain 17 Post-



Pain 16 Pre-



Empirical keys of HRV profiles

- LFpp>eHFpp: excessive SNS (PR<0.4)
- eHFpp>LFpp and RMSSD>SDNN: excessive SNS with PNS (PR le ~0.4)
- eHFpp>LFpp and SDNN>RMSSD: PNS dominant (PR gt ~0.4)
- PR and CR are useful for comparing intra-individual change

CR vs PR after HRVB in a Sample of Veterans with Chronic Pain						
	Pre-	Post-	TP2 PR-TP2 PR (SE)	TP2 CR-TP1 CR1 (SE)		
	n	n	'Diff CR'	'Diff PR'		
Active	26	22	0.53 (0.23) ¹	0.62 (0.22) ¹		
Sham	29	19	0.06 (0.03) ²	0.05 (0.05) ²		
¹ p< .005 ² ns						

Predictive Strength of CR vs PR					
Active HRVB Group					
Rho (Diff PR with Diff Outcome) vs Rho (Diff CR with Diff Outcome)					
toff (bol	d means changed hs to sig	gnificant)			
Minutes weekly practice GT Perceived Stress Scale ND					
GT	BPI Severity	ND			
GT	BPI Interference	ND			
GT	Pain Catastrophizing	ND			
GT (-)	Physical Fatigue	ND			
GT (-)	Mental Fatigue	ND			
ND	Total Fatigue	ND			
ND					
	ive Strei active HR tcome) v toff (bold GT GT GT GT (-) GT (-) ND ND	ive Strewyth of CR vs PRActive HB GroupCome)Rho (Diff CR with Diff OCome)Weass changed ns to signGTPerceived Stress ScaleGTBPI SeverityGTBPI InterferenceGTPain CatastrophizingGT (-)Mental FatigueNDTotal Fatigue			

GT=Greater Than; ND=No Difference

<u>Take-aways</u>

- HRV from resting HR tachygram can be quantified in a number of ways
- SNS HRV power above 0.07 Hz is minimal and becomes negligible by 0.1 Hz
 - PNS power below 0.07 Hz is minimal
- HRV power below 0.07 Hz is common in clinical populations and may indicate pathology
 - Reflected by eHF peak power vs LF peak Power
- Interpretation of a single tachygram requires both graphic display and several indices
- Intra-person comparisons are generally informative
 - Coherence and Parasympathetic Ratios are both promising
- Inter-person comparisons require both graphic display and several indices
 - eHFpp vs LFpp and SDNN vs RMSSD
 - Parasympathetic Ratio may be more predictive




REFERENCES AND READINGS

Readings on RSA: Respiration and Vagal Tone

Eckberg, D. L. (1997). Sympathovagal balance: a critical appraisal. *Circulation*, *96*(9), 3224-3232. (republished March 2018)

Grossman P. (2017). Comment on heart rate variability and cardiac vagal tone in psychophysiological research – recommendations for experiment planning, data analysis, and data reporting. *Front Psychol* 8:213. doi:10.3389/fpsyg.2017.00213

Grossman, P., & Kollai, M. (1993). Respiratory sinus arrhythmia, cardiac vagal tone, and respiration: Within-and between-individual relations. *Psychophysiology*, *30*(5), 486-495.

Grossman, P., & Taylor, E. W. (2007). Toward understanding respiratory sinus arrhythmia: relations to cardiac vagal tone, evolution and biobehavioral functions. *Biological psychology*, 74(2), 263-285.

Laborde, S., Mosley, E., & Thayer, J. F. (2017). Heart rate variability and cardiac vagal tone in psychophysiological research–recommendations for experiment planning, data analysis, and data reporting. *Frontiers in psychology*, *8*, 213.

Shaffer, F., & Ginsberg, J. P. (2017). An overview of heart rate variability metrics and norms. *Frontiers in public health*, *5*, 258.

Readings on Traube-Herring and Mayer Waves

Halliburton, W.D. (1919). Traube waves and Mayer waves. King's College, London, <u>https://physoc.onlinelibrary.wiley.com/doi/pdf/10.1113/expphysiol.1919.sp000262</u>

Visscher, M., Rupp, A., and Scott, F.H. (1924). The respiratory wave in arterial blood pressure. Department of Physiology, University of Minnesota <u>https://www.physiology.org/doi/pdf/10.1152/ajplegacy.1924.70.3.586</u>

Davidson, N. S., Goldner, S., & McCloskey, D. I. (1976). Respiratory modulation of baroreceptor and chemoreceptor reflexes affecting heart rate and cardiac vagal efferent nerve activity. *The Journal of physiology*, *259*(2), 523-530. <u>https://physoc.onlinelibrary.wiley.com/doi/pdf/10.1113/jphysiol.1976.sp011480</u>

Eckberg, D. L., & Sleight, P. (1992). *Human baroreflexes in health and disease* (No. 43). Oxford University Press.

Karemaker, J. M. (1999). Autonomic integration: the physiological basis of cardiovascular variability. *The Journal of physiology*, *517*(2), 316-316.

Blood pressure (BP) and heart rate (HR) are continually varying. The nervous mechanisms behind this variability have been studied extensively in non-human animal models (for reviews, see Eckberg & Sleight, 1992). Since the 1980s, the combined availability of non-invasive blood pressure measurement by the Peñáz-Wesseling Finapres, and more and more powerful computers in the laboratory, have revived research into prevalence and physiological meaning of this variability in humans. When Fourier analysis was applied to analysis of BP variability (BPV) and HR variability (HRV), two frequency peaks stood out: one around the respiratory frequency and one around 0.1 Hz, or one oscillation in 10 s. These frequencies had been observed in blood pressure recordings before, actually over 130 years ago: Traube-Hering waves (coupled to respiration) and Mayer waves, the 0.1 Hz and slower oscillations. In the earlier research it had been established that oscillating sympathetic activity causes the Mayer waves in blood pressure. The respiration-coupled blood pressure oscillations were partly explained by mechanical effects of respiration and possibly by the vagally induced heart period oscillations coupled to respiration, known as respiratory sinus arrhythmia (RSA) (Eckberg & Sleight, 1992).

Bernardi, L., Porta, C., Gabutti, A., Spicuzza, L., & Sleight, P. (2001). Modulatory effects of respiration. *Autonomic neuroscience*, *90*(1-2), 47-56.

Respiration is a powerful modulator of heart rate variability, and of baro- and chemoreflex sensitivity. Abnormal respiratory modulation of heart rate is often an early sign of autonomic dysfunction in a number of diseases. In addition, increase in venous return due to respiration may help in maintaining blood pressure during standing in critical situations. This review examines the possibility that manipulation of breathing pattern may provide beneficial effects in terms not only of ventilatory efficiency, but also of cardiovascular and respiratory control in physiologic and pathologic conditions, such as chronic heart failure. This opens a new area of future research in the better management of patients with cardiovascular autonomic dysfunction. Julien, C. (2006). The enigma of Mayer waves: facts and models. *Cardiovascular research*, *70*(1), 12-21.

Mayer waves are oscillations of arterial pressure occurring spontaneously in conscious subjects at a frequency lower than respiration (~ 0.1 Hz in humans). Mayer waves are tightly coupled with synchronous oscillations of efferent sympathetic nervous activity and are almost invariably enhanced during states of sympathetic activation. For this reason, the amplitude of these oscillations has been proposed as a surrogate measure of sympathetic activity, although in the absence of a clear knowledge of their underlying physiology. Some studies have suggested that Mayer waves result from the activity of an endogenous oscillator located either in the brainstem or in the spinal cord. Other studies, mainly based on the effects of sino-aortic baroreceptor denervation, have challenged this view. Several models of dynamic arterial pressure control have been developed to predict Mayer waves. In these models, it was anticipated that the numerous dynamic components and fixed time delays present in the baroreflex loop would result in the production of a resonant, self-sustained oscillation of arterial pressure. Recent analysis of the various transfer functions of the rat baroreceptor reflex suggests that Mayer waves are transient oscillatory responses to hemodynamic perturbations rather than true feedback oscillations. Within this frame, the amplitude of Mayer waves would be determined both by the strength of the triggering perturbations and the sensitivity of the sympathetic component of the baroreceptor reflex.

Readings on Baroreflex: RFB and Coherence

- Luecken, L. J., & Gallo, L. C. (2008). *Handbook of physiological research methods in health psychology*. Sage.
- Vaschillo, E., Lehrer, P., Rishe, N., & Konstantinov, M. (2002). Heart rate variability biofeedback as a method for assessing baroreflex function: a preliminary study of resonance in the cardiovascular system. *Applied Psychophysiology and Biofeedback*, 27(1), 1-27.
- Vaschillo, E. G., Vaschillo, B., Buckman, J. F., Pandina, R. J., & Bates, M. E. (2010, January). The investigation and clinical significance of resonance in the heart rate and vascular tone baroreflexes. In *International Joint Conference on Biomedical Engineering Systems and Technologies* (pp. 224-237). Springer, Berlin, Heidelberg.
- Vaschillo, E., Vaschillo, B., & Lehrer, P. (2004). Heartbeat synchronizes with respiratory rhythm only under specific circumstances. *Chest*, *126*(4), 1385-1386; author reply 1386-1387.
 Yasuma, F., & Hayano, J.I. (2004) Respiratory snus arrhythmia: why does the heart beat synchronize with respiratory rhythm? *Chest 125:*683-690.