Cardiovascular Psychophysiology
Facts and Functions

- The busy heart
  - Six quarts of blood pumped per minute
  - 100,000 beats per day
  - Try it!

- Functions
  - Transport oxygen from lungs and nutrients from gut
  - Transport waste products
  - Transport regulatory substances (e.g. endocrines)
  - Thermal exchange between core and periphery
Metabolic Demands
Anatomy of the Heart

- Cardiac Muscle (myocardium)
  - not striated, not smooth
  - four features distinguish from smooth or striate
    - Muscle has unstable resting potential – basis for intrinsic and rhythmic contraction
    - Action potential freely conducted from one cell to another (lattice-like syncytial) network of cardiac fibers
    - Repolarization lasts about 100 msec
    - Contraction phase = duration of cardiac action potentials (initial depolarization followed by sustained depolarization phase of 0.2-0.3 secs)

- Four chambers
  - Right Atrium
  - Right Ventricle
  - Left Atrium
  - Left Ventricle
Anatomy of the Heart

- Arch of the Aorta
- Superior Vena Cava
- Right Atrium
- Pulmonary Trunk
- Left Atrium
- Pulmonary Vein
- Inferior Vena Cava
- Mitral Valve
- Tricuspid Valve
- Papillary Muscle
- Right Ventricle
- Left Ventricle
- Ventricular Septum
Human Circulatory System
Circulation in a bit more realistic detail

Figure 8.1. Systemic and pulmonary circulation. In keeping with usual depictions of the heart, the right side of the heart is on the left side of the picture. Lighter gray areas indicate oxygenated blood and darker gray areas indicate deoxygenated blood.
Anatomy of the Heart

- Superior Vena Cava
- Aorta
- Pulmonary Arteries
- Pulmonary Veins
- Right A-V Valve
- Chordae Tendinae
- Pulmonary Veins
- Left A-V Valve
- Inferior Vena Cava
- Intraventricular Septum
- Papillary Muscle
More Valves

- Aortic and Pulmonary Valves
  - Respond to relative pressure difference between ventricles and aorta or pulmonary artery
  - As ventricles contract, pressure builds, and forces valves open when pressure exceeds arterial pressure
- “Dub” in the Lub-Dub sound (sounds are valves closing or “slamming” shut)
Neural Conduction of the Heart

- Two Nodes
  - Sino-Atrial (SA) node – “Primary Pacemaker”
  - Atrial-Ventricular (AV) node – “Yoked”

- Nodes have intrinsic rhythmicity
  - SA node: 105 bpm
  - AV node: 40-60 bpm

- Denervated heart would still beat at over 100 bpm
  - Must be extrinsic influences to slow or speed heart
Neural Conduction of the Heart

- Hierarchy ensures that normally the SA node “drives” the system
  - AV nodes provide a critical delay (allows atria to fully contract before ventricles do)
  - AV nodes have important refractory period to prevent rapid successive ventricular contractions
- A coordinated wave of depolarization
  - Contraction of 4 chambers of heart must be precisely choreographed
The SA and AV Nodes in Action
The Schematized EKG waveform

P = Atrial depolarization
QRS = Ventricular depolarization
T = Ventricular repolarization
Note that Atrial repolarization is not visible
The EKG waveform

- P-wave
- P-R (or P-Q) interval
- P-wave duration
- Isoelectric line
- QRS complex duration
- S-T interval
- T-wave duration
- T-P segment
- QT interval
- P-P interval
The Cardiac Cycle

Lead II electrocardiogram:
- P wave
- R wave

Muscle contraction:
- Atrial
- Ventricular

Pressure, mmHg:
- Aortic pressure
- Pulse pressure
- Left ventricular pressure
- Left atrial pressure

Aortic valve:
- Closed
- Open
- Closed

Mitral valve:
- Open
- Closed
- Open

Heart sounds:
- S1
- S2
- S2

Left ventricular volume, ml:
- End-diastolic volume
- End-systolic volume

Aortic flow:
- Inflow
- Outflow

Time, s:
- 0.6
- 0.8

Systole

See also Fig 8.2 in Text
Cardiac Output

\[ CO = HR \times SV \]
Cardiac Chronoptropy

- Heart rate regulated extrinsically
- Vagal (PNS) influence
  - Slows HR
  - So too will dripping ACH on SA node 😊
  - Likely that all changes below 100 bpm are predominately vagally induced
- SNS influence
  - Speeds HR, but impact not as strong as PNS
  - Main effect is to increase contractility
**Figure 8.6.** General pattern of pharmacology of the autonomic innervations. Abbreviations refer to the relevant postsynaptic receptor populations: $N_N$ – nicotinic cholinergic; $M$ – muscarinic cholinergic; $\beta_1$ – beta1 adrenergic.
SNS and PNS influences

CARDIAC PARASYMPATHETIC NERVE ACTIVITY LEVEL -> HEART RATE
- negative chronotropic

CARDIAC SYMPATHETIC NERVE ACTIVITY LEVEL -> CARDIAC OUTPUT
- positive chronotropic
- contractility (positive inotropic)

ARTERIAL PRESSURE
- afterload

FILLING PRESSURE
- preload
(Starling's law)

STROKE VOLUME
- +
- +
HR change to simultaneous vagal and sympathetic stimulation

Figure 8.9. Autonomic space. (A) Continuum model of autonomic control, wherein the status of the system can be depicted along a single continuum extending from parasympathetic dominance to sympathetic dominance. (B) A more comprehensive model of autonomic control, characterized by an autonomic plane (representing the fact that parasympathetic and sympathetic systems can change reciprocally, cooperatively, or independently) and an overlying effector surface which illustrates the end organ state (heart period) for any location on the underlying autonomic plane. Beta illustrates the intrinsic heart period in the absence of autonomic control.
Integrated Control Mechanisms

- **Baroreceptor Reflex**
  - Pressure sensitive receptors
  - Located in the arch of the aorta and carotid sinus nerves
  - Join Vagal and Glossopharangeal nerves
  - Terminate in regulatory centers in medulla
  - With increase in BP, causes compensatory decrease in HR, contractility, and SV
  - Quickly adjusts to maintain BP

- **Valsalva Maneuver**
Figure 8.7. General organization of the baroreceptor heart rate reflex. Reflex originates in mechanoreceptors in the heart and the carotid and other great arteries. The NTS excites (+ symbol) the parasympathetic motor neurons (PMN) and inhibits (− symbol) relay neurons to the sympathetic motor neuron pool (SMN). Insert illustrates the relationship between blood pressure (BP) and heart rate (HR). PG and SG depict parasympathetic and sympathetic ganglia, respectively. Other abbreviations are as in Figure 8.6.
Valsalva Maneuver
Integrated Control Mechanisms

- **Respiratory Effects**
  - Respiratory Sinus Arrhythmia (RSA)
    - This arrhythmia is not a bad thing!
  - HR acceleration linked to inspiration
  - HR deceleration linked to expiration

- **RSA**
  - Indexes strength of Vagal influence
  - More later…
Cardiac Inotropy

- Contractility is predominately sympathetically mediated
- Often measured invasively, but can be measured noninvasively
  - EKG plus phonocardiogram
  - Impedance cardiography
SNS and PNS Integration: A Caveat

- Relatively easy to measure PNS: RSA or other metrics of HRV
- Relatively easy to measure SNS: Contractility via PEP
- BUT... one is measure of chronotropy, other is measure of inotropy
  - Changes in contractility can occur independently of changes in rate
  - SNS inputs for inotropy primarily controlled by left-sided inputs to AV node
  - SNS inputs of chronotropy primarily controlled by right-sided inputs to SA node
- Thus, like “mixing apples and oranges”
Cardiovascular Measures

- Electrocardiogram (EKG)
- Phonocardiogram (PCG)
- Impedance cardiography
- Photoplethysmography
- Ballistocardiography
- Blood Pressure
EKG

AC signal
Sample 200-500 Hz

Frontal plane

Bipolar

<table>
<thead>
<tr>
<th>Bipolar limb leads: ECG voltage measurements between pairs of limbs:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lead I: between RA and LA</td>
</tr>
<tr>
<td>Lead II: between RA and LL</td>
</tr>
<tr>
<td>Lead III: between LA and LL</td>
</tr>
</tbody>
</table>

Einthoven triangle, showing relation of the bipolar limb leads
EKG

AC signal
Sample 200-500 Hz
Which Time?

- Real time
  - Heart Rate
  - Expressed as beats per time (usually bpm)
- Cardiac time
  - Heart Period; interbeat interval (IBI)
  - Expressed in msec
- Converting

\[
HR = \frac{1}{HP} \times 60,000
\]

\[
HR = \frac{1}{1000} \times 60,000 = 60\text{bpm}
\]
Phonocardiography

- Position microphone over heart
- Lub-Dub is transduced to electrical signal
Photoplethysmography

Three methods, all involve measuring light absorbed by peripheral vasculature.
The Photoplethysmographic Output

Increase in Pressure due to opening of Aortic Valve

Dichrotic Notch; closing of valve, end of ejection

~LVET
Photoplethysmograph: Peripheral Vasoconstriction

T1 is onset of constriction
Top Panel: Pulse Volume (recorded with 1 sec time constant)
Lower Panel: Blood Volume (no filter)
Measuring contractility with EKG, PCG, and Photoplethysmography

PEP = Pre-ejection period
LVET = Left Ventricular Ejection Time
   = Upswing of pressure wave to S2
Electromechanical Systole = Q to S2
PEP = EMS – LVET

PEP reflects sympathetic influence on cardiac contractility
Measuring Blood Pressure

Auscultatory Technique
- Not good for instantaneous readings
- Not good for repeated readings
Ballistocardiography

- Imagine
  - On a chair on a platform on an air hockey table
  - Cardiac events cause movement of platform
- New applications:
  - Finding individuals hiding in vehicles
  - Finding individuals stuck in rubble
Impedance Cardiography

- Low energy high-frequency AC passed through thoracic region
- Changes in impedance to signal created by mechanical events of cardiac cycle, especially changes in thoracic blood volume
- $\Delta Z$ is change in impedance
- $dz/dt$ is 1st derivative of impedance signal $Z$
- $R-Z$ is time from r-wave to peak ventricular contraction indicated in $Z$ signal
- The “Heather” index – divide $dz/dt$ by $R-Z$ interval; putative measure of heart’s ability to respond to stress
Measuring Vagal Influence

- Descending Vagal Influence slows HR
- Respiration interrupts this vagal influence
- The size of periodic oscillations due to respiration can therefore index the strength of the Vagal influence
  - Note, however, that under some circumstances, there can be dissociation between RSA and presumed central cardiac vagal efferent activity (cf., Grossman & Taylor, 2007)
  - Concerns over changes in rate, and to lesser extent depth
  - See special issue of *Biological Psychology*, 2007 for more in depth treatment of these issues and more!

- **Demo** with QRSTool
Abbreviated History of HR Variability
(with thanks to Porges, 2007)

- Physiology treated HR as DV, similar to behavior
- Dominance of behaviorism emphasized control over the DV (behavior)
- Changes in HR unrelated to the manipulation considered noise
  - Lacey (1967) and Obrist (1981) had models related to attention, and metabolic demand, but HR *variability* did not fit in either model
  - Via appropriate experimental design, HR should be entirely under the control of experimental or environmental demands
- Nonetheless, history of quantifying HR variability dates to the 1950’s with case report long before that:
  - 1958: Lacey and Lacey, greater HRV associated with greater impulsivity
  - 1915: Eppinger and Hess, described a vagotonic syndrome with clinical features that included an exaggerated RSA
  - Interest in HRV as an *individual difference* variable, however, really starts with the work of Steve Porges
IBI Series (real time)

.12-.40 Hz filtered IBI Time Series
High Variability Subject

<table>
<thead>
<tr>
<th>Rate</th>
<th></th>
<th>Total Variability</th>
<th></th>
<th>&quot;Sympathetic&quot;</th>
<th></th>
<th>&quot;Parasympathetic&quot;</th>
</tr>
</thead>
<tbody>
<tr>
<td>73.3</td>
<td>HR</td>
<td>9.2</td>
<td>HRV</td>
<td>1.4</td>
<td>CSI</td>
<td></td>
</tr>
<tr>
<td>832.3</td>
<td>IBI</td>
<td>112.4</td>
<td>SDNN</td>
<td>57.1</td>
<td>PNN50</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>132.8</td>
<td>RMSSD</td>
<td>97.6</td>
<td>MCD</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>5.3</td>
<td>CVI</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>8.8</td>
<td>RSA</td>
<td></td>
</tr>
</tbody>
</table>

Low Variability Subject
- Reptilian “Dumb”: Dorsal Motor Nucleus
  - Massive reduction in HR & conservation of oxygen.
  - Dive reflex -- cold water on the face during breath hold
- Phylogentically newer “smart” Vagus
  - Originates from Nucleus Ambiguous
  - Modulates influence to:
    - Promote attentional engagement, emotional expression, and communication.
    - Mobilizes organism to respond to environmental demands
      - Phasically withdraws inhibitory influence, increasing HR
      - Upon removal of the environmental stressor, resumes its efferent signal
        - Slowing heart rate
        - Allows the organism to self-sooth

This polyvagal theory is not without its critics (e.g., Grossman & Taylor, 2007).
Bradycardia observed in a diving seal. Data adapted from R.S. Elsner (1998), courtesy of http://www.deeperblue.net/article.php/225
<table>
<thead>
<tr>
<th></th>
<th>ANS Component</th>
<th>Behavioral Function</th>
<th>Lower motor neurons</th>
</tr>
</thead>
<tbody>
<tr>
<td>III</td>
<td>Myelinated vagus (ventral vagal complex)</td>
<td>Social communication, self-soothing and calming, inhibit “arousal”</td>
<td>Nucleus ambiguus</td>
</tr>
<tr>
<td>II</td>
<td>Sympathetic-adrenal system</td>
<td>Mobilization (active avoidance)</td>
<td>Spinal cord</td>
</tr>
<tr>
<td>I</td>
<td>Unmyelinated vagus (dorsal vagal complex)</td>
<td>Immobilization (death feigning, passive avoidance)</td>
<td>Dorsal motor nucleus of the vagus</td>
</tr>
</tbody>
</table>

Fig. 1. Phylogenetic stages of the polyvagal theory.

Porges, 2007
Tonic Vs Phasic

- Tonic Level indexes capacity
- Phasic change indexes actualization of that capacity

**Attention**
- higher vagal “tone” was associated with faster reaction time to a task requiring sustained attention
  - attentional skills improved
  - appropriate task-related suppression of heart rate variability was observed while performing the task requiring sustained attention

**Emotion**
- Beauchaine (2001):
  - low baseline vagal “tone” is related to negative emotional traits
  - high vagal withdrawal is related to negative emotional states
Task-related and Emotion-related modulation

Movius & Allen, 2001
Individual Differences in Cardiac Vagal Control (aka “Trait Vagal Tone”) 

- **Infants**
  - Various sick infants have lower vagal tone (Respiratory Distress Syndrome, Hydrocephalic)
  - Infants with higher vagal tone (Porges, various years)
    - More emotionally reactive (both + & -)
    - More responsive to environmental stimuli (behaviorally and physiologically)

- **Anxiety Disorders**
  - Lower Vagal Tone in GAD ([Thayer et al., 1996](#))
  - Lower Vagal Tone in Panic Disorder ([Friedman & Thayer, 1998](#))

- **Depression**
  - Depression characterized by lower Vagal tone?
  - Gender may moderate ([Thayer et al., 1998](#))
    - Note small sample: 15 depressed, 11 controls
  - State dependent? ([Chambers & Allen, 2002](#))
Figure 1. Power in the high frequency (respiratory) component of heart period variability in GAD patients and controls during relaxation and worry.
<table>
<thead>
<tr>
<th>Variable</th>
<th>Panic (mean, S.D.)</th>
<th>Blood phobic (mean, S.D.)</th>
<th>Control (mean, S.D.)</th>
<th>T ratio, df, p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>IBI (ms)</td>
<td>761.8 (141.0)</td>
<td>837.1 (92.4)</td>
<td>905.2 (132.5)</td>
<td>P &lt; B 4.59 (215), p &lt; 0.001</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>P &lt; C 7.65 (214), p &lt; 0.001</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>B &lt; C 4.30 (207), p &lt; 0.001</td>
</tr>
<tr>
<td>VAR (ms²)</td>
<td>3942 (4009)</td>
<td>4334 (2663)</td>
<td>6112 (4563)</td>
<td>P &lt; C 3.70 (214), p &lt; 0.001</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>B &lt; C 3.44 (207), p &lt; 0.001</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>P = B N.S.</td>
</tr>
<tr>
<td>MSD (ms)</td>
<td>44.4 (31.2)</td>
<td>55.6 (22.7)</td>
<td>71.4 (32.1)</td>
<td>P &lt; B 3.05 (215), p &lt; 0.001</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>P &lt; C 6.34 (214), p &lt; 0.001</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>B &lt; C 4.11 (207), p &lt; 0.001</td>
</tr>
<tr>
<td>HF power (ms²</td>
<td>991 (1225)</td>
<td>1385 (1073)</td>
<td>2239 (1911)</td>
<td>P &lt; B 2.49 (212), p &lt; 0.01</td>
</tr>
<tr>
<td>Hz⁻¹)</td>
<td></td>
<td></td>
<td></td>
<td>P &lt; C 5.67 (212), p &lt; 0.001</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>B &lt; C 3.90 (203), p &lt; 0.001</td>
</tr>
<tr>
<td>LF/HF</td>
<td>2.1 (2.5)</td>
<td>1.3 (1.8)</td>
<td>1.0 (1.5)</td>
<td>P &lt; B 2.41 (209), p &lt; 0.005</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>P &lt; C 3.64 (203), p &lt; 0.001</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>B = C N.S.</td>
</tr>
</tbody>
</table>

P, panic; B, blood phobic; C, control.
Data from Thayer et al., 1998, *Bio Psychiatry*
Change in Vagal Tone

Change in HRSD Score

Chambers and Allen (2002) *Psychophysiology*
Trait Vagal Tone (cont’)

- Defensive Coping *(Movius & Allen, 2001)*
- Integrative Developmental Model

---

**Table 1. Patterns of autonomic nervous system functioning in common psychopathologies and personality types**

<table>
<thead>
<tr>
<th>Motivational Predisposition</th>
<th>Behavioral Manifestation</th>
<th>Motivational System (SNS)</th>
<th>Regulative System (PNS)</th>
<th>Emotional Trait (RSA)</th>
<th>Emotional State (RSA Reactivity)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Disinhibition</td>
<td>Impulsivity (ADHD)</td>
<td>High</td>
<td>Low</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>Aggression (UACD)</td>
<td>High</td>
<td>Low</td>
<td>Low</td>
<td>High</td>
</tr>
<tr>
<td></td>
<td>Panic</td>
<td>High</td>
<td>High</td>
<td>Low</td>
<td>High</td>
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<tr>
<td></td>
<td>Extraversion</td>
<td>High</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Inhibition</td>
<td>Anxiety</td>
<td>—</td>
<td>High</td>
<td>Low</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>Depression</td>
<td>Low</td>
<td>High</td>
<td>Low</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>Panic</td>
<td>High</td>
<td>High</td>
<td>Low</td>
<td>High</td>
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<tr>
<td></td>
<td>Introversion</td>
<td>—</td>
<td>High</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>None</td>
<td>Emotional stability</td>
<td>—</td>
<td>—</td>
<td>High</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>Emotional lability</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>High</td>
</tr>
</tbody>
</table>

*Note: High, atypically high activity; Low, atypically low activity. Dashes represent normal activity. Entries in the BIS, RSA, and RSA reactivity columns are supported by the literature reviewed herein. Entries in the BAS column are more speculative and require empirical confirmation.*
Beauchaine et al (2001)
Journal of Abnormal Psychology

Figure 4. Baseline respiratory sinus arrhythmia (RSA; top panel), and RSA reactivity (bottom panel) for aggressive CD/ADHD participants, hyperactive/impulsive ADHD participants, and controls. CD = conduct disorder; ADHD = attention-deficit/hyperactivity disorder.
Baseline Suppress Recovery

Low Def

High Def

Movius & Allen, 2001
Trait Vagal Tone as Moderator of Response following Bereavement

- Bereavement as a period of cardiovascular risk
- Disclosure as an intervention for Bereavement (O’Connor, Allen, Kaszniak, 2005)
- Overall, all folks get better, but no differential impact of intervention
- BUT… Vagal Tone as moderator

Intervention Group

Control Group
Orienting, Attention, and Defense

Emotional reactivity

- Pleasant
- Neutral
- Unpleasant

Heart rate

Change (BPM)

Time (s)
SCR (by contrast)

Emotional reactivity

- Pleasant
- Neutral
- Unpleasant

Skin conductance

Change (μSiemens)

Time (s)
OR Vs DR

Diagram showing the relationship between emotional intensity and physiological responses.

- **Sweat Glands**
  - Startle inhibition begins
  - SCL increase begins

- **Startle Reflex**
  - Cardiac deceleration begins
  - Startle potentiation begins

- **Heart Rate**
  - Cardiac acceleration begins

- **Response Amplitude**

- **Emotional Intensity**
  - Calm
  - Aroused

- **Freezing**
  - Fight, Flight

- **Overt Action**