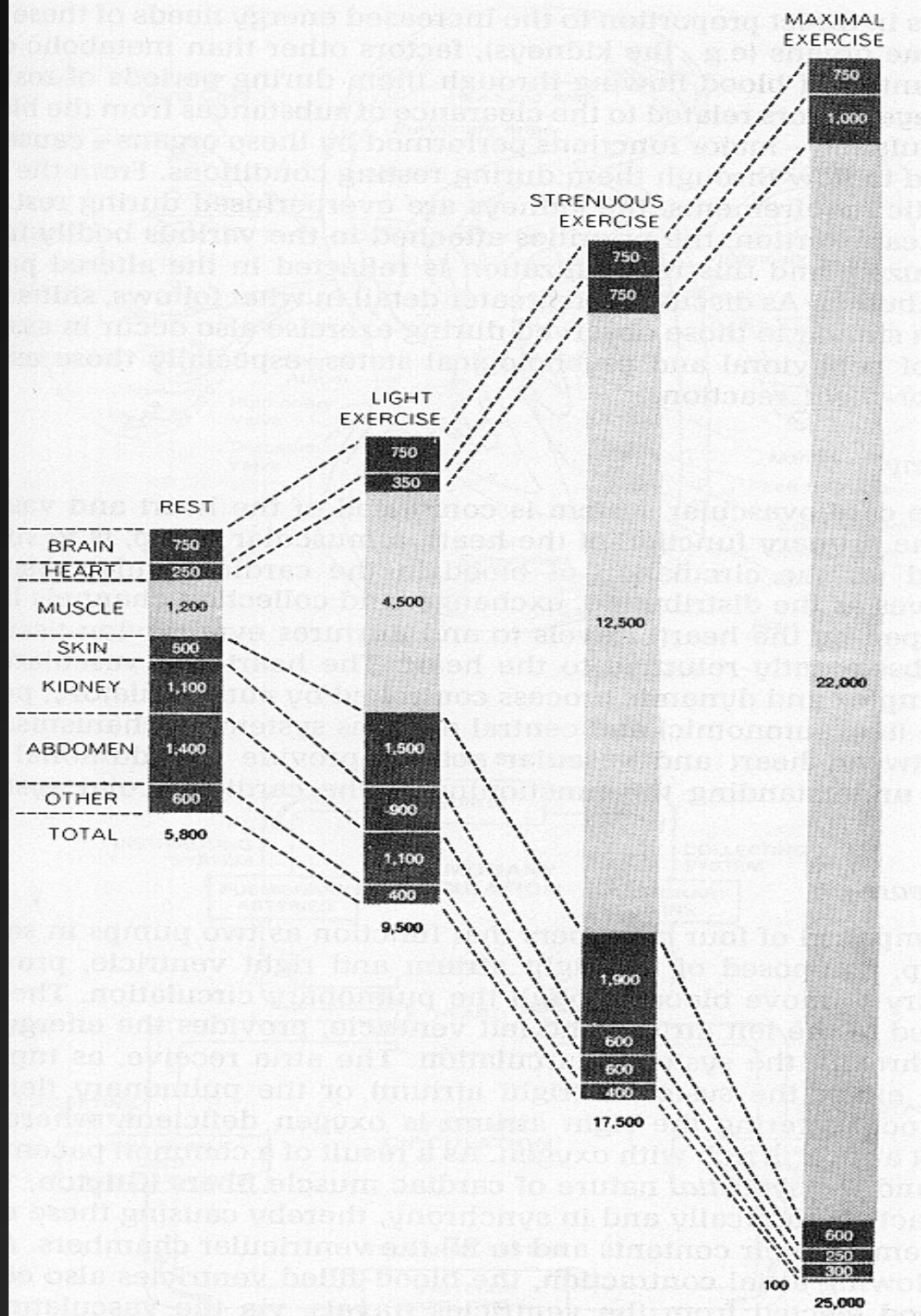


*Cardiovascular
Psychophysiology*

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

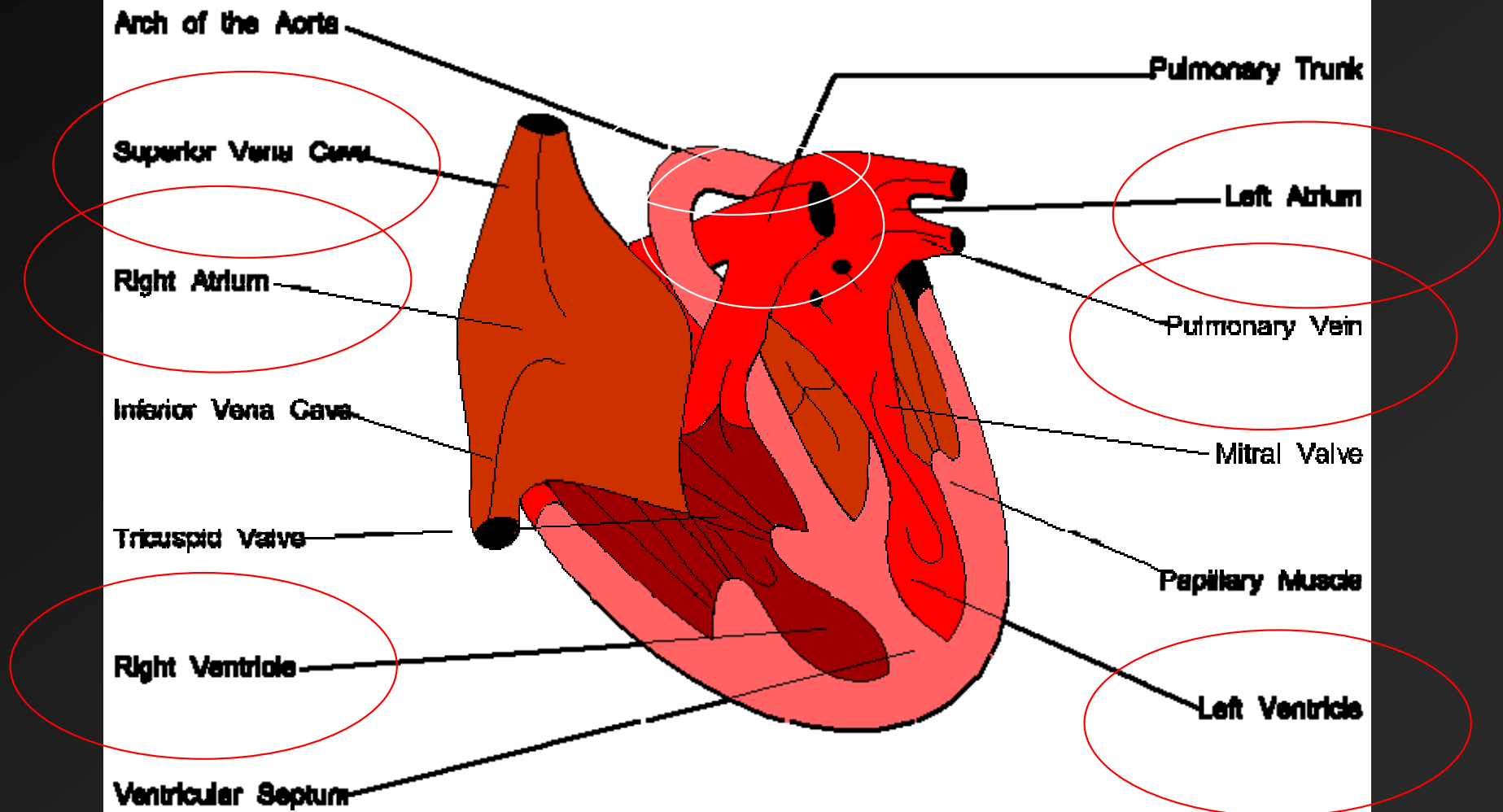


Anatomy of the Heart

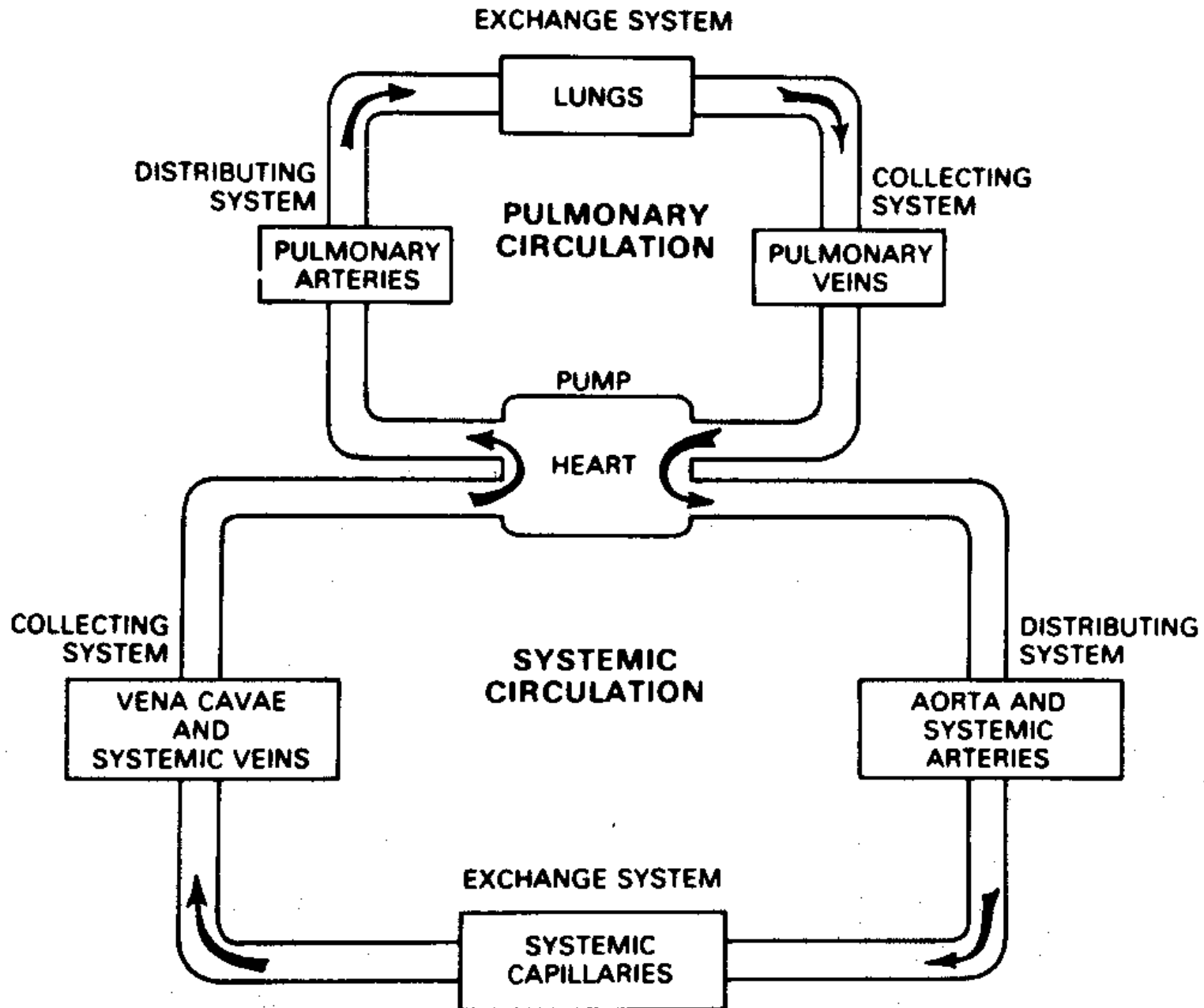
- Cardiac Muscle (myocardium)
 - not striated, not smooth
 - four features distinguish from smooth or striate
 - Muscle has unstable resting potential – basis for intrinsic and rhythmic contraction
 - Action potential freely conducted from one cell to another (lattice-like syncytial) network of cardiac 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

Heart



Human Circulatory System



Circulation in a bit more realistic detail

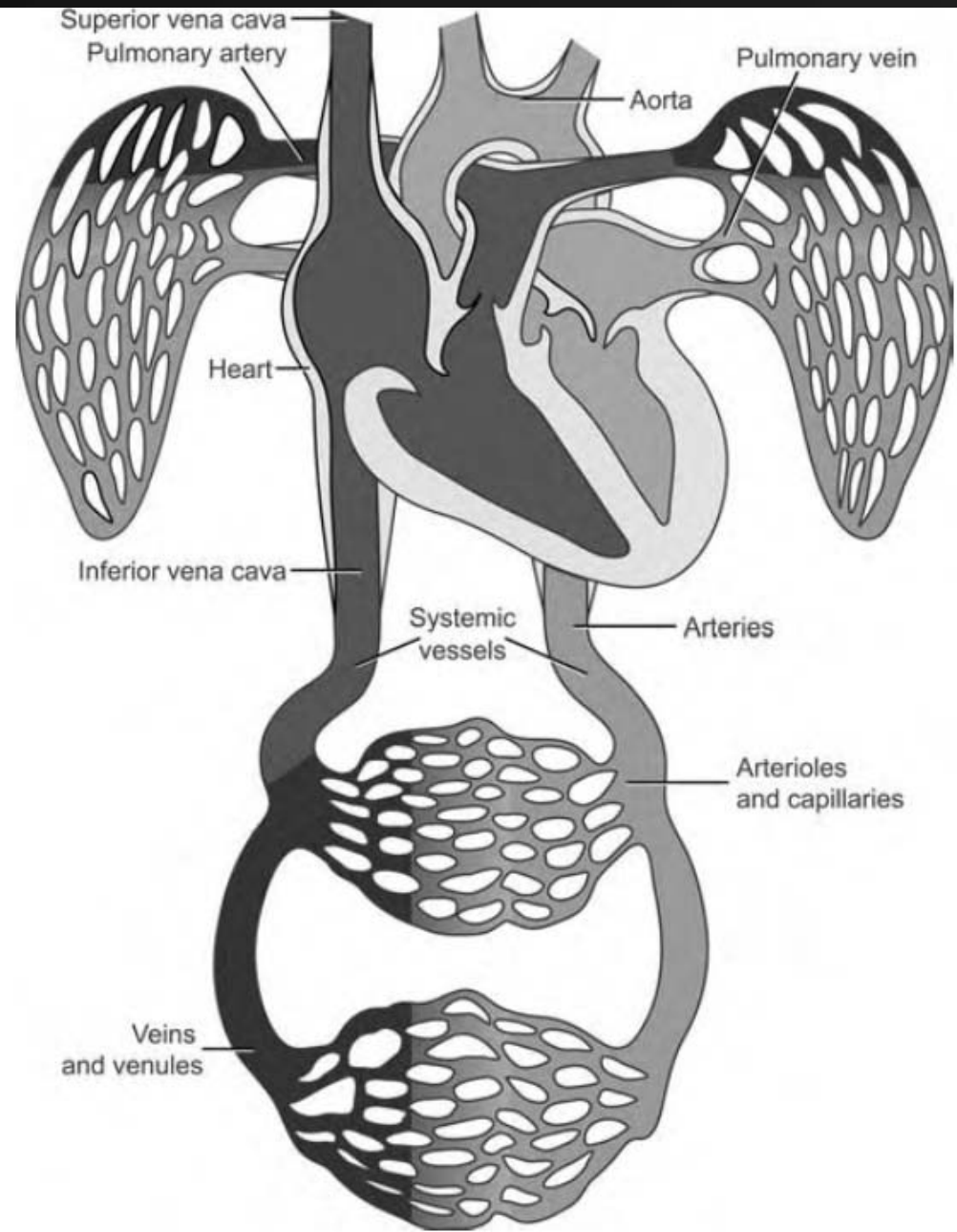
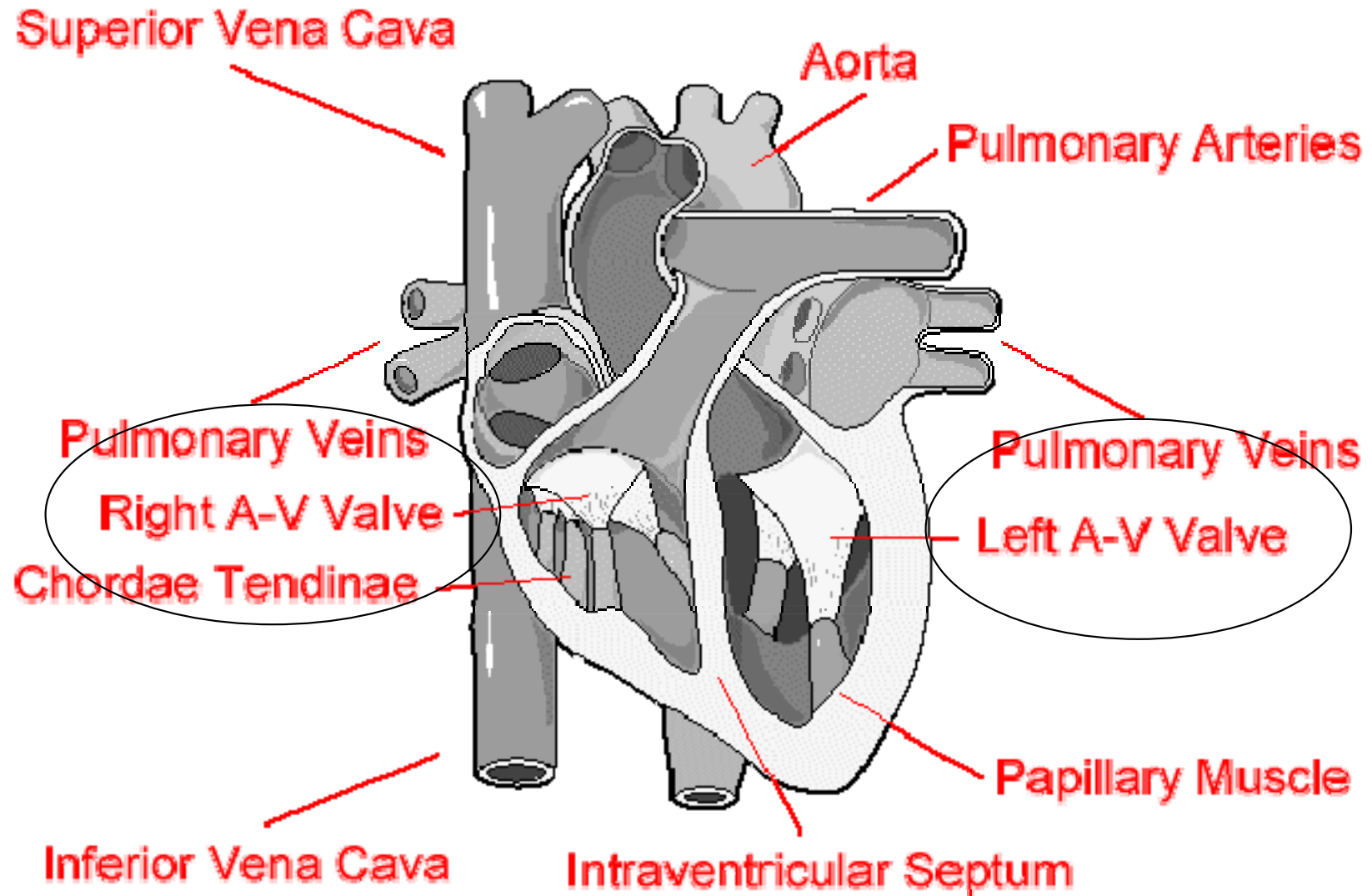


Figure 8.1. Systemic and pulmonary circulation. In keeping with usual depictions of the heart, the right side of the heart is on the left side of the picture. Lighter gray areas indicate oxygenated blood and darker gray areas indicate deoxygenated blood.

Anatomy of the Heart



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)



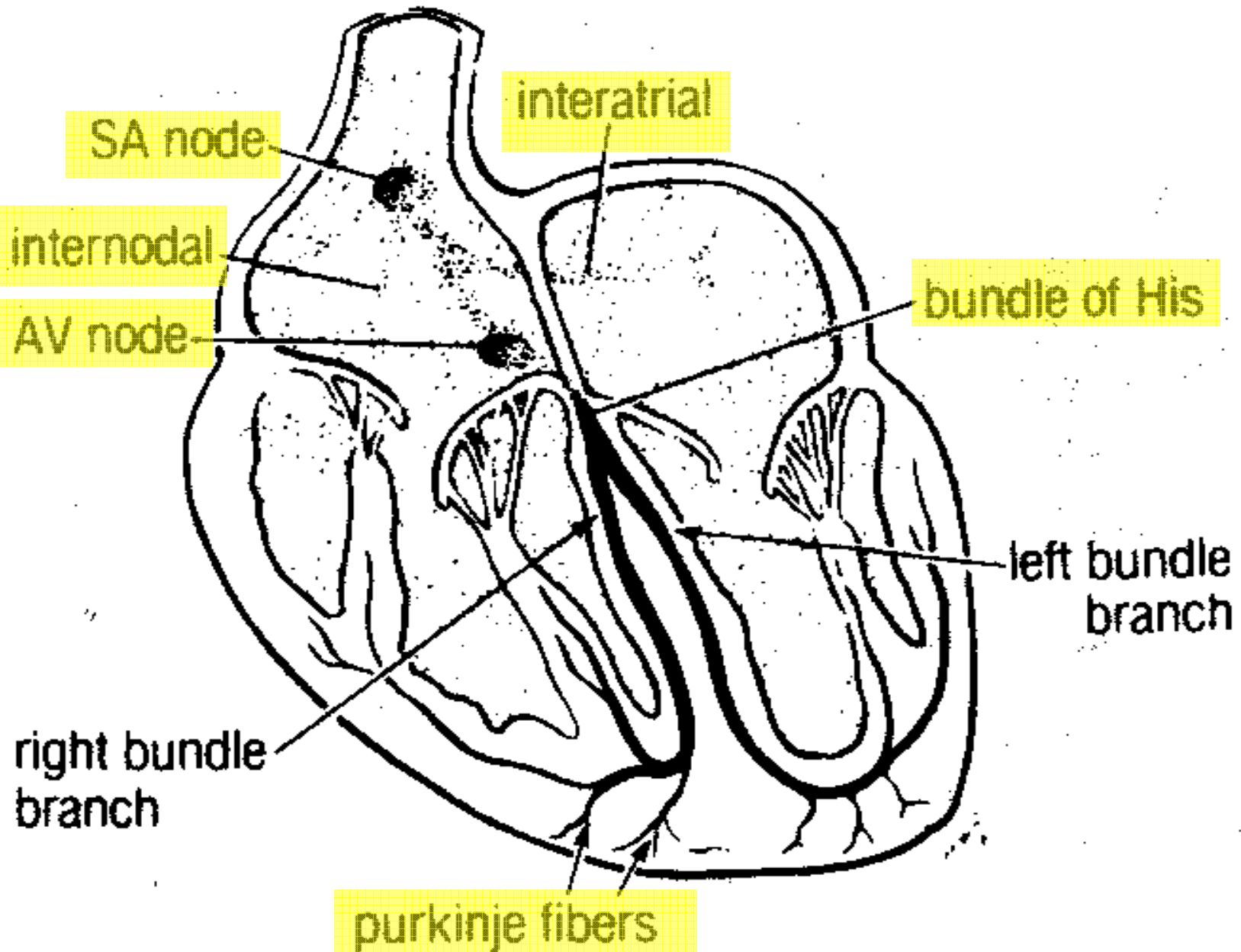
Neural Conduction of the Heart

- 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

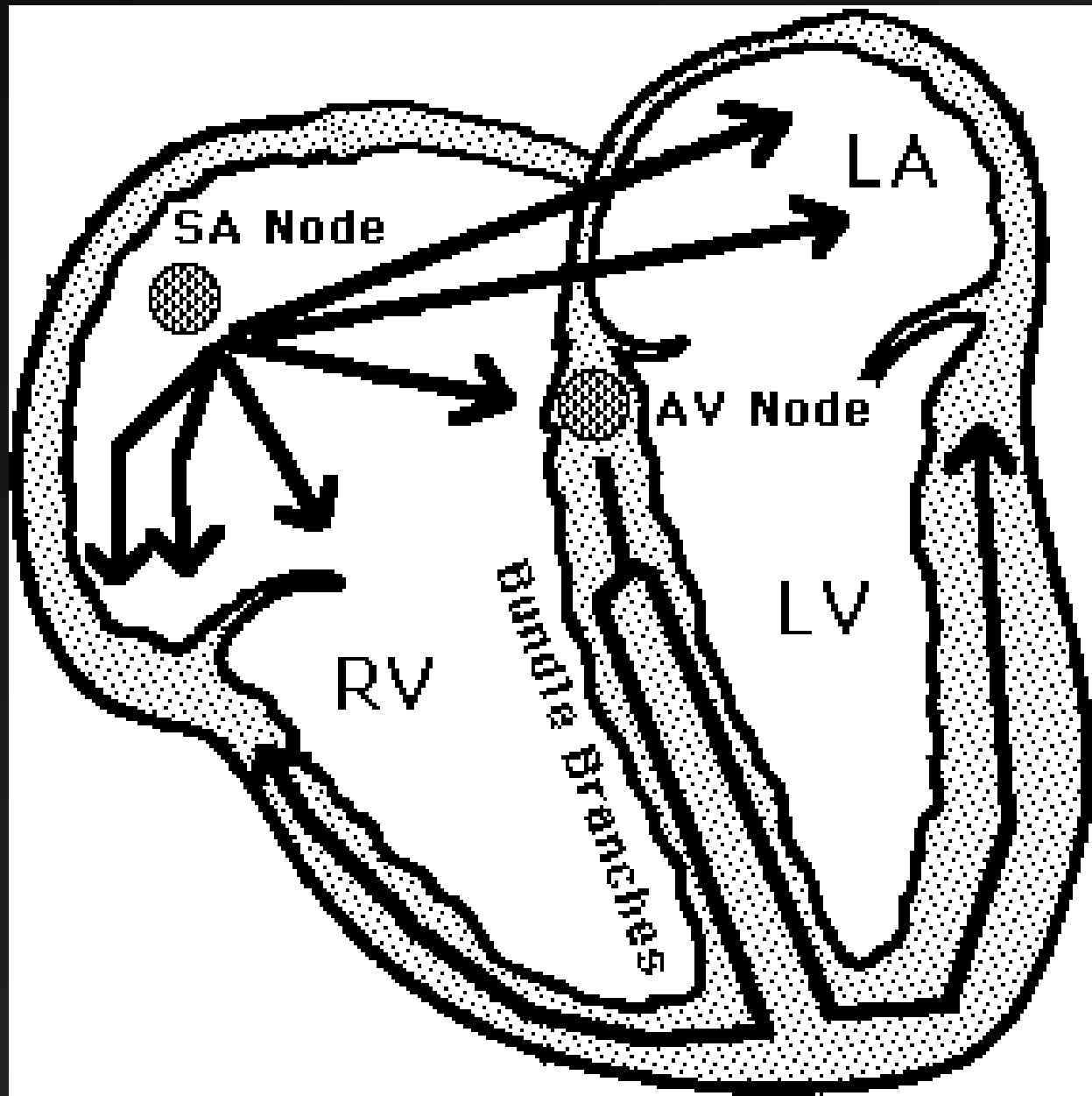
Neural Conduction of the Heart

- 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

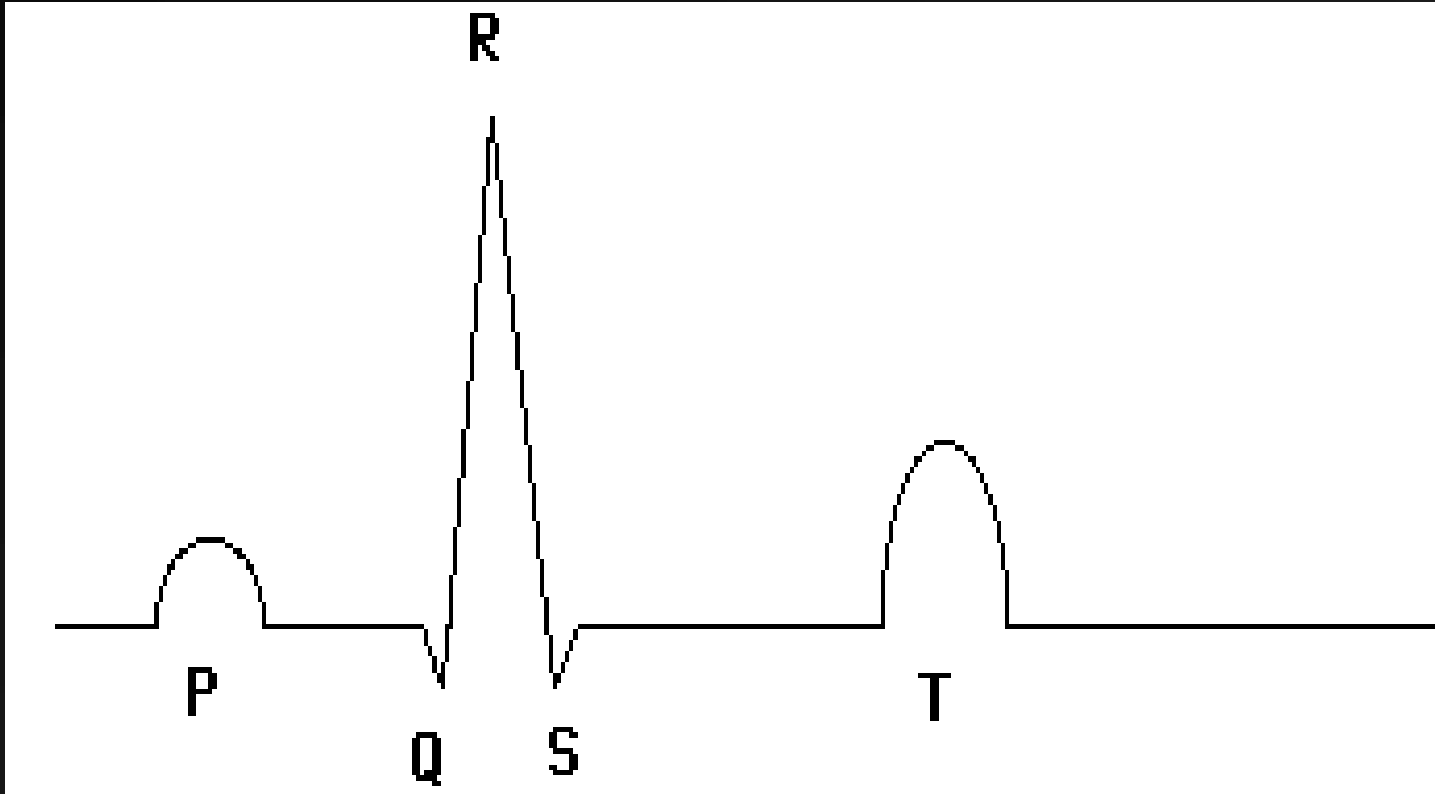
Nodes and Fibers



The SA and AV Nodes in Action



The Schematized EKG waveform



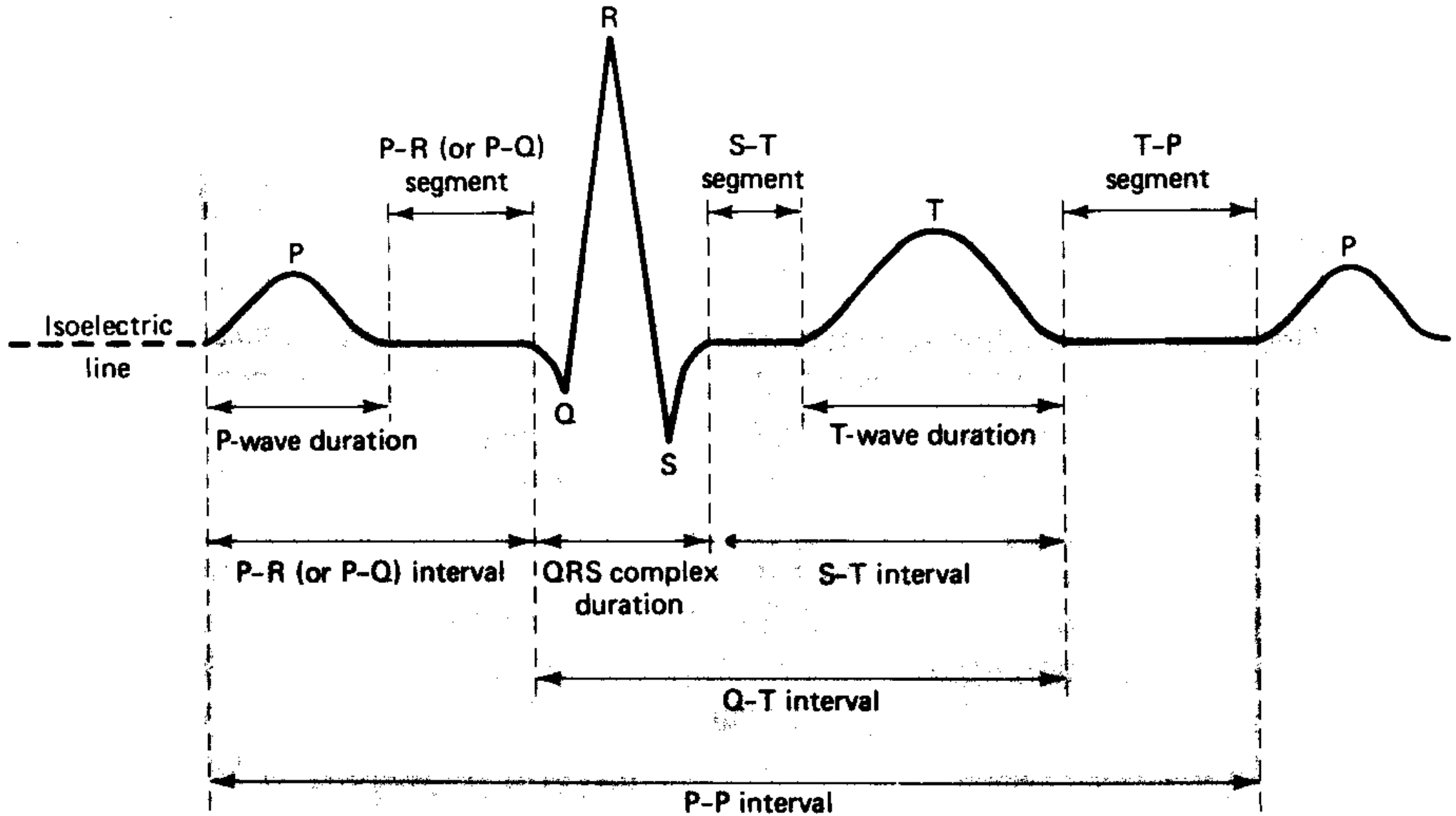
P = Atrial depolarization

QRS = Ventricular depolarization

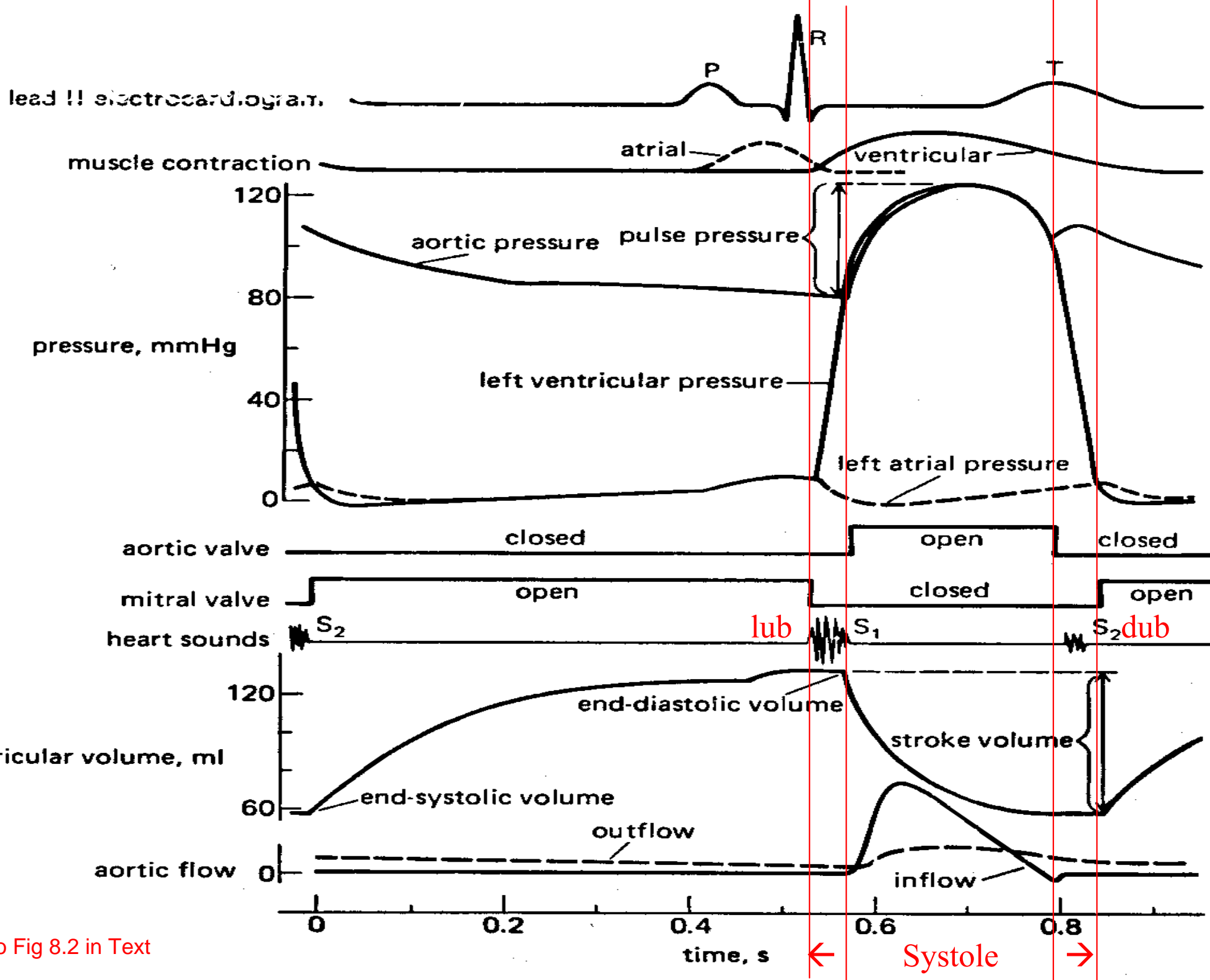
T = Ventricular repolarization

Note that Atrial repolarization is not visible

The EKG waveform



<http://mcdb.colorado.edu/courses/2115/units/Other/heartbeat%20animation.html>



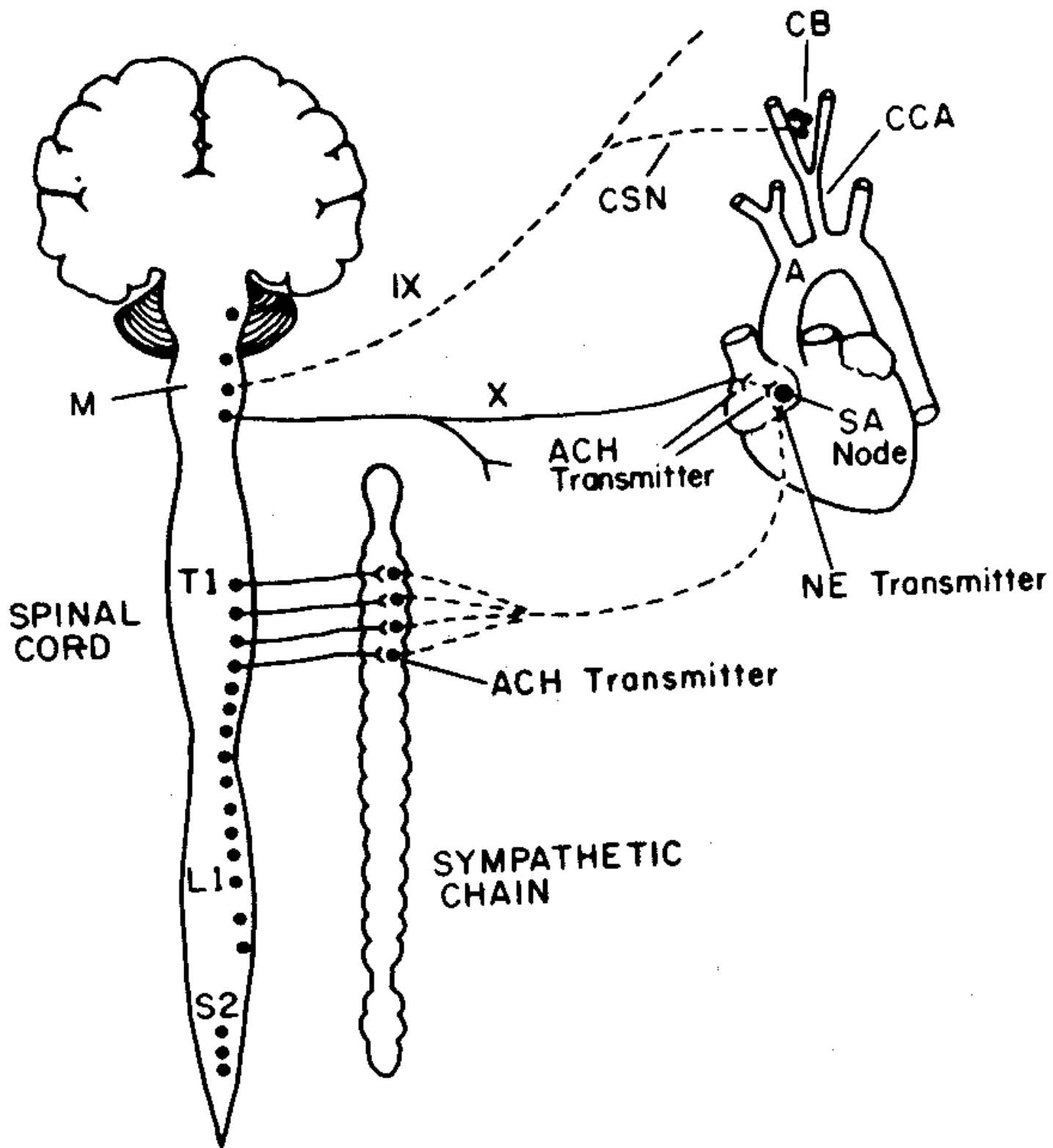
See also Fig 8.2 in Text

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



Autonomic Innervation

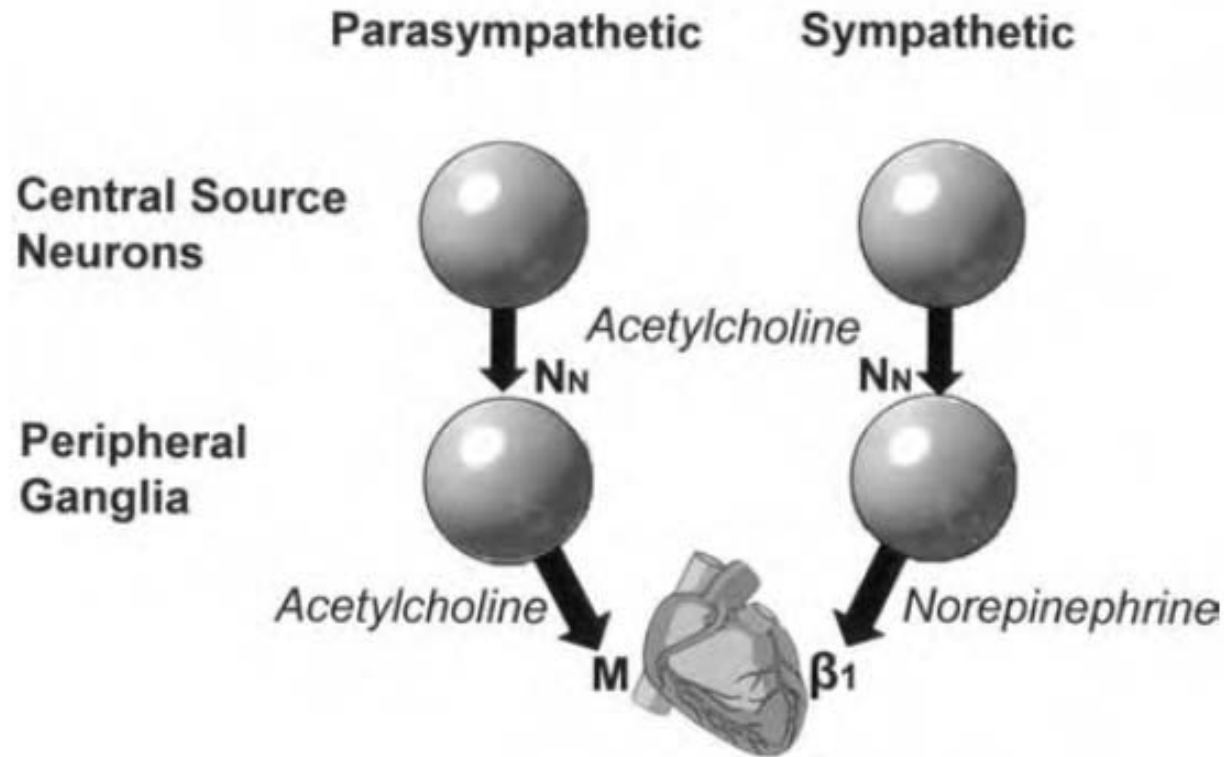
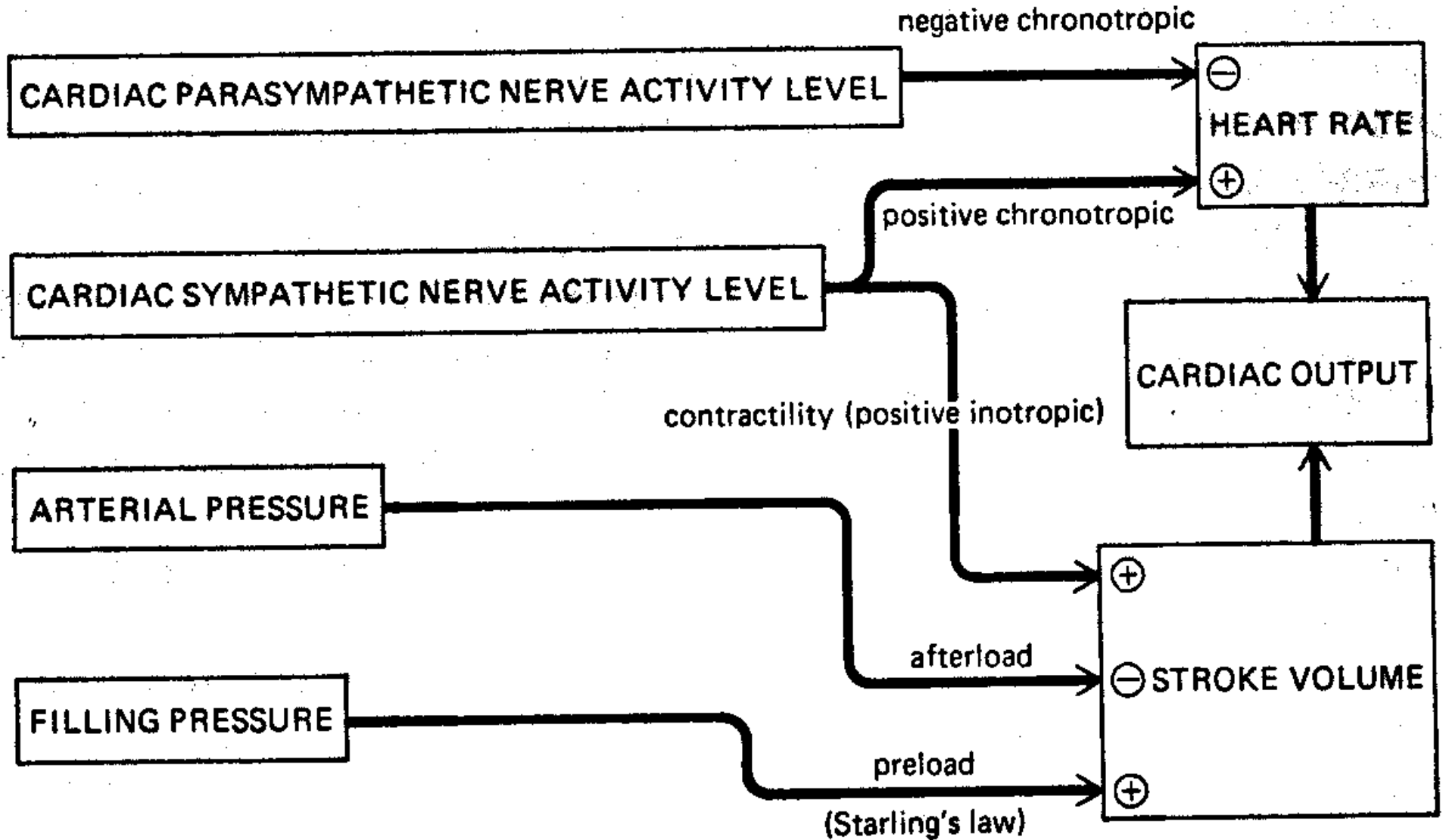
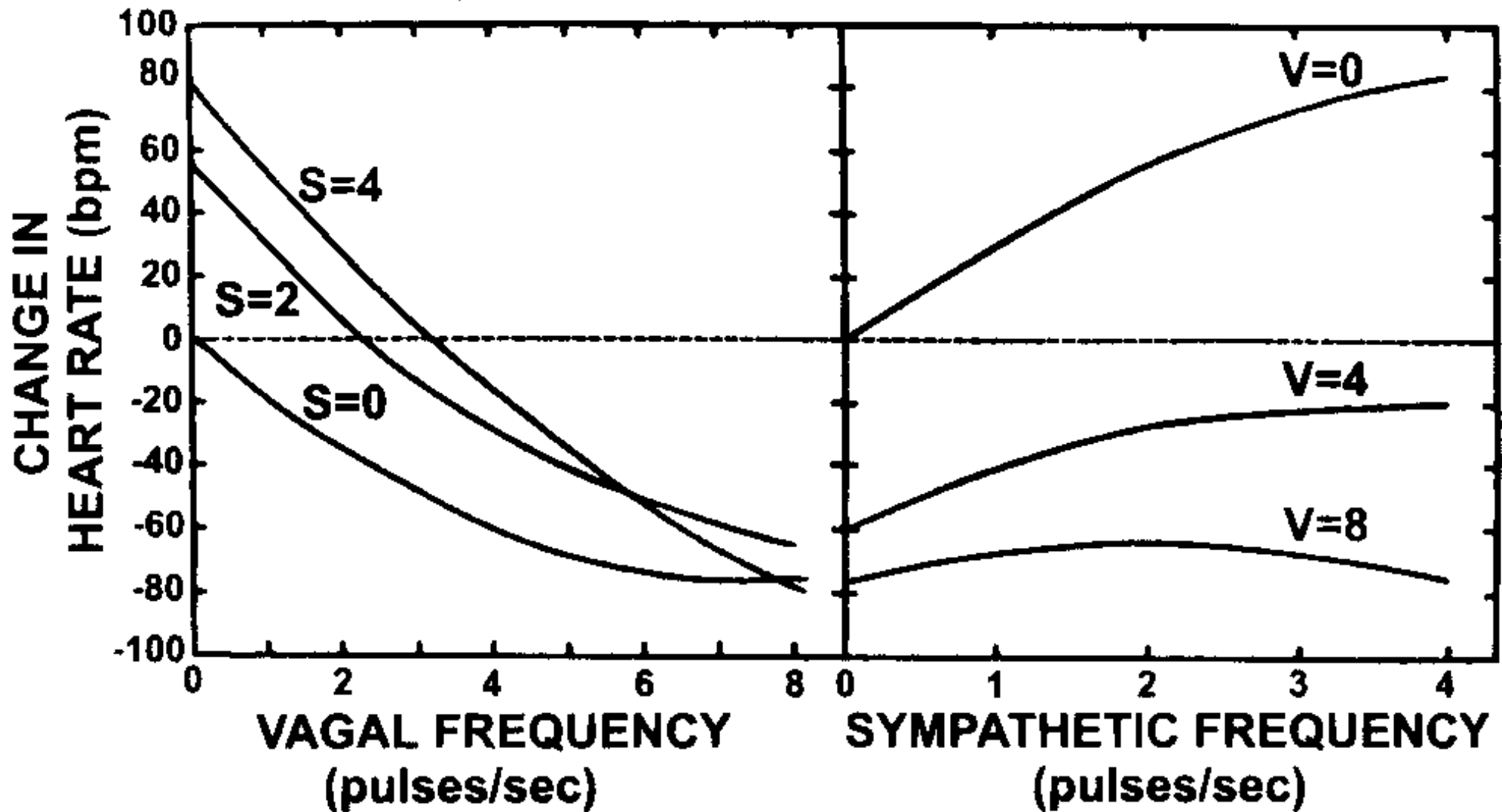


Figure 8.6. General pattern of pharmacology of the autonomic innervations. Abbreviations refer to the relevant postsynaptic receptor populations: N_N – nicotinic cholinergic; M – muscarinic cholinergic; β_1 – beta1 adrenergic.

SNS and PNS influences



HR change to simultaneous vagal and sympathetic stimulation



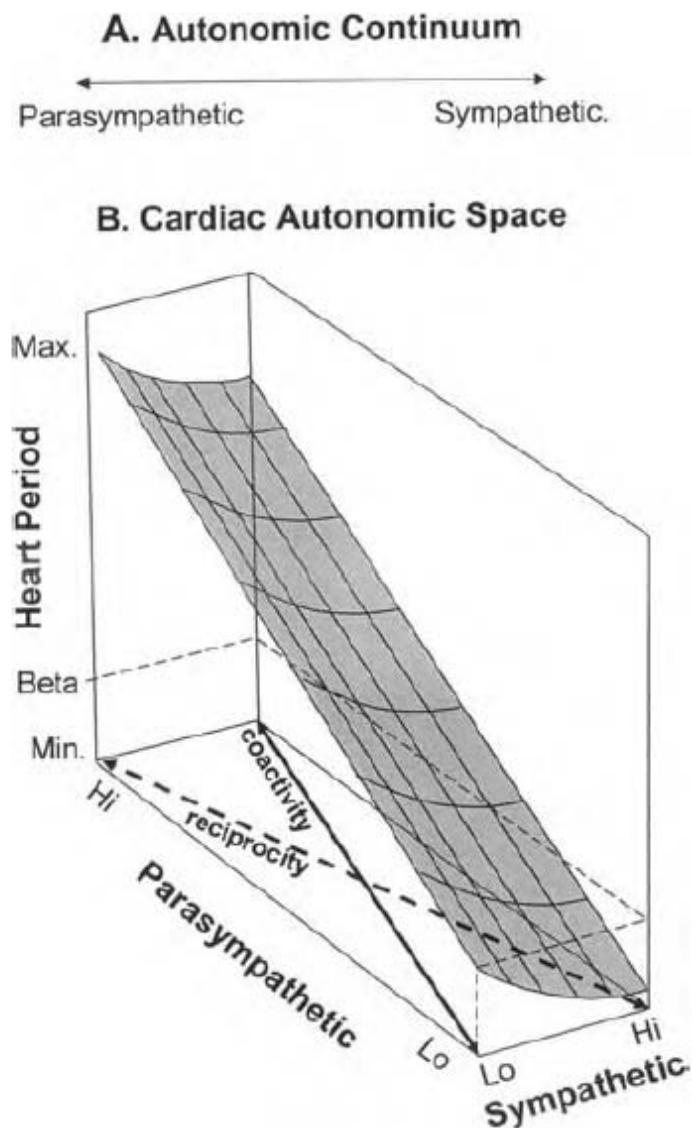


Figure 8.9. Autonomic space. (A) Continuum model of autonomic control, wherein the status of the system can be depicted along a single continuum extending from parasympathetic dominance to sympathetic dominance. (B) A more comprehensive model of autonomic control, characterized by an autonomic plane (representing the fact that parasympathetic and sympathetic systems can change reciprocally, coactively, 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

Baroreceptor Reflex

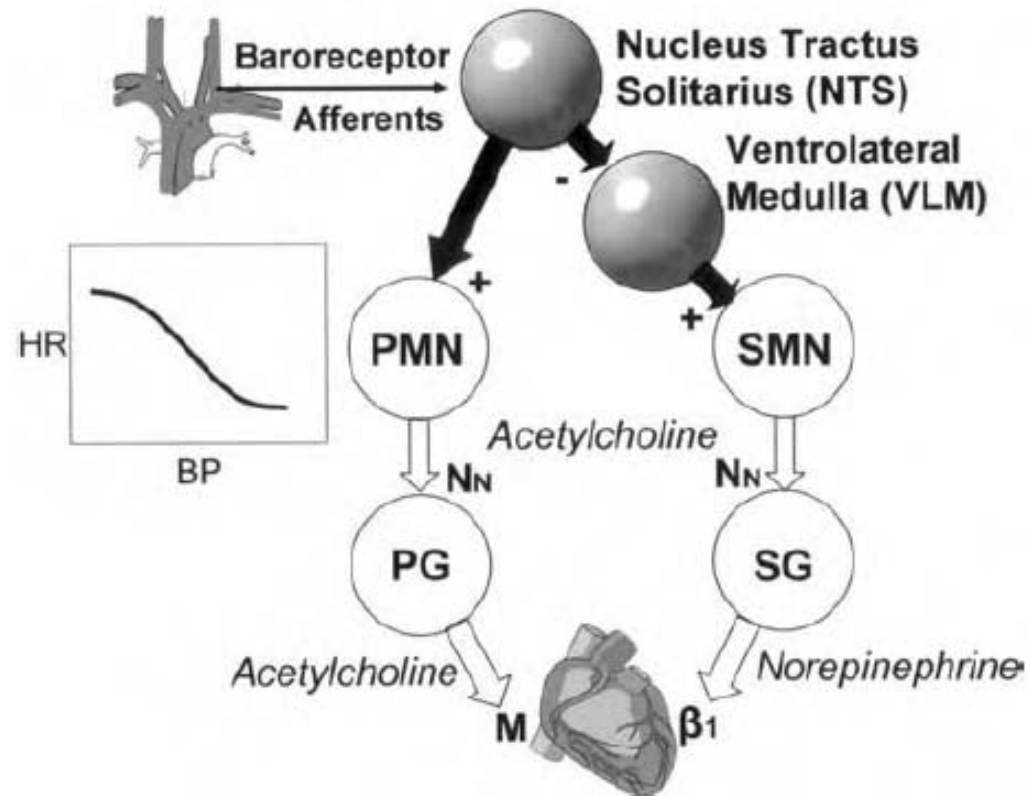
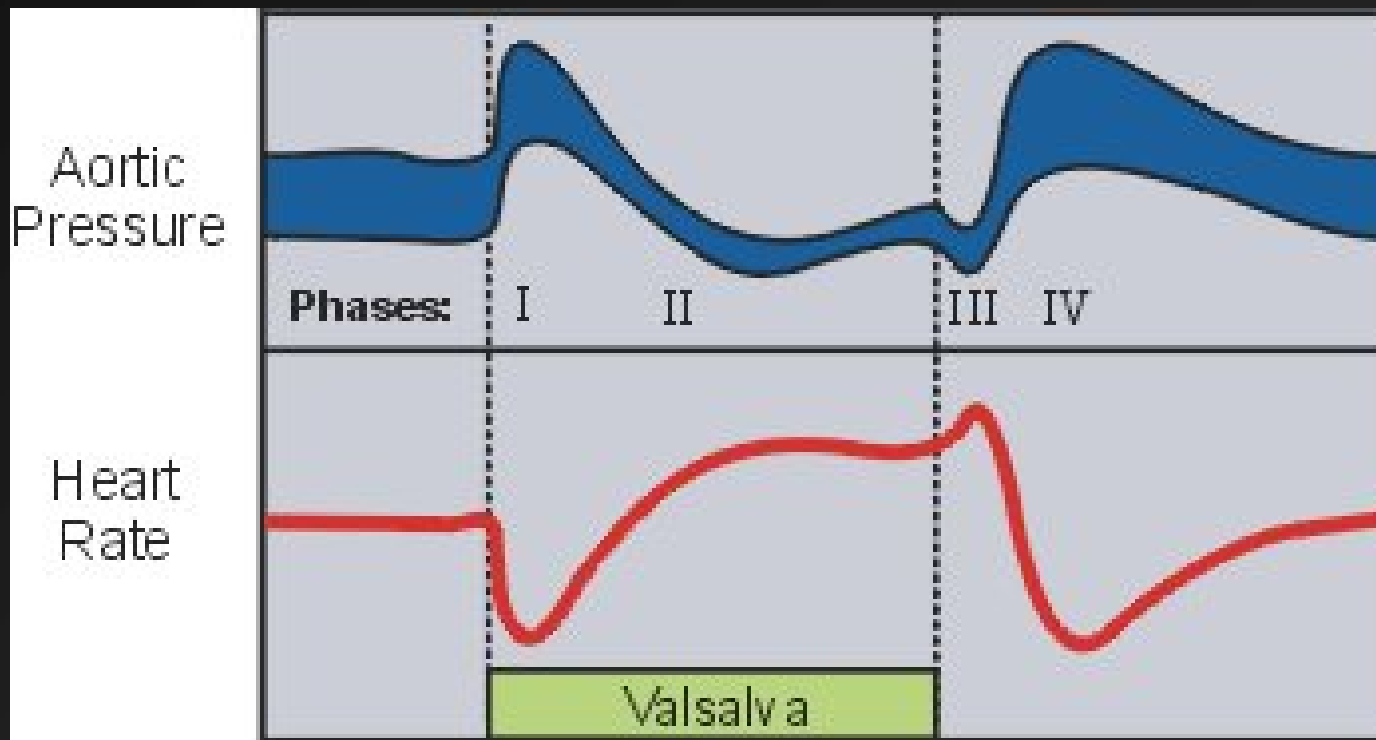


Figure 8.7. General organization of the baroreceptor heart rate reflex. Reflex originates in mechanoreceptors in the heart and the carotid and other great arteries. The NTS excites (+ symbol) the parasympathetic motor neurons (PMN) and inhibits (- symbol) relay neurons to the sympathetic motor neuron 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

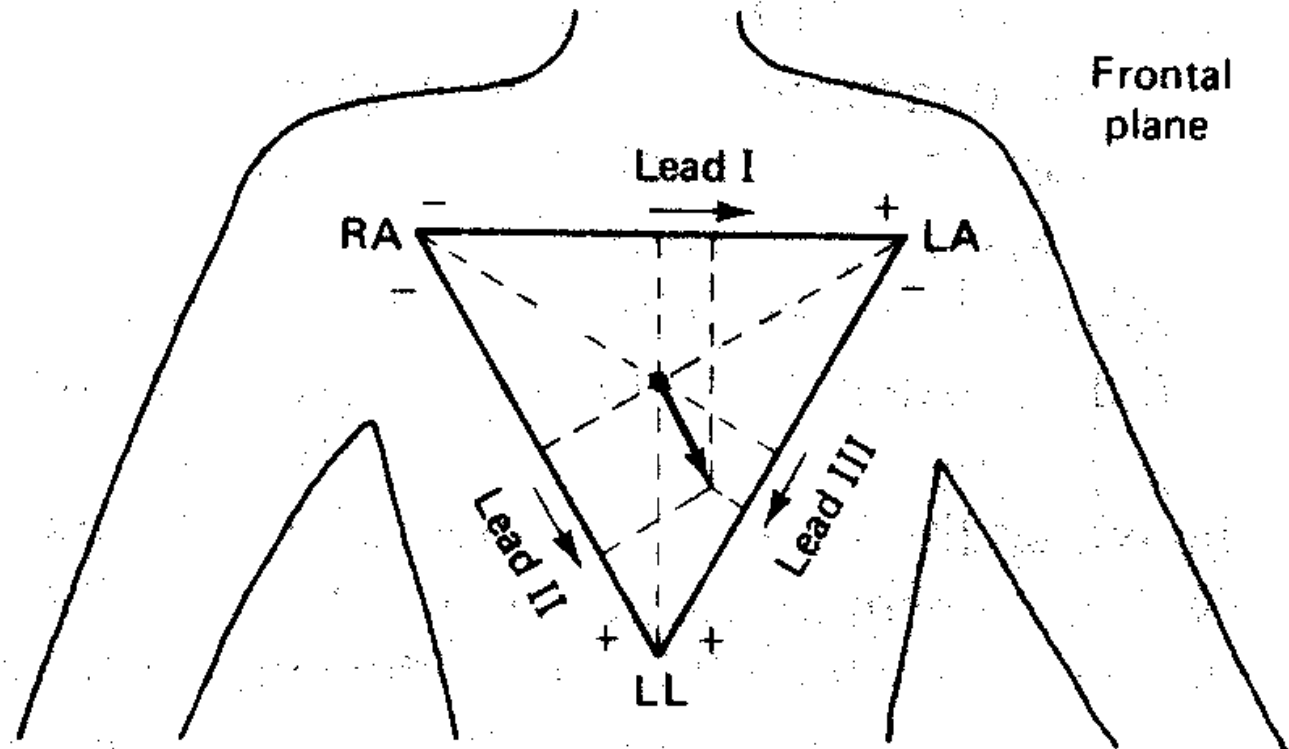
- Relatively easy to measure PNS: RSA or other metrics of HRV
- Relatively easy to measure SNS: Contractility via PEP
- BUT... one is measure of chronotropy, other is measure of inotropy
 - Changes in contractility can occur independently of changes in rate
 - SNS inputs for inotropy primarily controlled 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



Bipolar

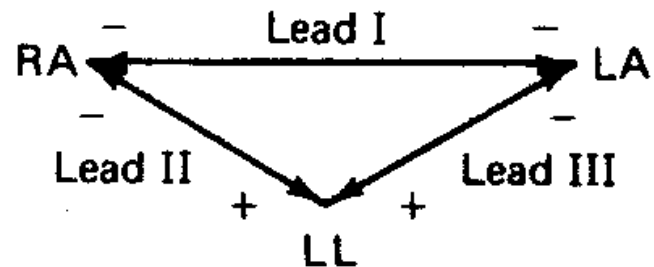
Bipolar limb leads: ECG voltage measurements between pairs of limbs:

Lead I: between RA and LA

Lead II: between RA and LL

Lead III: between LA and LL

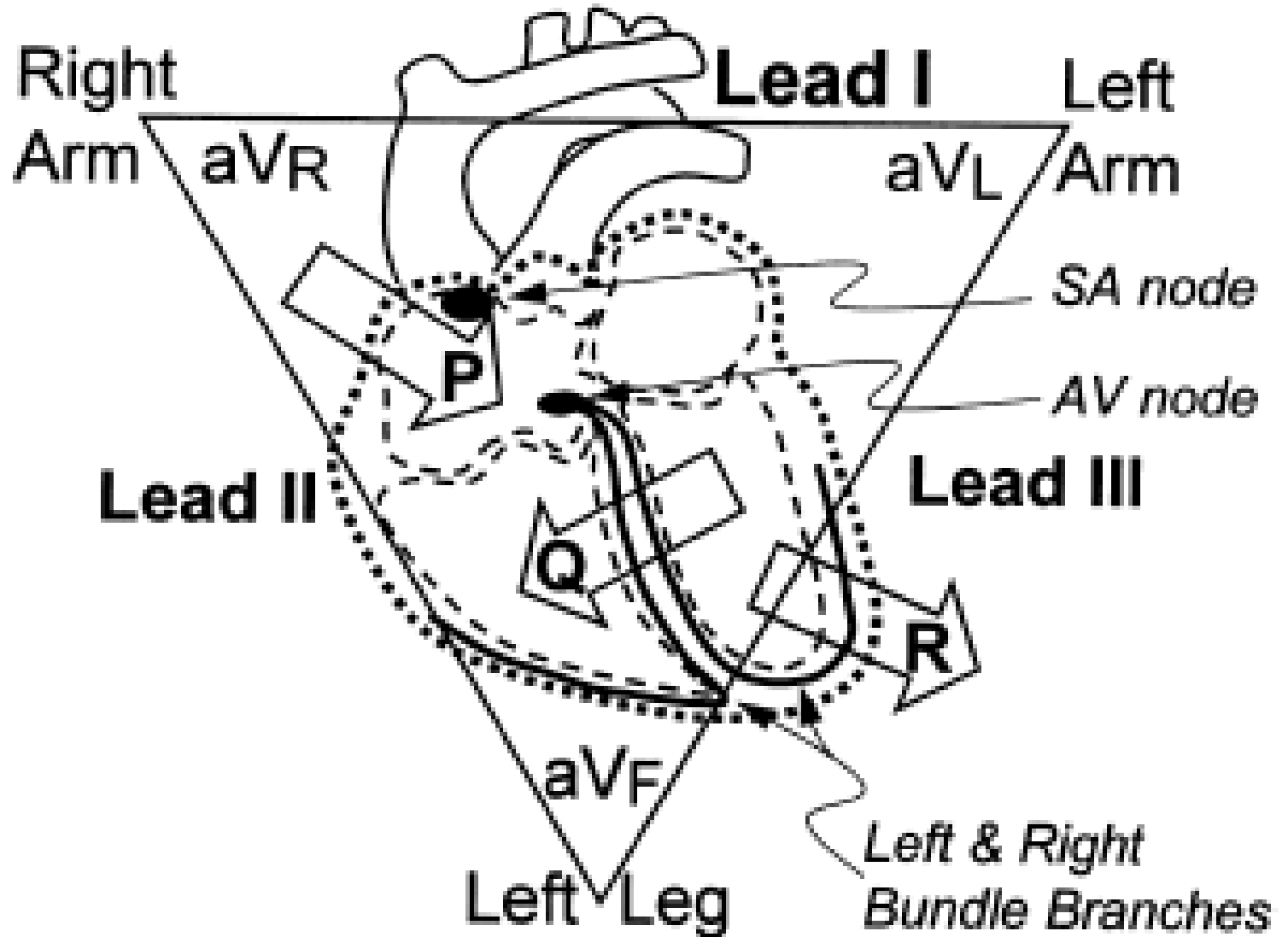
Einthoven triangle, showing relation of the bipolar limb leads



EKG

AC signal
Sample 200-500 Hz

B. Einthoven's triangle



Which Time?

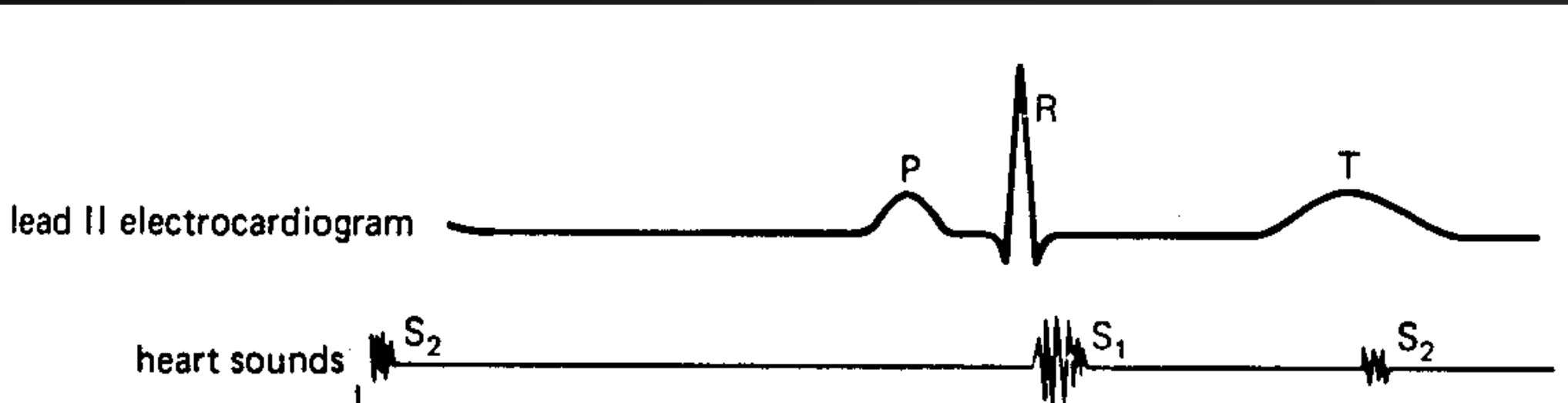
- Real time
 - Heart Rate
 - Expressed as beats per time (usually bpm)
- Cardiac time
 - Heart Period; interbeat interval (IBI)
 - Expressed in msec
- Converting

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

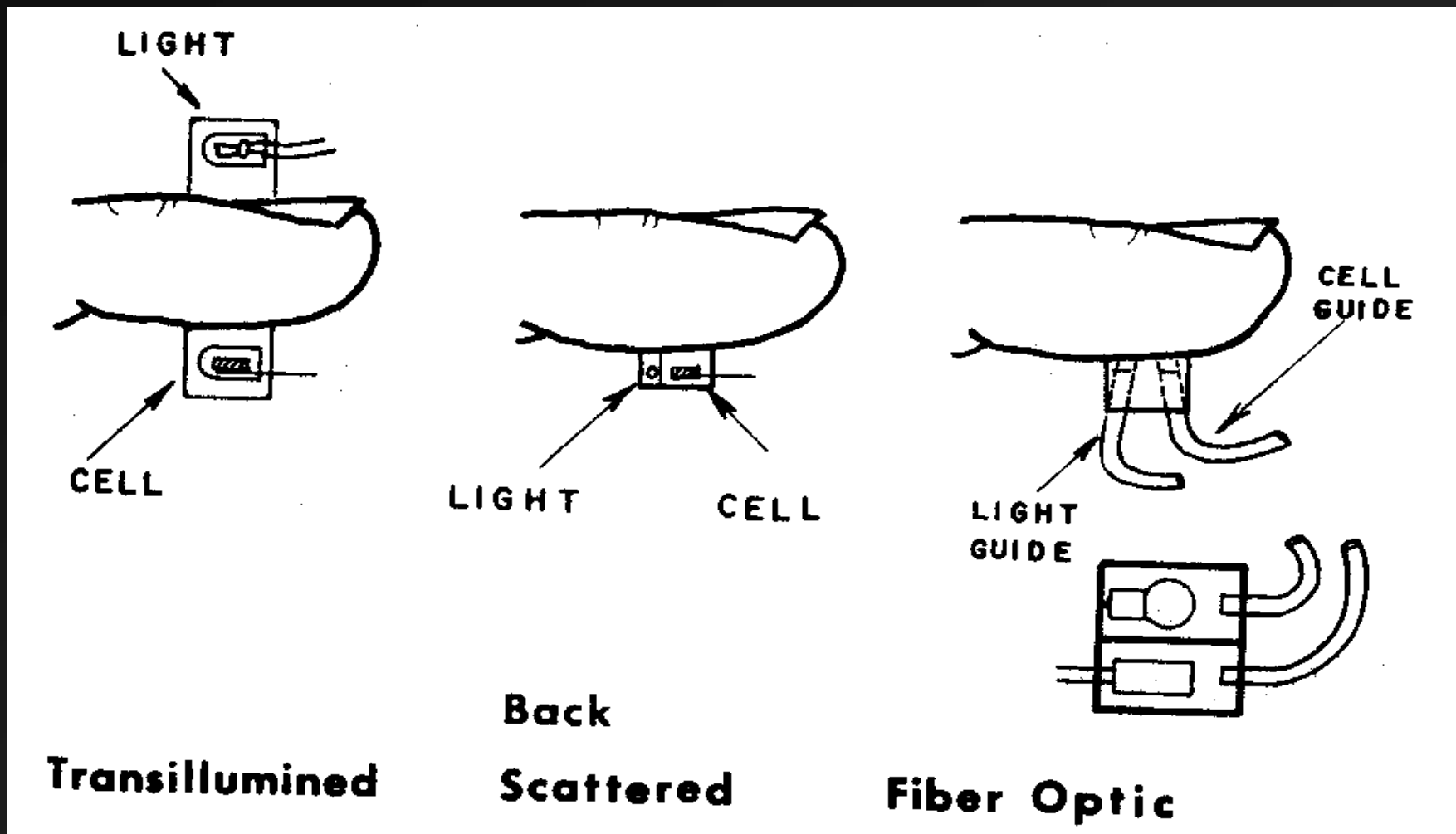
$$HR = \frac{1}{1000} \times 60,000 = 60bpm$$

Phonocardiography

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

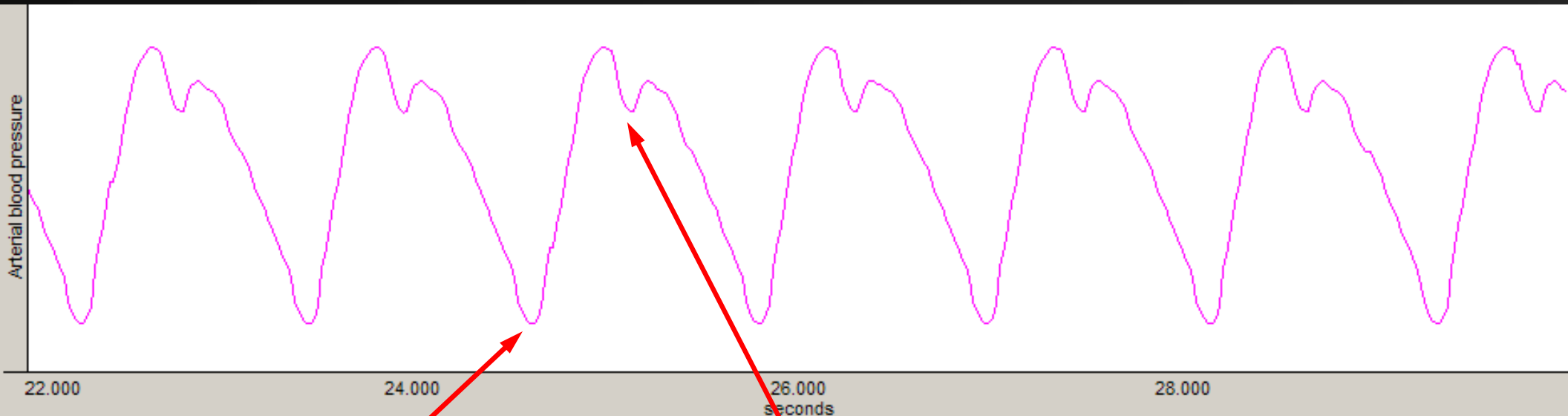


Photoplethysmography



Three methods, all involve measuring light absorbed by peripheral vasulature

The Photoplethysmographic Output

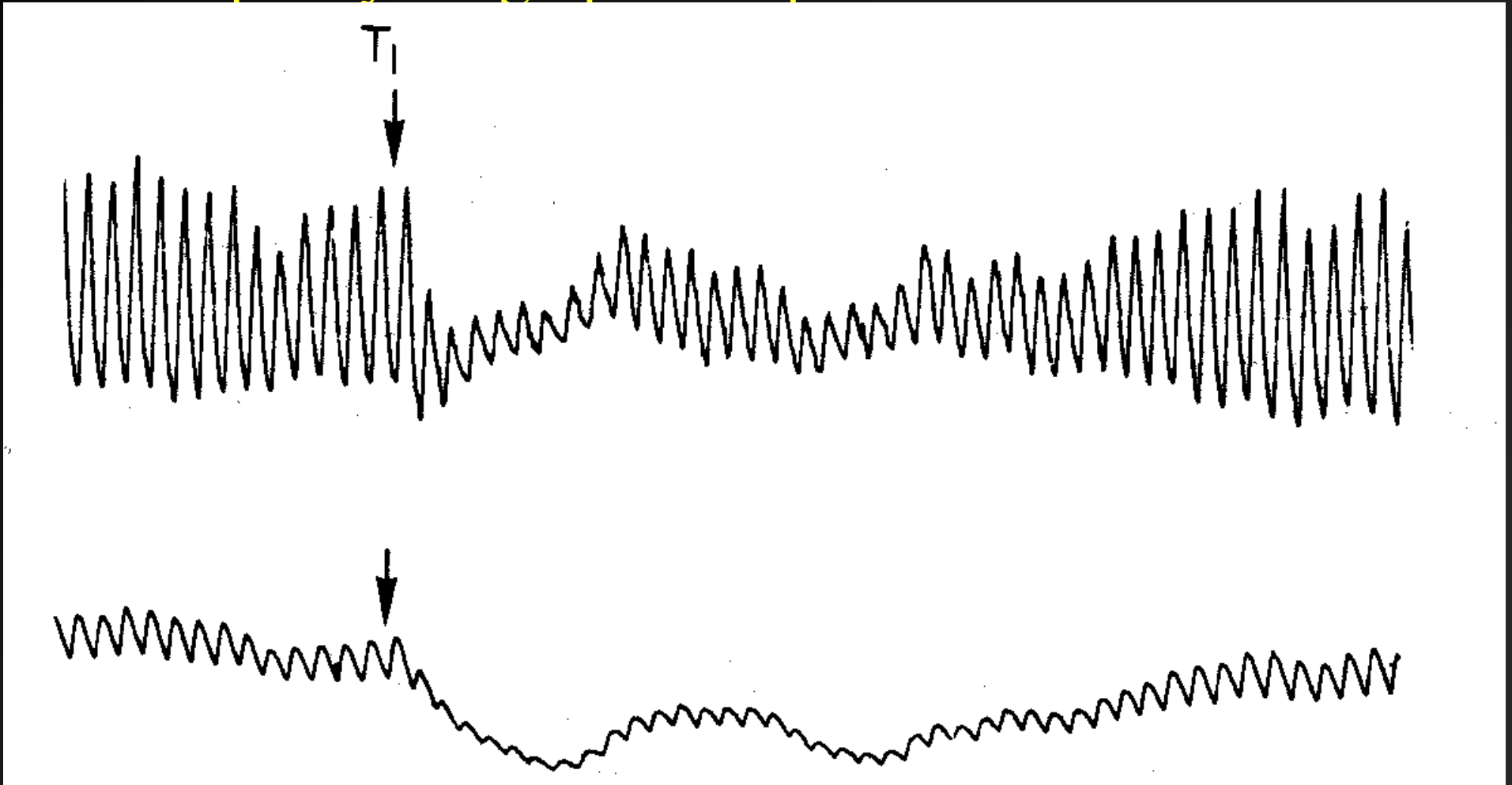


Increase in Pressure due to opening of Aortic Valve

~LVET

Dichrotic Notch; closing of valve, end of ejection

Photoplethysmograph: Peripheral Vasoconstriction



T_1 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

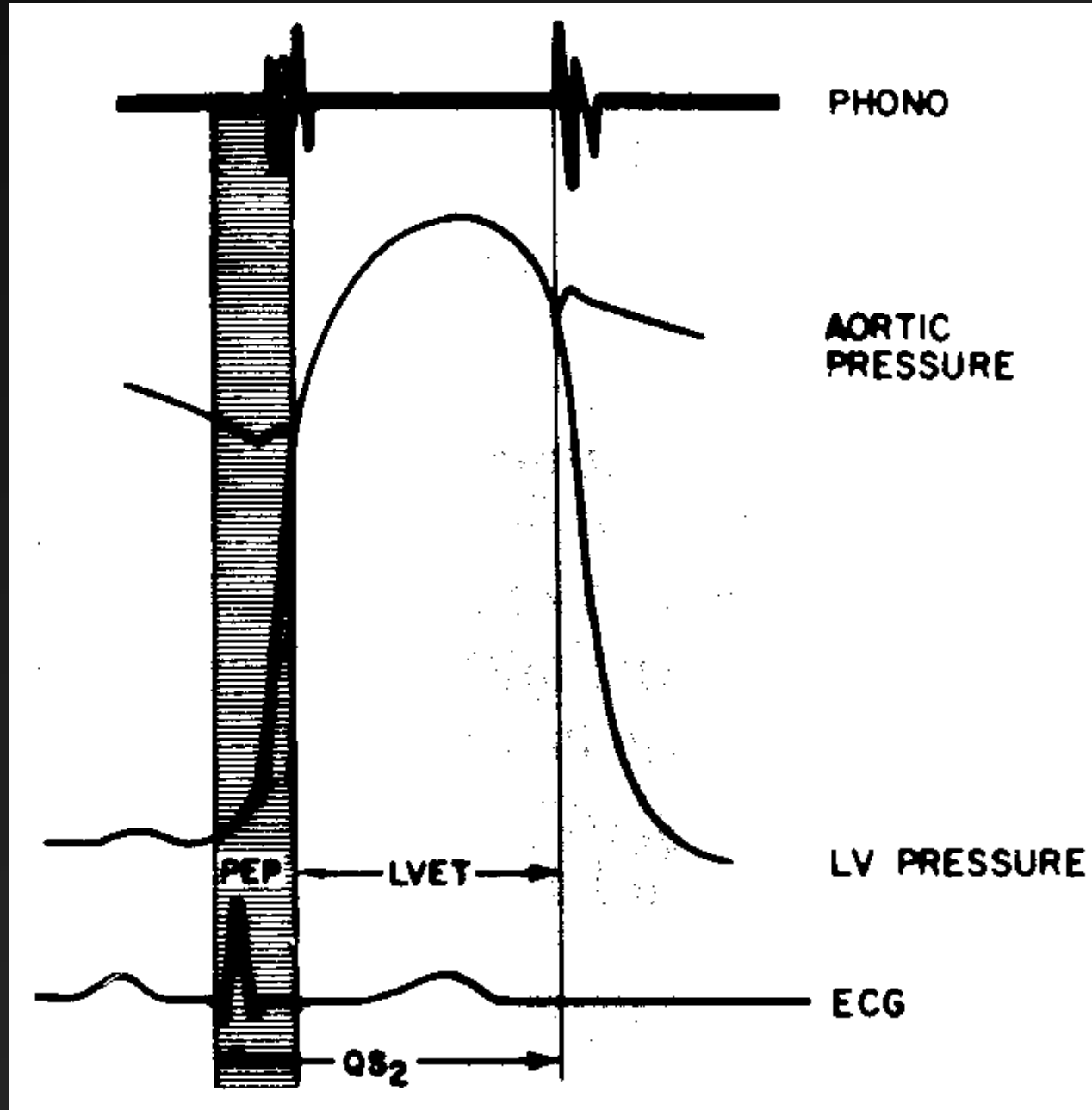
LVET = Left Ventricular Ejection Time

= Upswing of pressure wave to S2

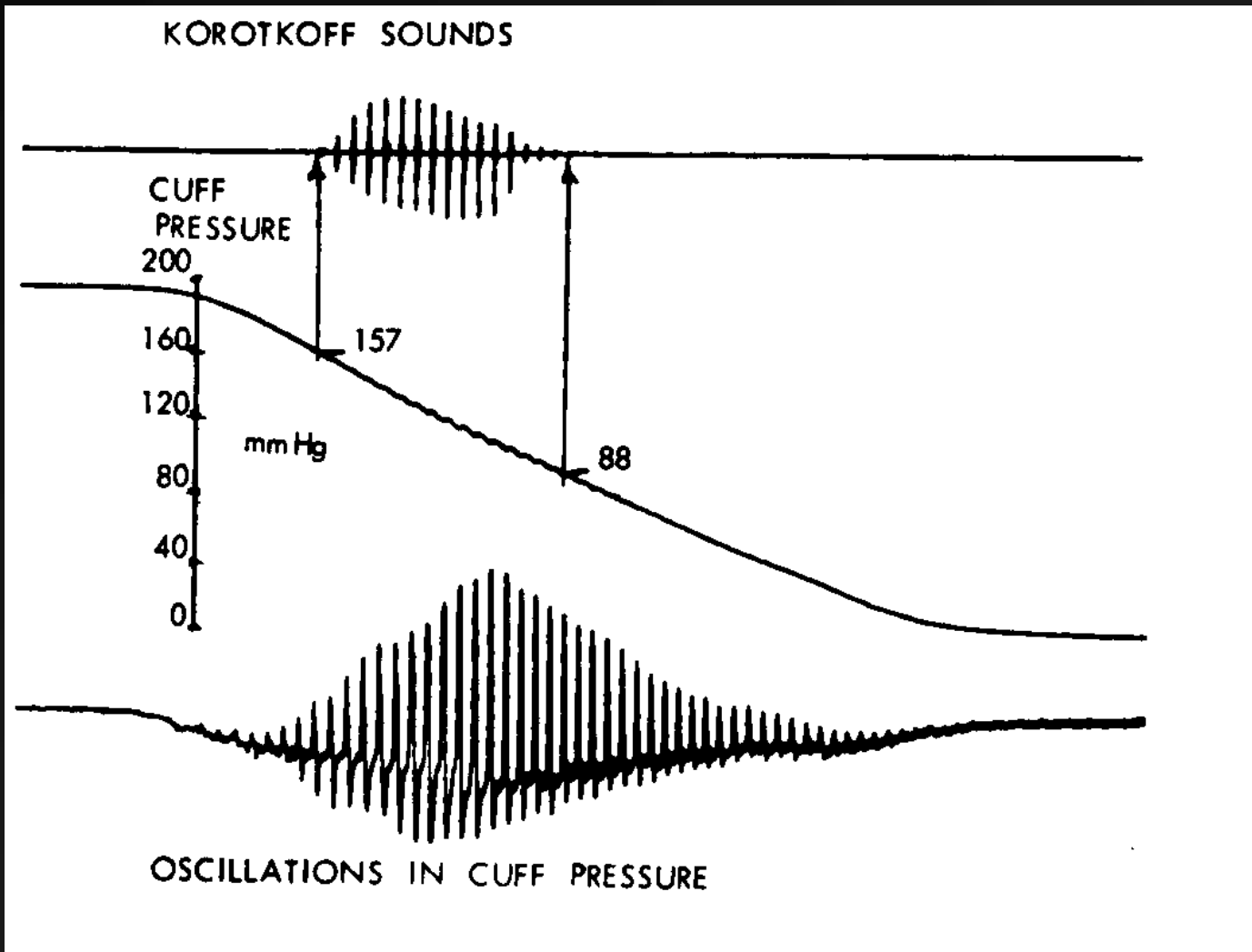
Electromechanical Systole = Q to S2

$PEP = EMS - LVET$

PEP reflects sympathetic influence on cardiac contractility



Measuring Blood Pressure



Auscultatory Technique

- Not good for instantaneous readings
- Not good for repeated readings

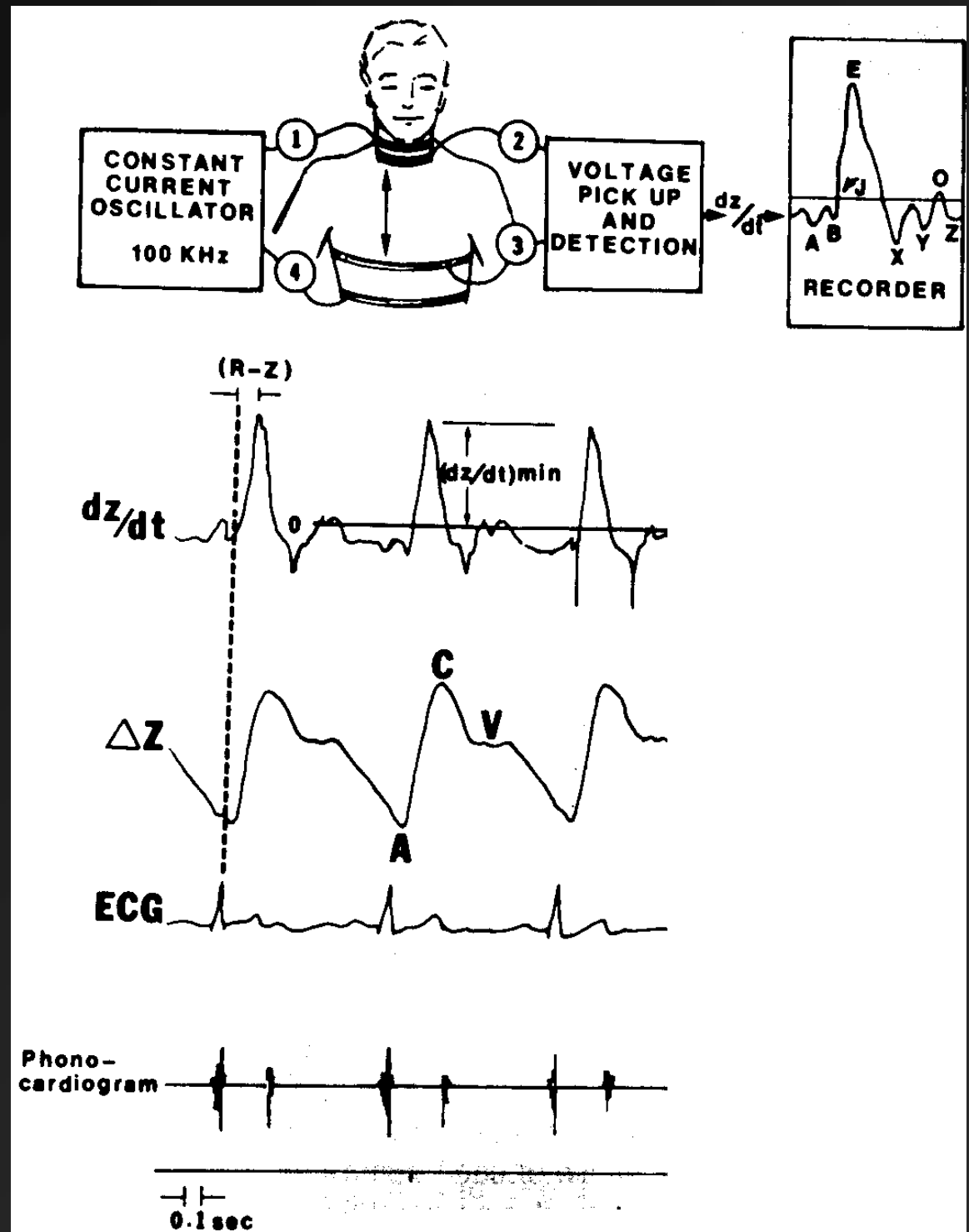
Ballistocardiography

- 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
- ΔZ 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



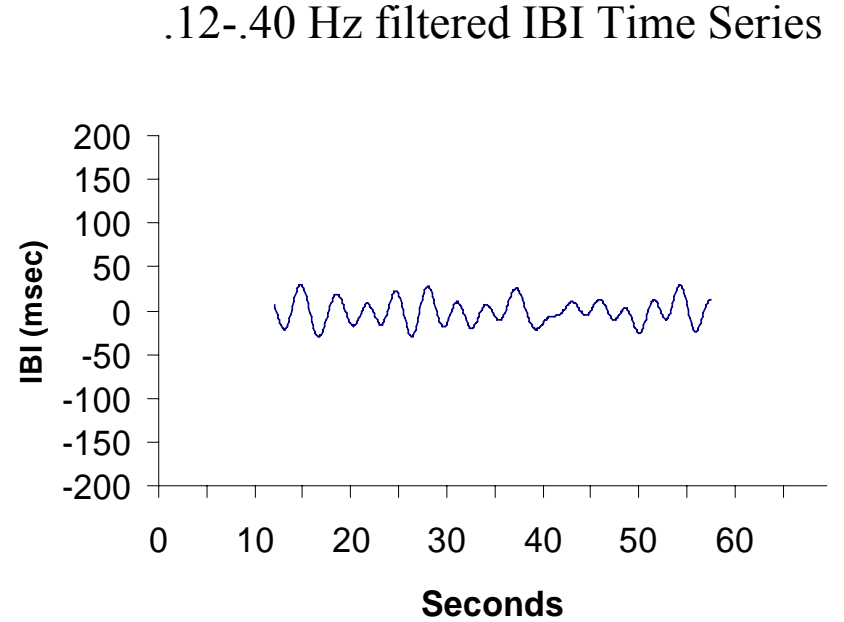
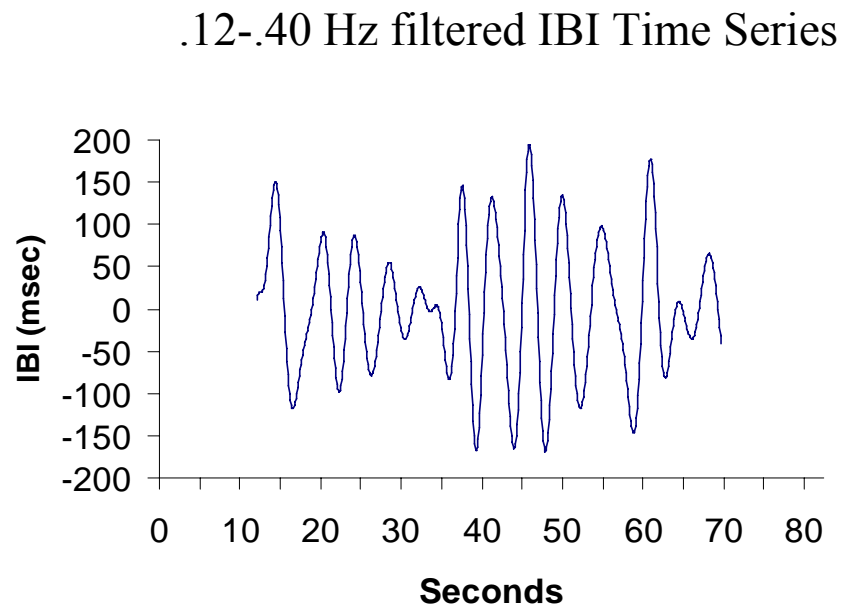
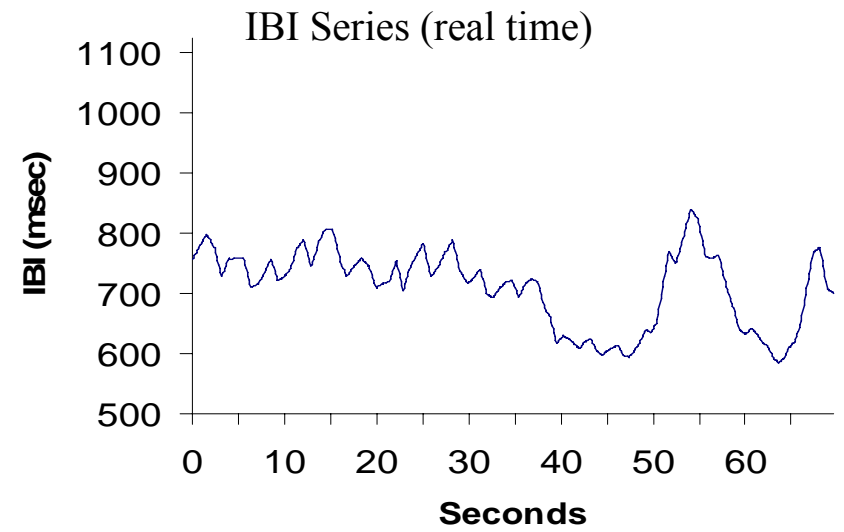
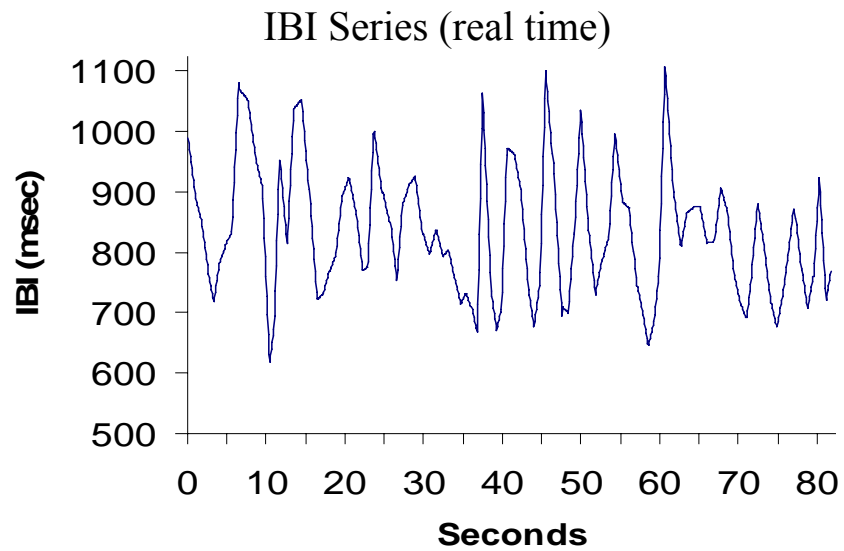
Measuring Vagal Influence

- 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

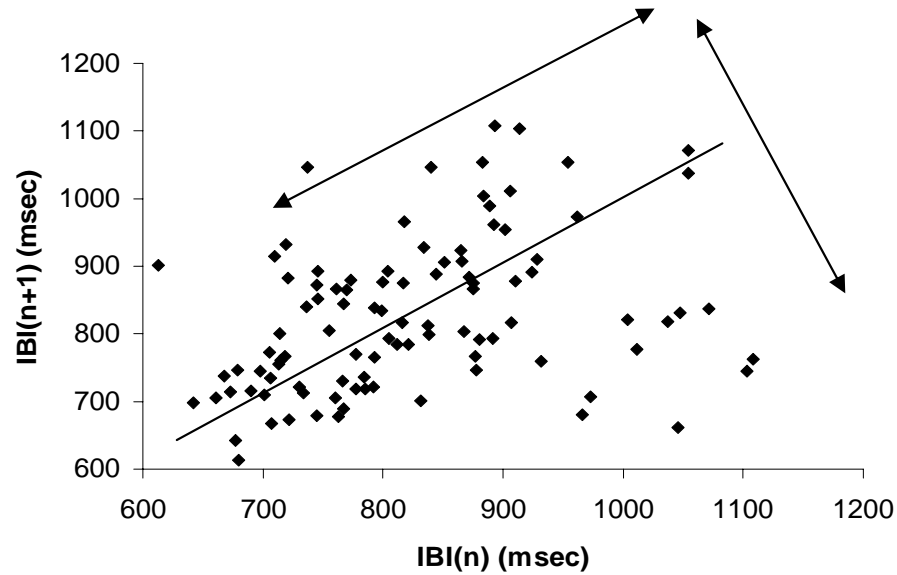
Abbreviated History of HR Variability

(with thanks to Porges, 2007)

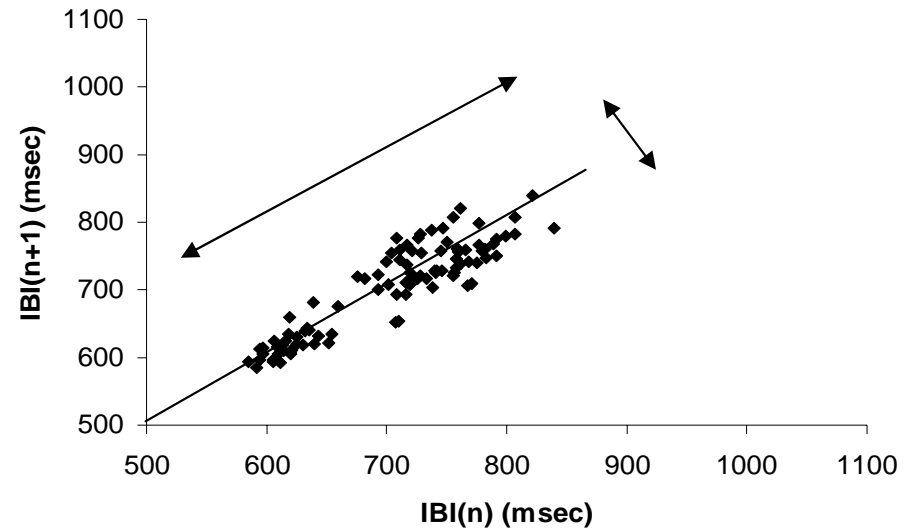
- Physiology treated HR as DV, similar to behavior
- Dominance of behaviorism emphasized control over the DV (behavior)
- Changes in HR unrelated to the manipulation considered noise
 - Lacey (1967) and Obrist (1981) had models related to attention, and metabolic demand, but HR *variability* did not 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



High Variability Subject



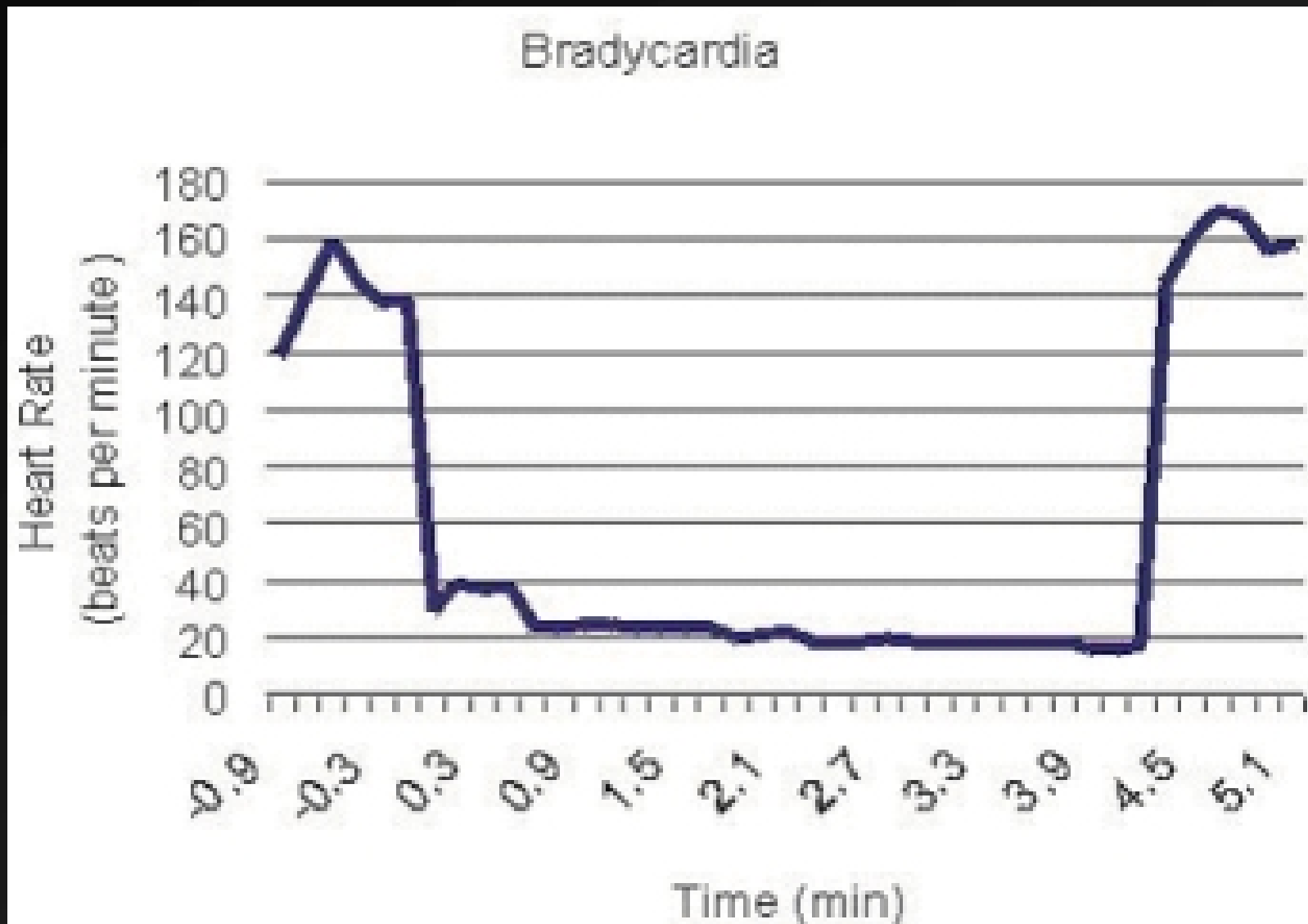
Low Variability Subject



Rate		
73.3	HR	85.7
832.3	IBI	707.7
Total Variability		
9.2	HRV	8.3
112.4	SDNN	66.3
132.8	RMSSD	27.7
"Sympathetic"		
1.4	CSI	4.7
"Parasympathetic"		
57.1	PNN50	10.8
97.6	MCD	22.0
5.3	CVI	4.5
8.8	RSA	5.3

Cardiac Vagal Control and Modulation

- Two Vagal Efferent Branches which terminate on SA Node (Porges 1995, 2003, 2007)
 - Reptilian “Dumb”: Dorsal Motor Nucleus
 - Massive reduction in HR & conservation of oxygen.
 - [Dive reflex](#) -- cold water on the face during breath hold
 - Phylogenetically newer “smart” Vagus
 - Originates from Nucleus Ambiguus
 - 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-soothe
- 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>

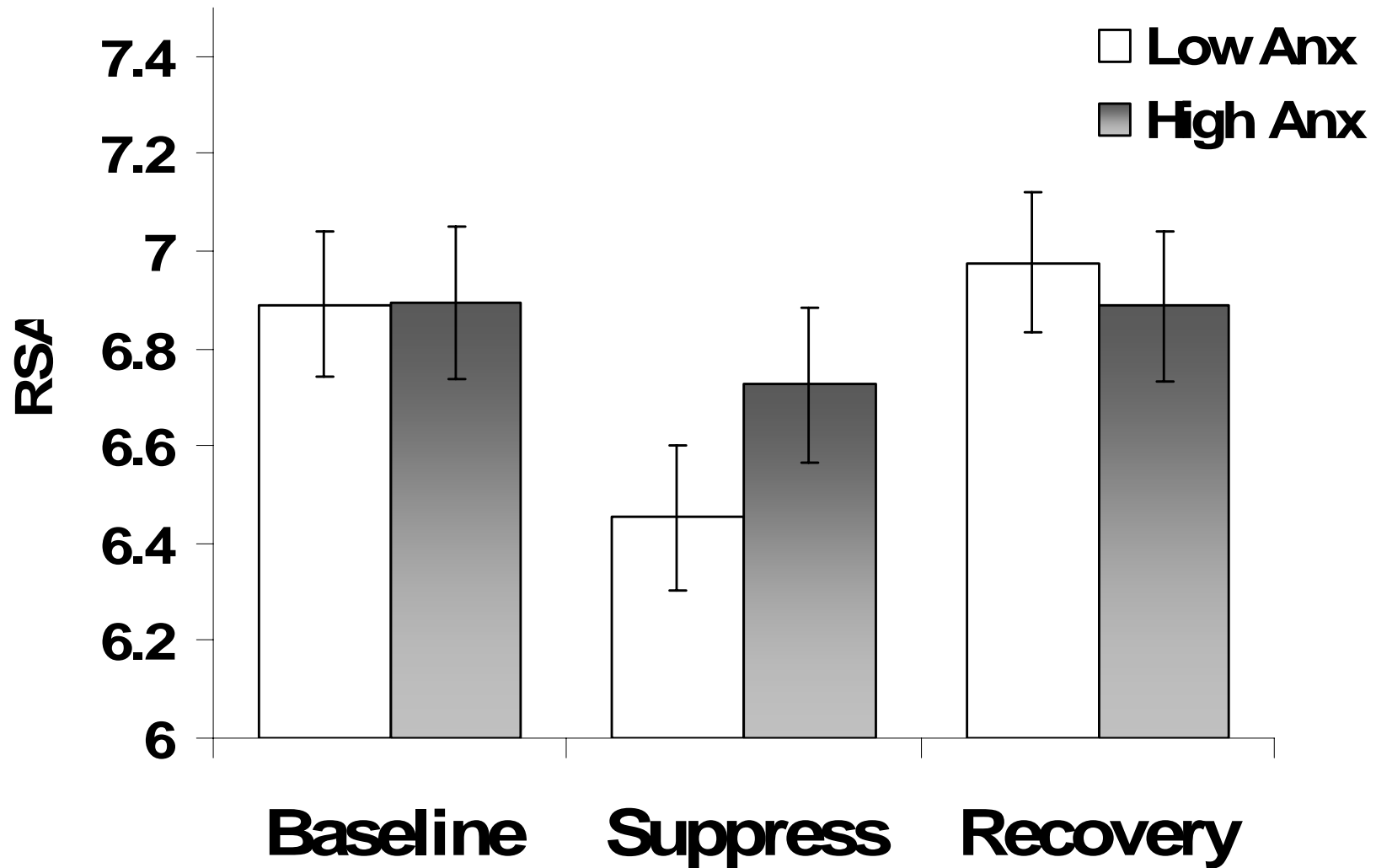
	ANS Component	Behavioral Function	Lower motor neurons
	Myelinated vagus <i>(ventral vagal complex)</i>	Social communication, self-soothing and calming, inhibit "arousal"	Nucleus ambiguus
	Sympathetic-adrenal system	Mobilization (active avoidance)	Spinal cord
	Unmyelinated vagus <i>(dorsal vagal complex)</i>	Immobilization (death feigning, passive avoidance)	Dorsal motor nucleus of the vagus

Fig. 1. Phylogenetic stages of the polyvagal theory.

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
 - Hyperactive kids treated with Ritalin (Porges, Walter, Korb, & Sprague, 1975).
 - 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



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](#))

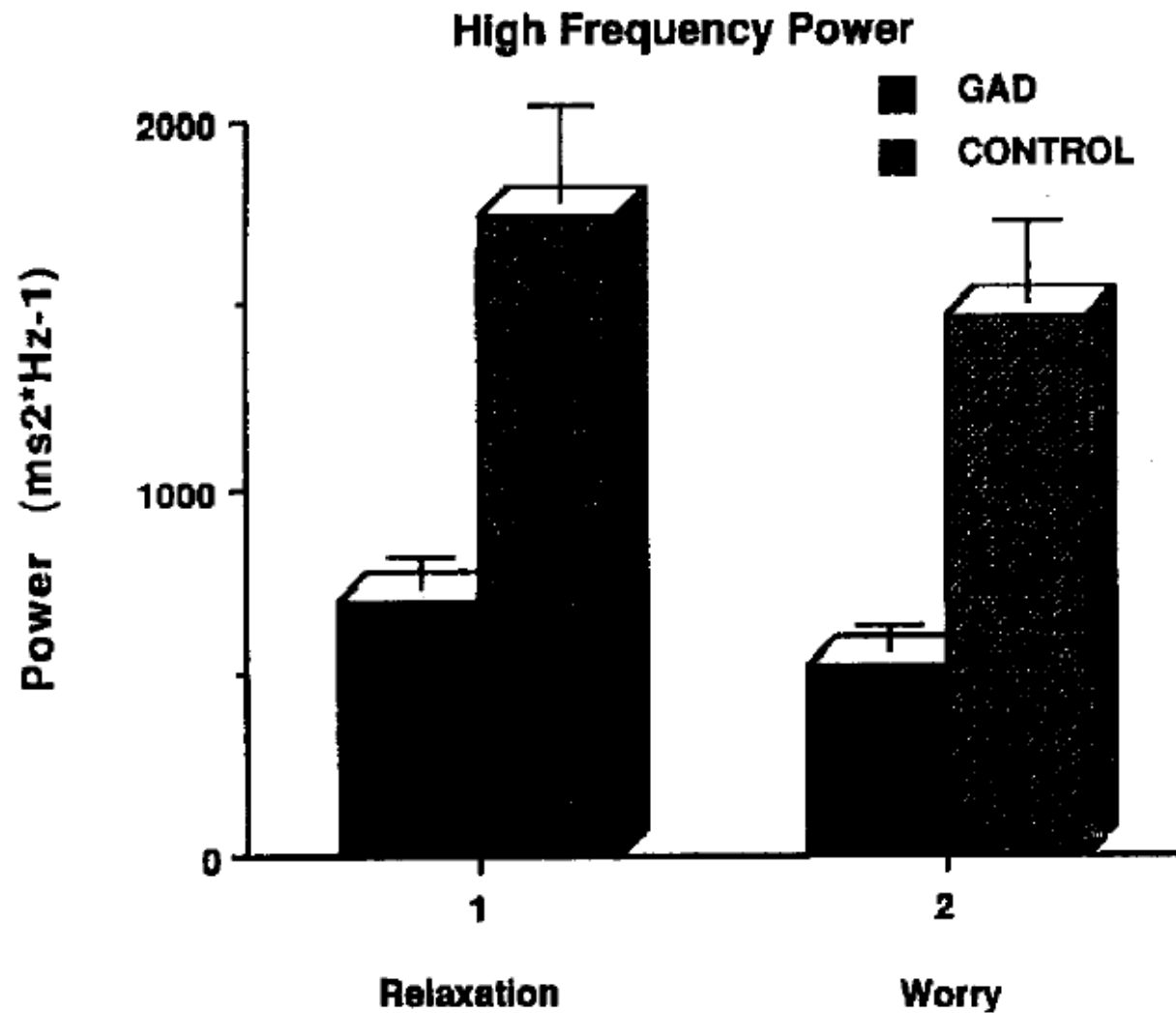
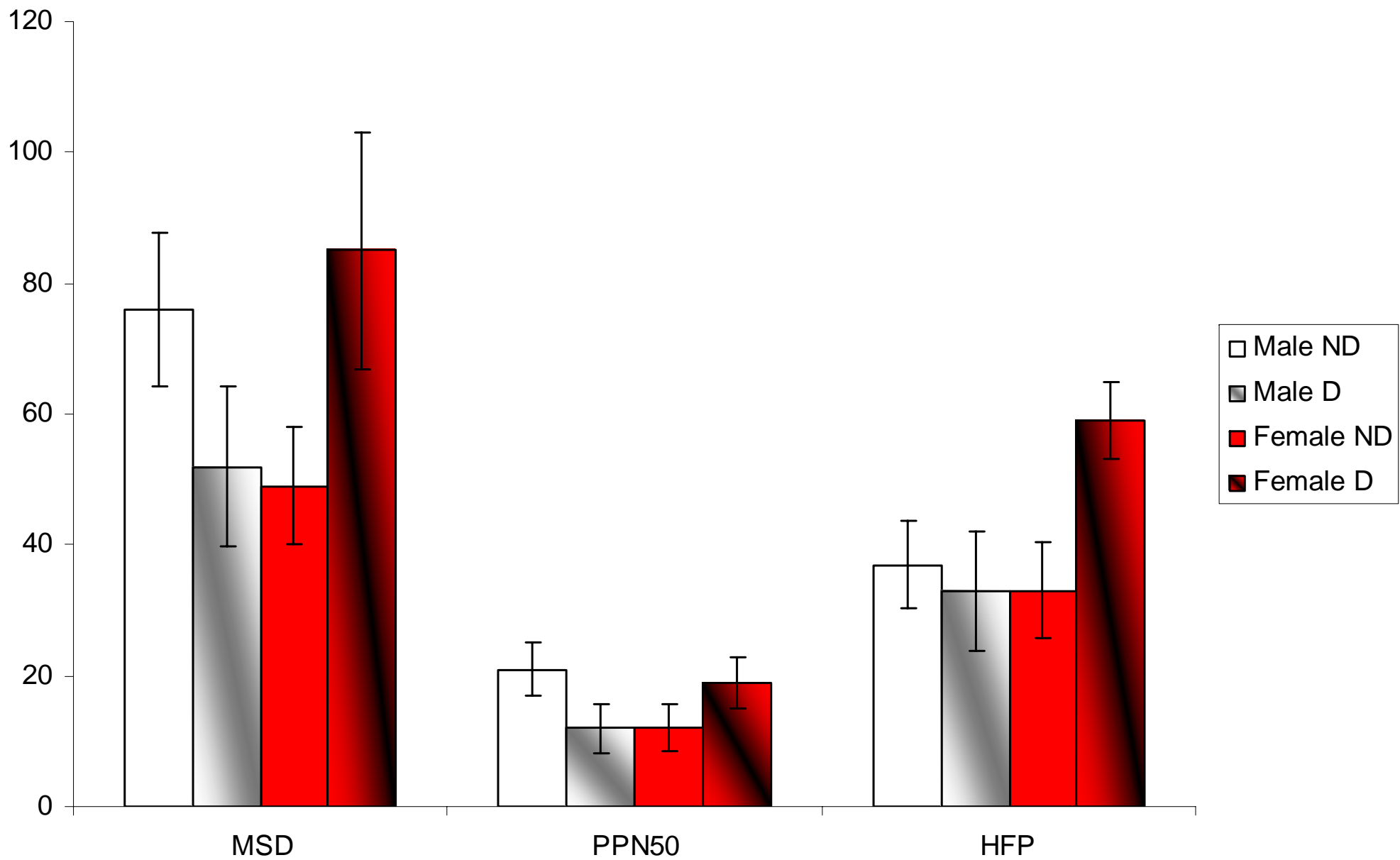


Figure 1. Power in the high frequency (respiratory) component of heart period variability in GAD patients and controls during relaxation and worry.

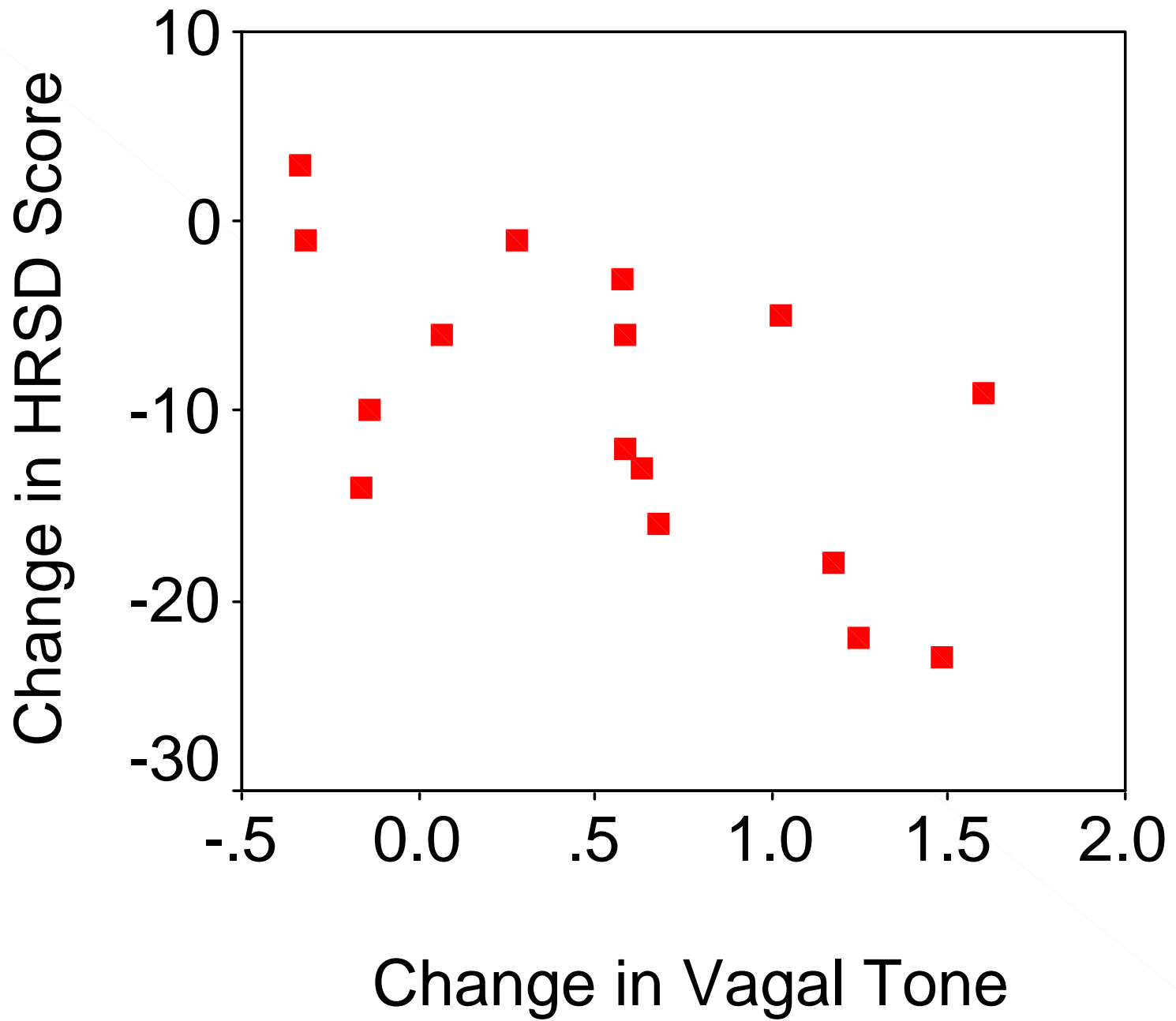
Table 1
Significant contrasts among panickers, blood phobics, and controls

Variable	Panic (mean, S.D.)	Blood phobic (mean, S.D.)	Control (mean, S.D.)	<i>T</i> ratio, df, <i>p</i> value
IBI (ms)	761.8 (141.0)	837.1 (92.4)	905.2 (132.5)	P < B 4.59 (215) <i>p</i> < 0.001 P < C 7.65 (214) <i>p</i> < 0.001 B < C 4.30 (207) <i>p</i> < 0.001
VAR (ms ²)	3942 (4009)	4334 (2663)	6112 (4563)	P < C 3.70 (214) <i>p</i> < 0.001 B < C 3.44 (207) <i>p</i> < 0.001 P = B N.S.
MSD (ms)	44.4 (31.2)	55.6 (22.7)	71.4 (32.1)	P < B 3.05 (215) <i>p</i> < 0.001 P < C 6.34 (214) <i>p</i> < 0.001 B < C 4.11 (207) <i>p</i> < 0.001
HF power (ms ² Hz ⁻¹)	991 (1225)	1385 (1073)	2239 (1911)	P < B 2.49 (212) <i>p</i> < 0.01 P < C 5.67 (212) <i>p</i> < 0.001 B < C 3.90 (203) <i>p</i> < 0.001
LF/HF	2.1(2.5)	1.3 (1.8)	1.0 (1.5)	P < B 2.41 (209) <i>p</i> < 0.005 P < C 3.64 (203) <i>p</i> < 0.001 B = C N.S.

P, panic; B, blood phobic; C, control.



Data from Thayer et al., 1998, *Bio Psychiatry*



Trait Vagal Tone (cont')

- Defensive Coping ([Movius & Allen, 2001](#))
- Integrative Developmental Model

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T. Beauchaine

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

Motivational Predisposition	Behavioral Manifestation	Motivational System (SNS)		Regulatory System (PNS)	
		Activation (BAS)	Inhibition (BIS)	Emotional Trait (RSA)	Emotional State (RSA Reactivity)
Disinhibition	Impulsivity (ADHD)	High	Low	—	—
	Aggression (UACD)	High	Low	Low	High
	Panic	High	High	Low	High
	Extraversion	High	—	—	—
Inhibition	Anxiety	—	High	Low	—
	Depression	Low	High	Low	—
	Panic	High	High	Low	High
	Introversion	—	High	—	—
None	Emotional stability	—	—	High	—
	Emotional lability	—	—	—	High

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 (2001),
*Development and
Psychopathology*

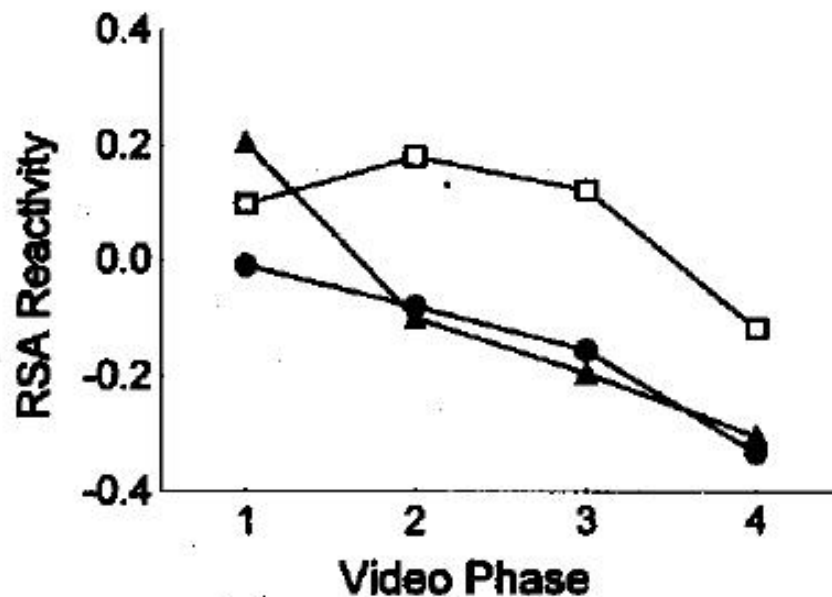
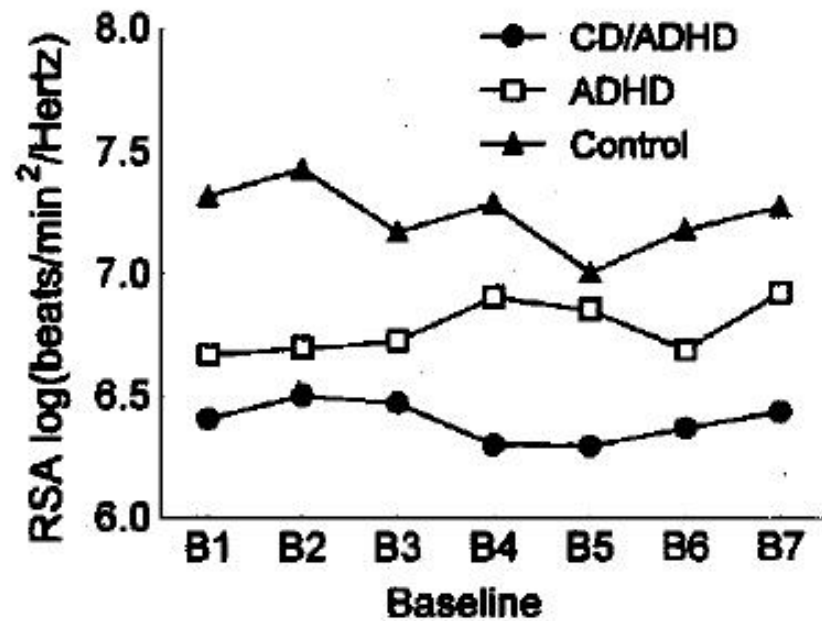
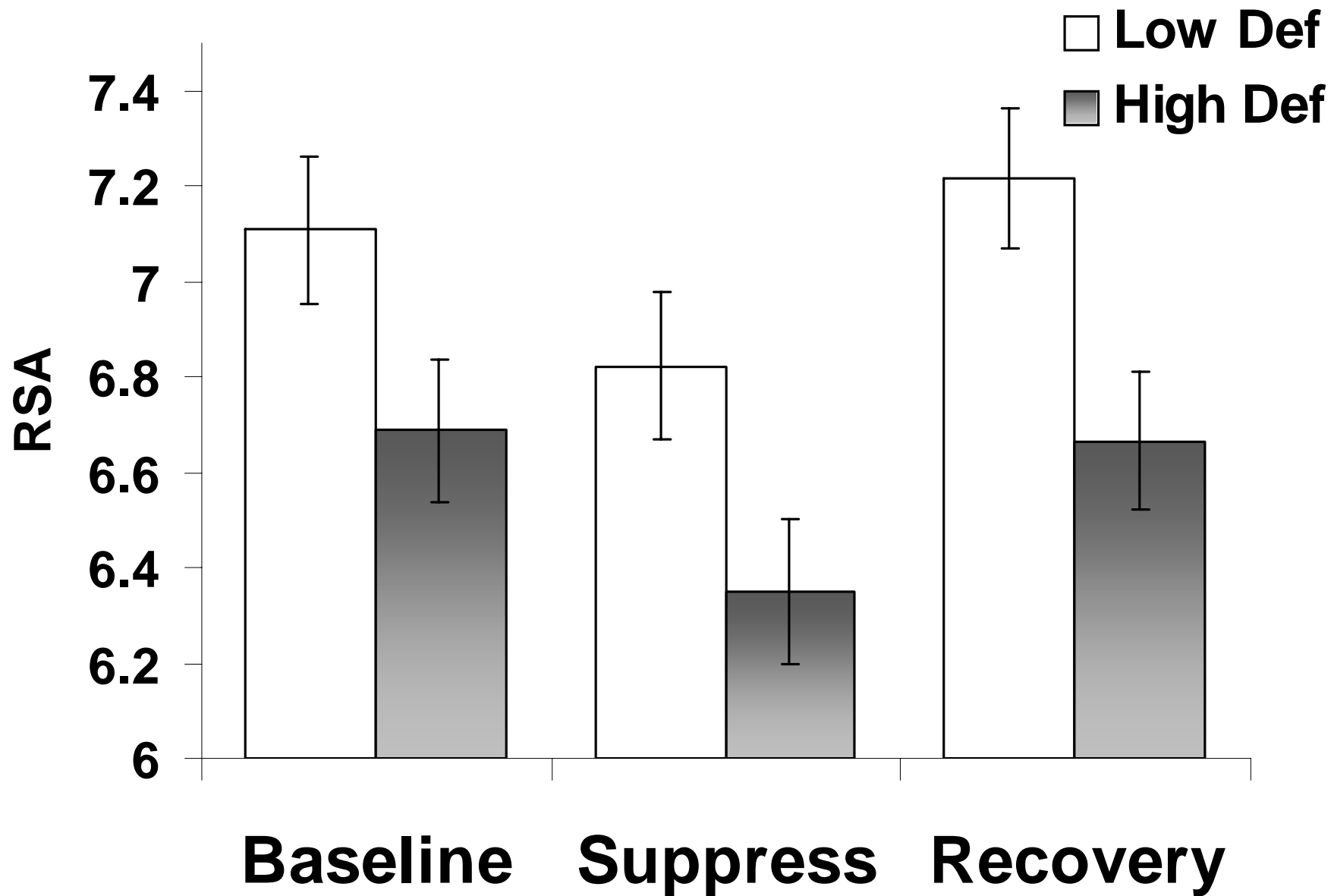


Figure 4. Baseline respiratory sinus arrhythmia (RSA; top panel), and RSA reactivity (bottom panel) for aggressive CD/ADHD participants, hyperactive/impulsive ADHD participants, and controls. CD = conduct disorder; ADHD = attention-deficit/hyperactivity disorder.

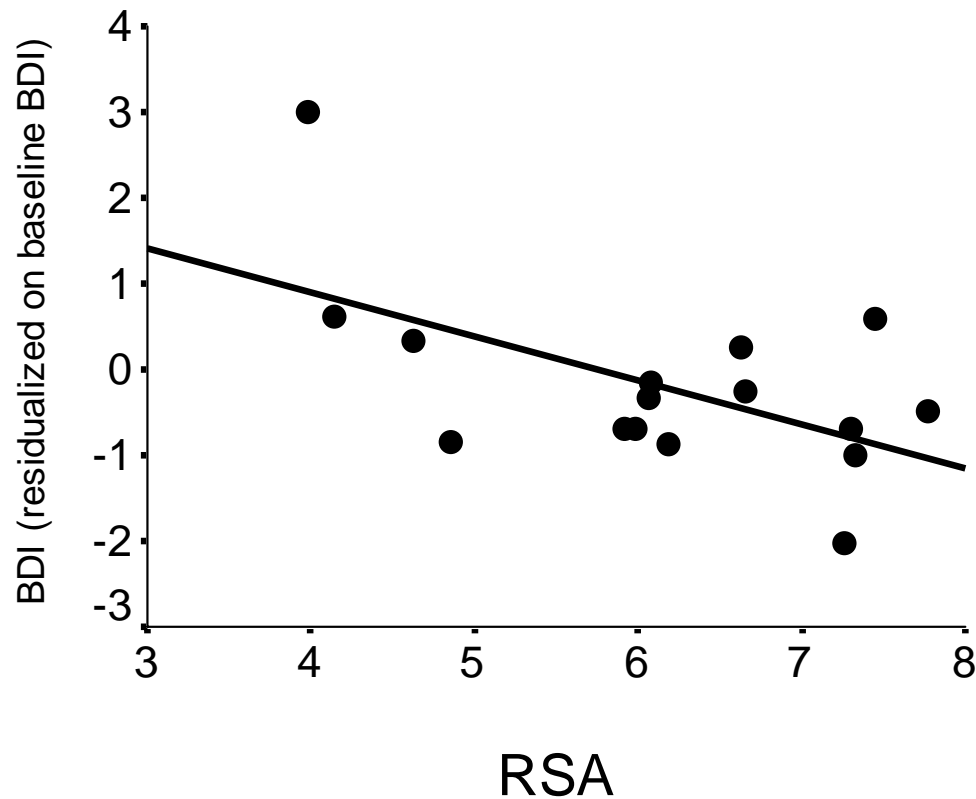
Beauchaine et al (2001)
Journal of Abnormal Psychology



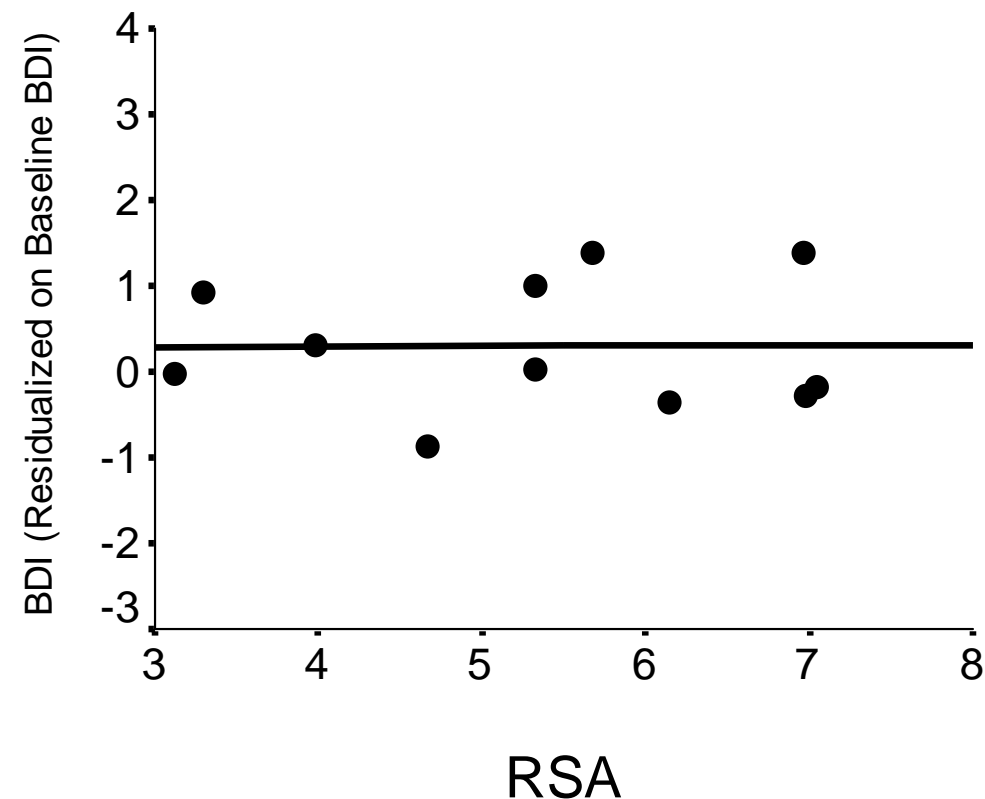
Trait Vagal Tone as Moderator of Response following Bereavement

- 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

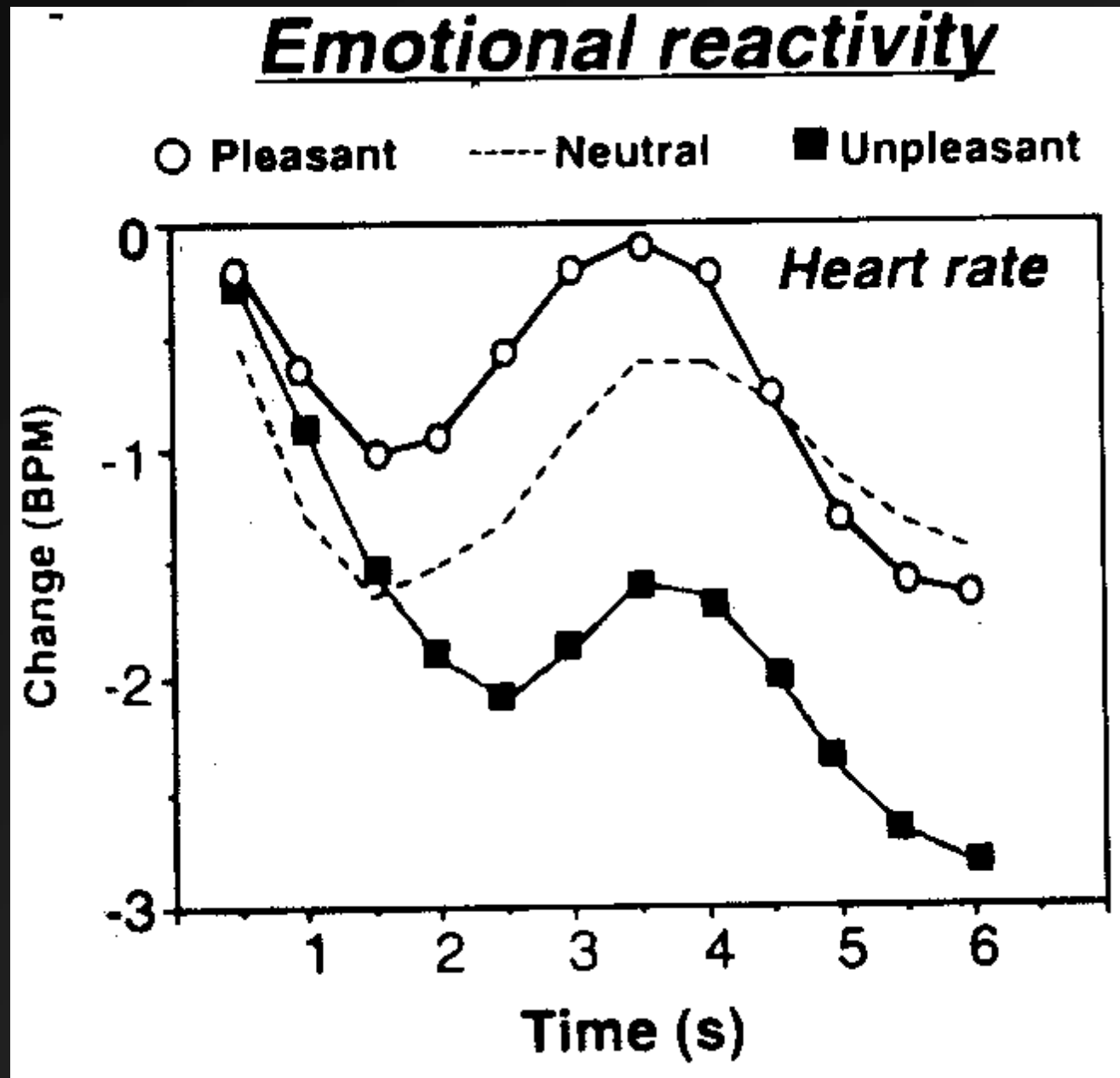
Intervention Group



Control Group



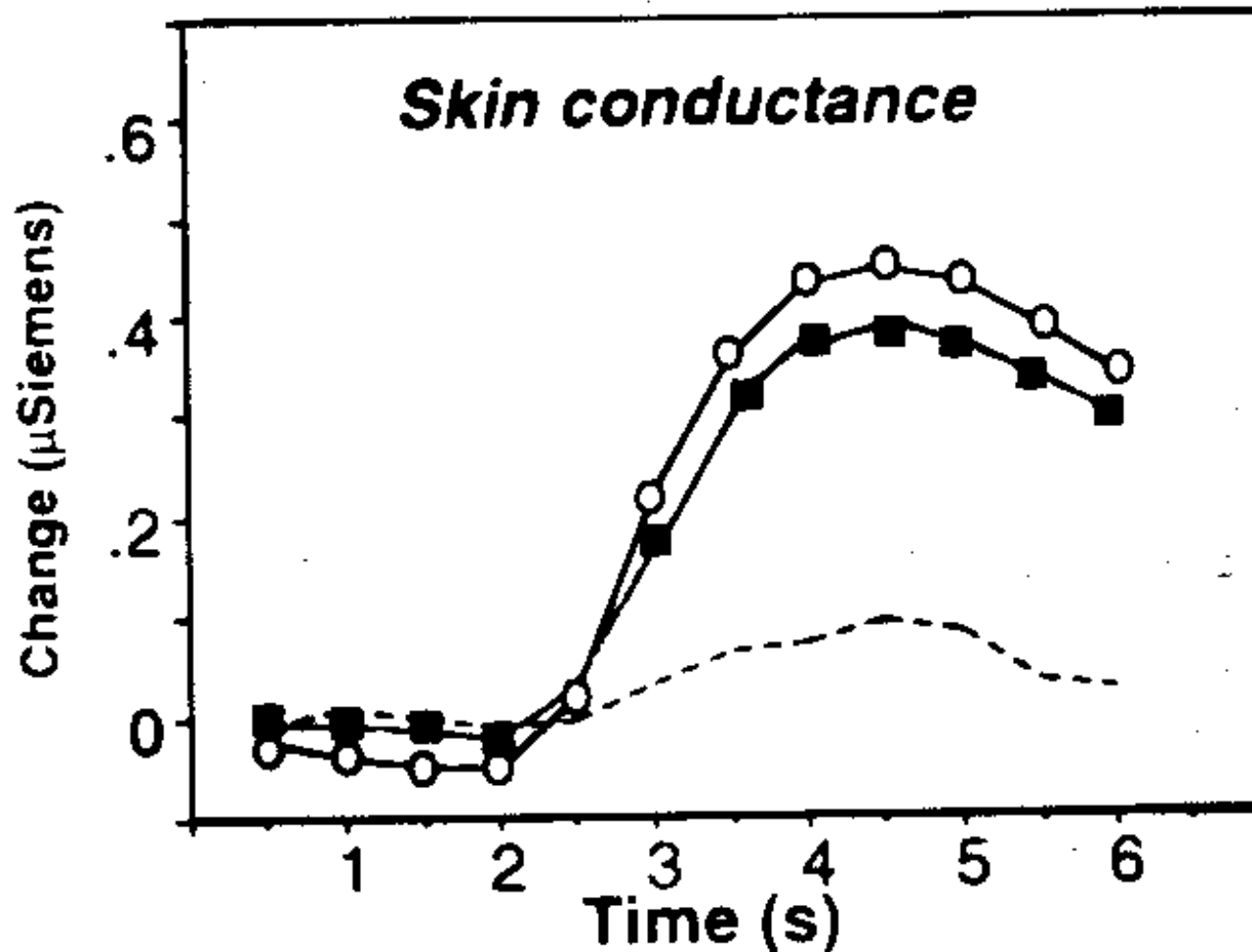
Orienting, Attention, and Defense

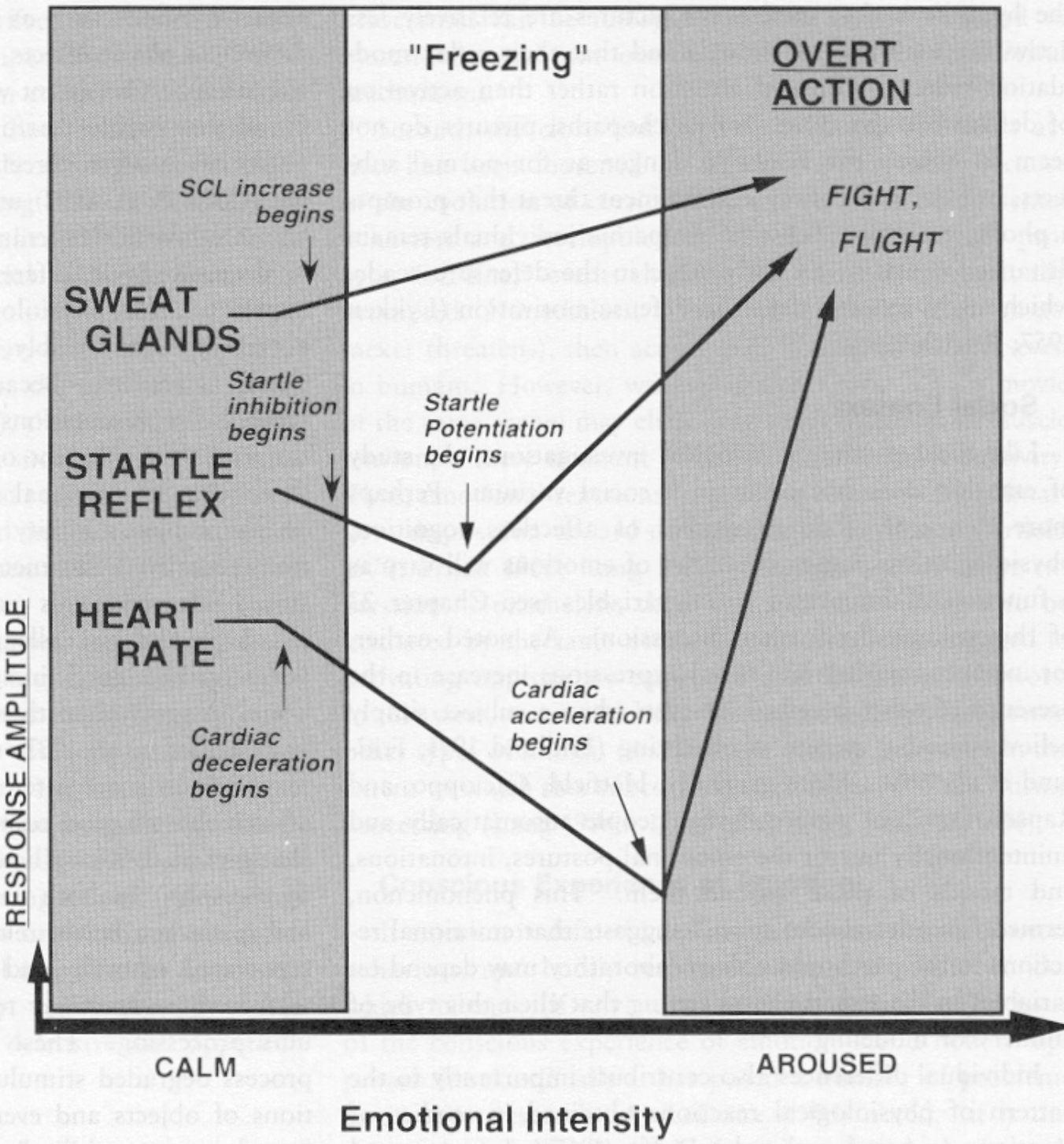


SCR (by contrast)

Emotional reactivity

○ Pleasant - - - - Neutral ■ Unpleasant





OR Vs DR