Cardiovascular Psychophysiology

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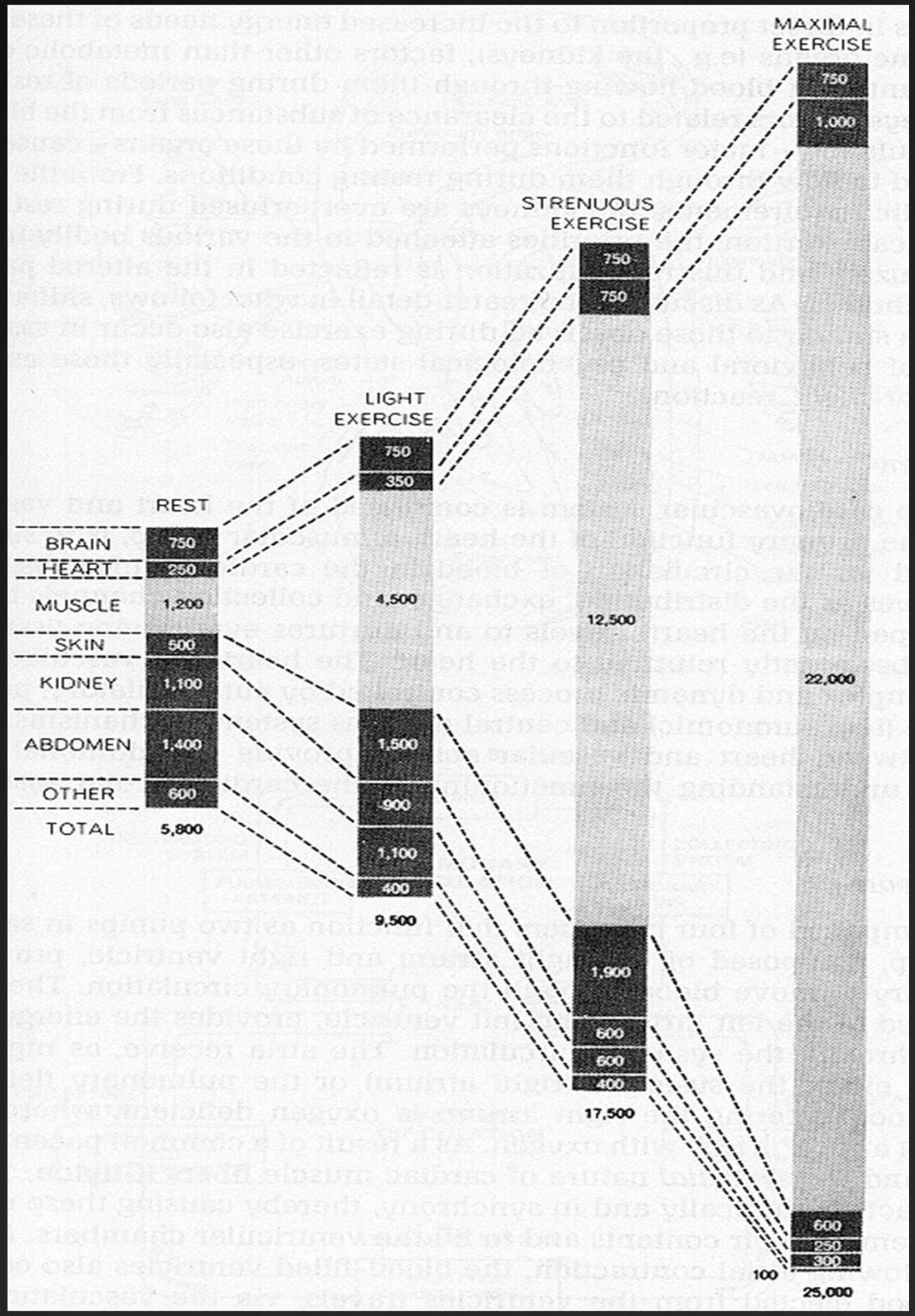
Facts and Functions

- 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

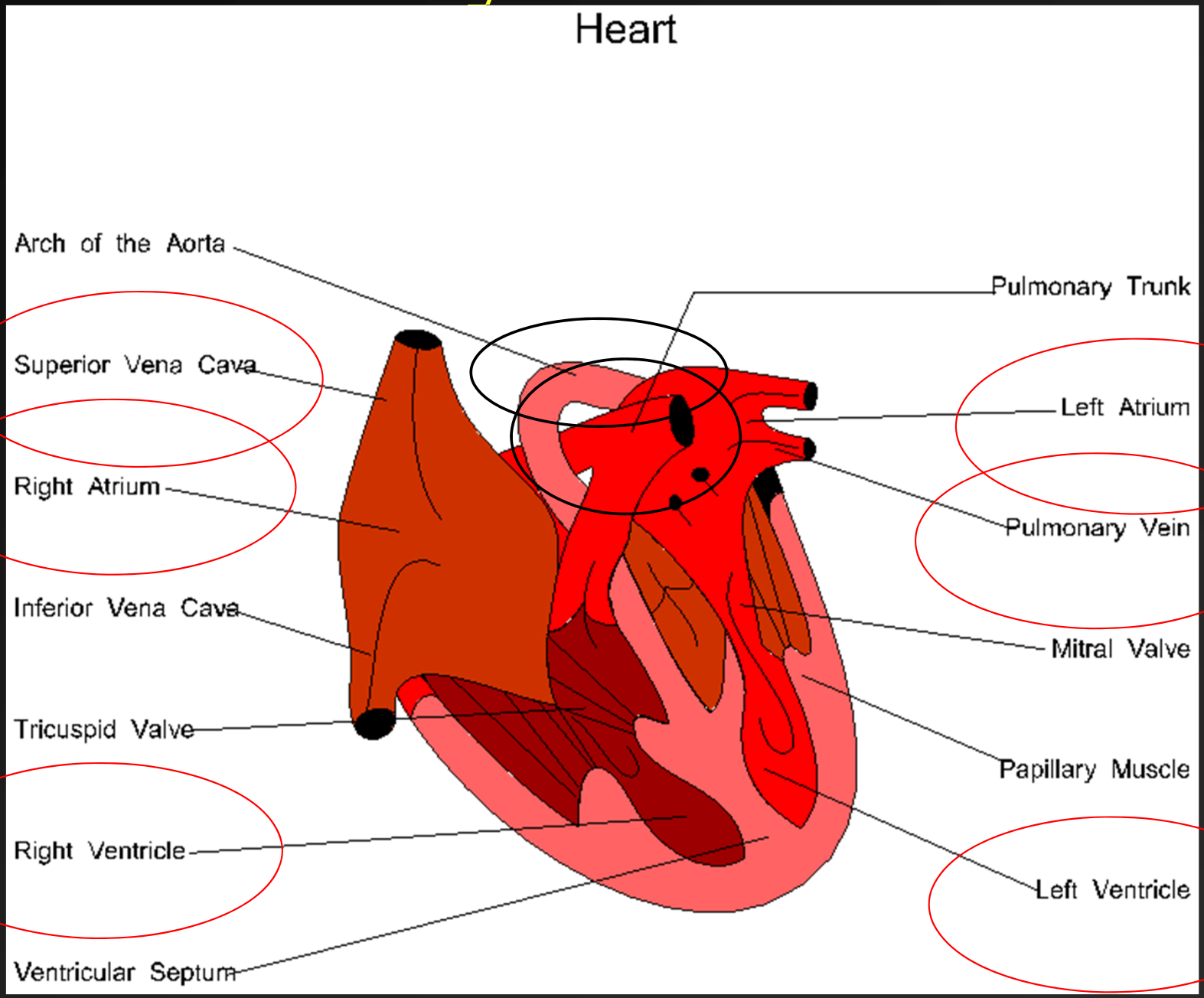


Anatomy of the Heart

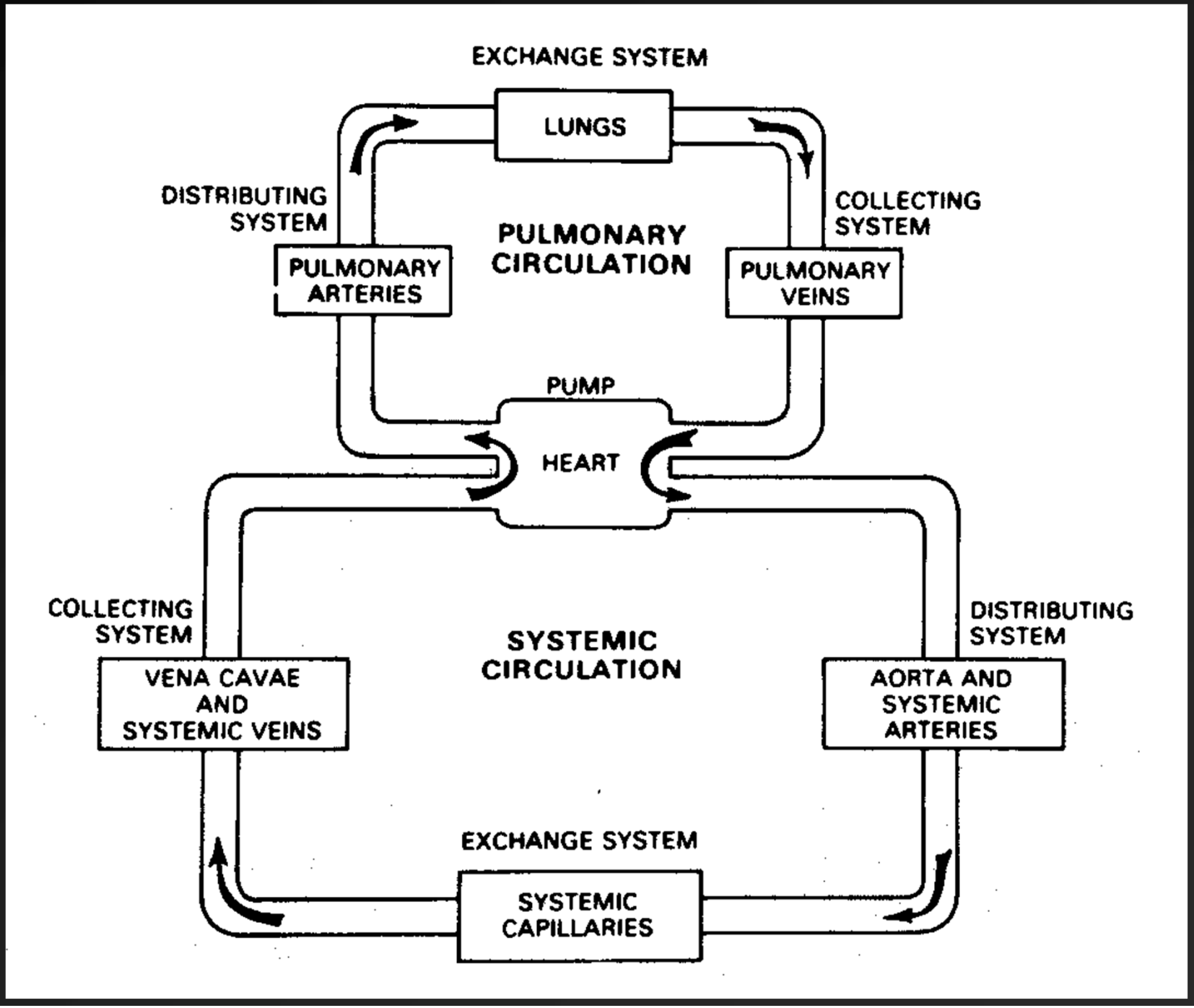
- Cardiac Muscle (myocardium)
  - not striated, not smooth
  - four features distinguish from smooth or striate
    - Muscle has unstable resting potential – basis for intrinsic and rhythmic contraction
    - Action potential freely conducted from one cell to another (lattice-like syncytial) network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



Human Circulatory System



Circulation in a bit more realistic detail

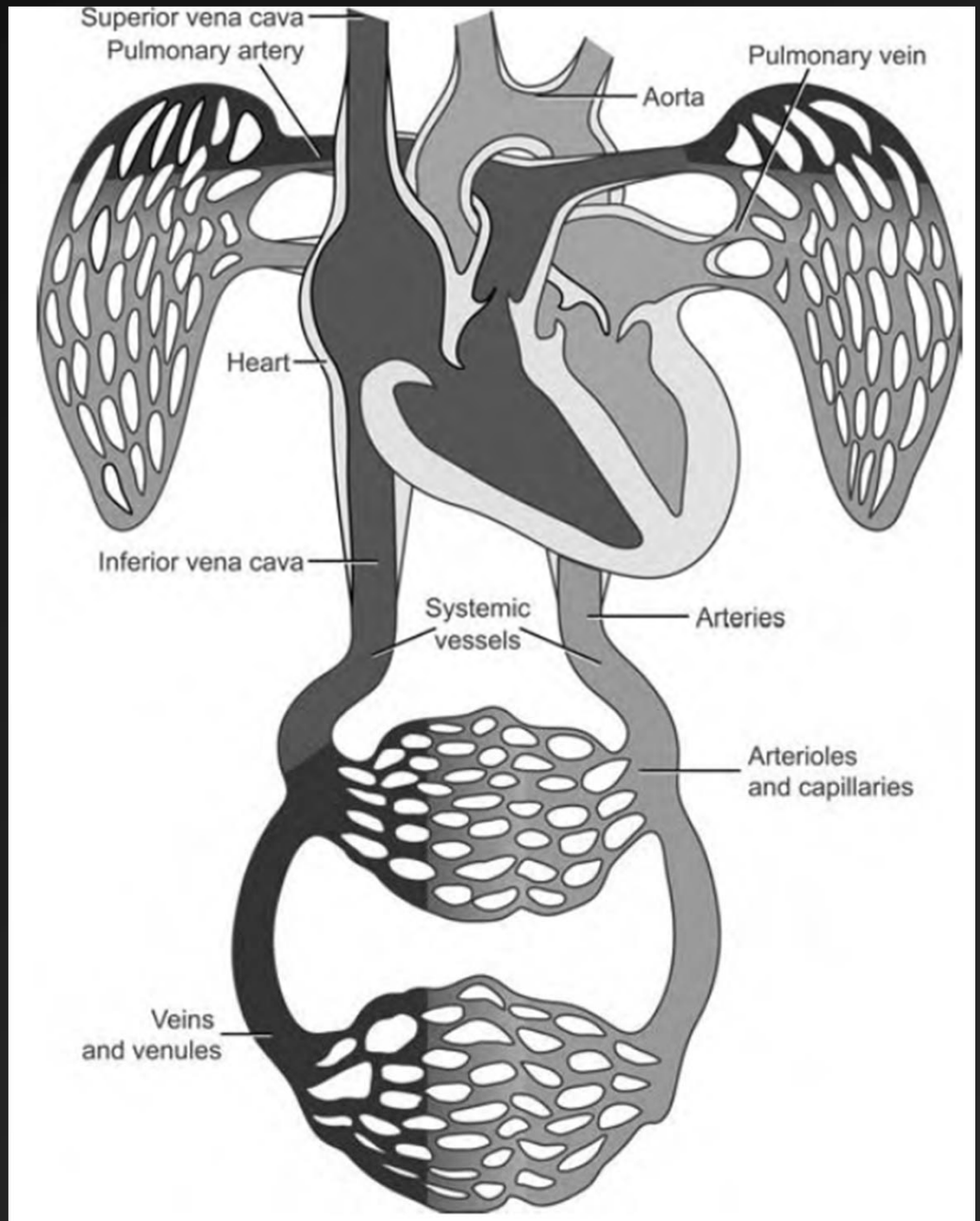
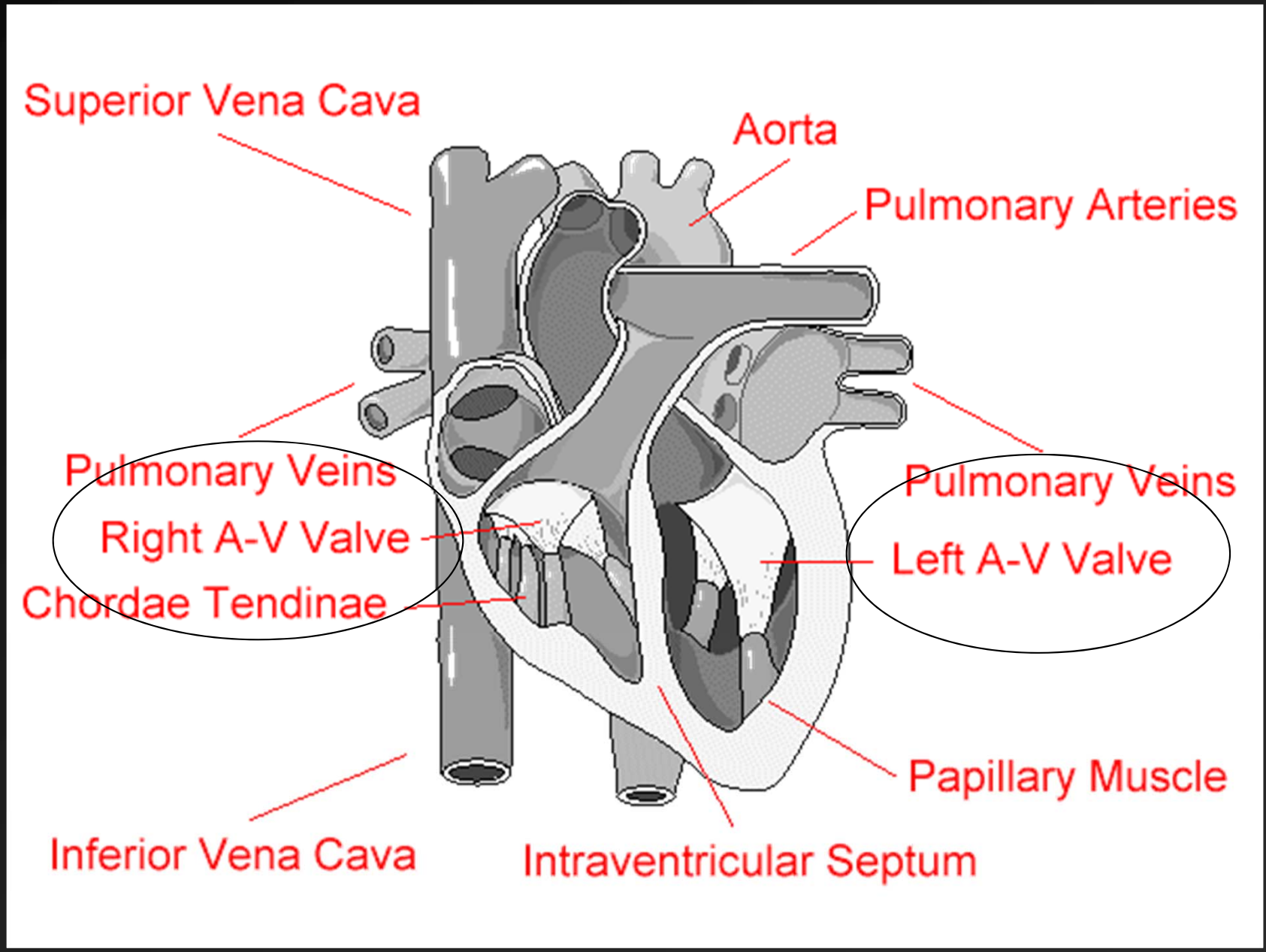


Figure 8.1. Systemic and pulmonary circulation. In keeping with usual depictions of the heart, the right side of the heart is on the left side of the picture. Lighter gray areas indicate oxygenated blood and darker gray areas indicate deoxygenated blood.

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"

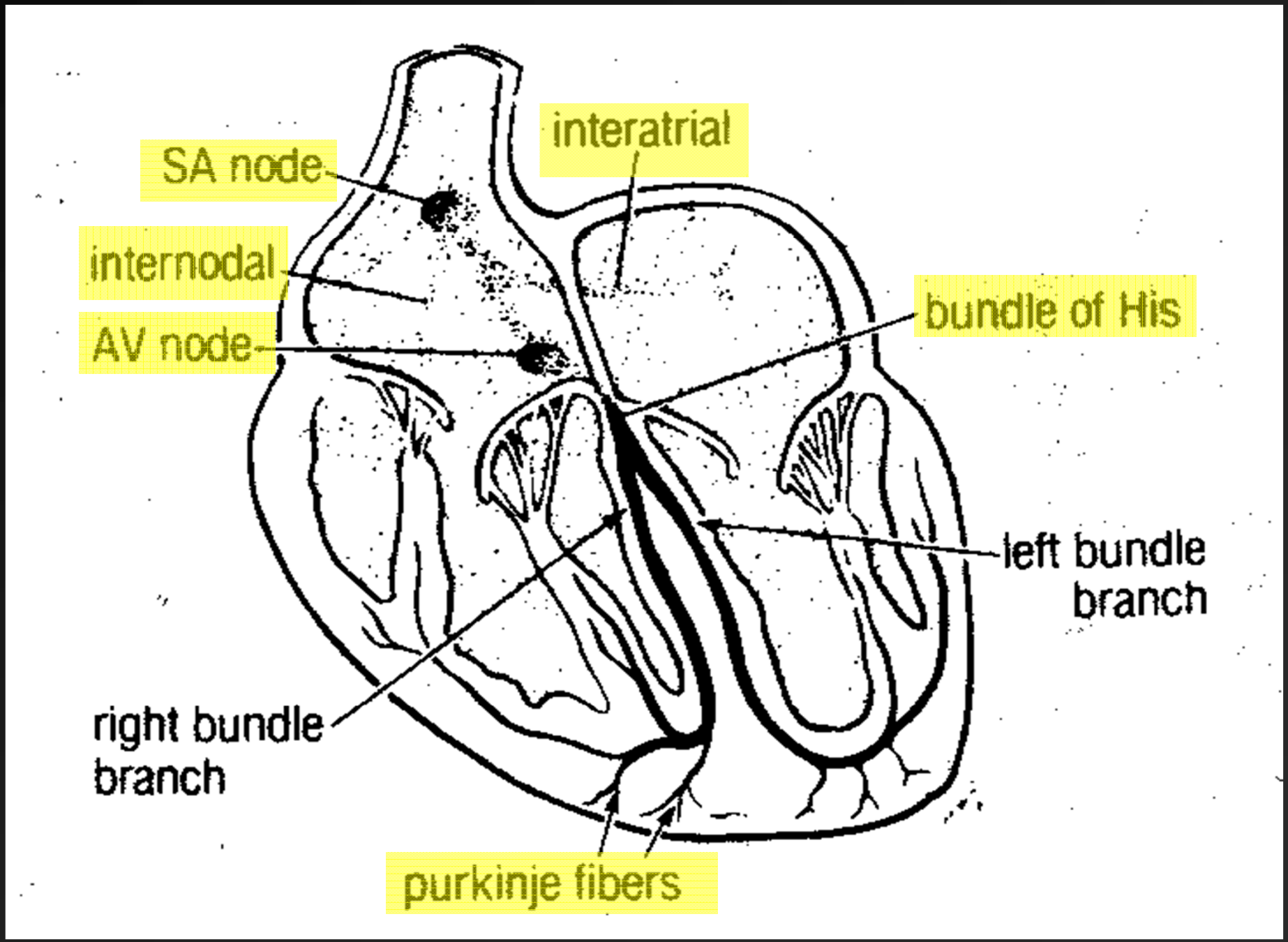
- Nodes have intrinsic rythmicity
  - SA node: 105 bpm
  - AV node: 40-60 bpm

- Denervated heart would still beat at over 100 bpm
  - Must be extrinsic influences to slow or speed heart

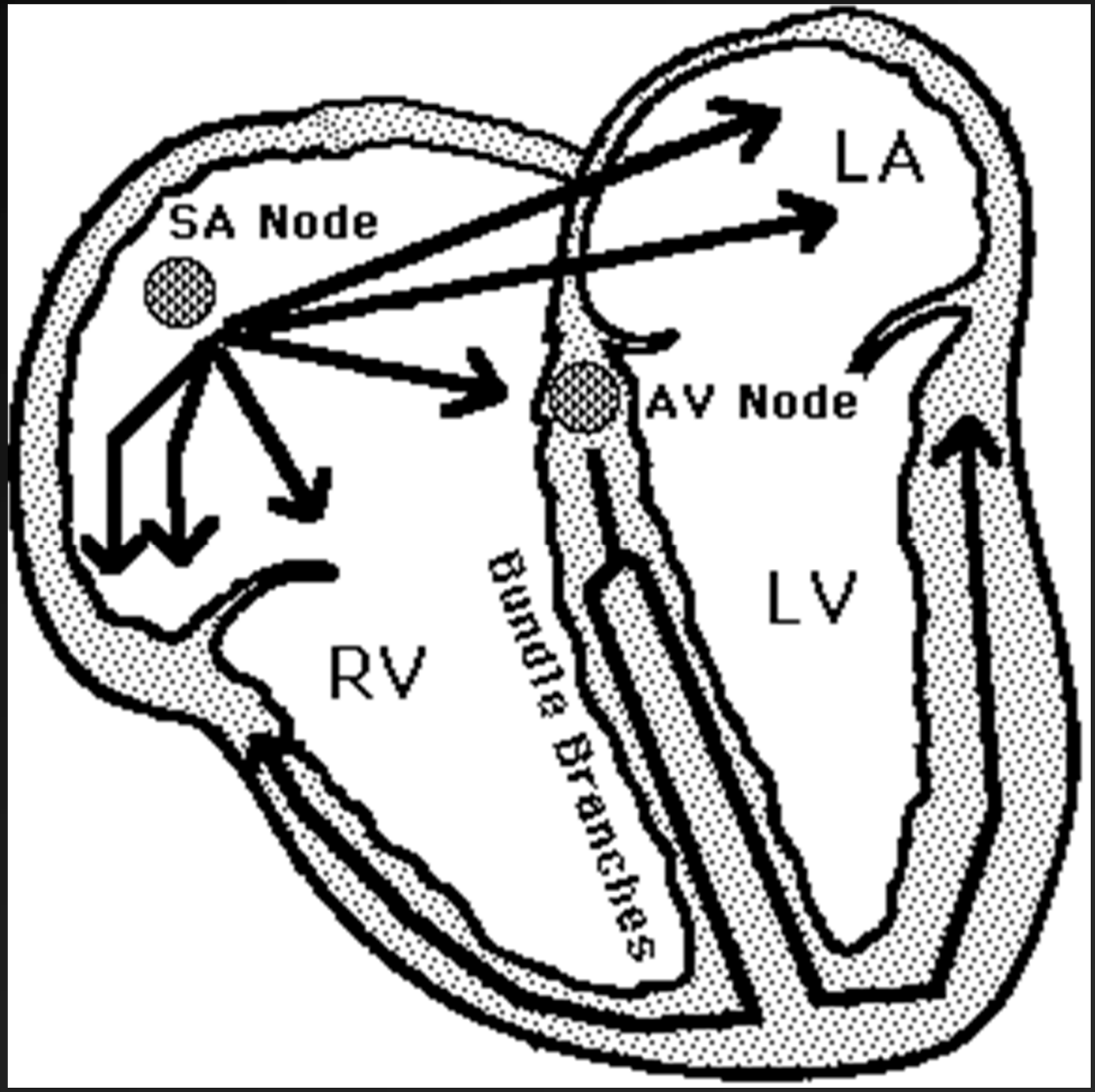
Neural Conduction of the Heart

- 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

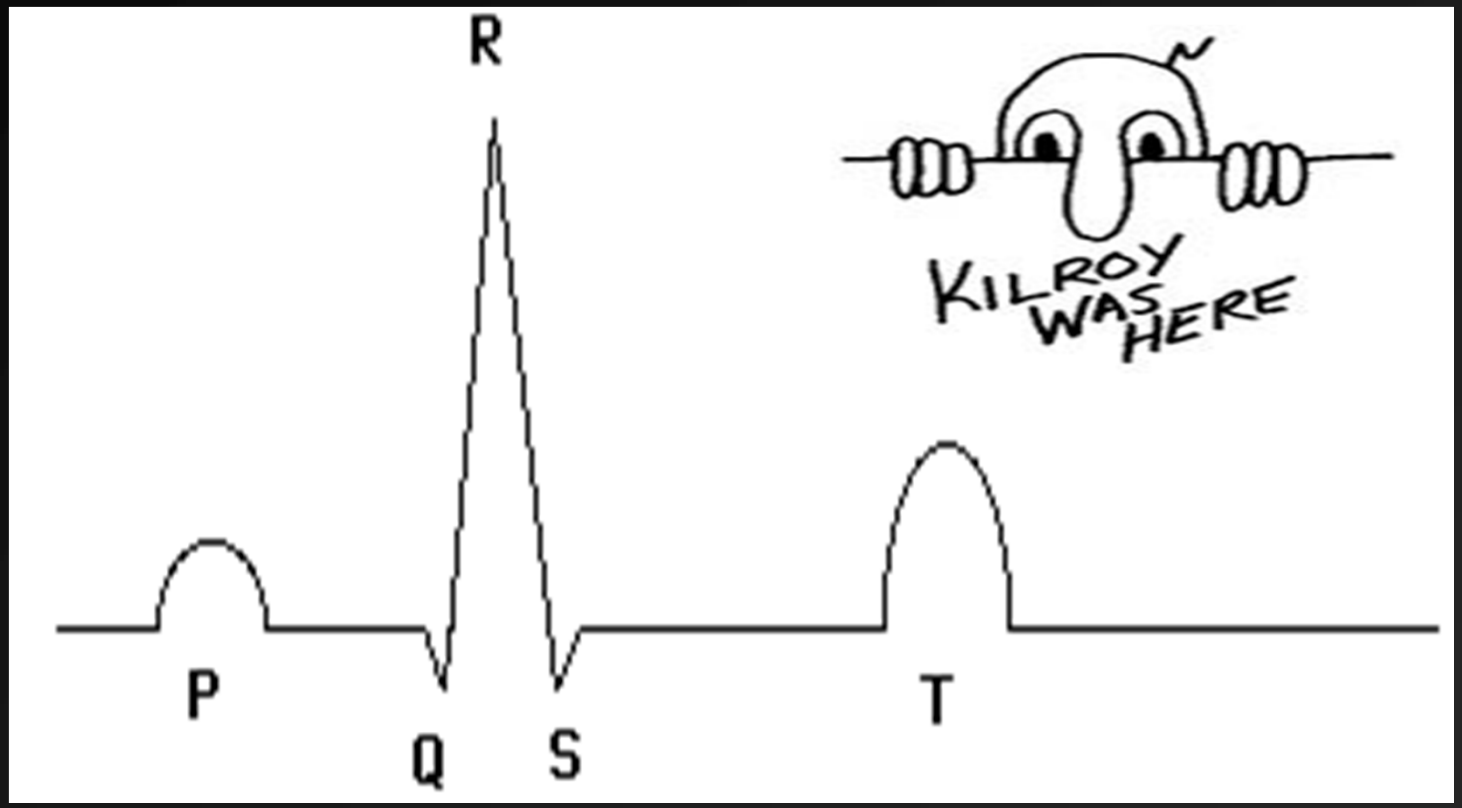


The SA and AV Nodes in Action



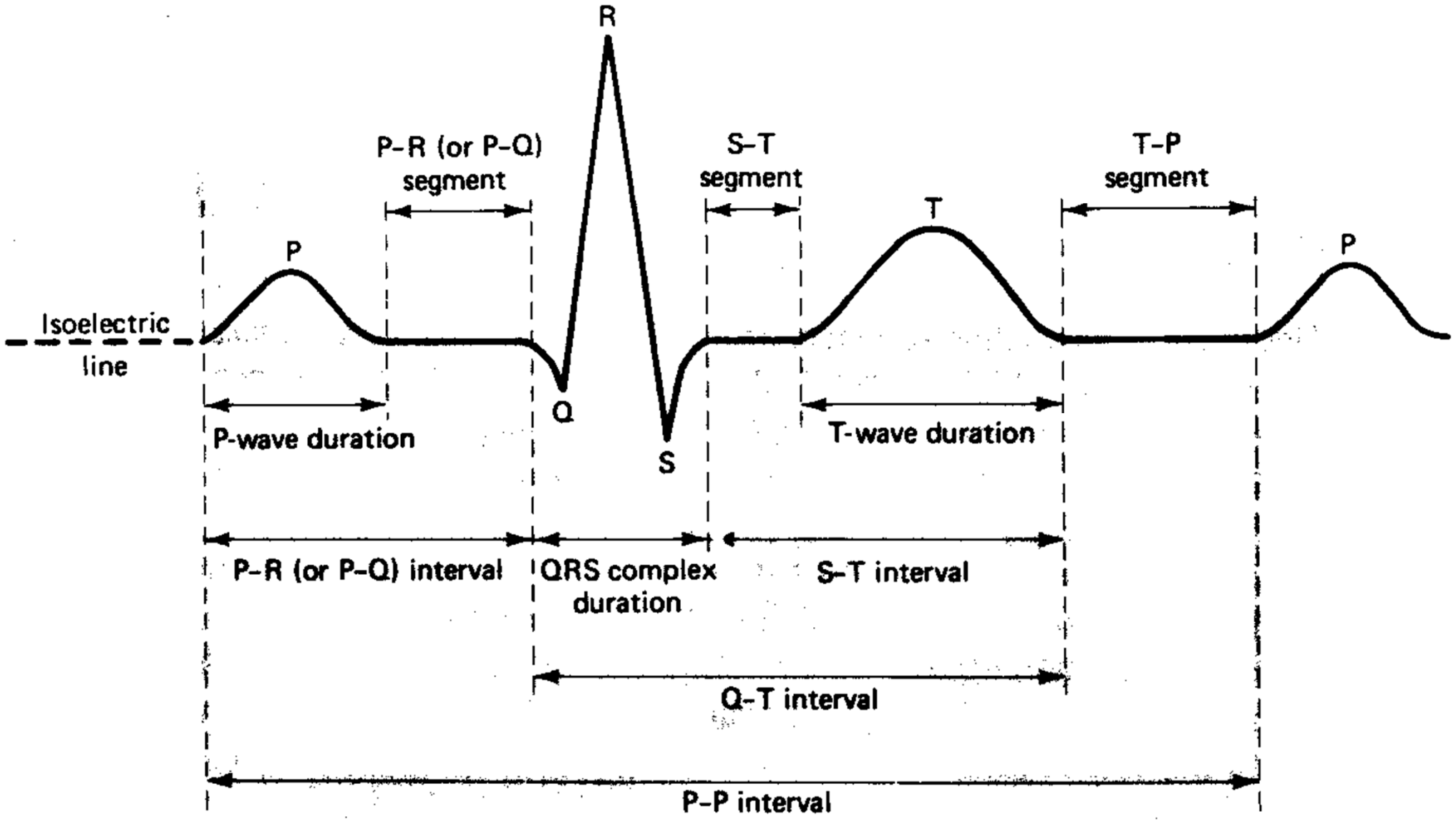
Important: refractory period of the AV node is longer then the time it takes the ventricles to contract

The Schematized EKG waveform

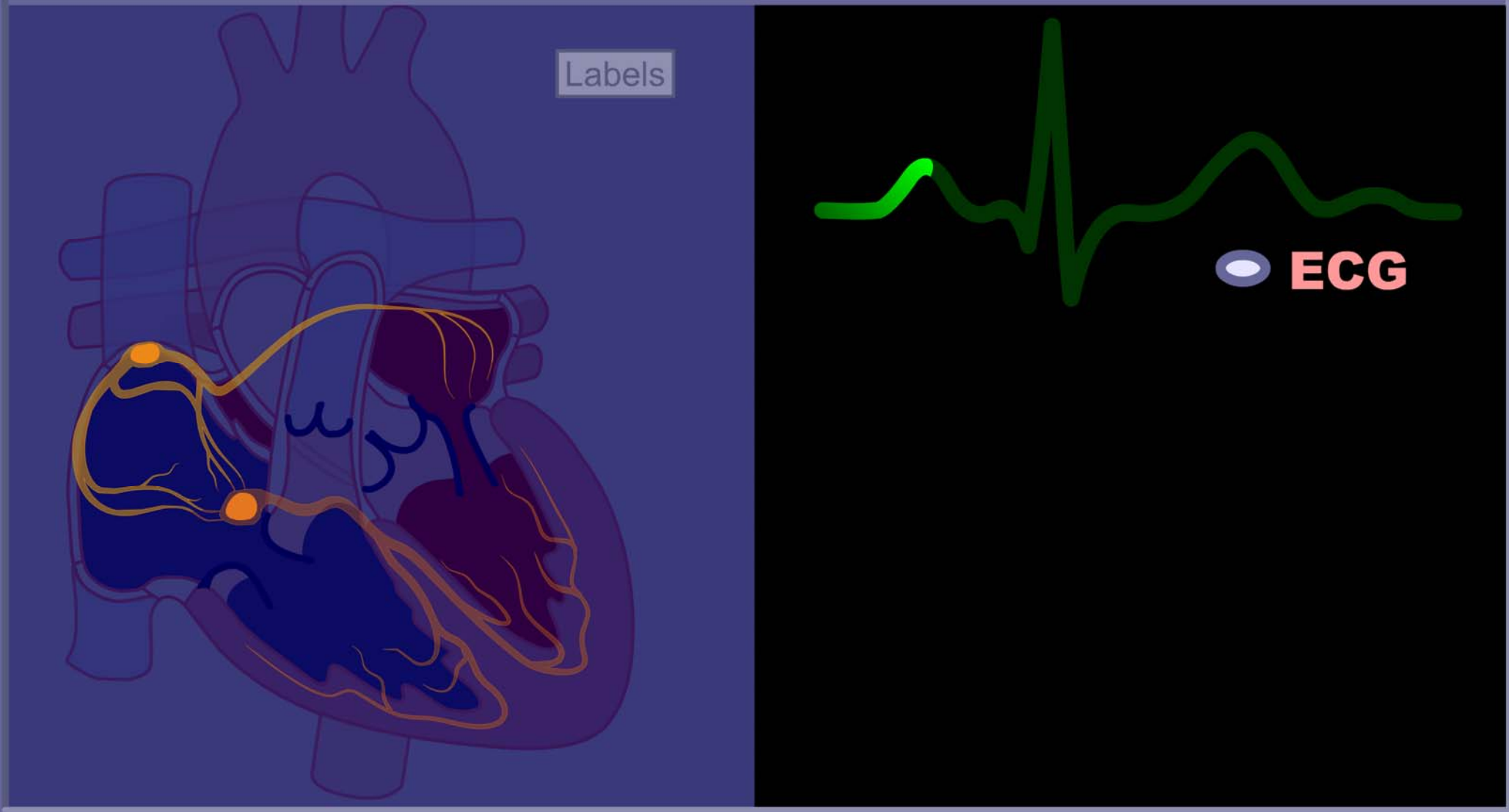


P = Atrial depolarization
QRS = Ventricular depolarization
T = Ventricular repolarization
Note that Atrial repolarization is not visible

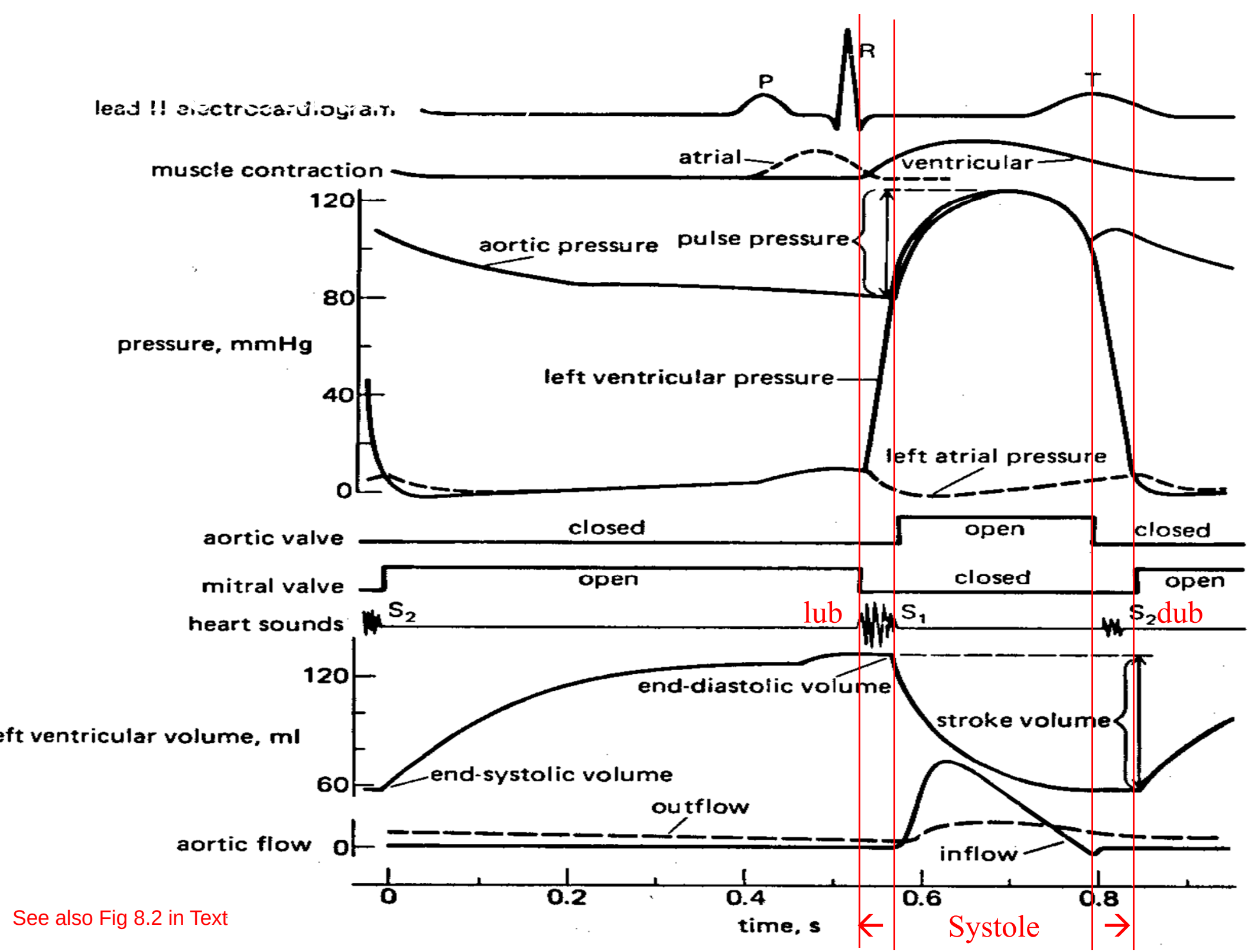
The EKG waveform



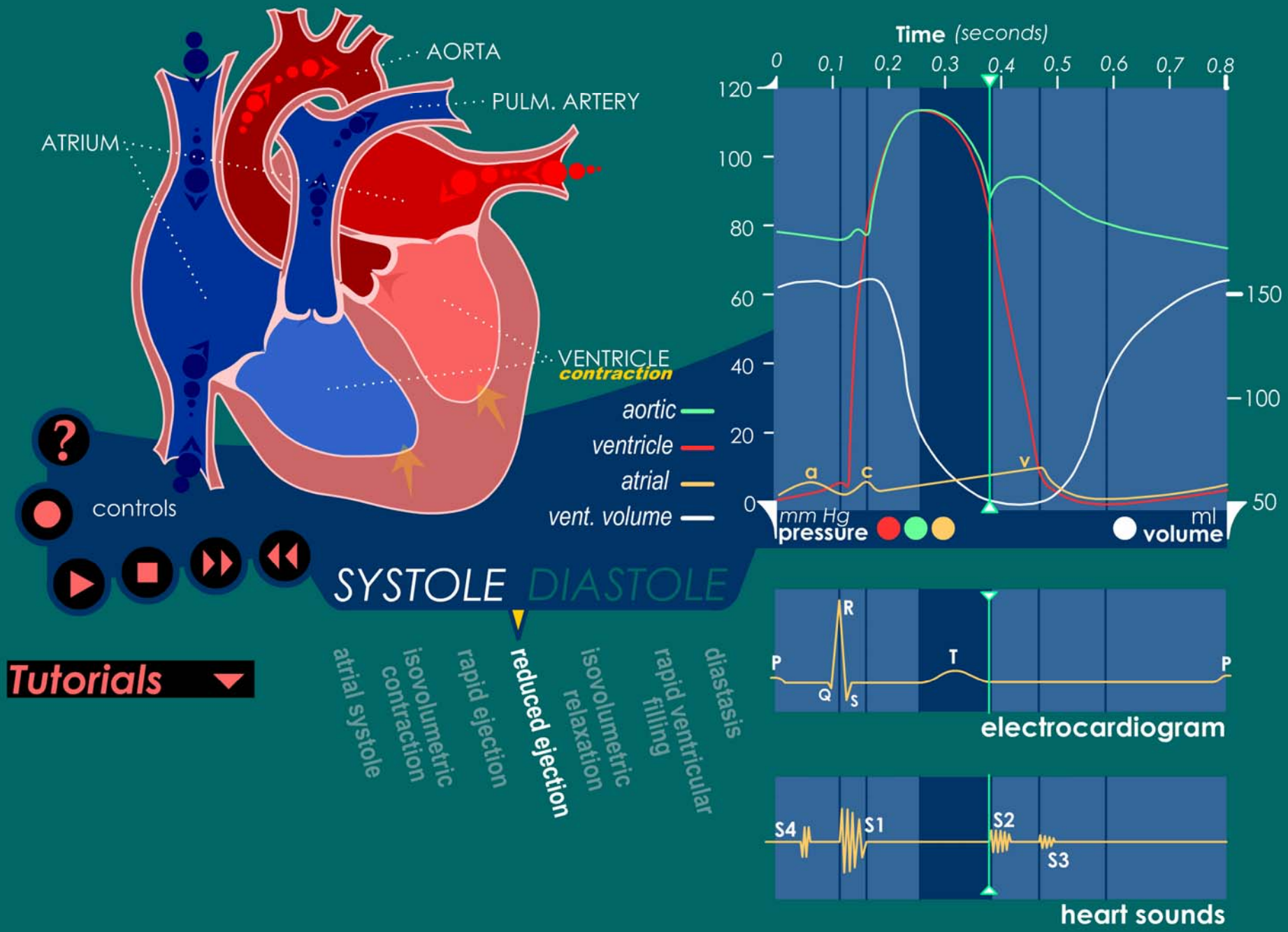
Electrical System of the Heart



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



See also Fig 8.2 in Text



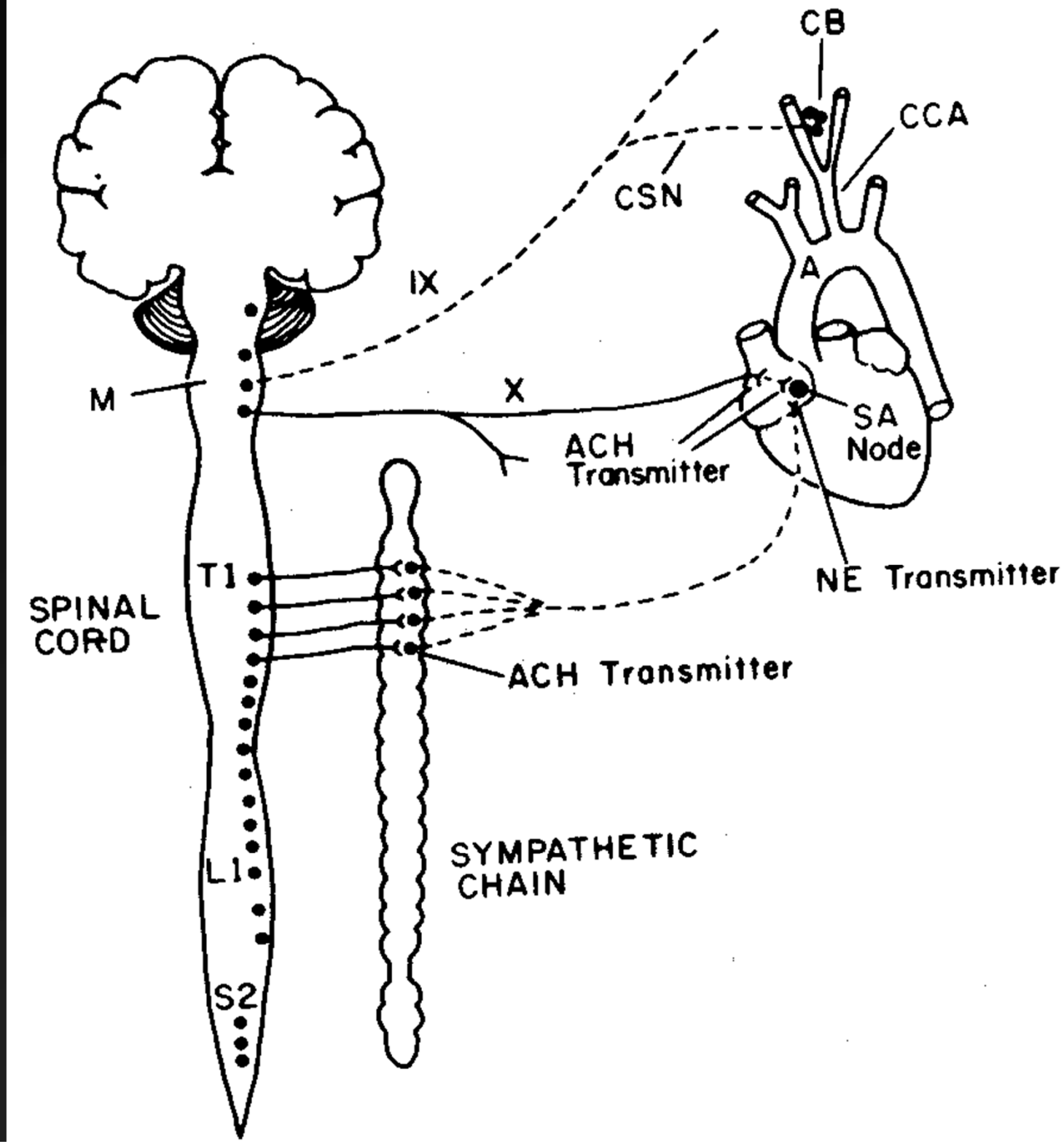
http://www-medlib.med.utah.edu/kw/pharm/hyper_heart1.html

Cardiac Output

$$CO = HR x SV$$

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


CB
CCA
CSN
IX
A
M
X
SA
Node
ACH
Transmitter
NE Transmitter
T1
SPINAL
CORD
ACH Transmitter
SYMPATHETIC
CHAIN
L1
S2

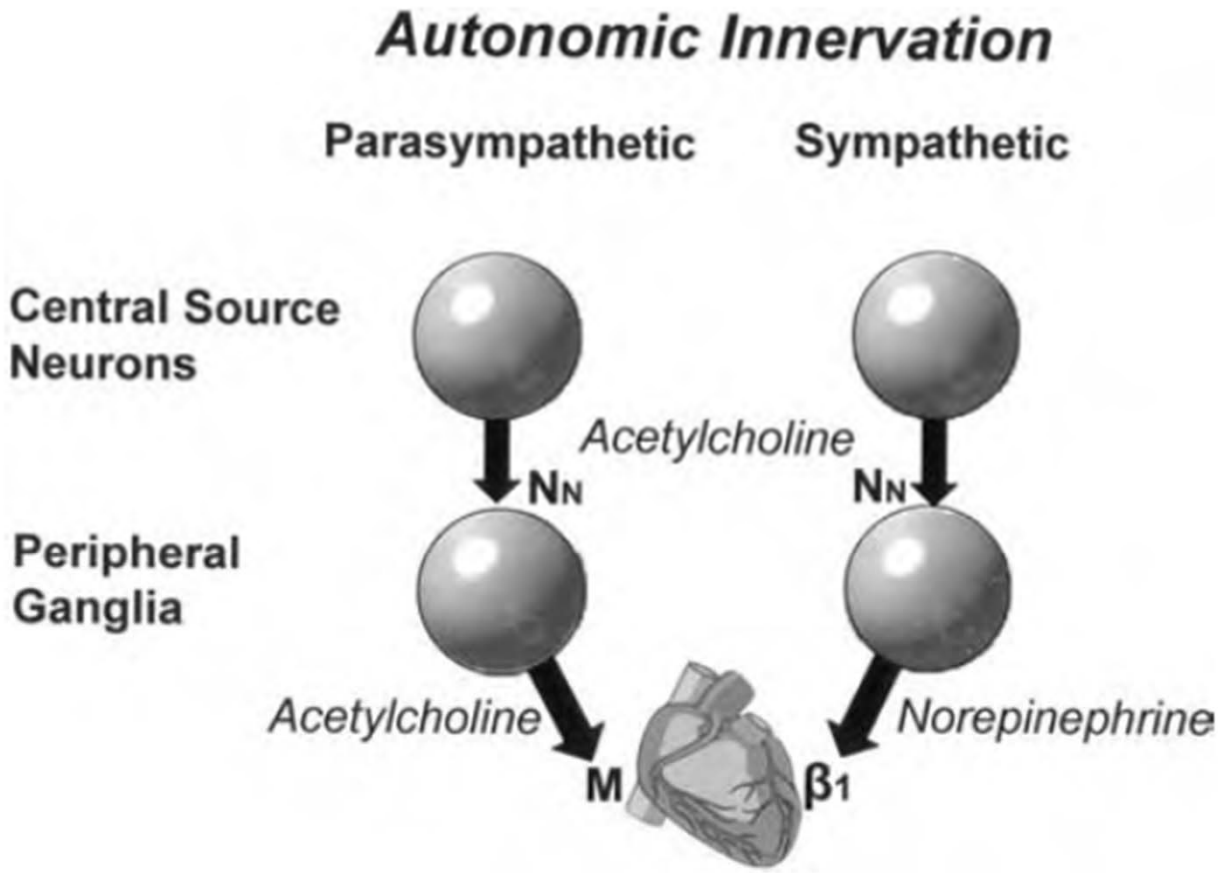
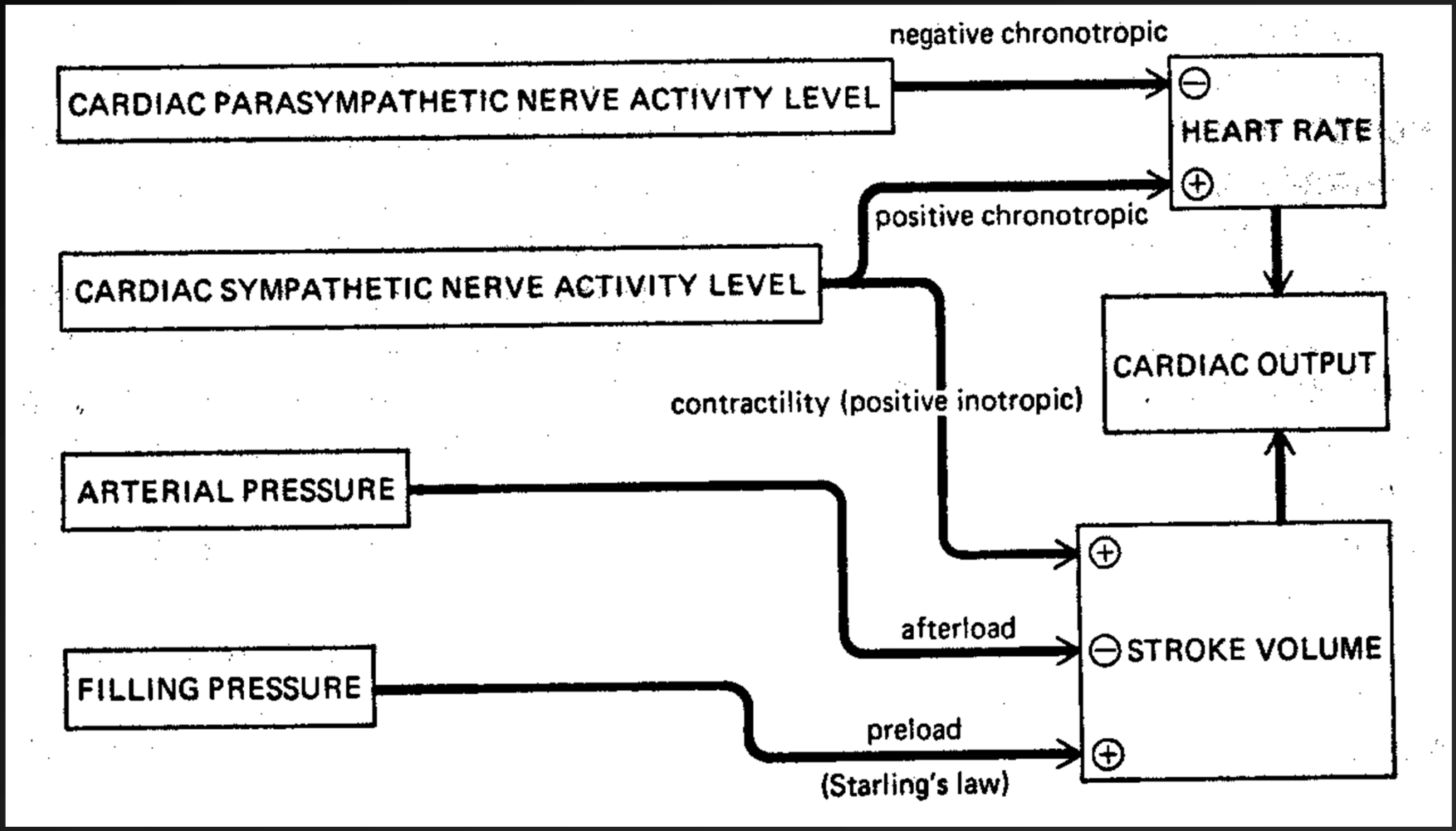
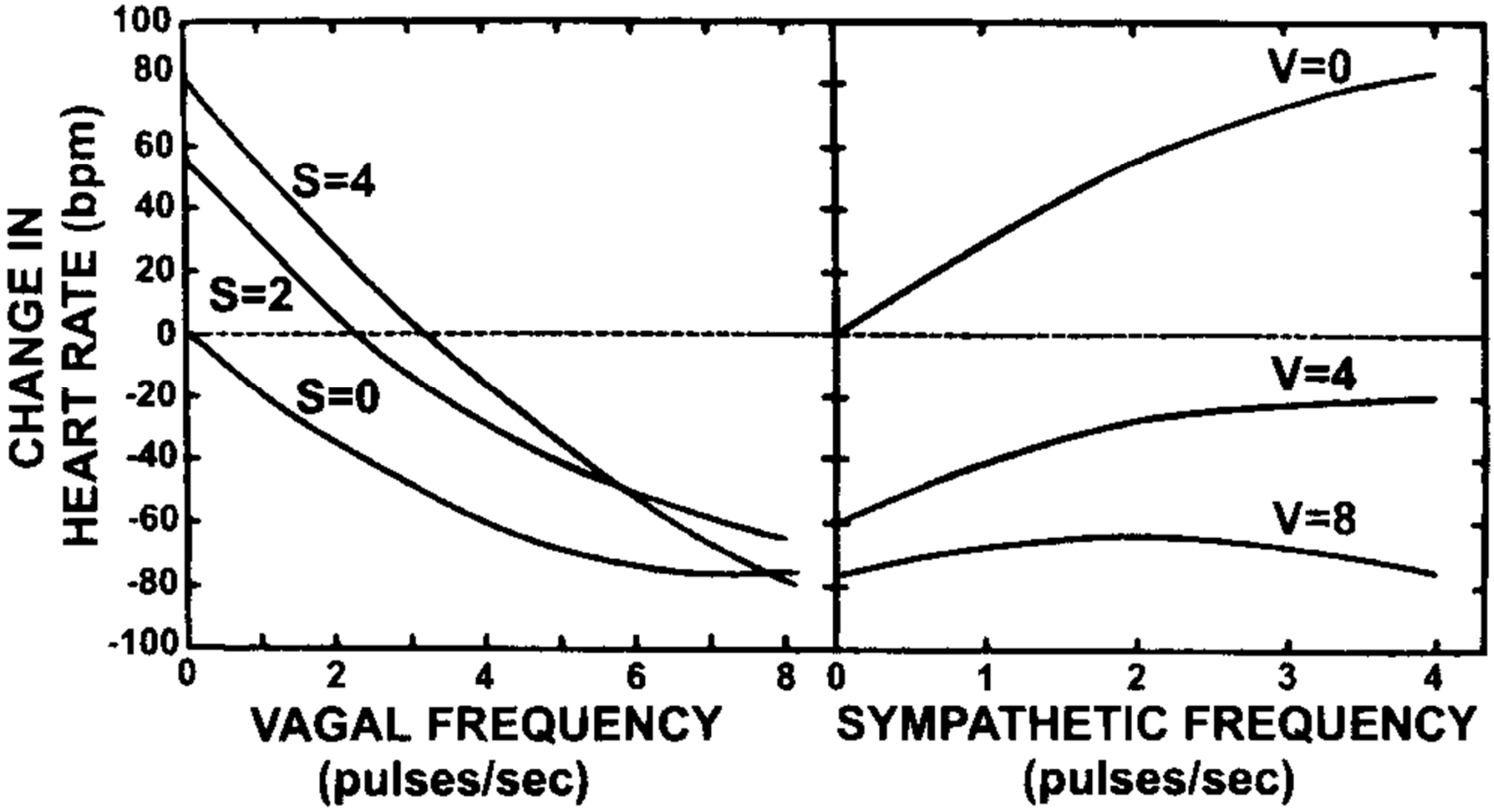


Figure 8.6. General pattern of pharmacology of the autonomic innervations. Abbreviations refer to the relevant postsynaptic receptor populations: N_N – nicotinic cholinergic; M – muscarinic cholinergic; $\beta 1$ – beta1 adrenergic.

SNS and PNS influences



HR change to simultaneous vagal and sympathetic stimulation



Levy & Zieske (1969). *J Applied Phsyio, 27*, 465-470

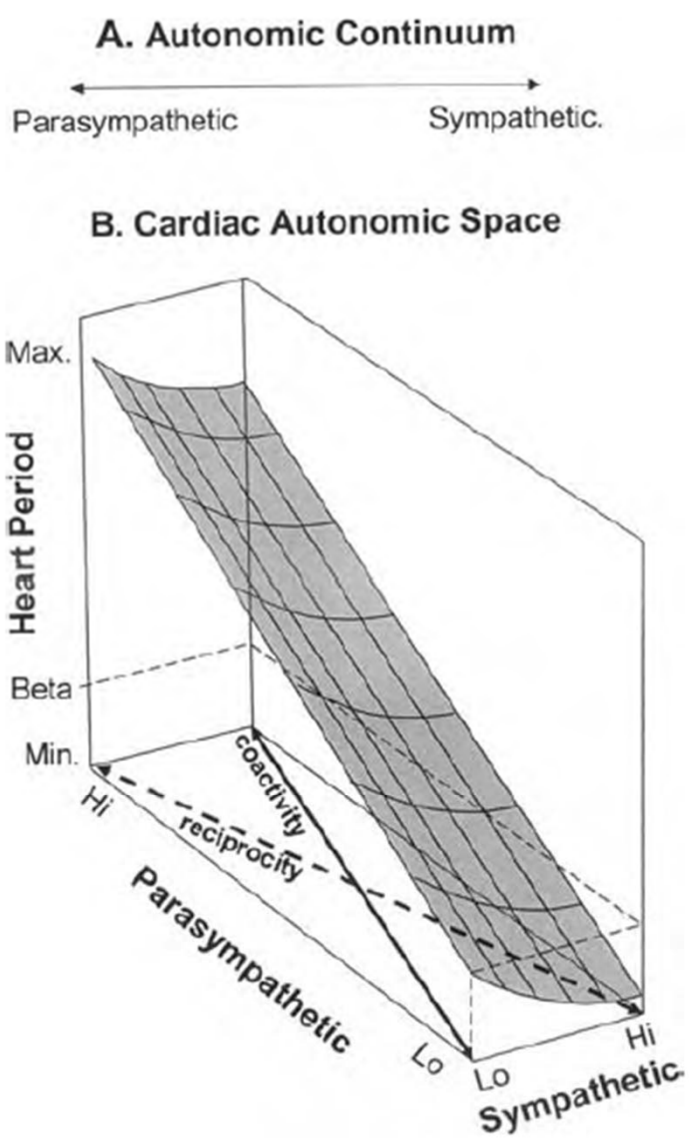


Figure 8.9. Autonomic space. (A) Continuum model of autonomic control, wherein the status of the system can be depicted along a single continuum extending from parasympathetic dominance to sympathetic dominance. (B) A more comprehensive model of autonomic control, characterized by an autonomic plane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

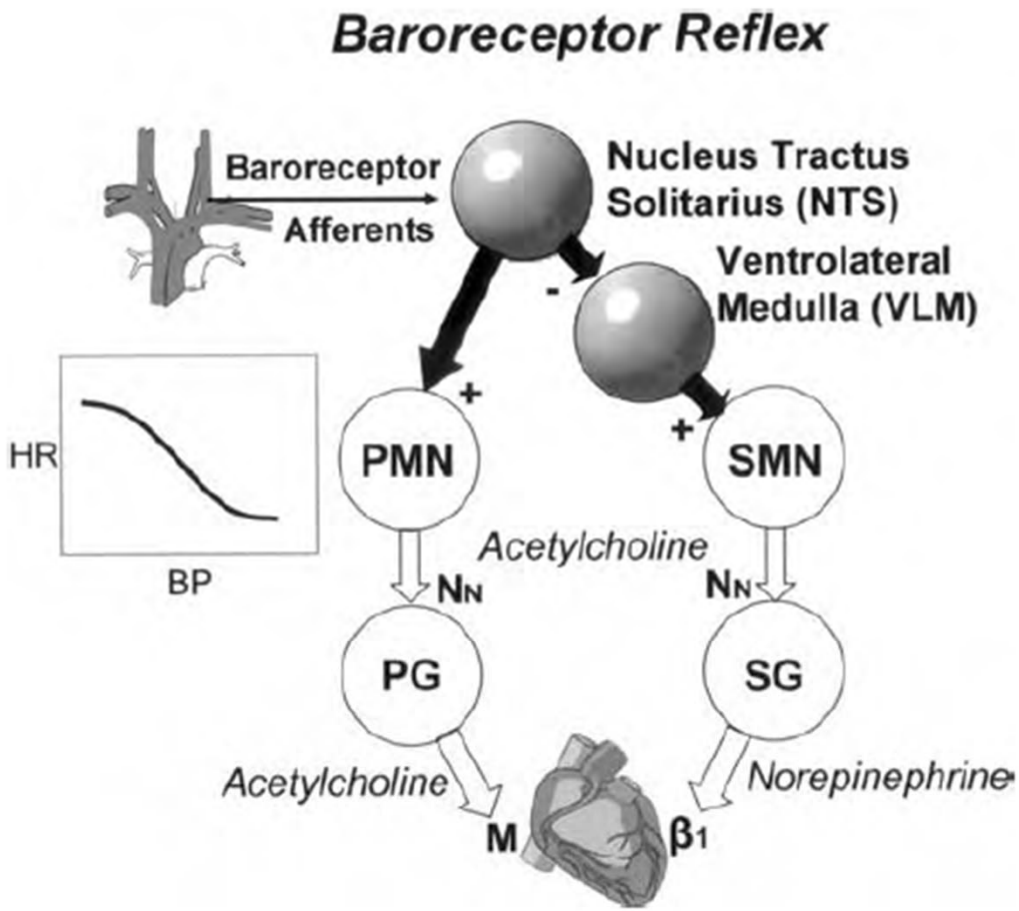
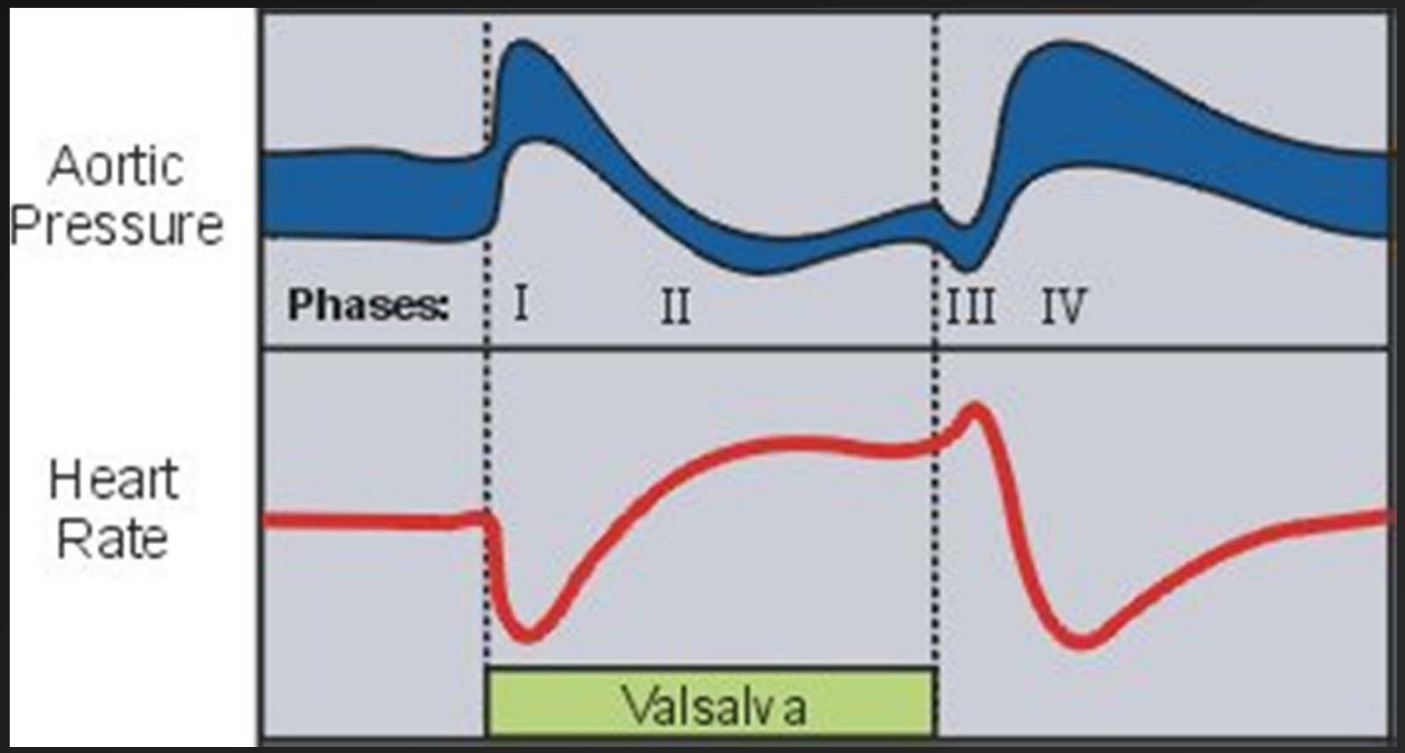


Figure 8.7. General organization of the baroreceptor heart rate reflex. Reflex originates in mechanoreceptors in the heart and the carotid and other great arteries. The NTS excites (+ symbol) the parasympathetic motor neurons (PMN) and inhibits (– symbol) relay neurons to the sympathetic motor neuron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

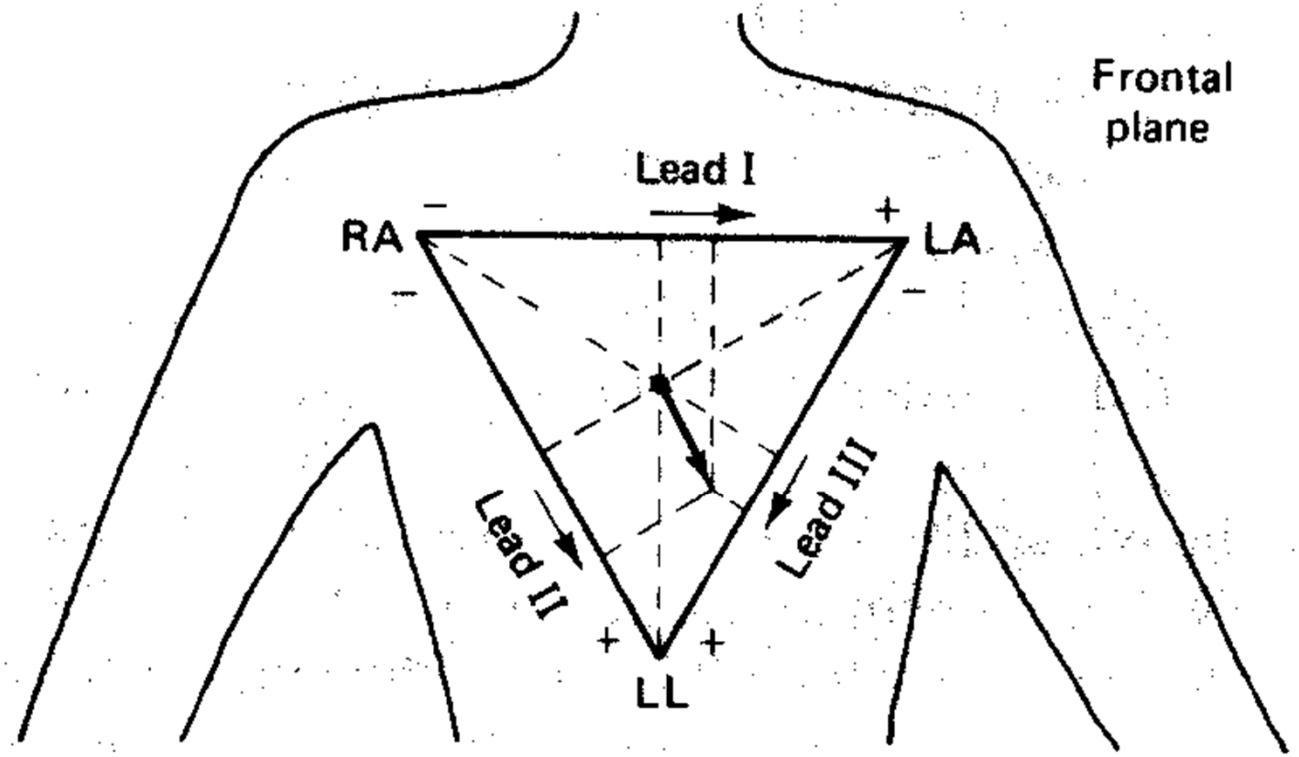
- Relatively easy to measure PNS: RSA or other metrics of HRV
- Relatively easy to measure SNS: Contractility via PEP
- BUT… one is measure of chronotropy, other is measure of inotropy
  - Changes in contractility can occur independently of changes in rate
  - SNS inputs for inotropy primarily controlled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

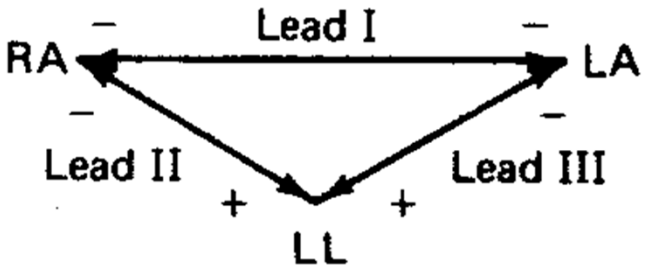


Bipolar

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

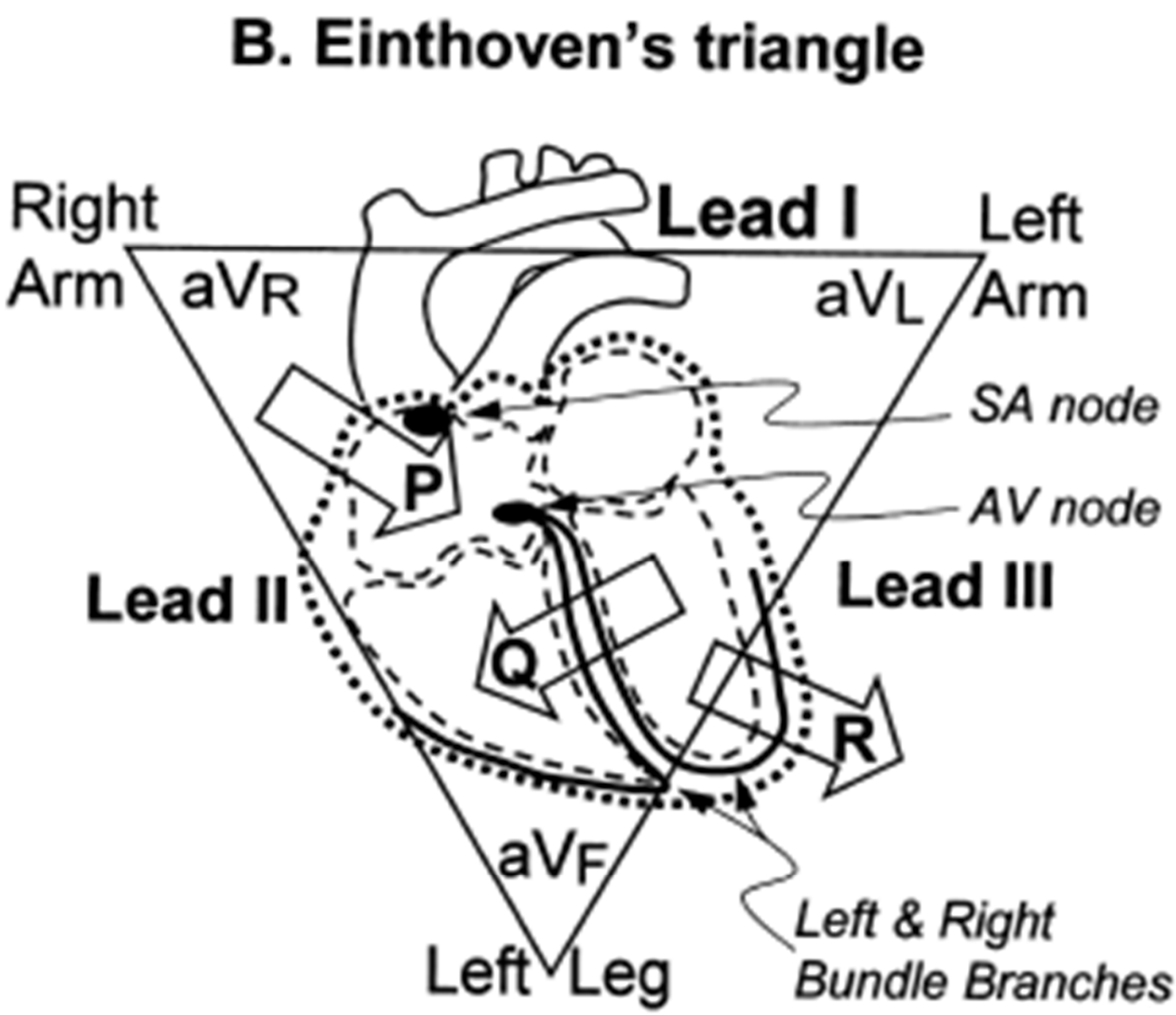
Lead I:	between RA and LA
Lead II:	between RA and LL
Lead III:	between LA and LL

Einthoven triangle, showing relation of the bipolar limb leads



EKG

AC signal
Sample 200-500 Hz



EKG Demo

Which Time?

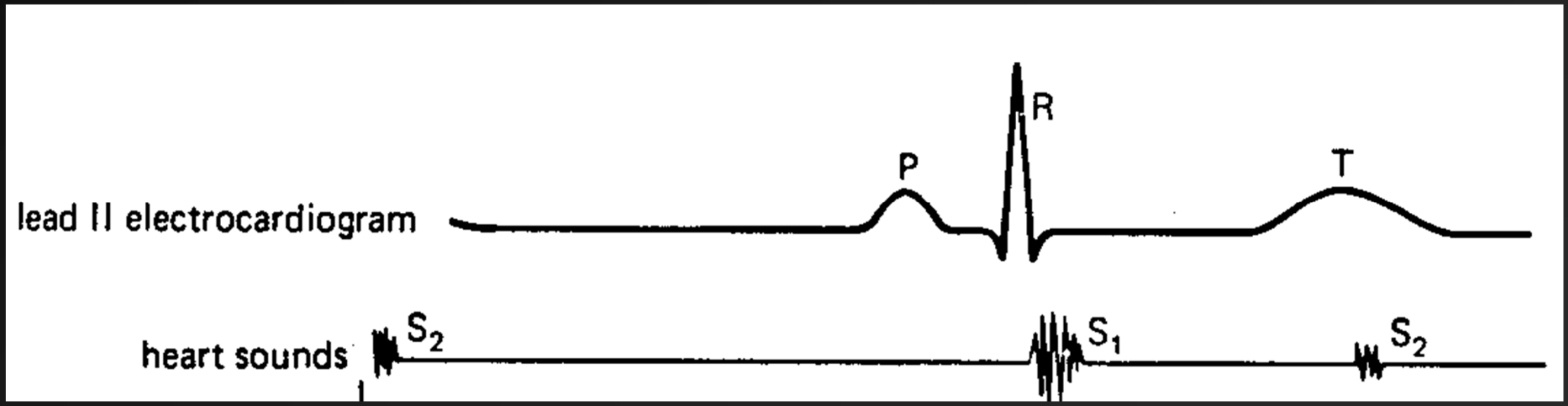
- Real time
  - Heart Rate
  - Expressed as beats per time (usually bpm)
- Cardiac time
  - Heart Period; interbeat interval (IBI)
  - Expressed in msec
- Converting

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

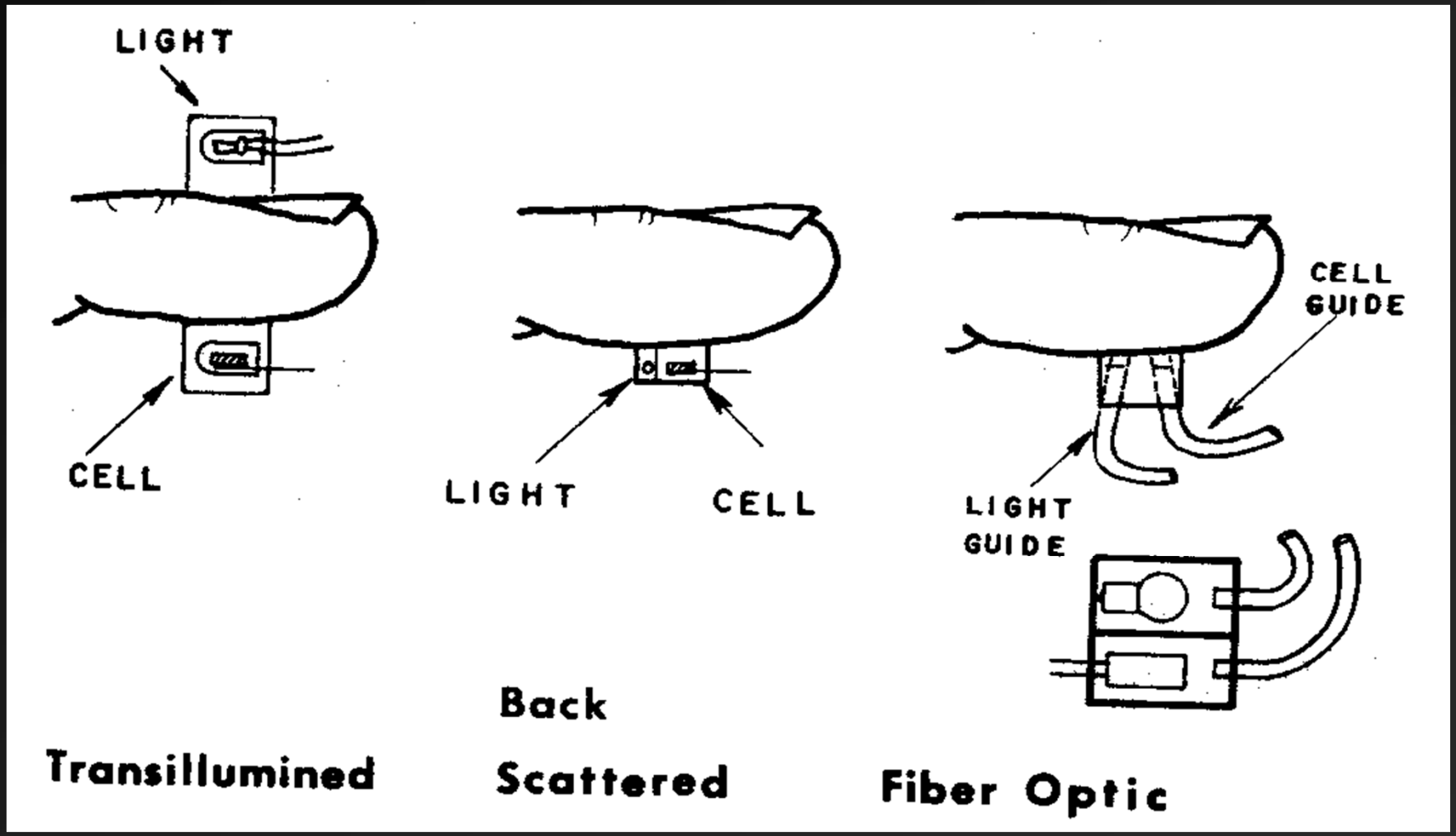
$$HR = \frac{1}{1000} X\, 60{,}000 = 60 bpm$$

Phonocardiography

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

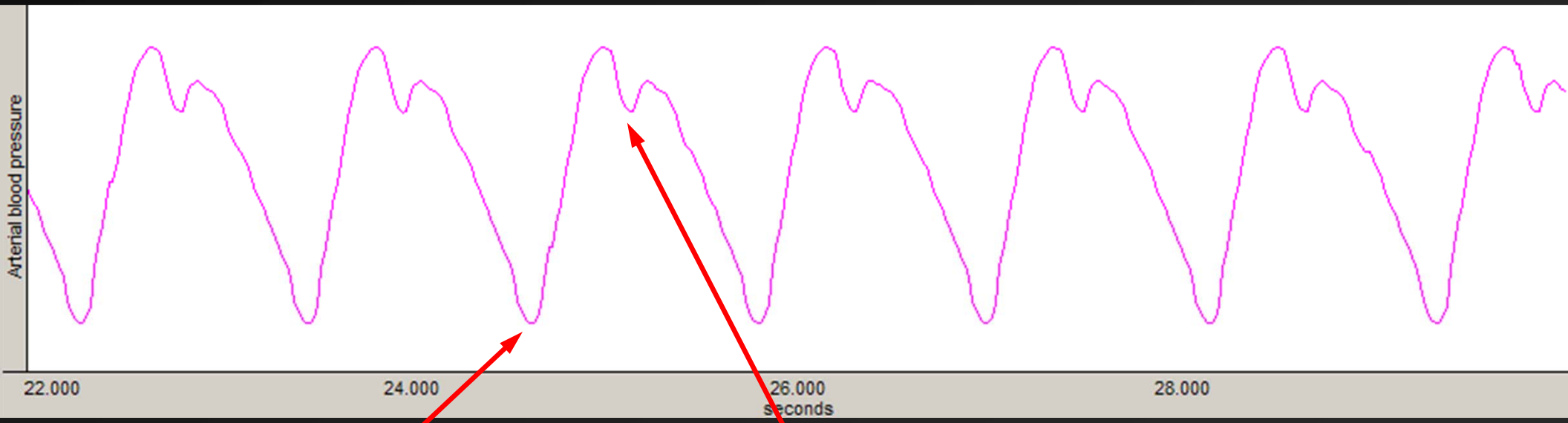


Photoplethysmography



Three methods, all involve measuring light absorbed by peripheral vasulature

The Photoplethysmographic Output

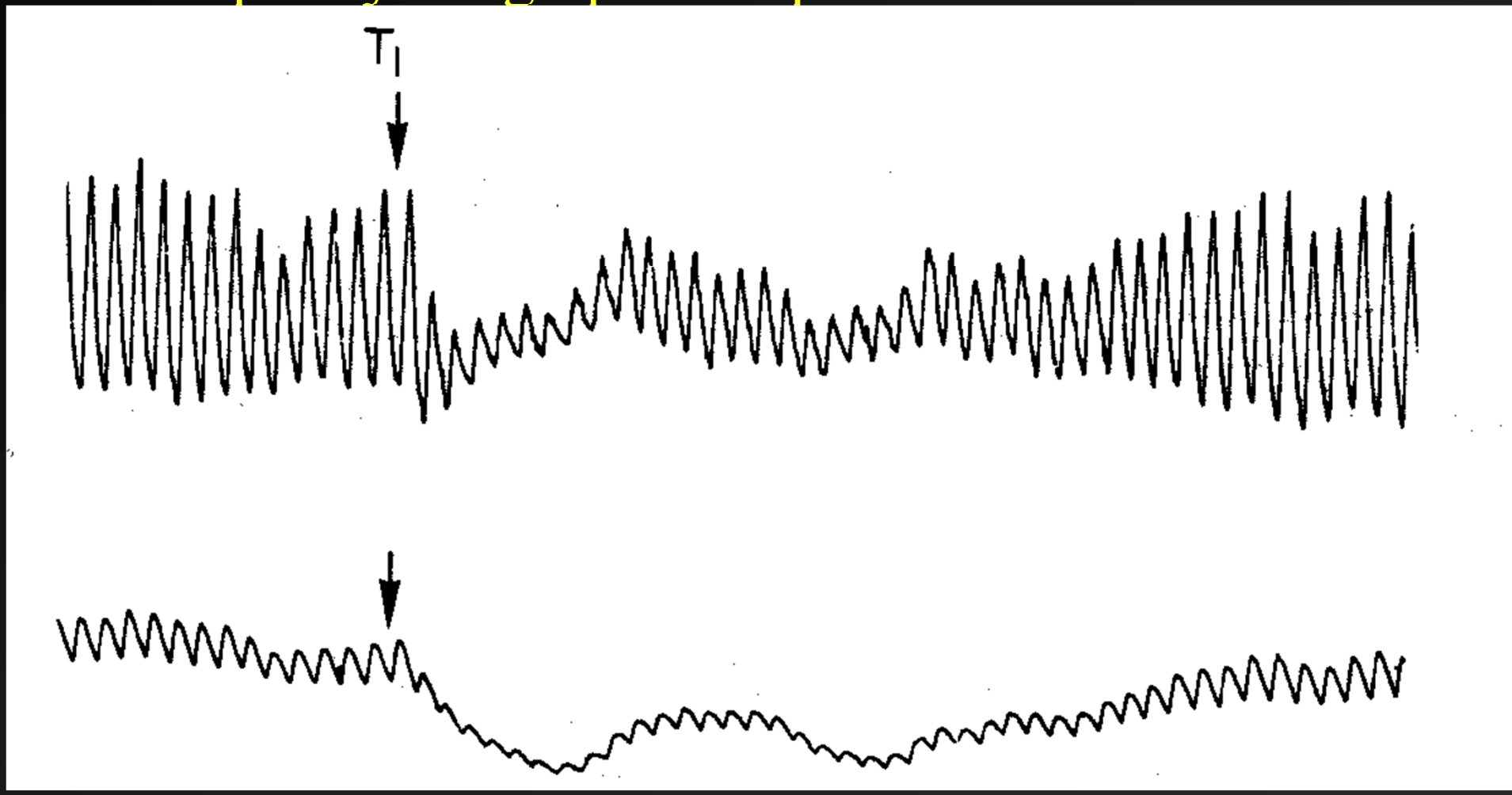


~LVET

Increase in Pressure due to opening of Aortic Valve

Dichrotic Notch; closing of valve, end of ejection

Photoplethysmograph: Peripheral Vasoconstriction

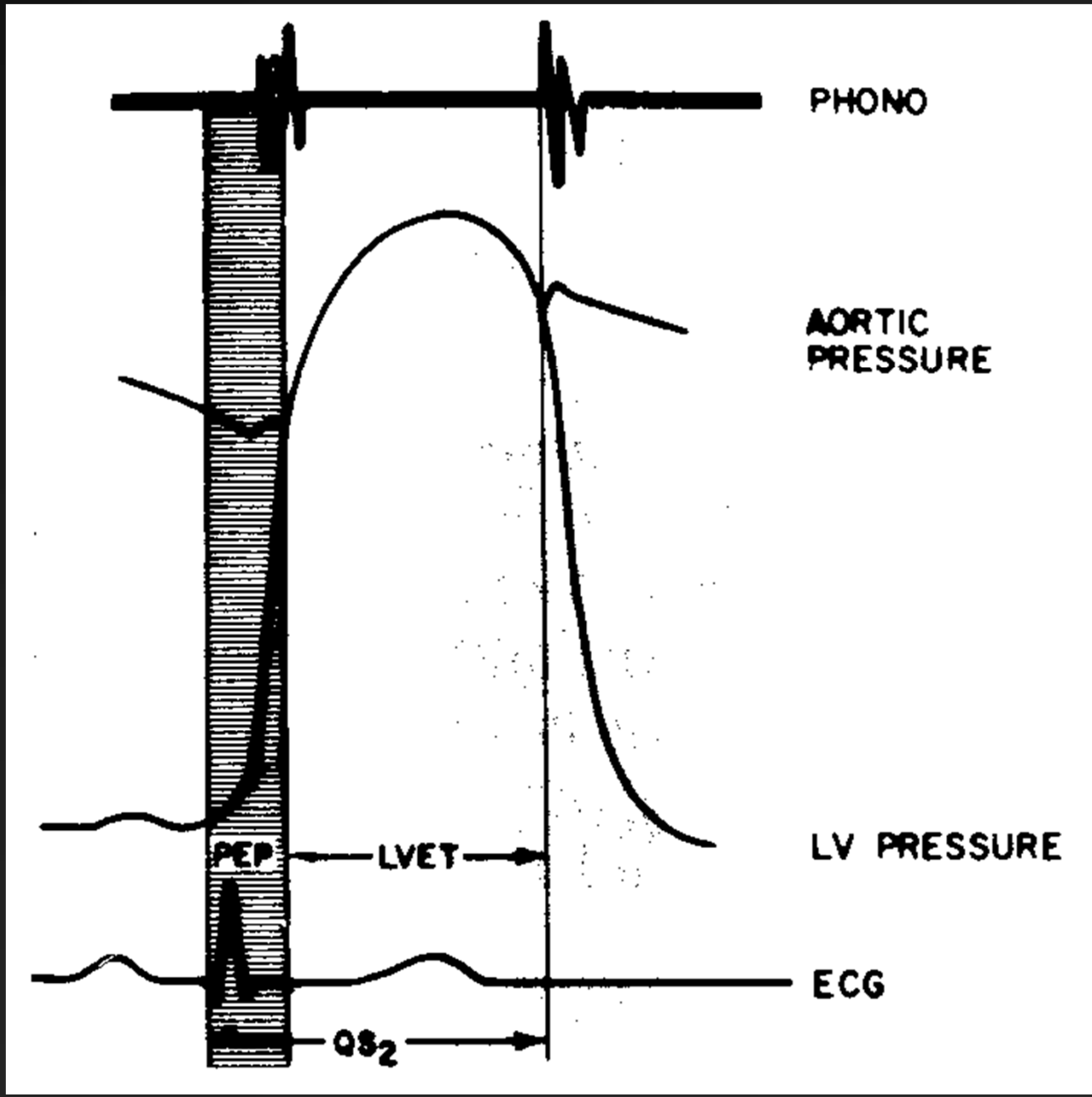


T1 is onset of constriction
Top Panel: Pulse Volume (recorded with 1 sec time constant)
Lower Panel: Blood Volume (no filter)

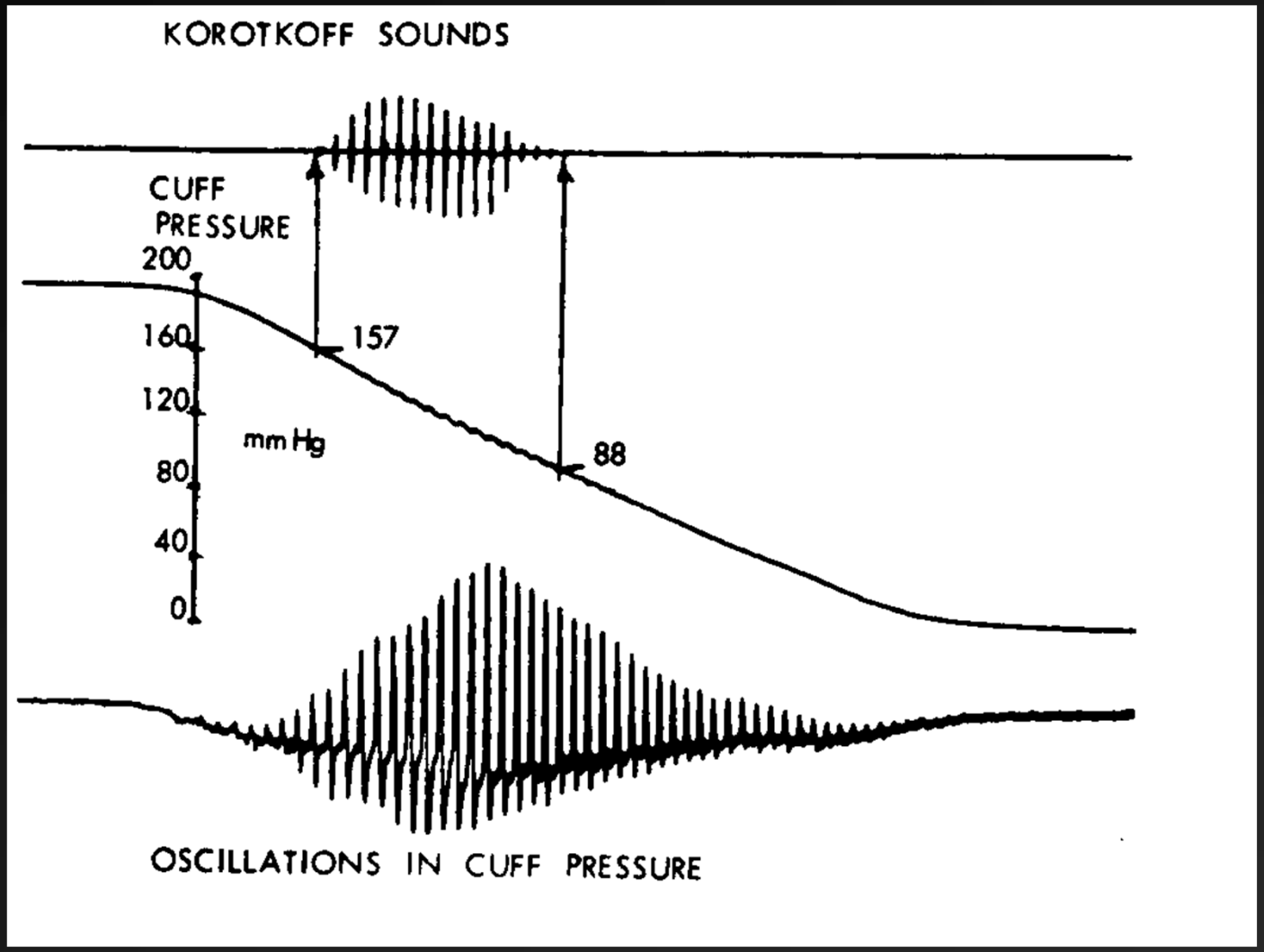
Measuring contractility with EKG, PCG, and Photoplethysmography

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

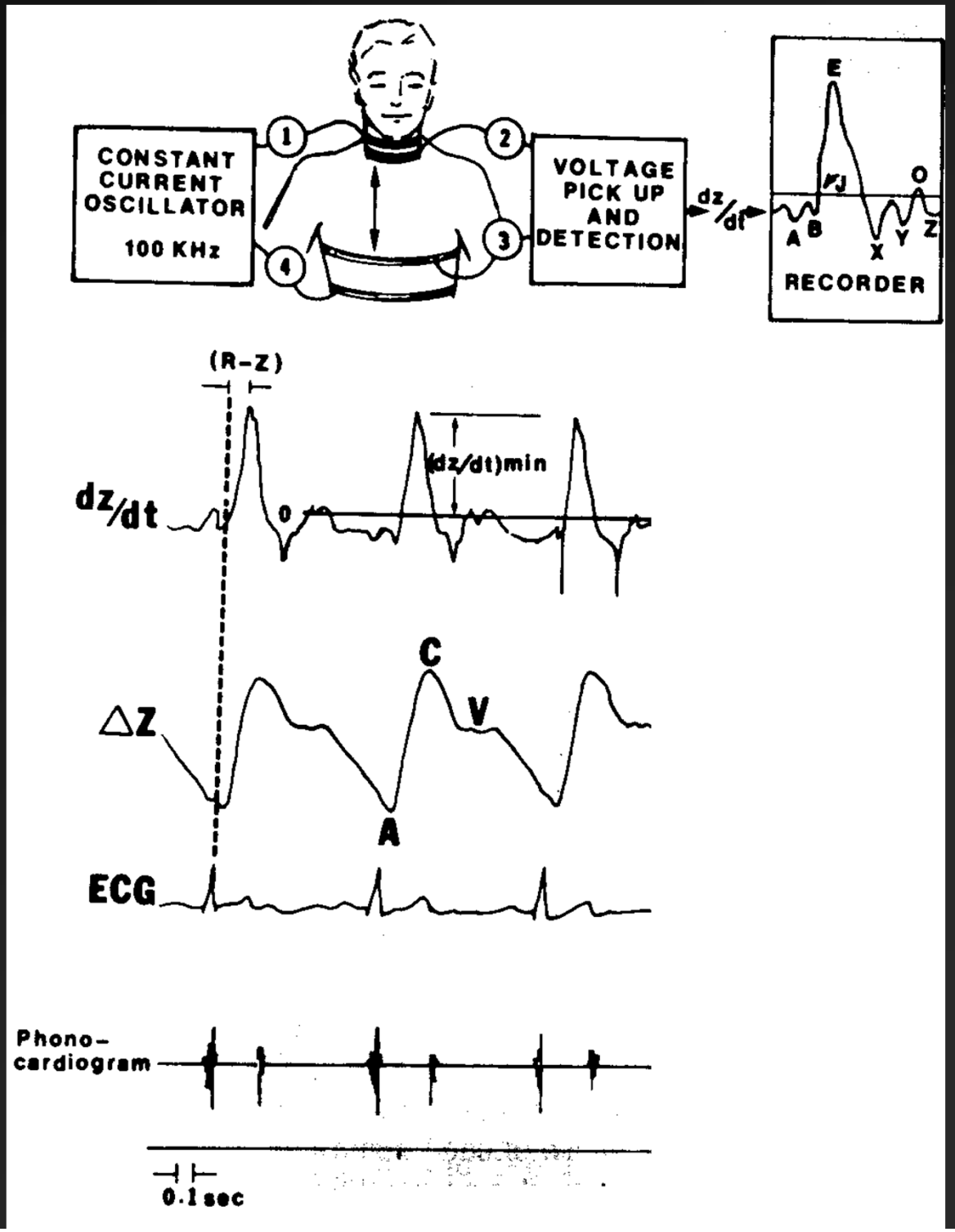
- Not good for instantaneous readings
- Not good for repeated readings

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
- 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

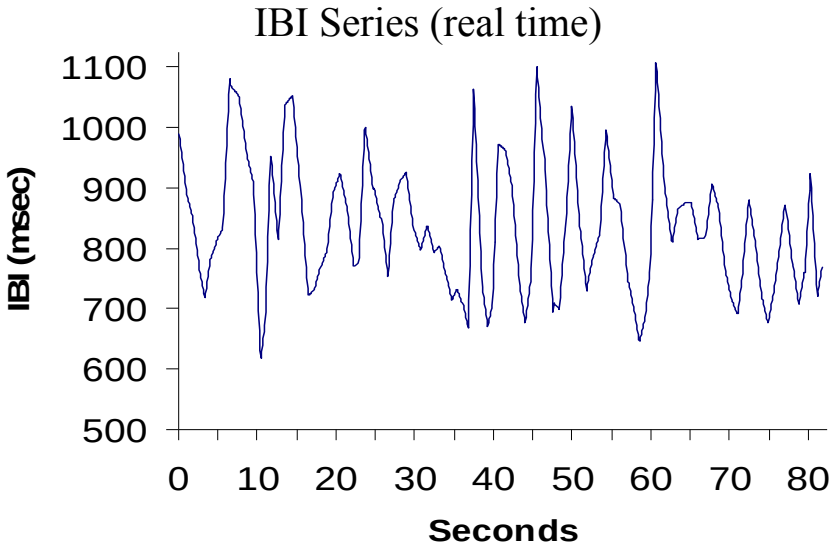


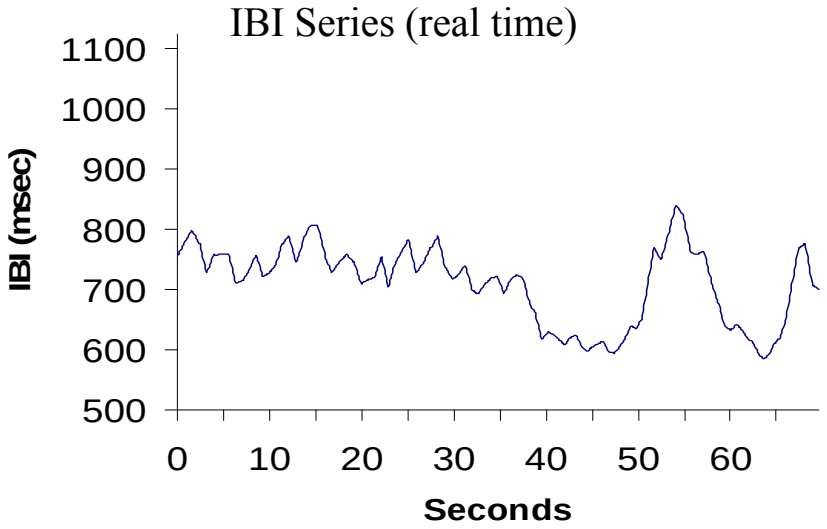
Measuring Vagal Influence

- 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


IBI Series (real time)
IBI (msec)
1100
1000
900
800
700
600
500
0
10
20
30
40
50
60
70
80
Seconds


IBI Series (real time)
IBI (msec)
1100
1000
900
800
700
600
500
0
10
20
30
40
50
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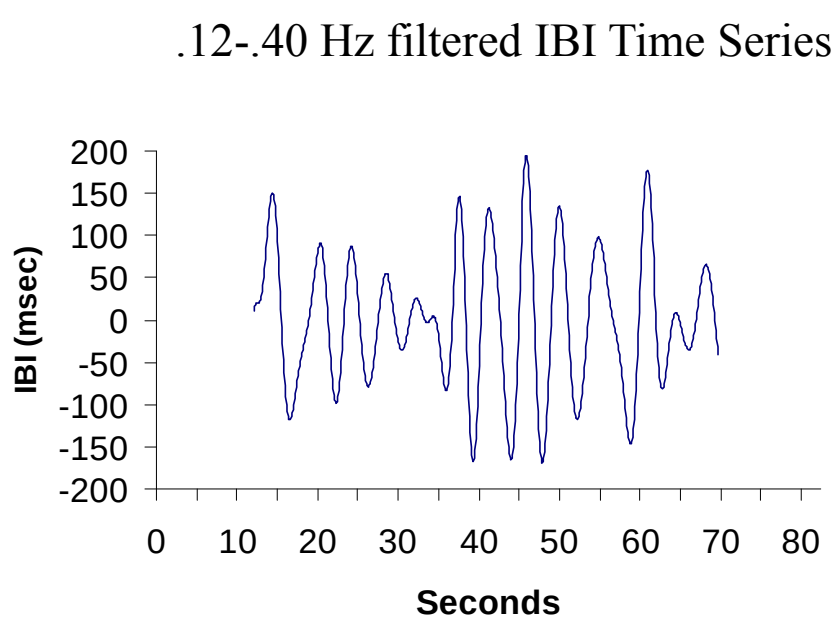
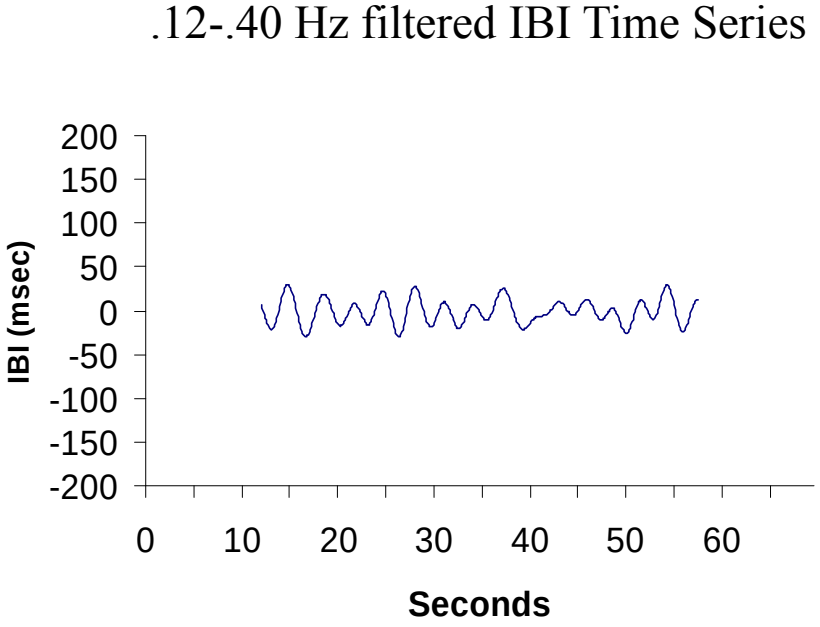
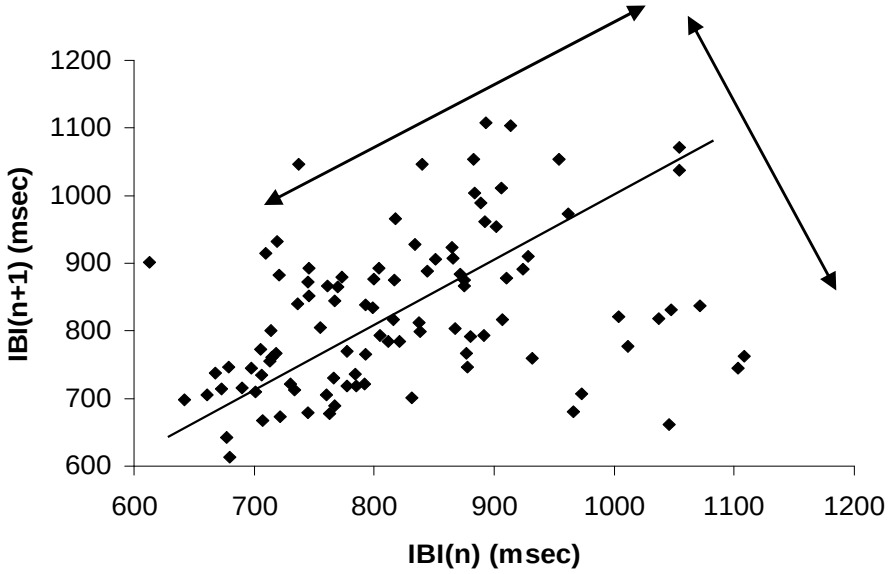
Seconds

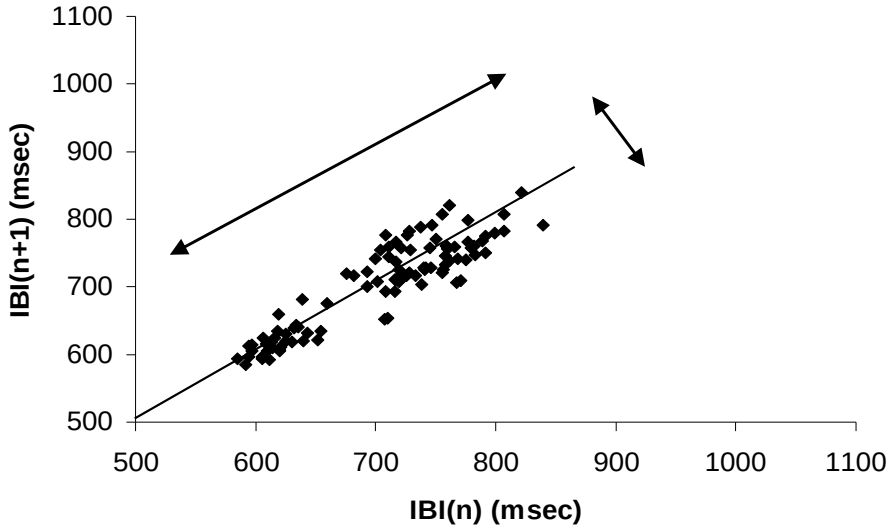

.12-.40 Hz filtered IBI Time Series
IBI (msec)
200
150
100
50
0
-50
-100
-150
-200
0
10
20
30
40
50
60
70
80
Seconds


.12-.40 Hz filtered IBI Time Series
IBI (msec)
200
150
100
50
0
-50
-100
-150
-200
0
10
20
30
40
50
60
Seconds

High Variability Subject



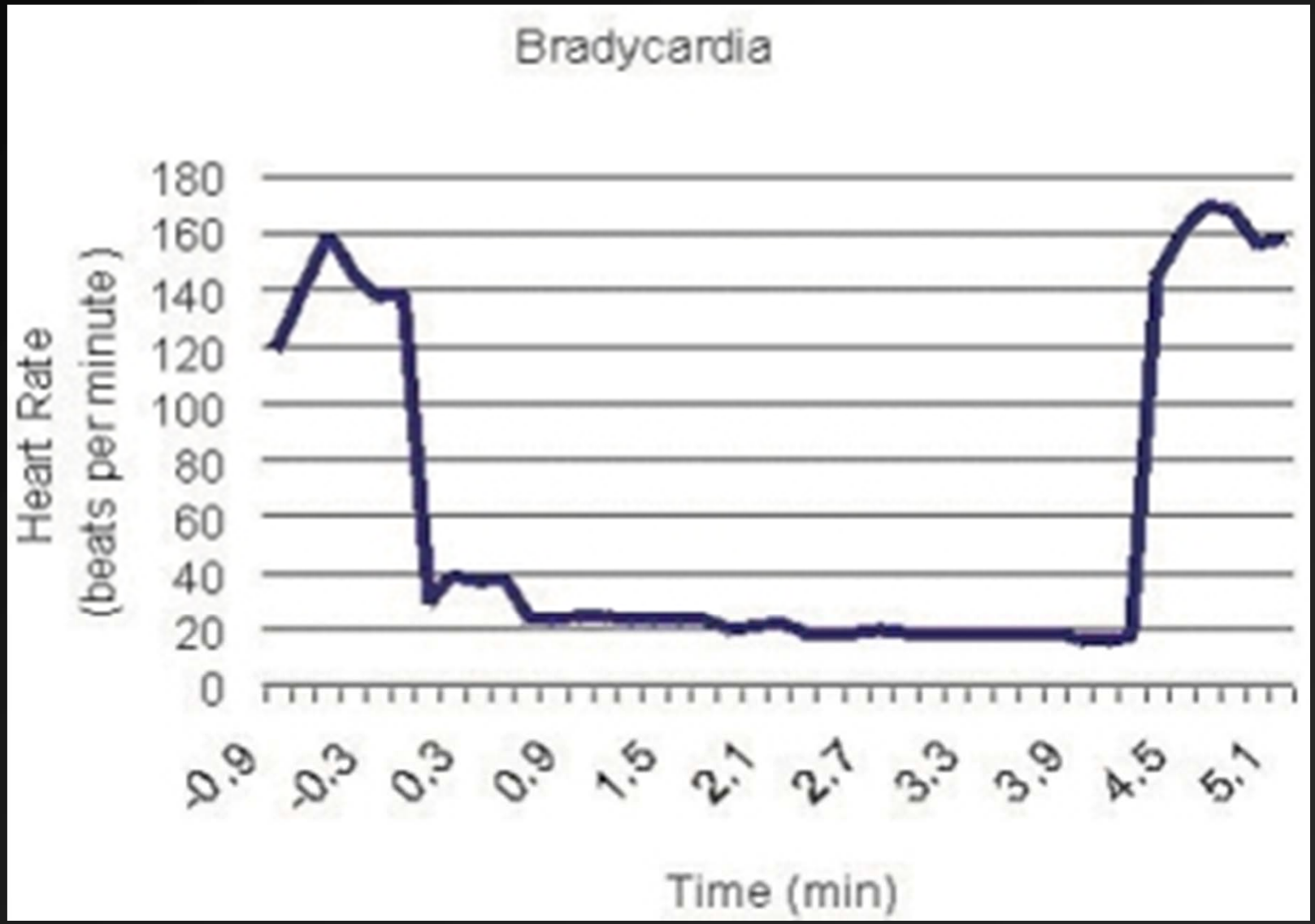
Low Variability Subject



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
  - Phylogentically newer "smart" Vagus
    - Orginates from Nucleus Ambiguous
    - Modualtes influence to:
      - Promote attentional engagement, emotional expression, and communication.
    - Mobilizes organism to respond to environmental demands
      - Phasicly 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**

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

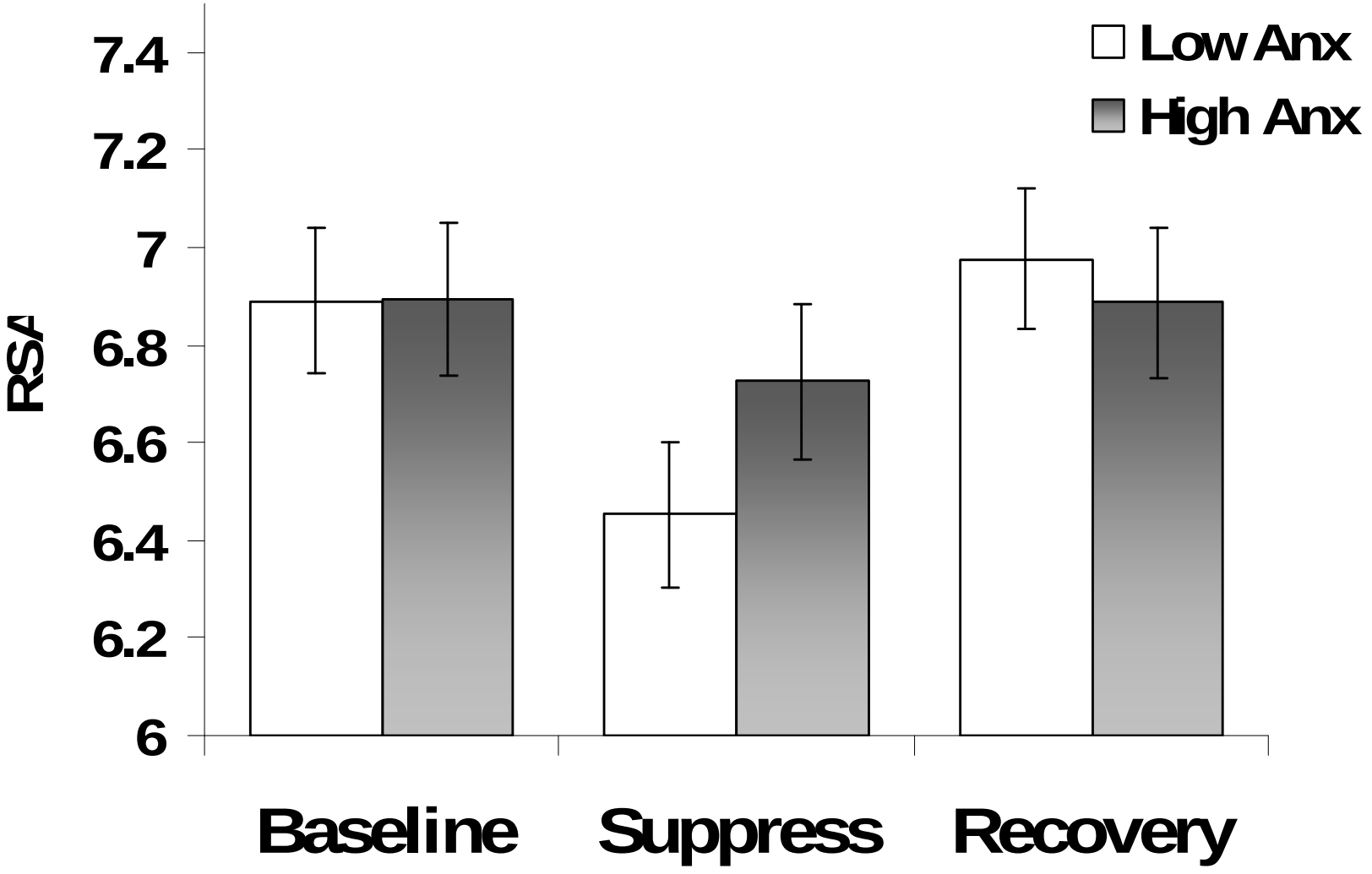
Fig. 1. Phylogenetic stages of the polyvagal theory.

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
  - 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



Movius & Allen, 2005

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)

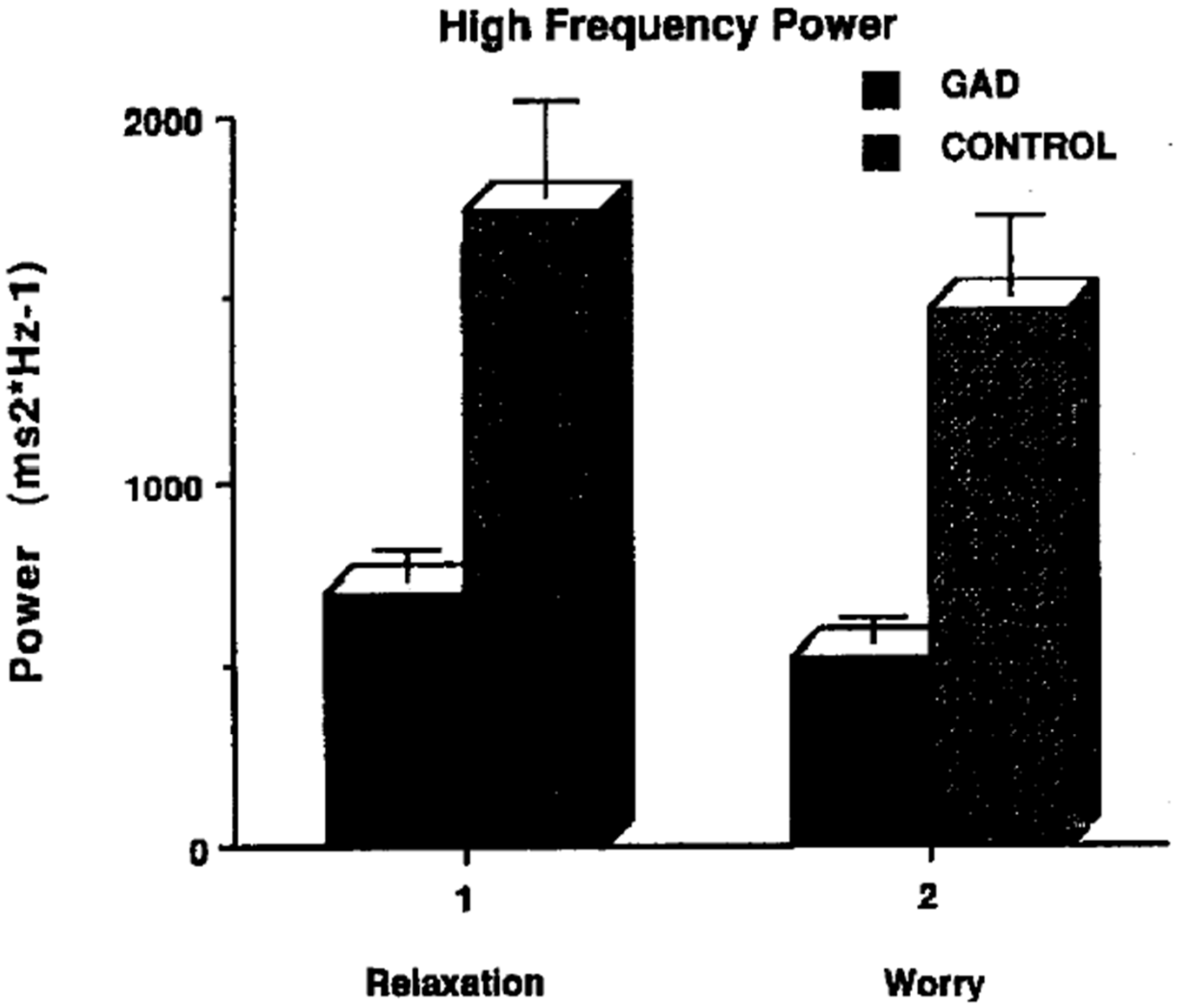
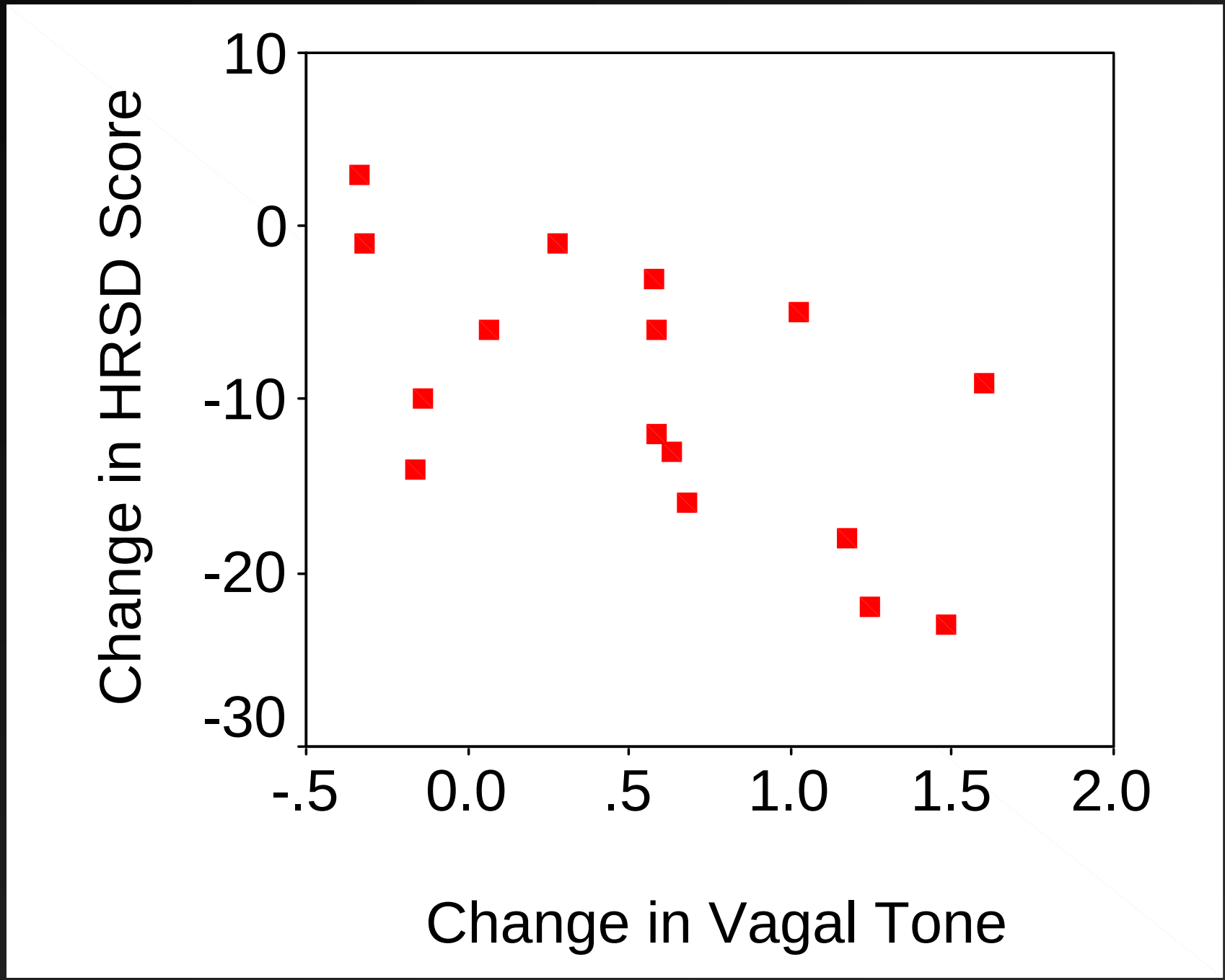


Figure 1. Power in the high frequency (respiratory) component of heart period variability in GAD patients and controls during relaxation and worry.

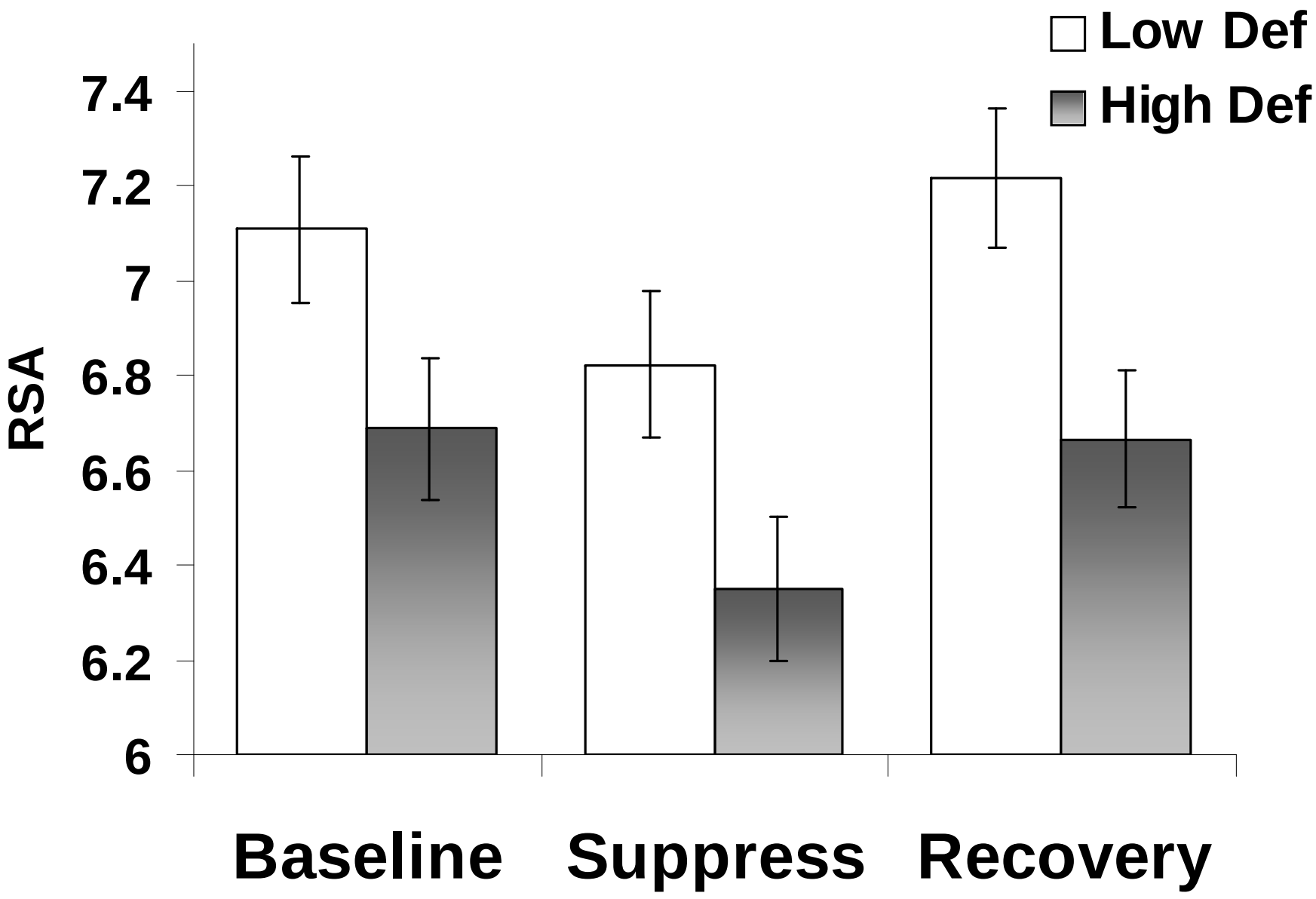
Table 1
Significant contrasts among panickers, blood phobics, and controls

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

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



Chambers and Allen (2002) *Psychophysiology*

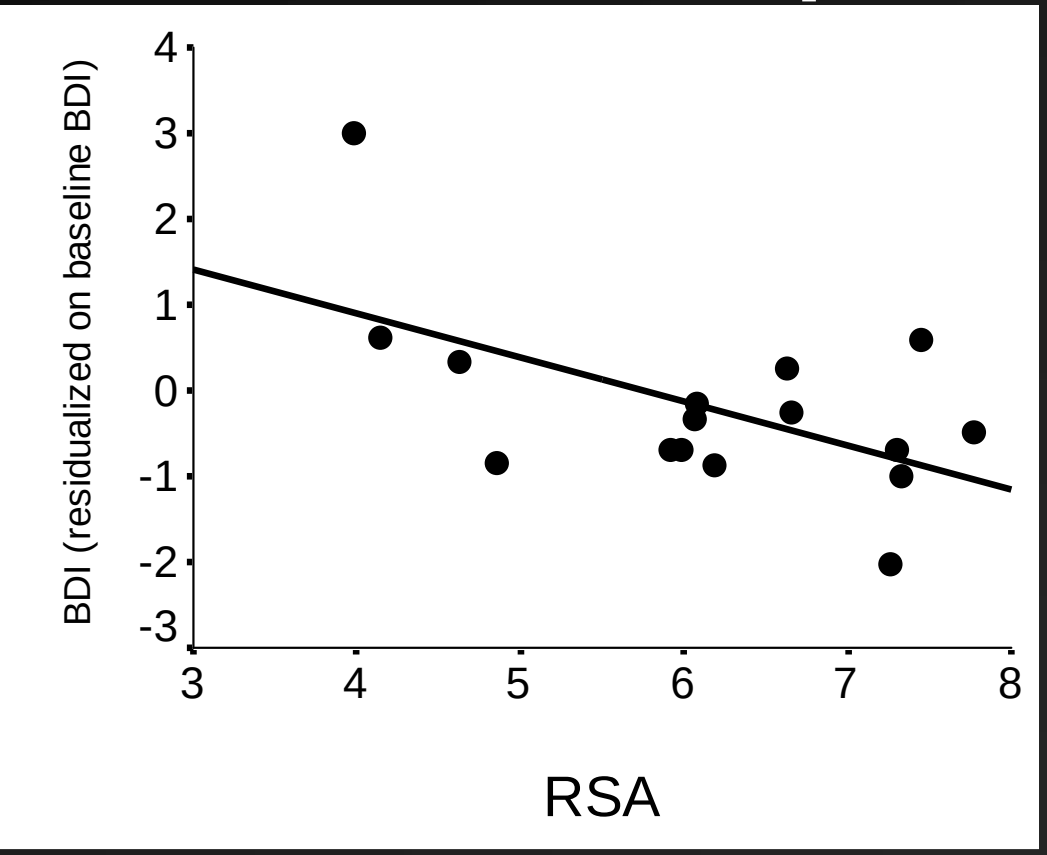


Movius & Allen, 2005

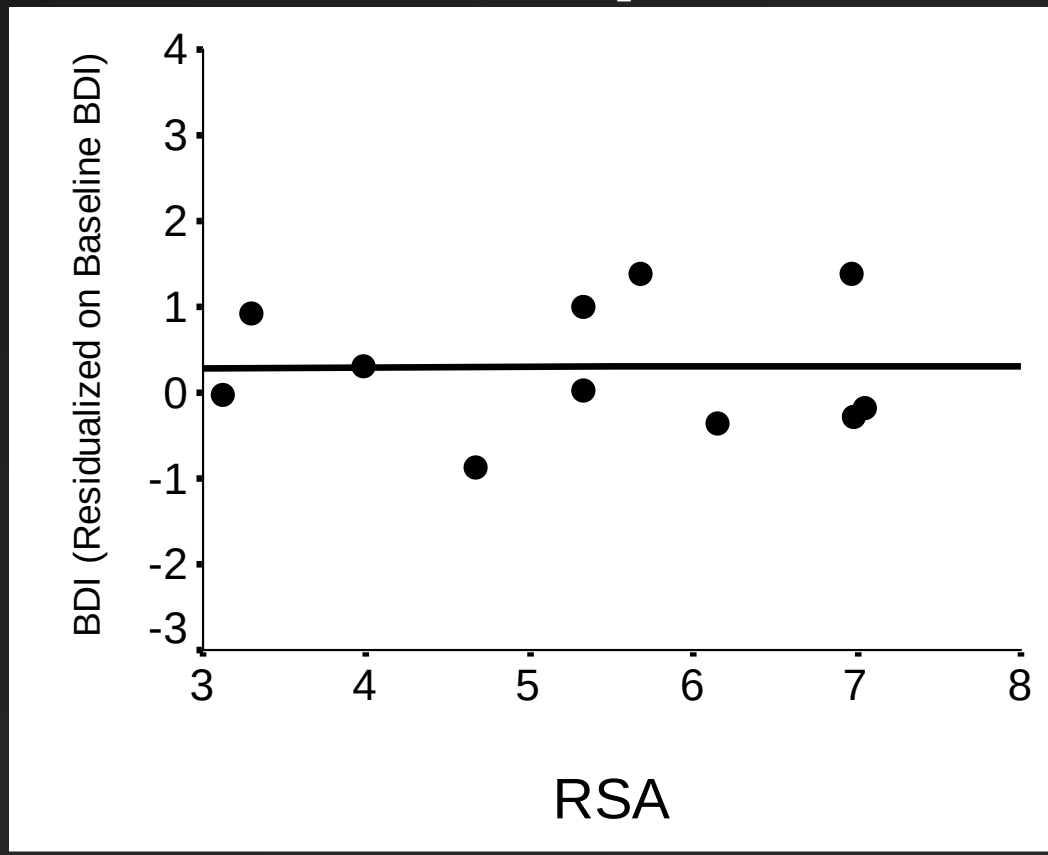
Trait Vagal Tone as Moderator of Response following Bereavement

- 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



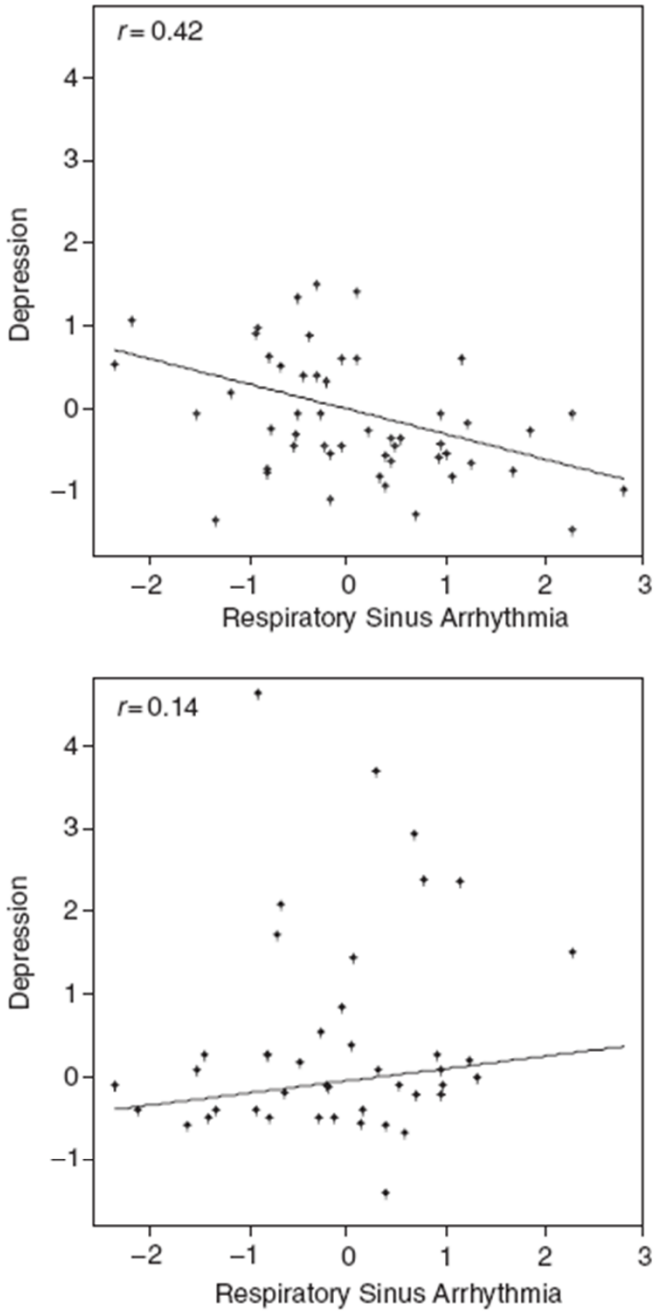


Figure 1. Scatterplot, prediction line, and prediction equation for the relationship between respiratory sinus arrhythmia (log of the variance of the band-limited [.12–.40 Hz] IBI series) and depression score (residualized on baseline depression score), for the disclosure group (top panel) and the control group (bottom panel). Negative depression score represents improvement from baseline to follow-up.

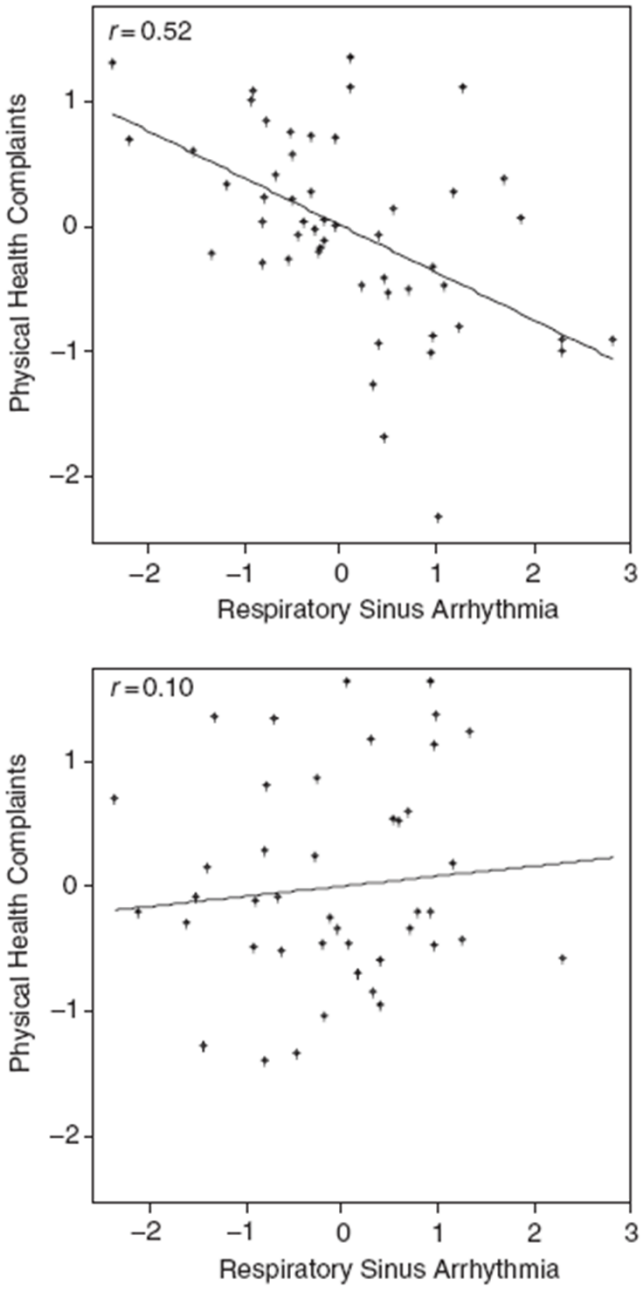
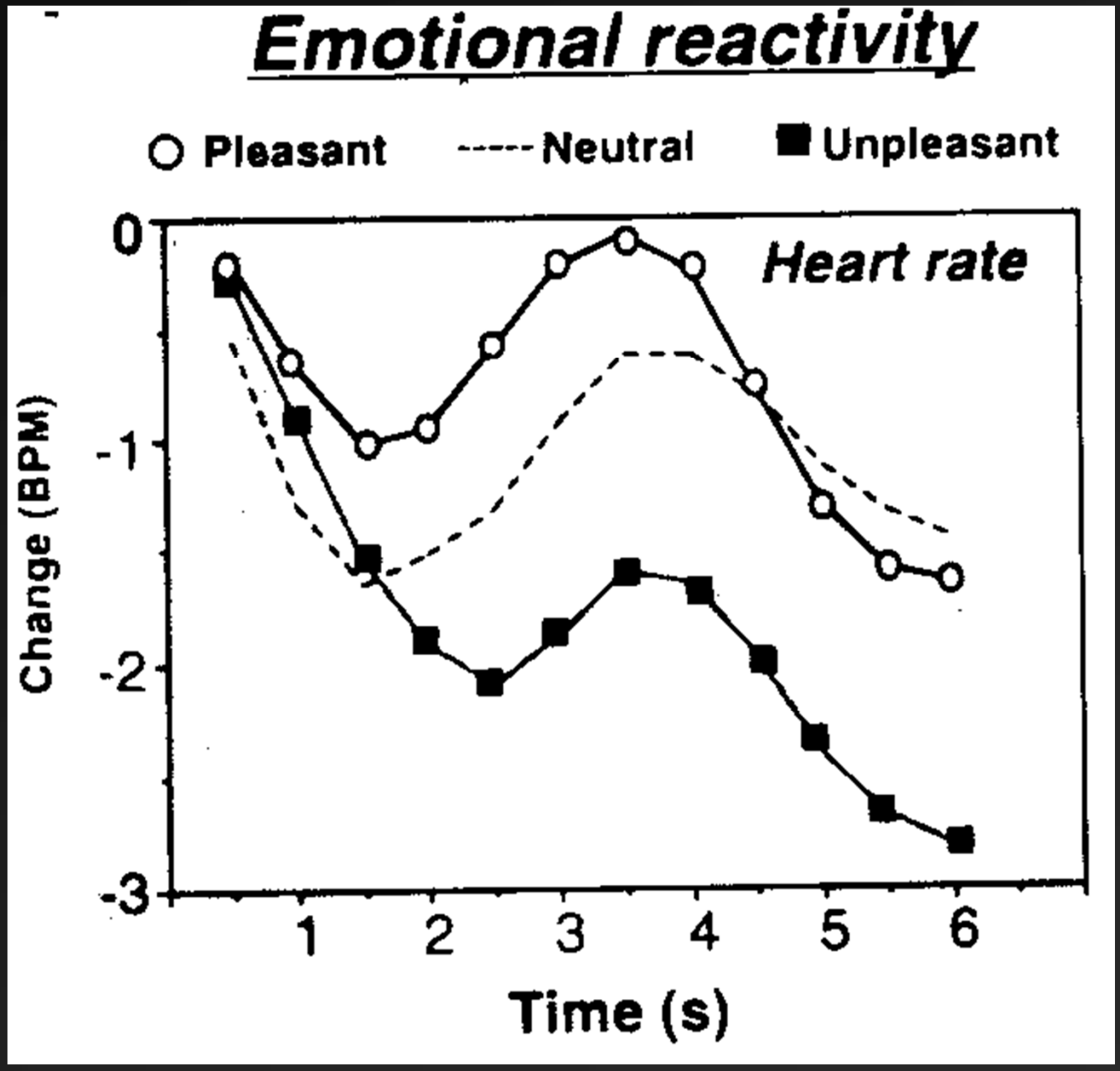
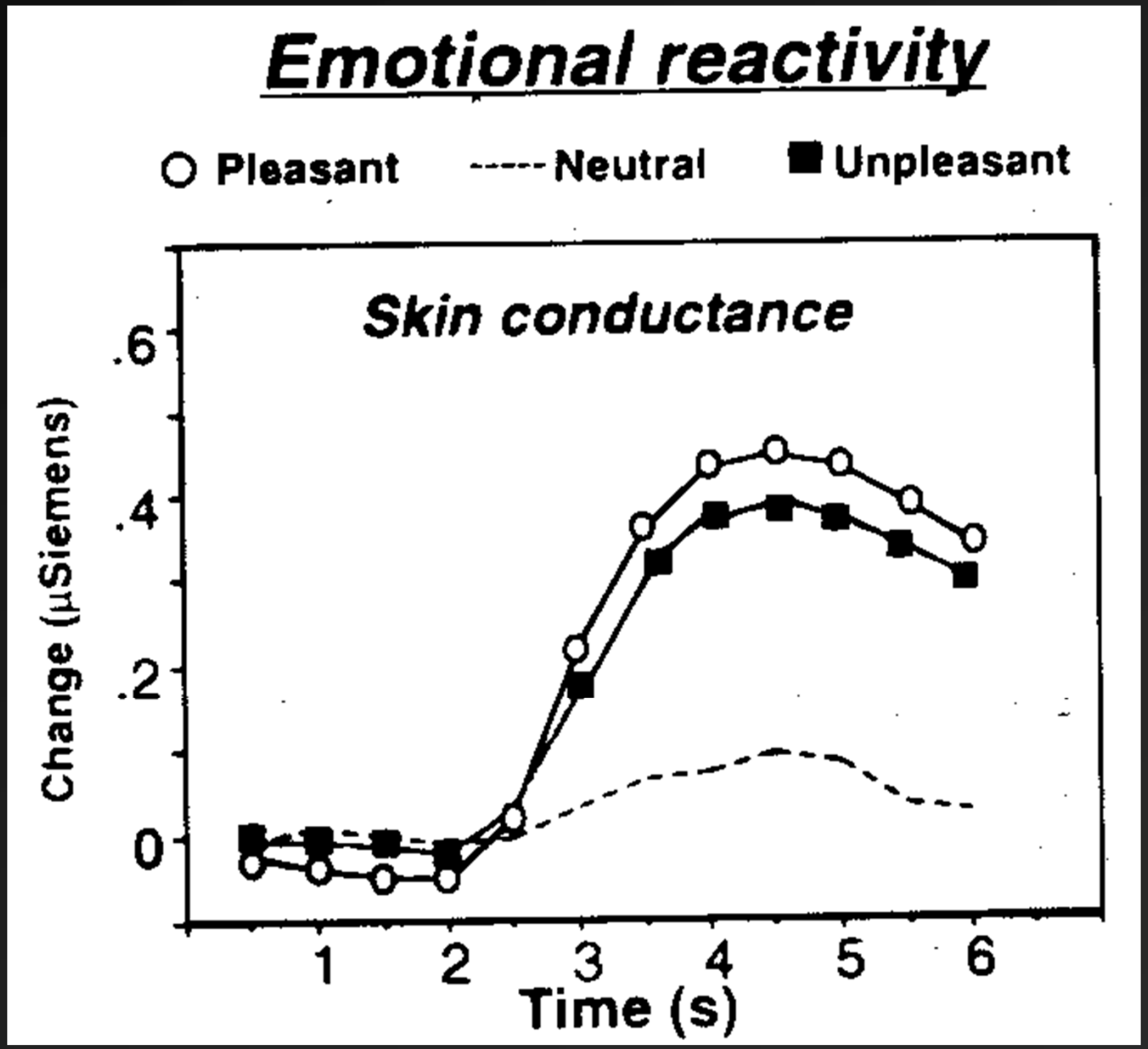


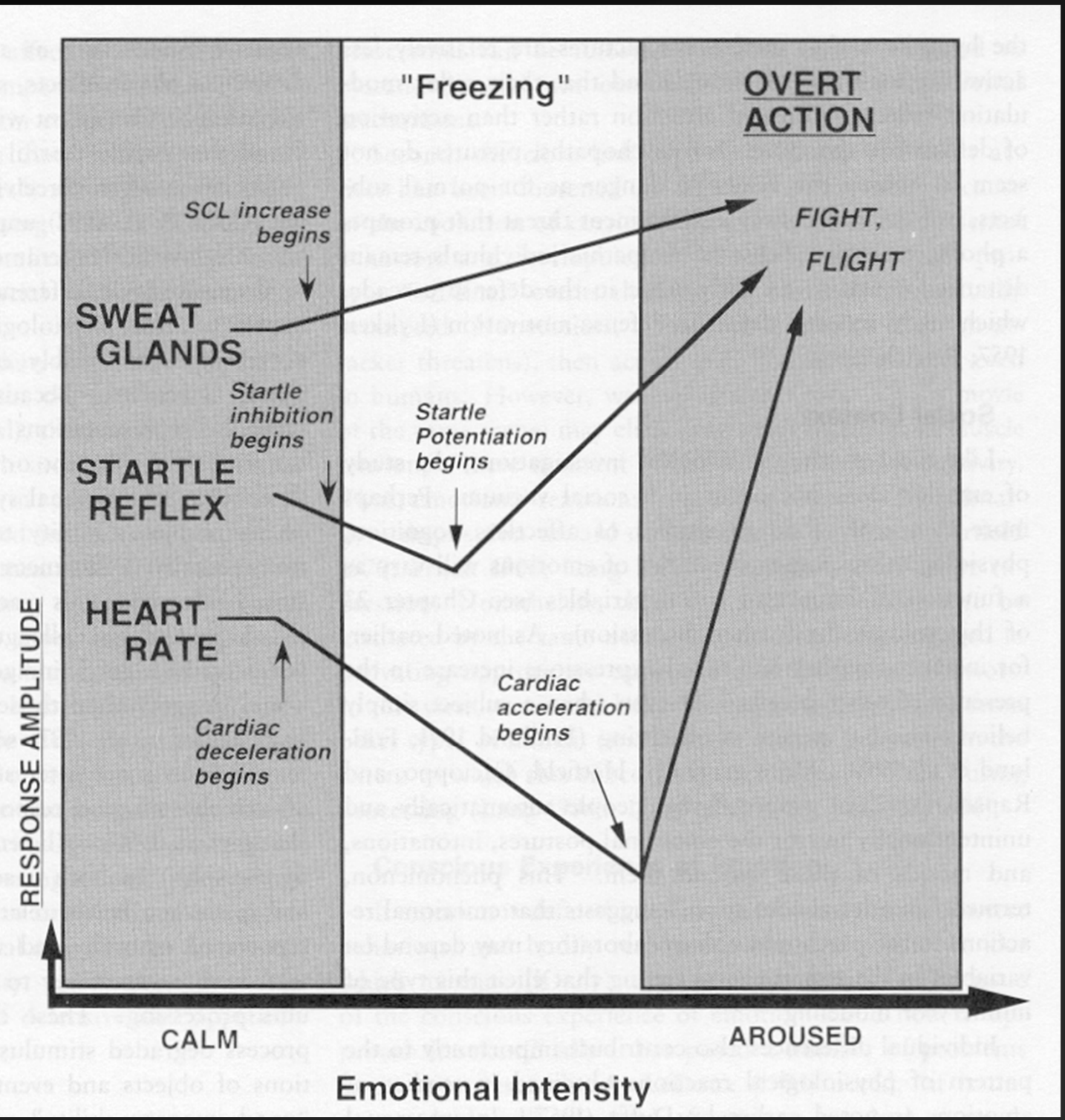
Figure 2. Scatterplot, prediction line, and prediction equation for the relationship between respiratory sinus arrhythmia (log of the variance of the band-limited [.12–.40 Hz] IBI series) and physical health complaint score (residualized on baseline physical health complaints score) for the disclosure group (top panel) and the control group (bottom panel). Negative physical health complaint score represents improvement from baseline to follow-up.

Orienting, Attention, and Defense



SCR (by contrast)




"Freezing"
OVERT ACTION
SCL increase begins
FIGHT, FLIGHT
SWEAT GLANDS
Startle inhibition begins
Startle Potentiation begins
STARTLE REFLEX
HEART RATE
Cardiac deceleration begins
Cardiac acceleration begins
RESPONSE AMPLITUDE
CALM
AROUSED
Emotional Intensity

OR Vs DR