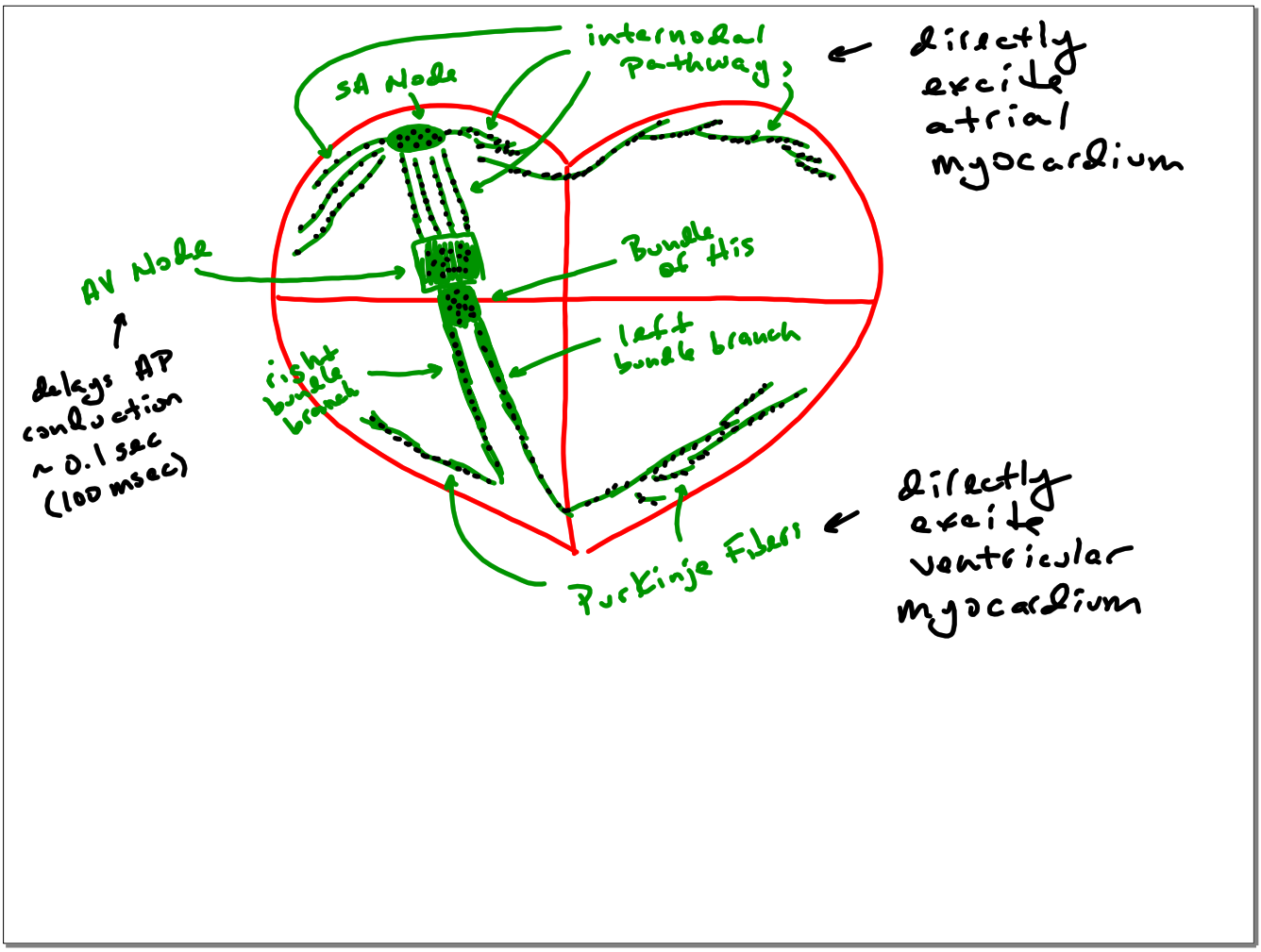
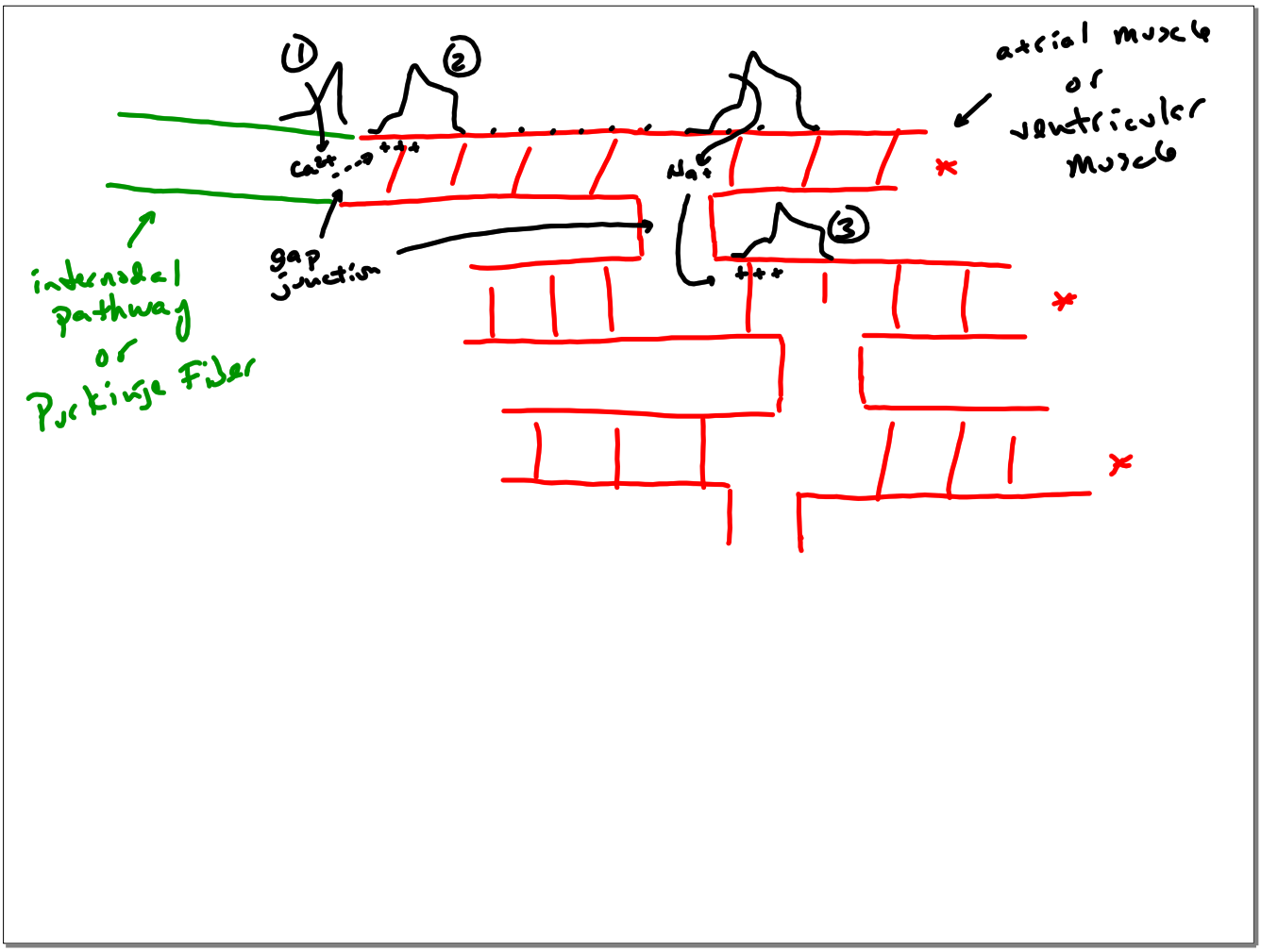
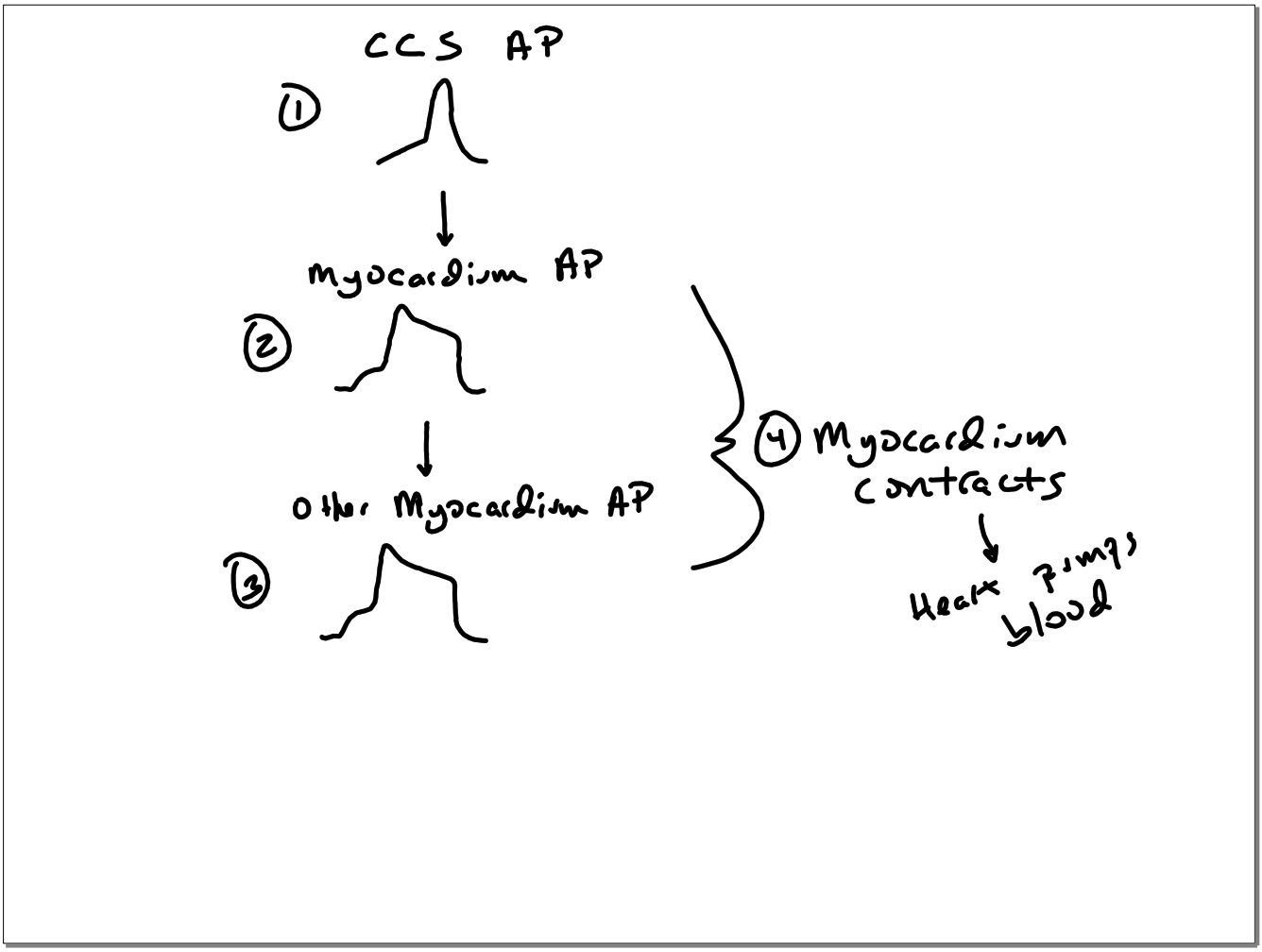

internodal pathways
directly excite atrial myocardium
SA Node
AV Node
delays AP conduction ~ 0.1 sec (100 msec)
Bundle of His
left bundle branch
right bundle branch
Purkinje Fibers
directly excite ventricular myocardium

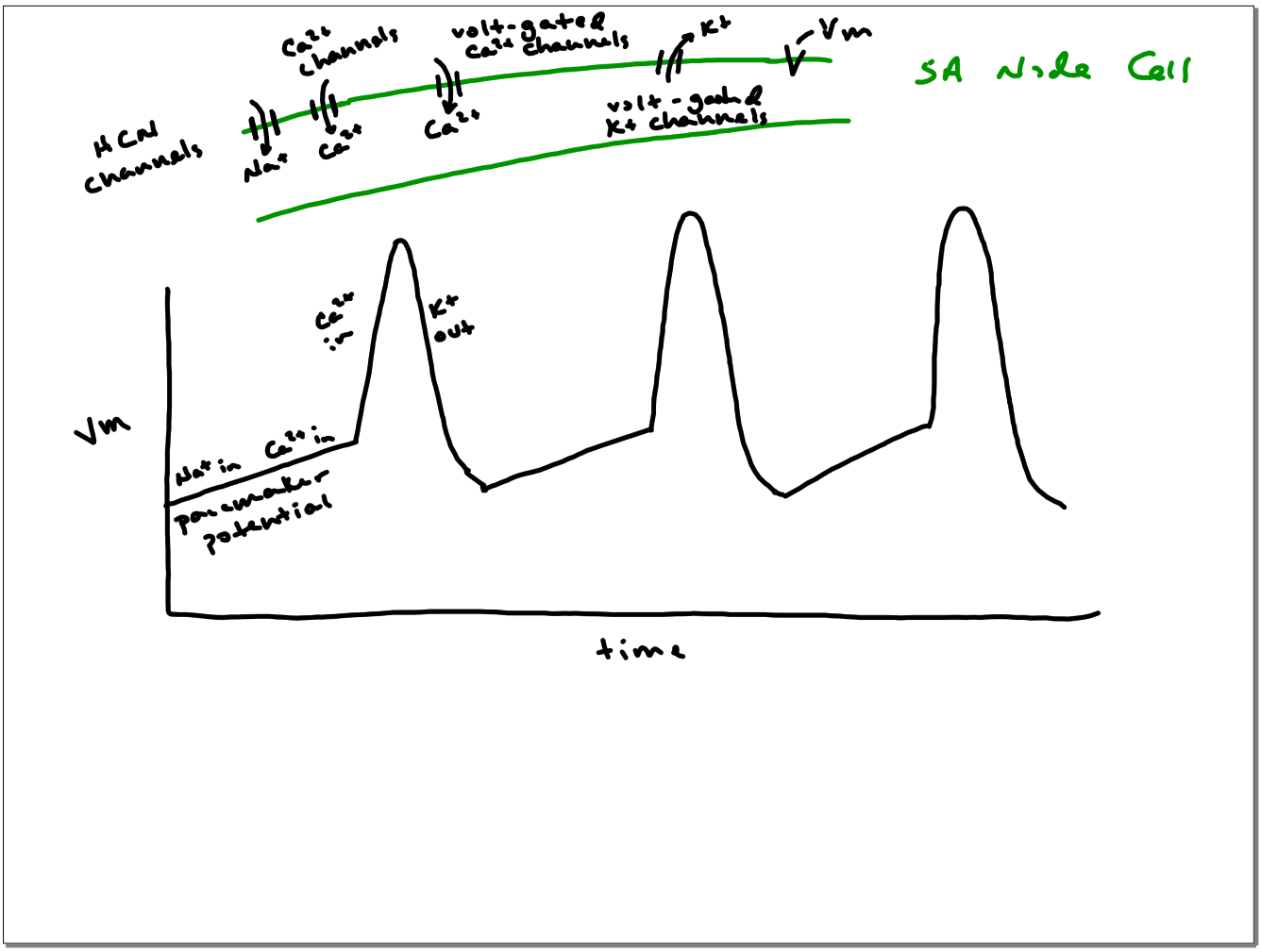
Feb 15-6:09 PM

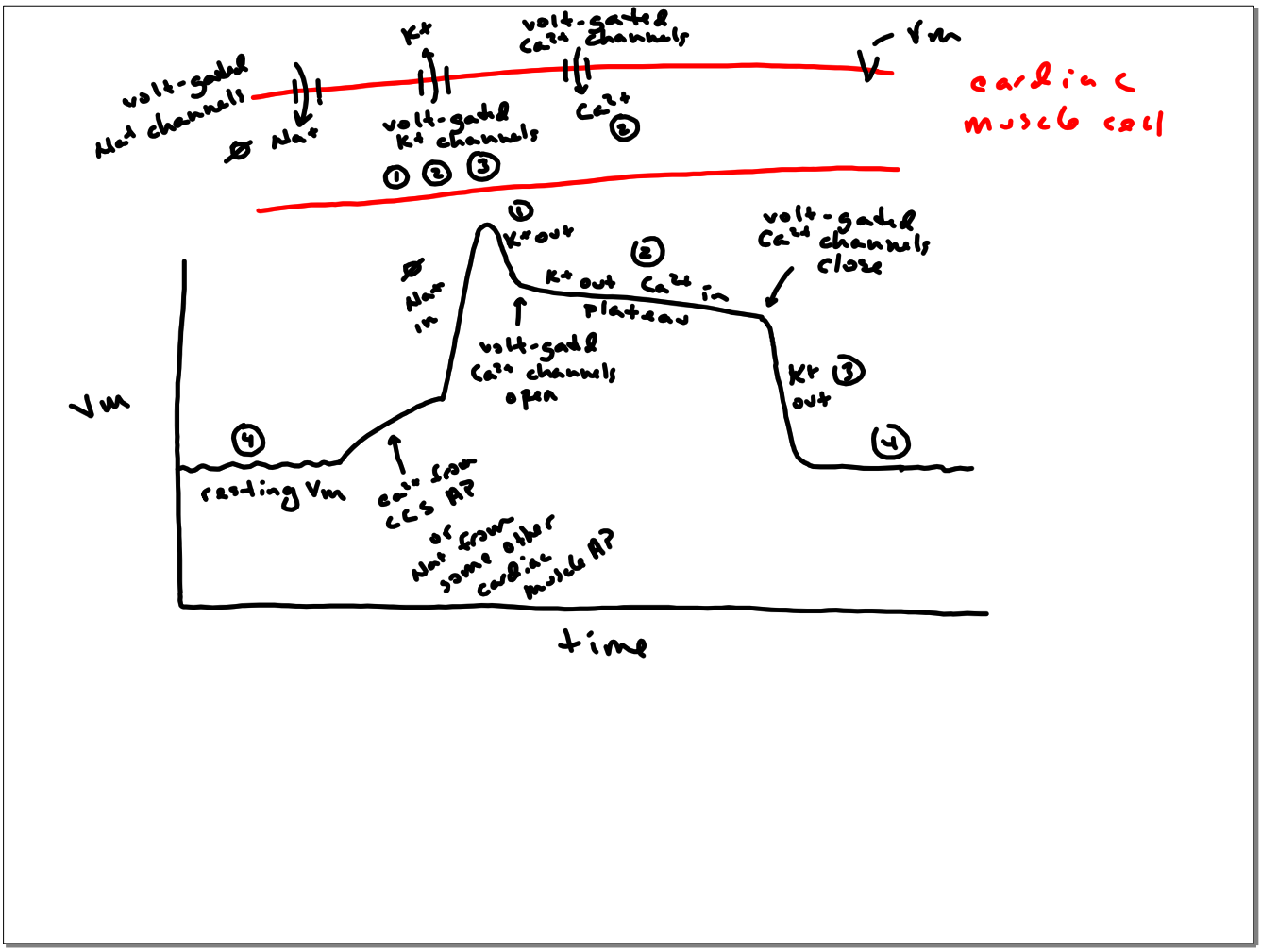

1
2
3
atrial muscle or ventricular muscle
gap junction
internodal pathway or Purkinje Fiber

Feb 15-6:10 PM

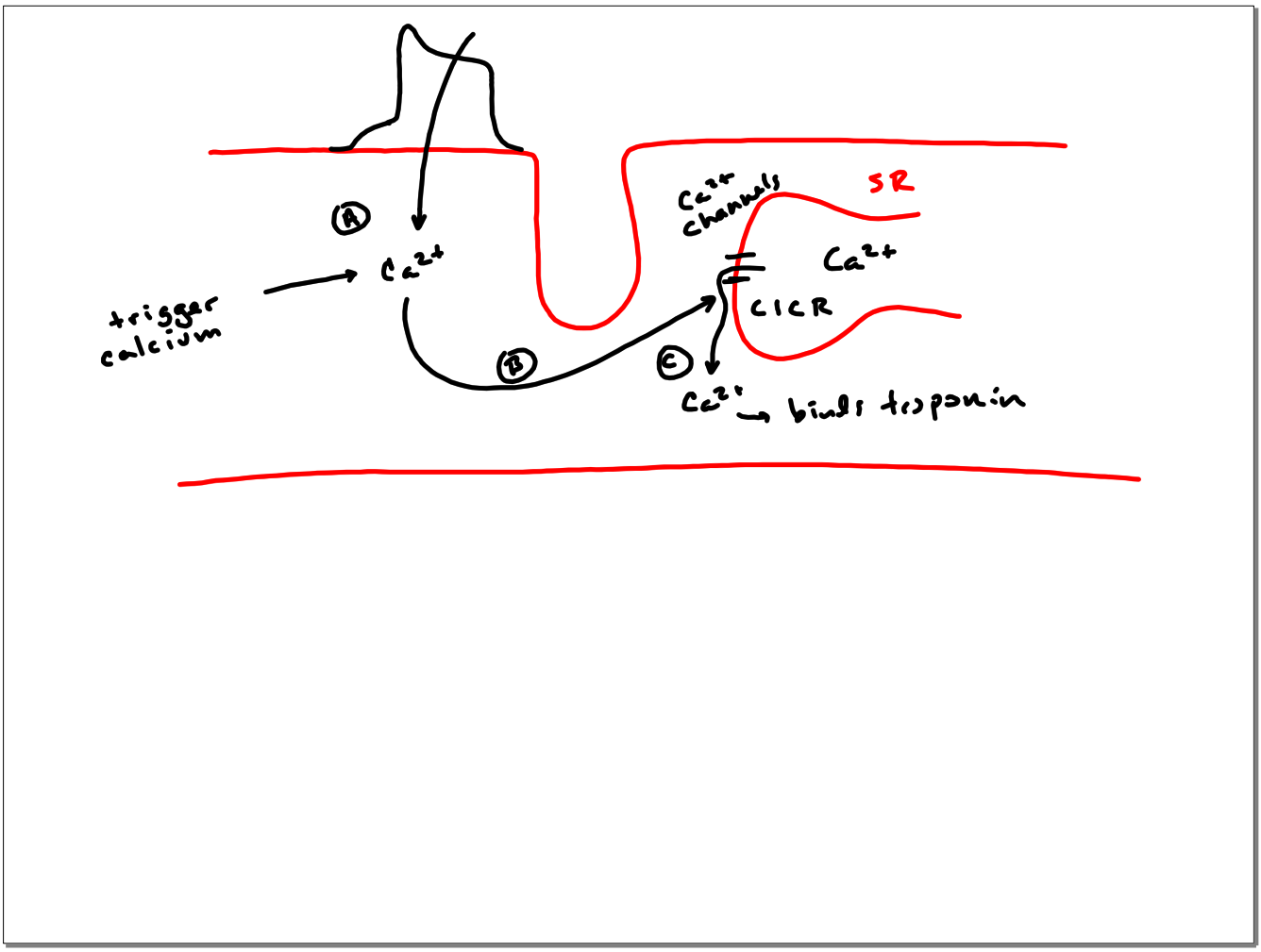

CCS AP
1
Myocardium AP
2
Other Myocardium AP
3
4 Myocardium contracts
Heart pumps blood

Feb 15-6:15 PM

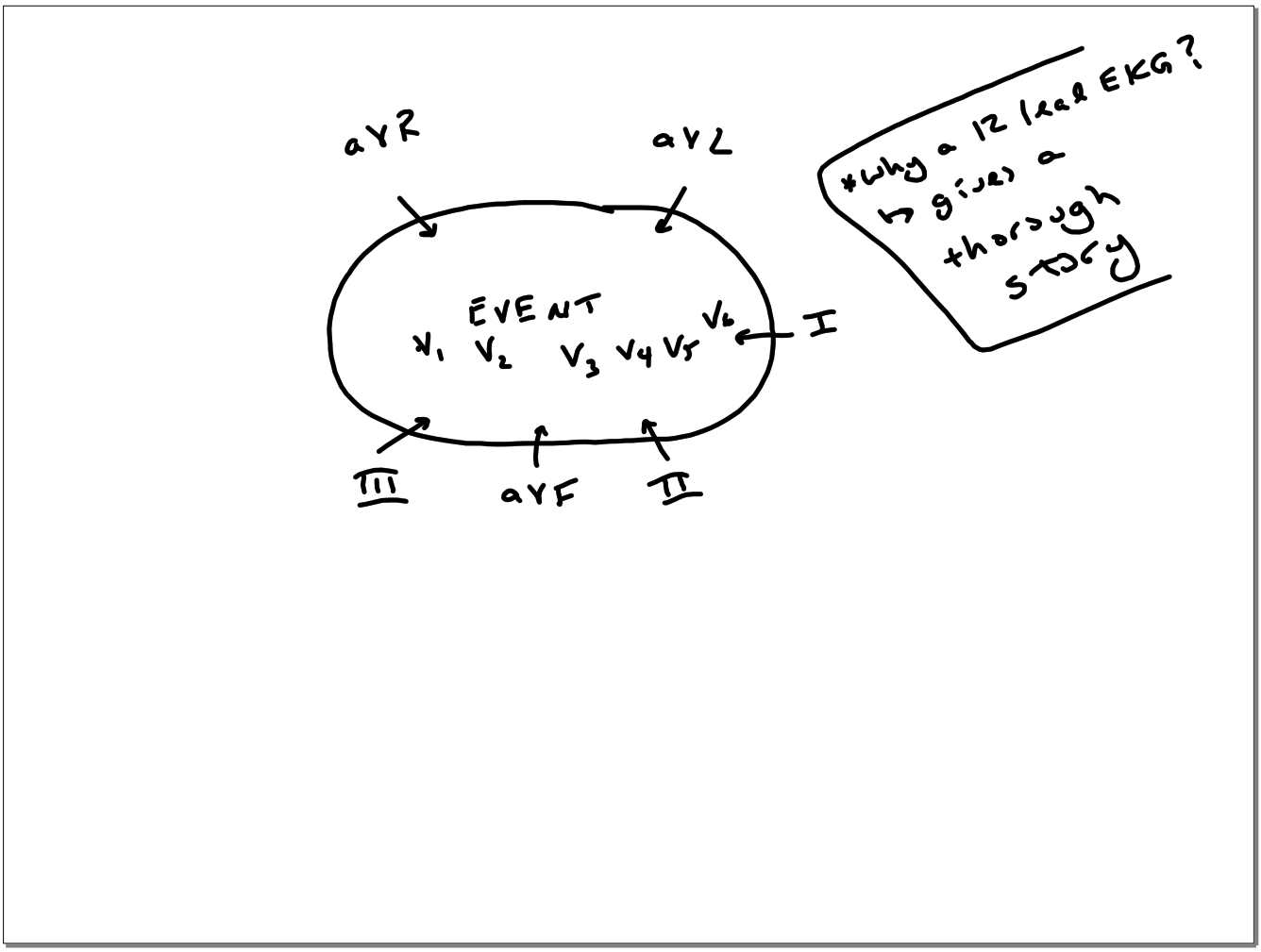

SA Node Cell
HCN channels
Ca2+ channels
volt-gated Ca2+ channels
K+
-Vm
volt-gated K+ channels
Na+
Ca2+
Ca2+
Ca2+ in
K+ out
Vm
Na+ in
Ca2+ in
pacemaker potential
time


volt-gated Na+ channels
Na+
K+
volt-gated K+ channels
volt-gated Ca2+ channels
Ca2+
Vm
cardiac muscle cell
1 2 3
1
K+ out
2
Na+ in
K+ out Ca2+ in
plateau
volt-gated Ca2+ channels close
volt-gated Ca2+ channels open
K+ 3
out
4
4
resting Vm
Ca2+ from CCs AP
or from Na+ from some other cardiac muscle AP
Vm
time

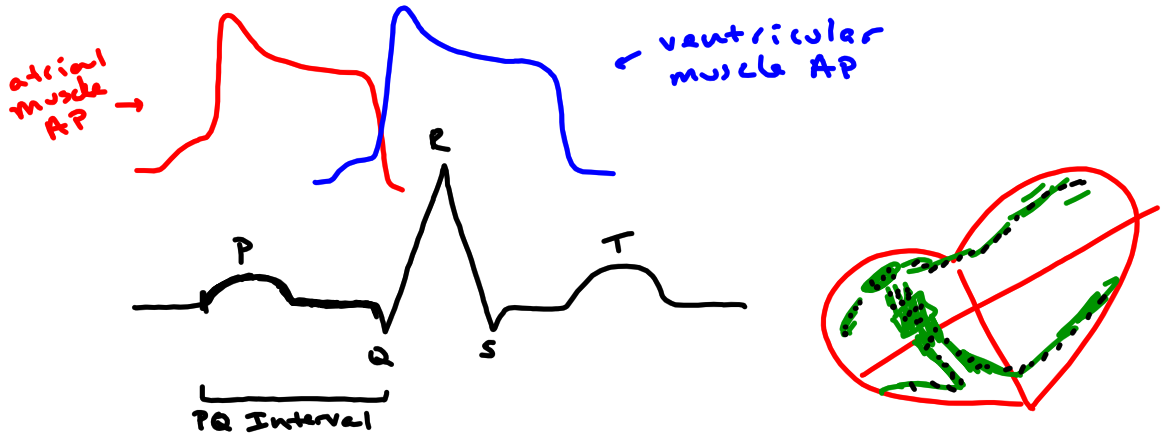
Feb 17-5:10 PM


A
Ca^{2+}
trigger calcium
Ca^{2+} channels
SR
Ca^{2+}
CICR
B
C
Ca^{2+} → binds troponin

Feb 17-5:22 PM

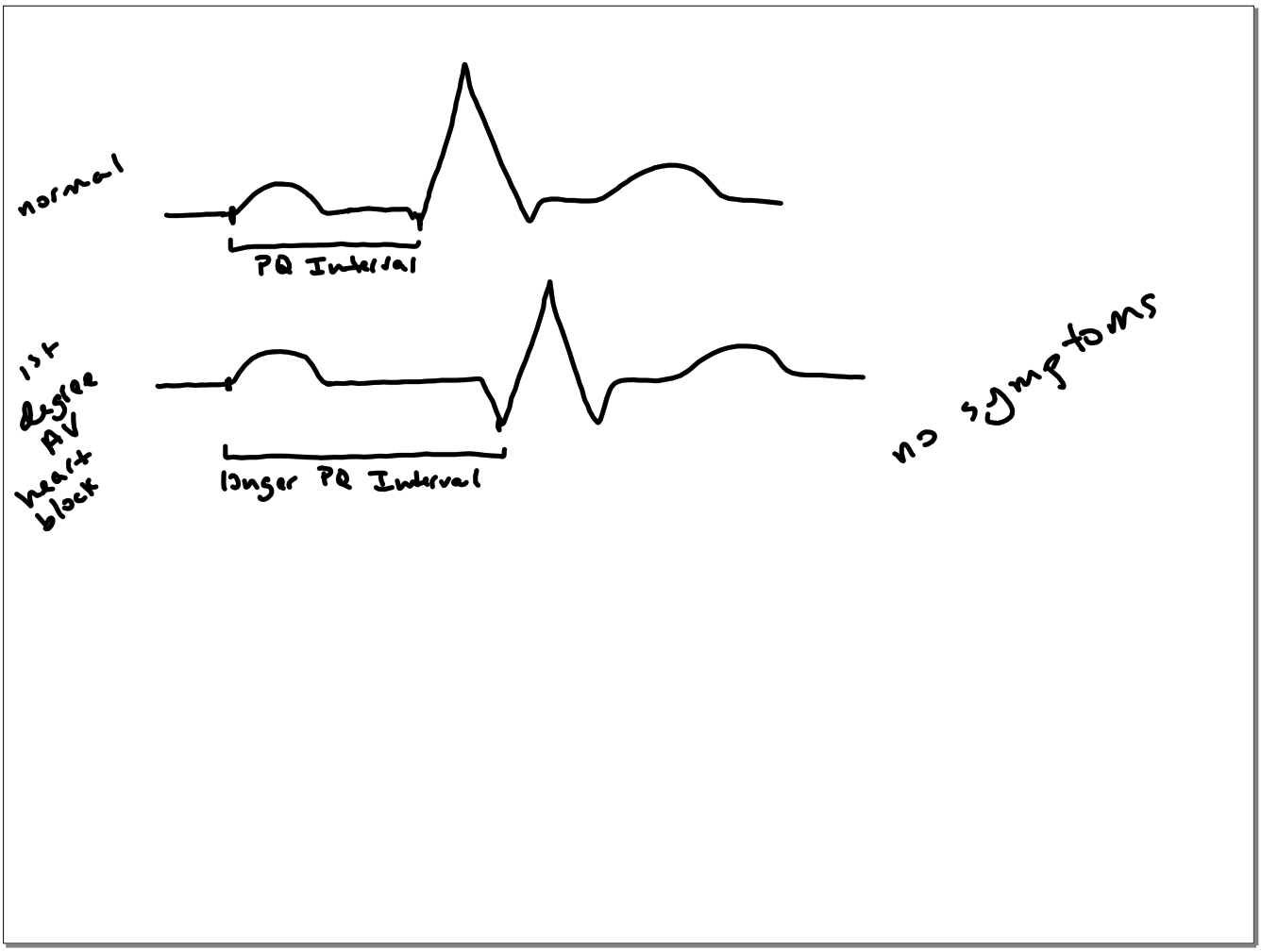

aVR
aVL
EVENT
V1 V2 V3 V4 V5 V6
I
III
aVF
II
*why a 12 lead EKG?
to give a
thorough
story

Feb 17-5:37 PM

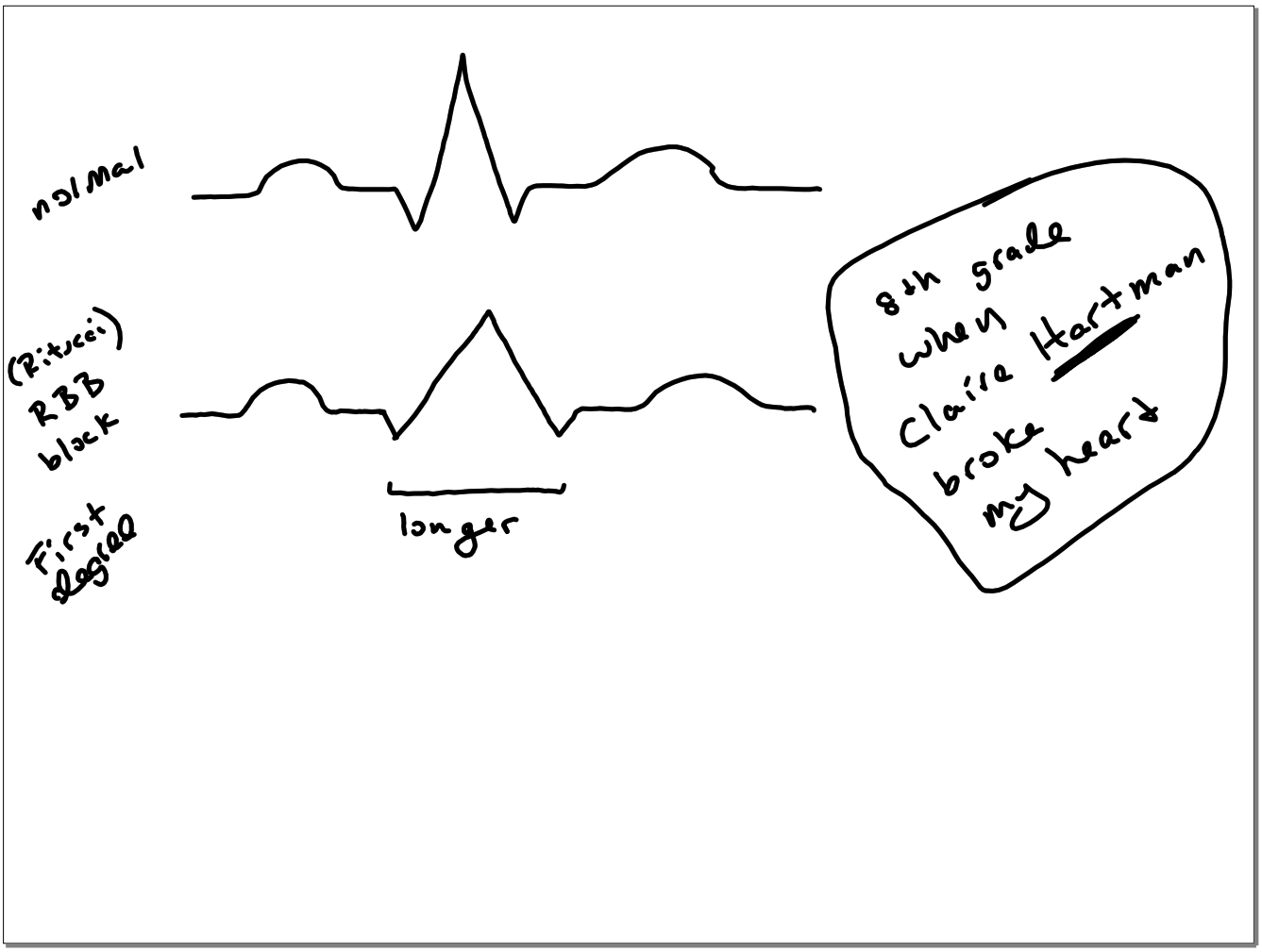


P wave: dep of atria
QRS : dep of ventricles
T wave : rep of ventricles
PQ interval: time it takes AP's to conduct through the CCS

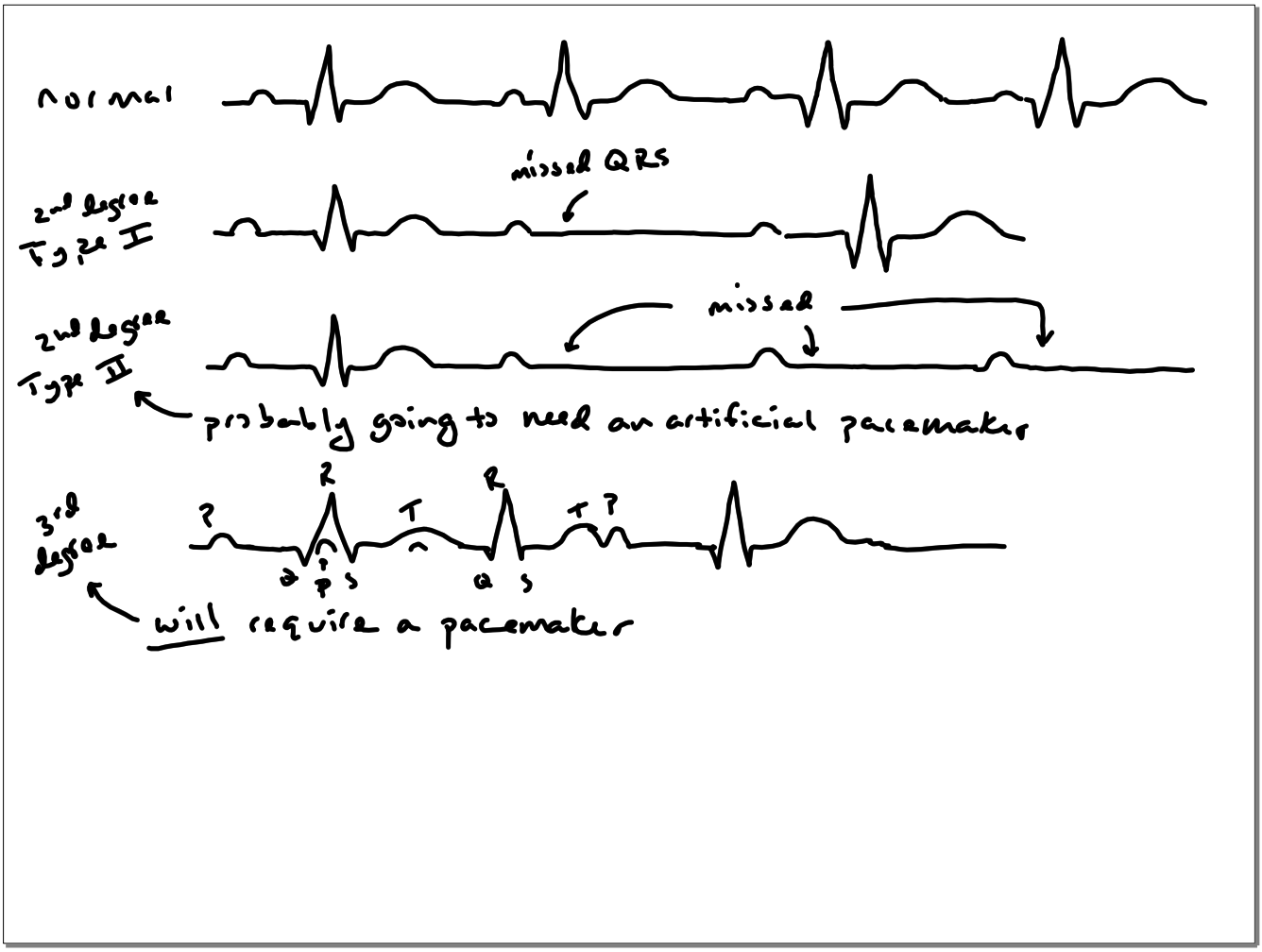
Feb 17-5:48 PM


normal
PQ Interval
1st degree AV heart block
longer PQ Interval
no symptoms

Feb 17-6:08 PM

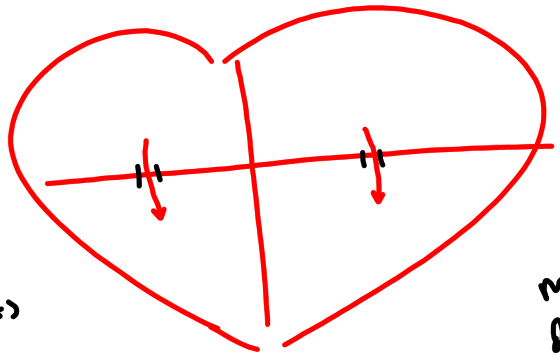

normal
(Ritucci)
RBB
block
First degree
longer
8th grade
when
Claire Hartman
broke
my heart

Feb 17-6:13 PM


normal
missed QRS
2nd degree Type I
missed
2nd degree Type II
probably going to need an artificial pacemaker
3rd degree
R
R
T
T
P
P
Q
S
S
will require a pacemaker

Feb 22-4:56 PM

A Fib

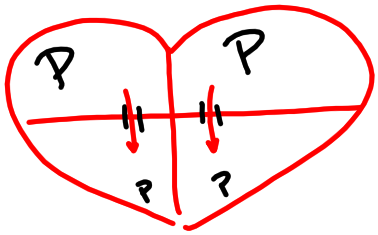
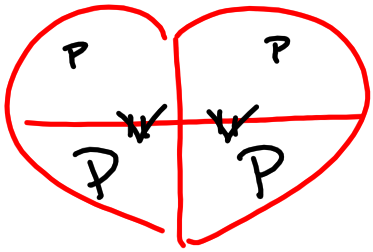


* might not present with symptoms, so why?

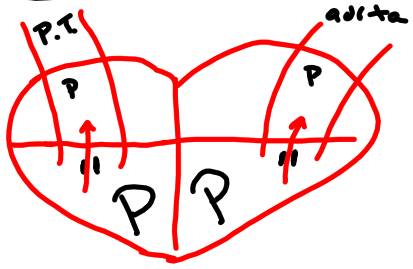
most blood is delivered from atria to ventricles w/o atria contracting

can lead to blood clots in atria

excess blood sits in atria (stasis)

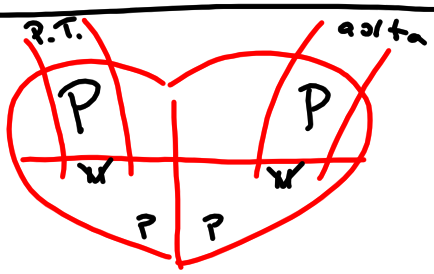
Opening of AV Values	Closing of AV values
	
atrial P > vent P	vent P > atria P first heart sound (S_1)

Opening of Semilunar Valves



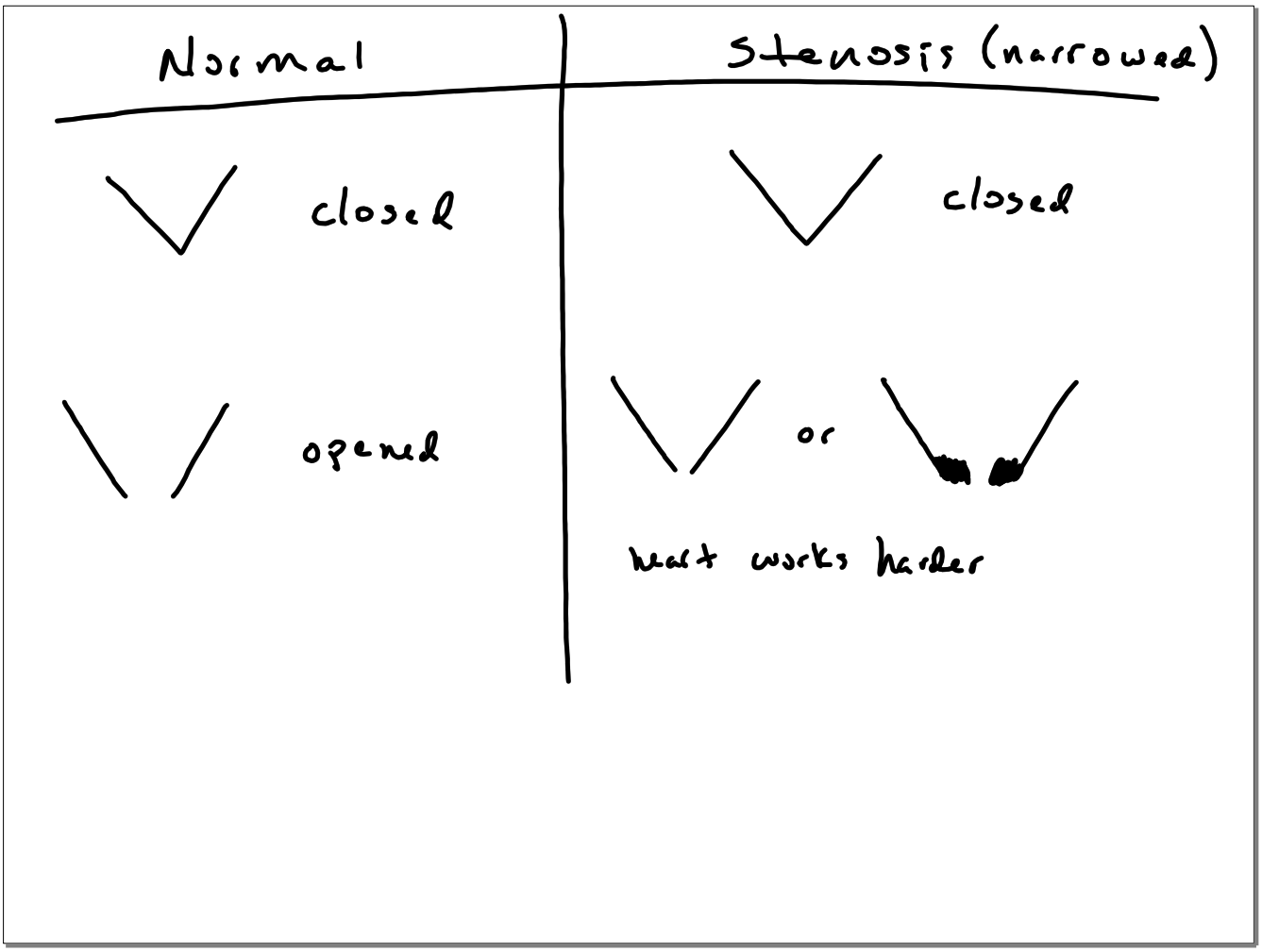
vent P > Great Artery P

Closing of Semilunar Valves

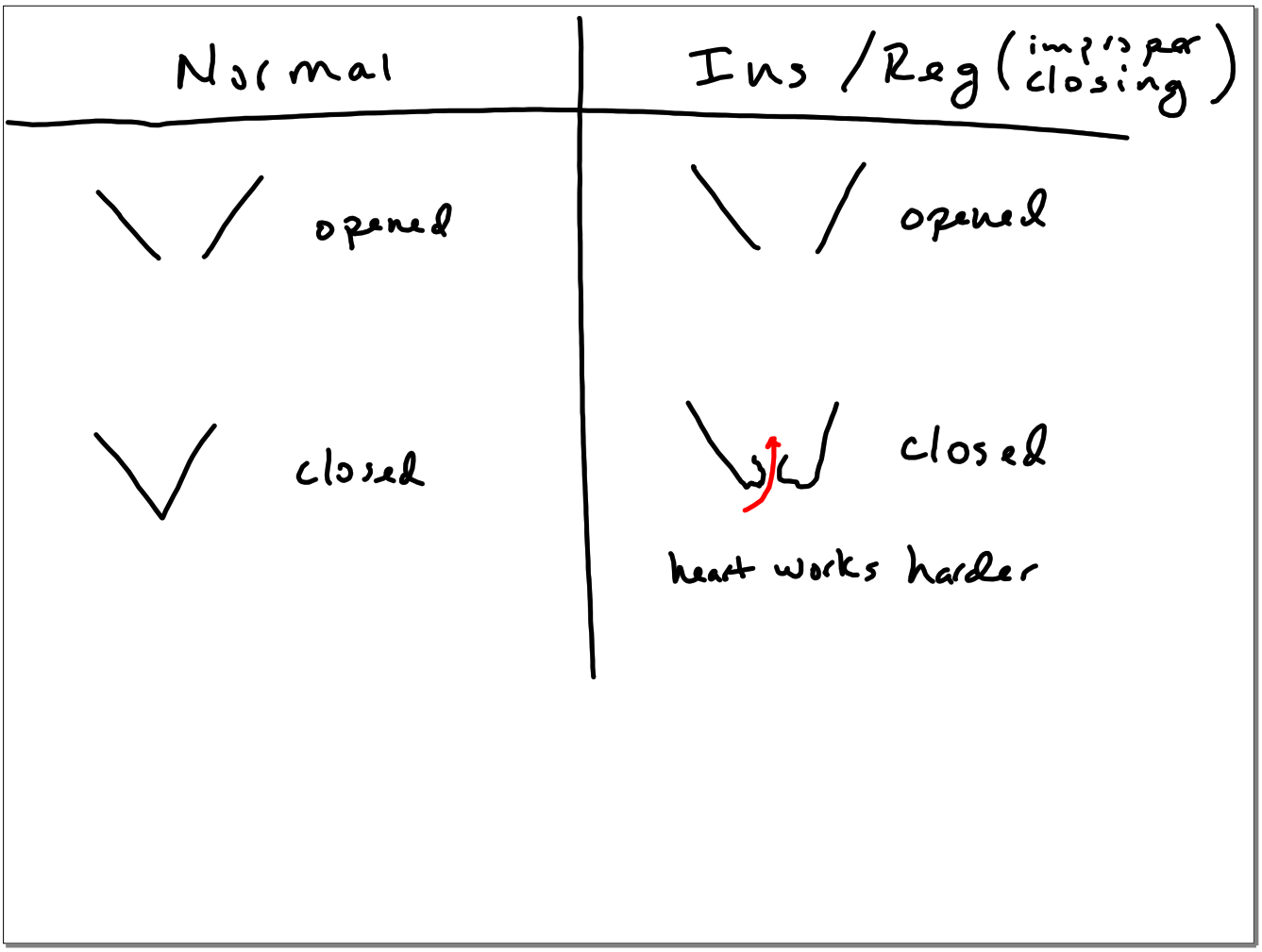


Great Artery P > vent P

second heart sound (S_2)


Normal
Stenosis (narrowed)
closed
closed
opened
or
heart works harder

Feb 22-5:25 PM


Normal
Ins / Reg (impairs per closing)
opened
opened
closed
closed
heart works harder

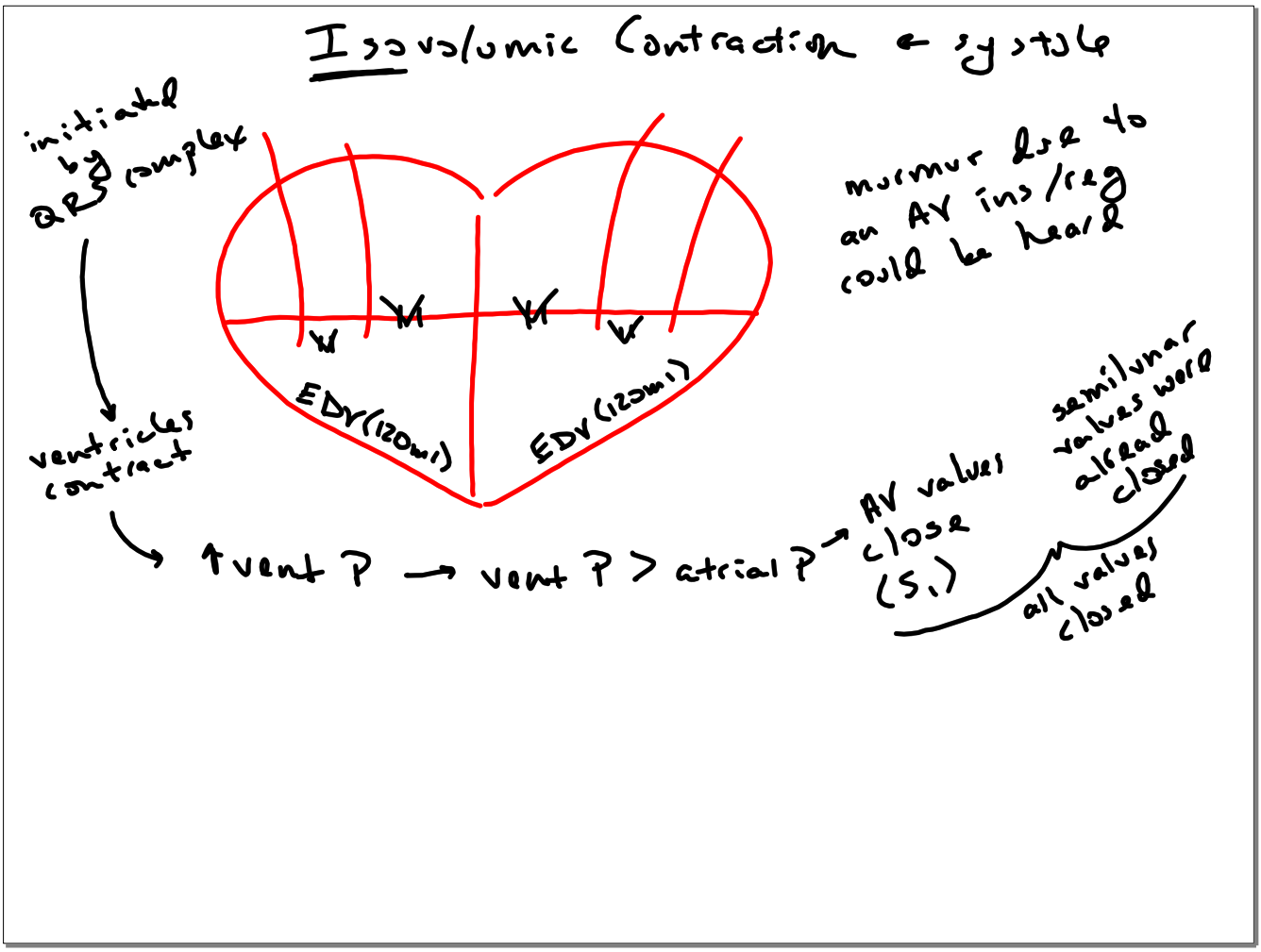
Feb 22-5:28 PM

* Cardiac Cycle

Isovolumic Contraction
Period of Eject
} Systolic

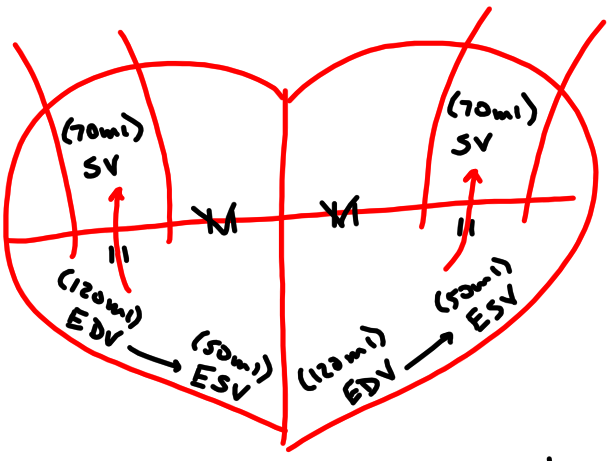
Isovolumic Relaxation
Passive Ventricular Filling
Active Ventricular Filling
} Diastolic

Feb 22-5:37 PM


Isovolumic Contraction ← systole
initiated by QRS complex
murmur due to an AV insuff could be heard
EDV (120ml)
EDV (120ml)
ventricles contract
↑vent P → vent P > atrial P → AV valves close (S1)
semilunar valves would already closed
all valves closed

Feb 22-5:44 PM

Period of Ejection ← systole



murmur due to a semilunar stenosis

ventricles continue to contract

↑↑Vent P → Vent P > Great Artery P → open semilunar valves

Feb 22-5:49 PM

$ESV = EDV - SV$

$SV = EDV - ESV$

$EDV = ESV + SV$

$$E.F. = \frac{SV}{EDV} \cdot 100$$

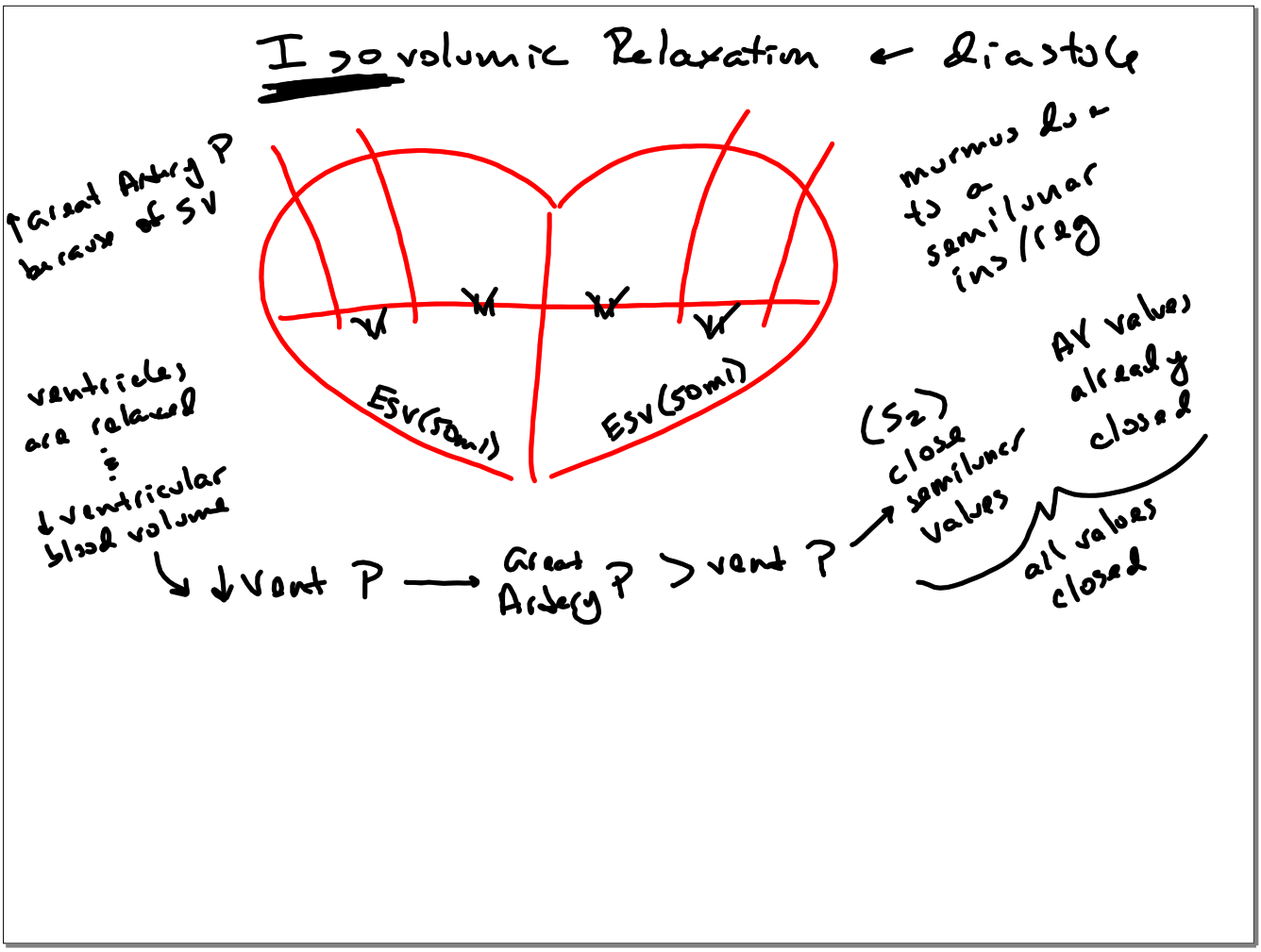
an E.F. greater than 50% is normal

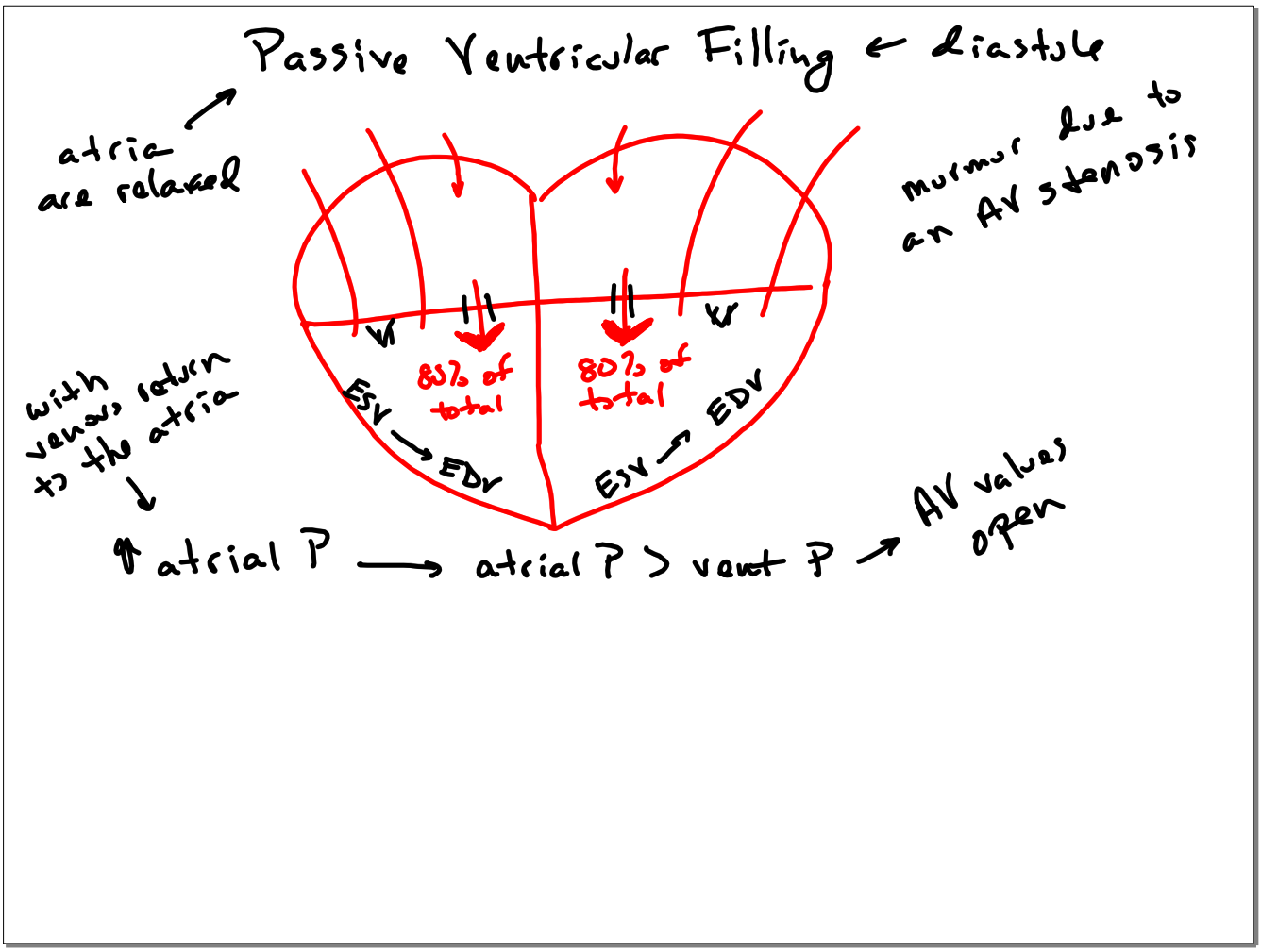
* E.F. is an indicator of how healthy the heart is

* unhealthy heart (i.e. heart failure)
  ↳ weak heart muscle

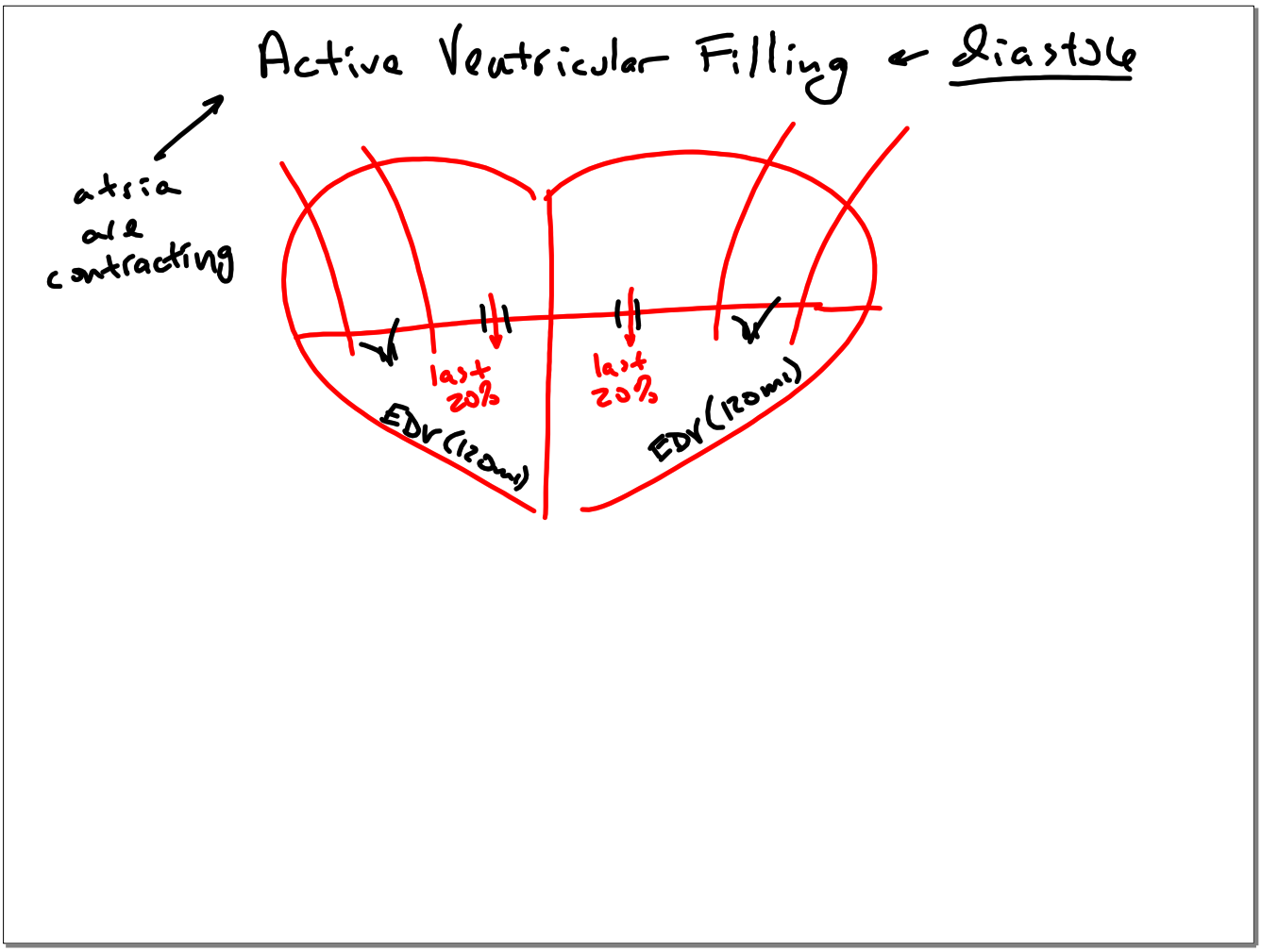
$$E.F. = \frac{30ml}{120ml} \cdot 100$$

$$= 25\%$$

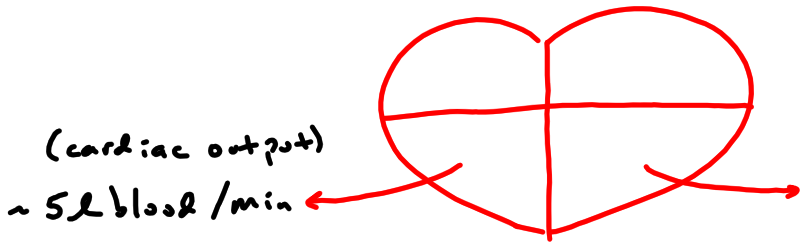

Isovolumic Relaxation ← diastole
murmus due to a semilunar insuff
great Artery P because of SV
ventricles are relaxed
↓ventricular blood volume
ESV(50ml)
ESV(50ml)
↓vent P → Great Artery P > vent P
(S2) close semilunar values
AV values already closed
all values closed


Passive Ventricular Filling ← diastole
atria are relaxed
murmur due to an AV stenosis
with veins return to the atria
80% of total
80% of total
ESV → EDV
ESV → EDV
↑ atrial P → atrial P > vent P → AV valves open

Feb 22-6:08 PM


Active Ventricular Filling ← diastole
atria are contracting
last 20%
last 20%
EDV (120ml)
EDV (120ml)

Feb 22-6:14 PM



$CO = HR \times SV$

$= \frac{70 \text{ beats}}{\text{min}} \times \frac{70 \text{ ml blood}}{\text{beat}}$

$= 4,900 \text{ ml blood/min} \rightarrow \sim 5\ell \text{ blood/min}$

* Efficient Heart:

↓ HR ∴ ↑ SV

↗

$$CO = \frac{50 \text{ beats}}{\text{min}} \times \frac{100 \text{ ml blood}}{\text{beat}}$$

$$= 5 \text{ l/min}$$

Feb 24-5:04 PM

* Positive inotrope is something that causes an increase in heart contractility (↑SV)
* Negative inotrope is something that causes a decrease in heart contractility (↓SV)
* Positive chronotrope is something that causes an increase in heart rate
* Negative chronotrope is something that cause a decrease in heart rate

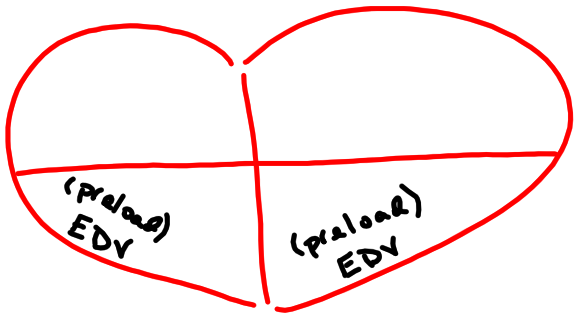
Feb 24-5:07 PM

* Unhealthy heart (i.e. heart failure)
  - ↓ contractility
    - ↓ SV
      - ↑ body responds by: ↑ HR

↑ weak heart muscle

inefficient heart

Feb 24-5:13 PM

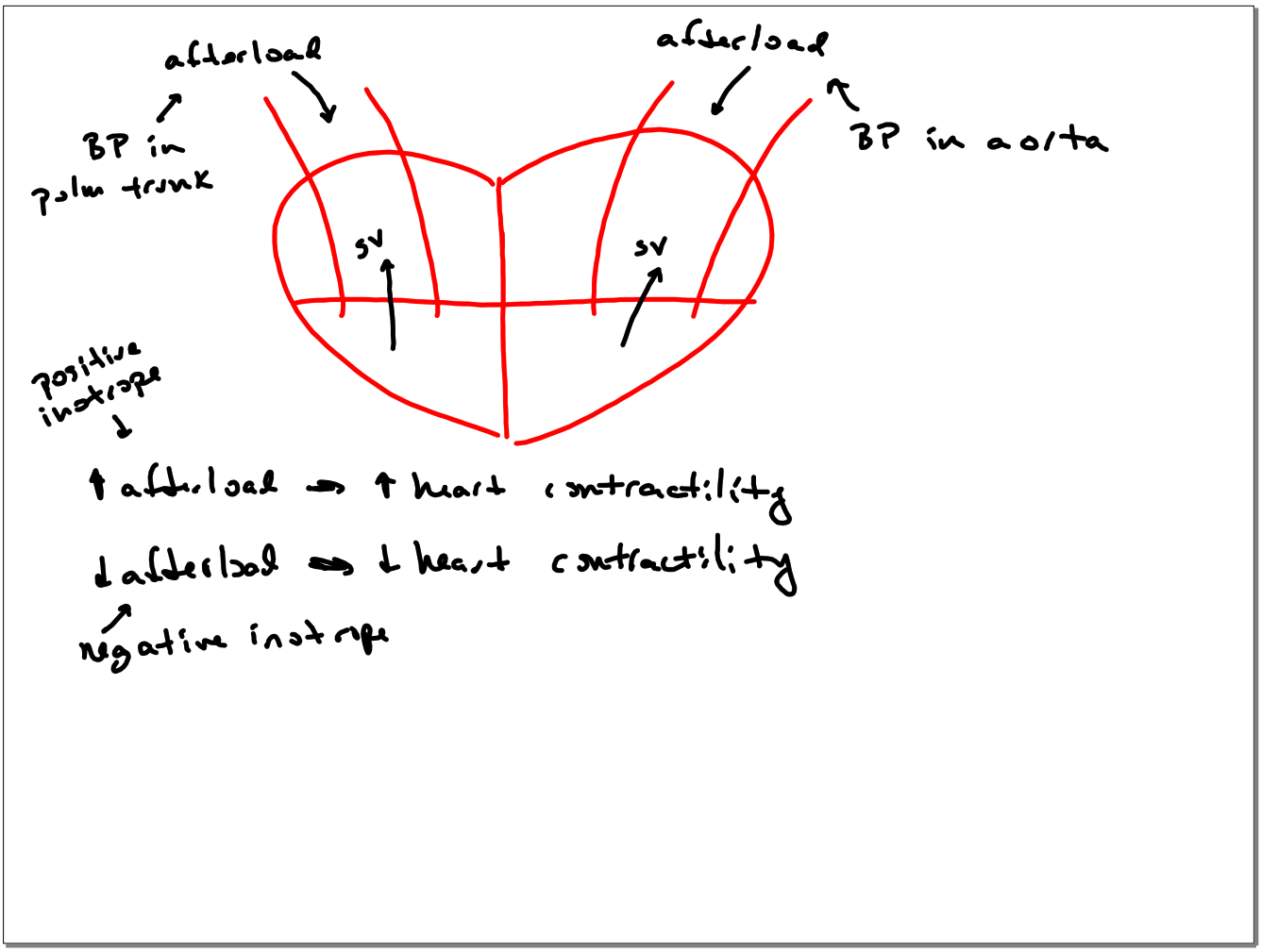


positive inotrope

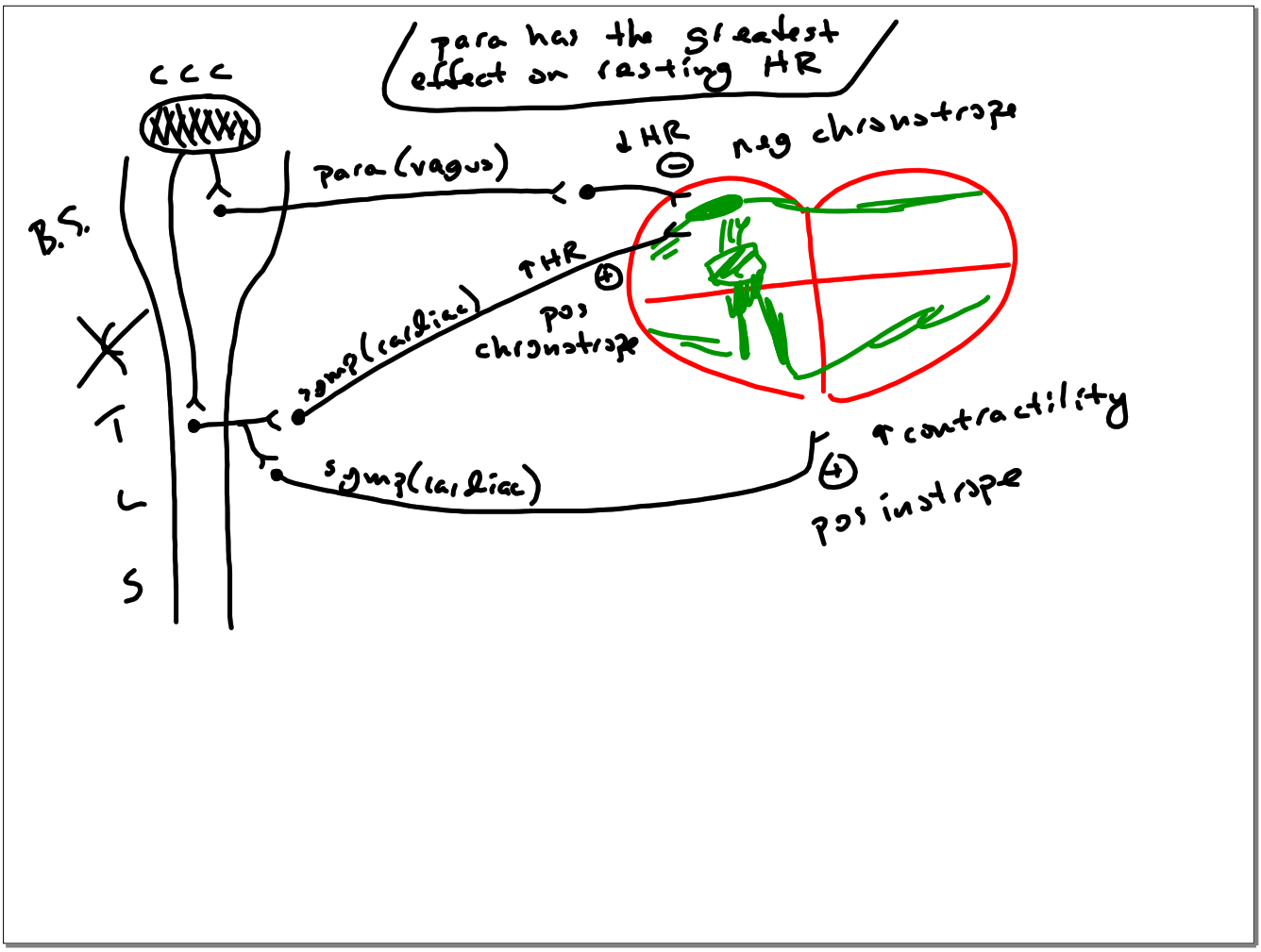
↑preload → ↑contractility → ↑SV

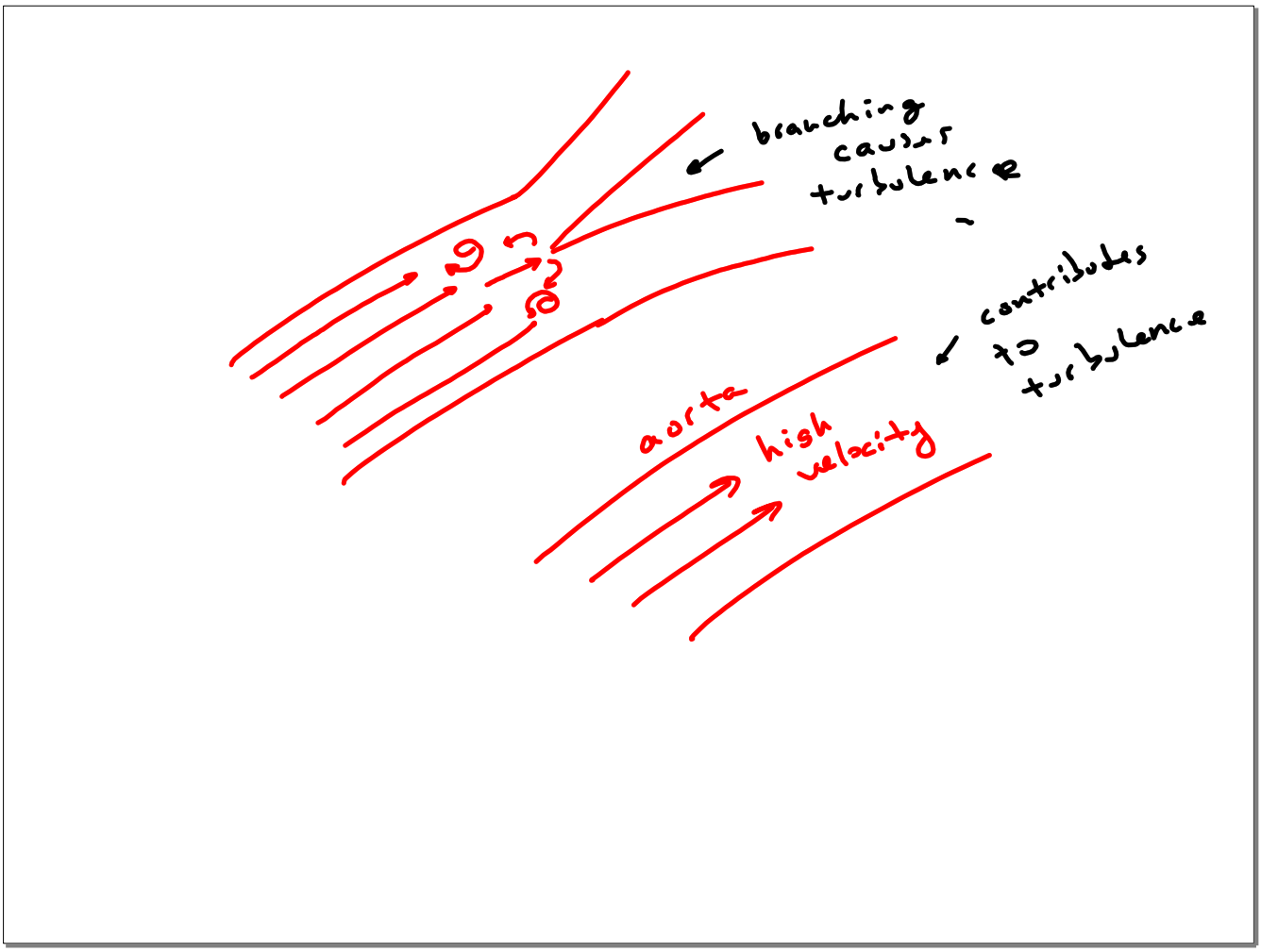
↓preload → ↓contractility → ↓SV

negative inotrope


afterload
afterload
BP in pulm trunk
BP in aorta
SV
SV
positive inotrope
↑ afterload ⇒ ↑ heart contractility
↓ afterload ⇒ ↓ heart contractility
negative inotrope

Feb 24-5:19 PM


para has the greatest effect on resting HR
CCC
B.S.
T
L
S
para (vagus)
↓HR
neg chronotrope
symp (cardiac)
↑HR
pos chronotrope
symp (cardiac)
↑ contractility
pos inotrope


branching
causes
turbulence
contributes
to
turbulence
aorta
high
velocity

Feb 24-5:34 PM

$$\text{Fluid Flow} = \frac{(P_1 - P_2)}{R}$$ ← pressure gradient

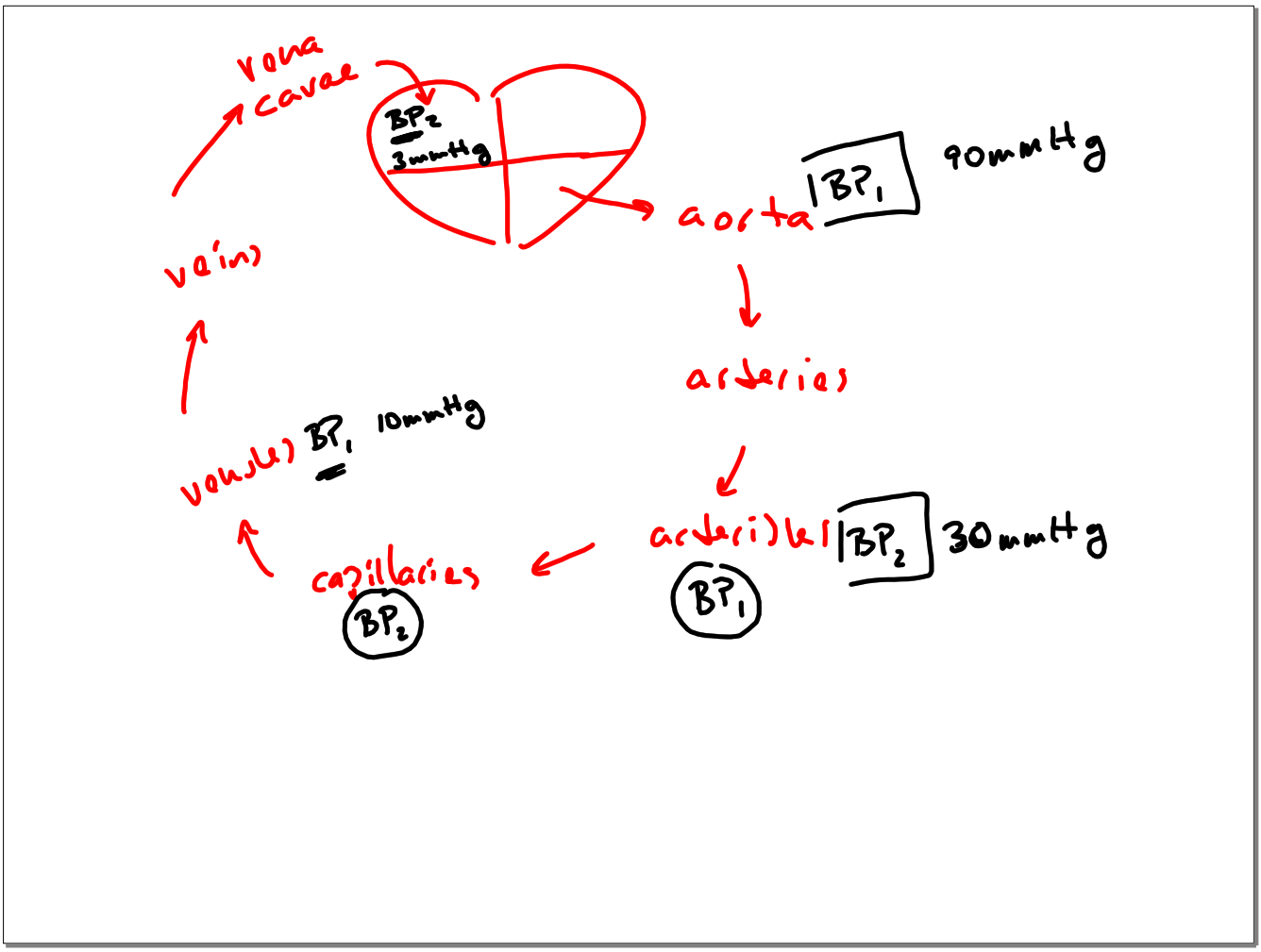
$$\text{Blood Flow} = \frac{(BP_1 - BP_2)}{R}$$

$$R \sim \frac{\nu}{r^4}$$

ν = viscosity of blood

r = radius of blood vessel

Feb 24-5:38 PM


vena cavae
BP_2 = 3mmHg
aorta
BP_1
90mmHg
veins
arteries
venules BP_1 = 10mmHg
arterioles
BP_2
30mmHg
BP_1
capillaries
BP_2

Feb 24-5:44 PM

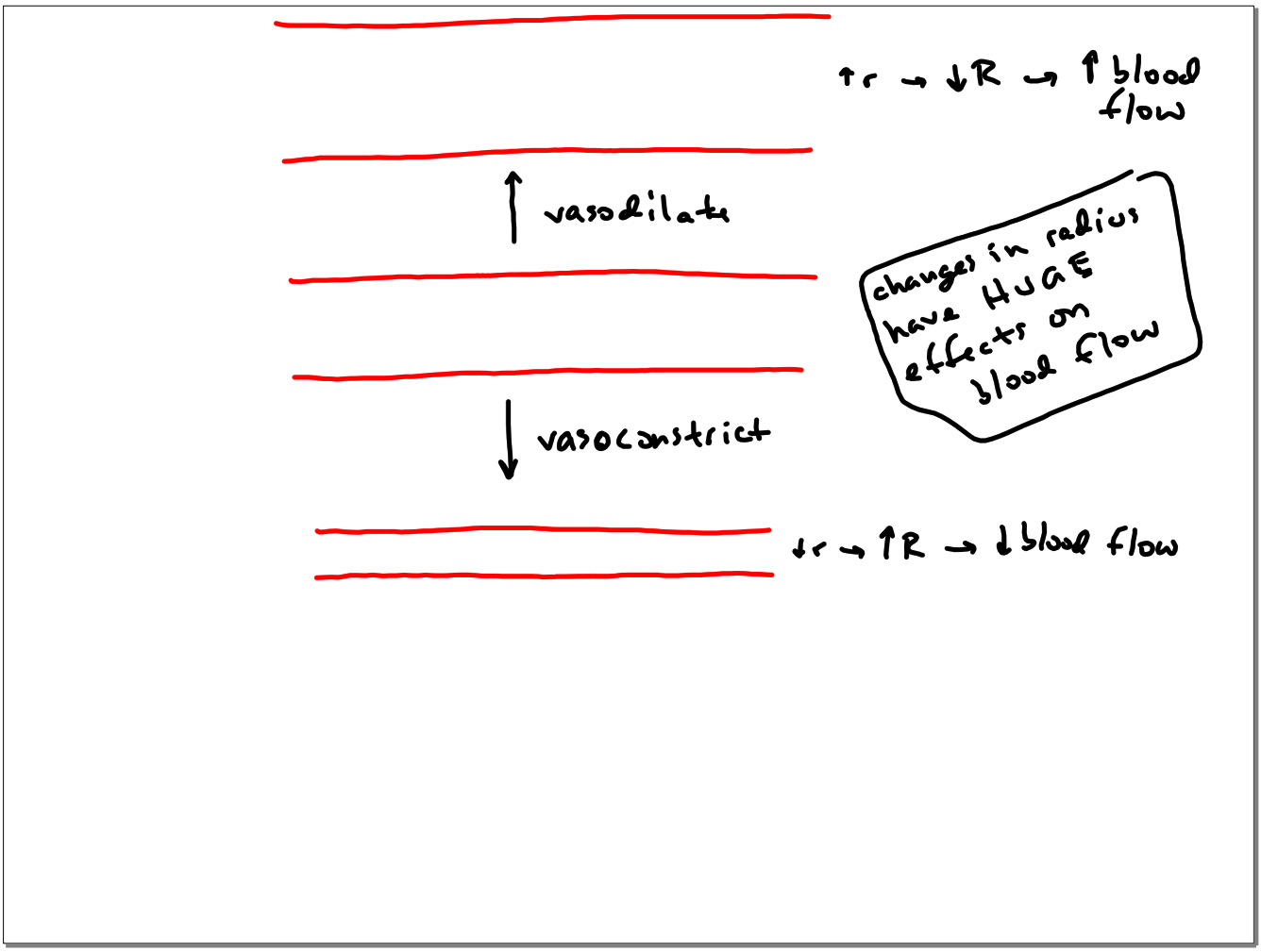
↑ $(BP_1 - BP_2)$ → ↑ blood flow

↓ $(BP_1 - BP_2)$ → ↓ blood flow

If BP in arterial system is too low

↳ $(BP_1 - BP_2)$ is too small

↳ ↓↓ blood flow ← risk organ failure


↑r → ↓R → ↑ blood flow
vasodilate
changes in radius have HUGE effects on blood flow
vasoconstrict
↓r → ↑R → ↓ blood flow

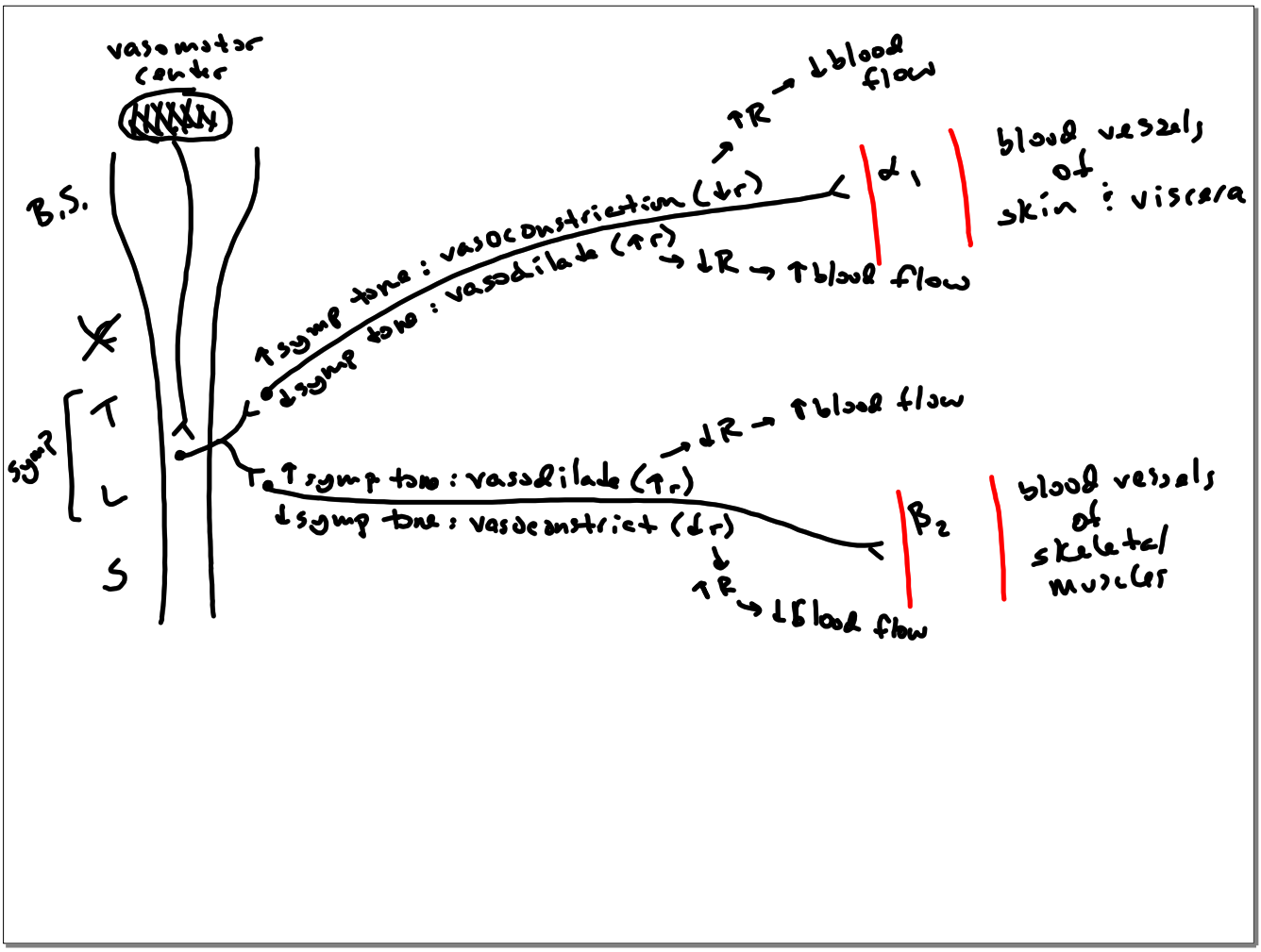
Feb 24-5:52 PM

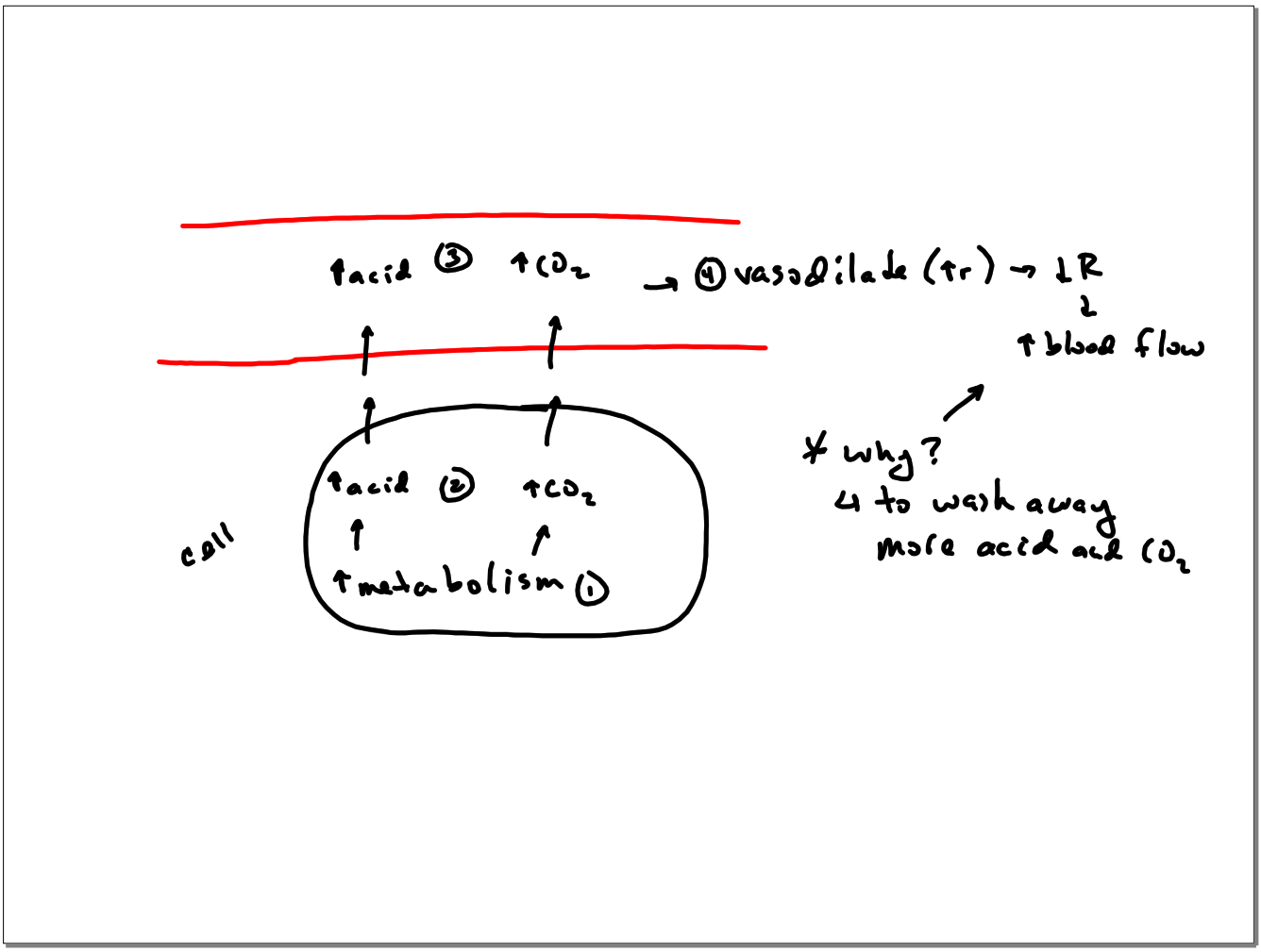
↑ viscosity → ↑R → ↓ blood flow

↓ viscosity → ↓R → ↑ blood flow

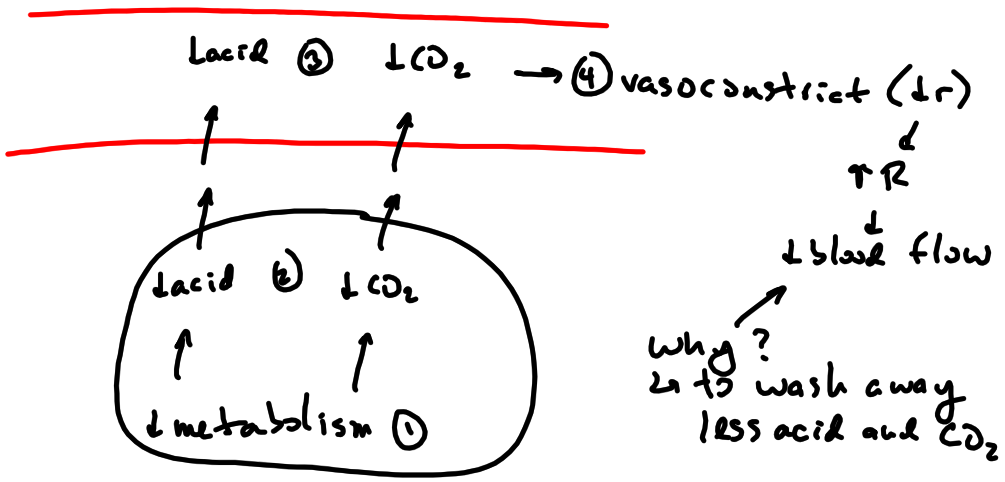
viscosity (thickness)

* what affects blood viscosity the most?
  - ↳ Hct
    - ↑ Hct → ↑ viscosity — polycythemia
    - ↓ Hct → ↓ viscosity

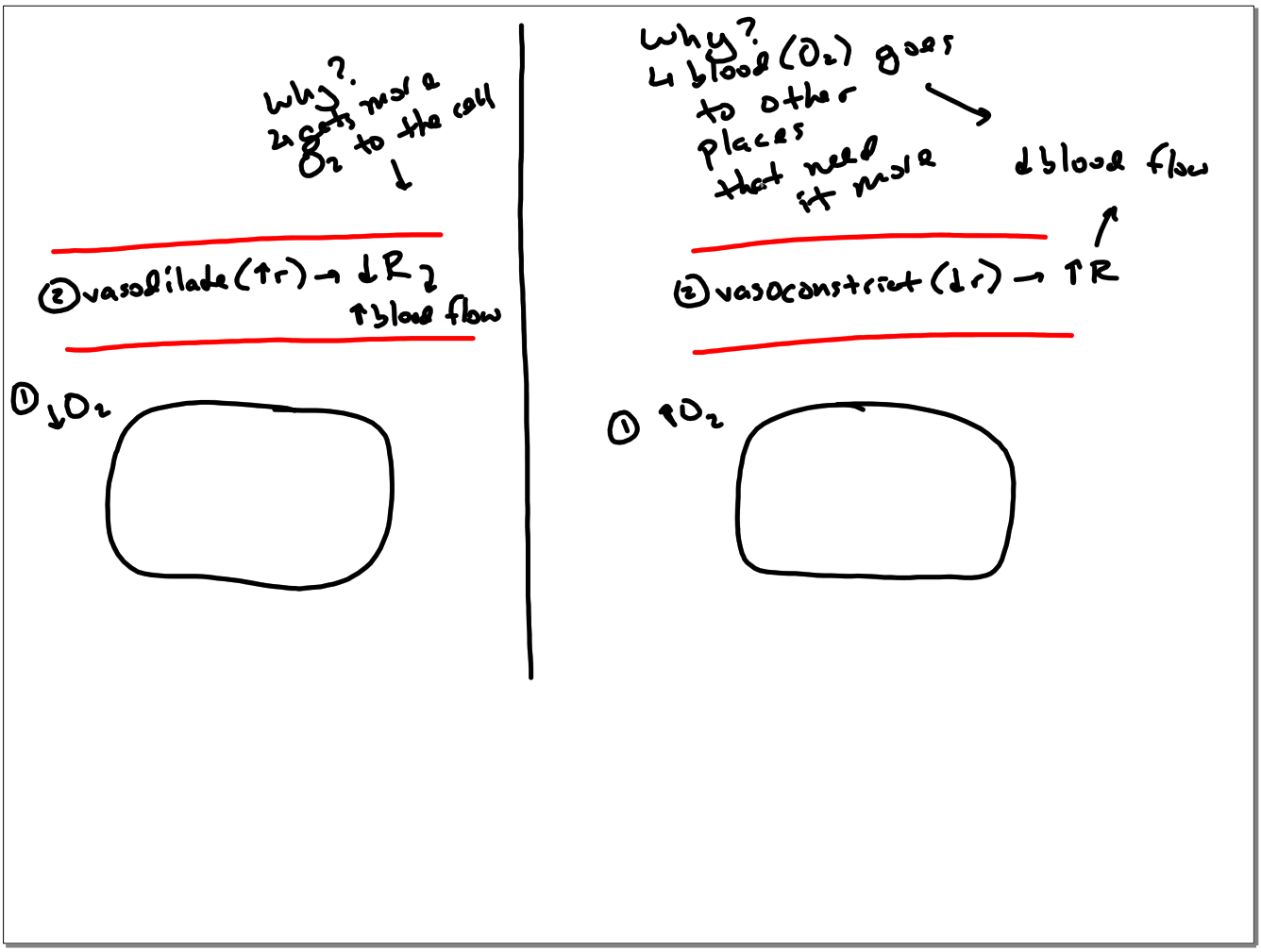

vasomotor center
B.S.
sympT
L
S
↑symp tone : vasoconstriction (↓r)
↓symp tone : vasodilate (↑r)
↑R → ↓blood flow
↓R → ↑blood flow
α_1
blood vessels of skin & viscera
↑symp tone : vasodilate ($\uparrow r_2$)
↓symp tone : vasoconstrict ($\downarrow r_2$)
↓R → ↑blood flow
↑R → ↓blood flow
β_2
blood vessels of skeletal muscles


↑acid ③ ↑CO_2 → ④ vasodilate (↑r) → ↓R
↓
↑blood flow
* why?
↳ to wash away
more acid and CO_2
↑acid ② ↑CO_2
cell
↑metabolism ①

Feb 24-6:03 PM


Lacid ③ ↓CO2 → ④ vasoconstrict (↓r)
↑R
↓ blood flow
why?
to wash away
less acid and CO2
↑acid ② ↓CO2
↑ metabolism ①

Feb 24-6:12 PM


Why?
↳ gets more O_2 to the cell
↓
② vasodilate (↑r) → ↓R_2
↑blood flow
① ↓O_2
Why?
↳ blood (O_2) goes to other places that need it more
↓blood flow
② vasoconstrict (↓r) → ↑R
① ↑O_2

Feb 24-6:14 PM

* systolic P : pressure during systole
* diastolic P : pressure during diastole

$$BP = \frac{110\ mmHg}{70\ mmHg}$$

* pulse pressure = (systolic P) - (diastolic P)

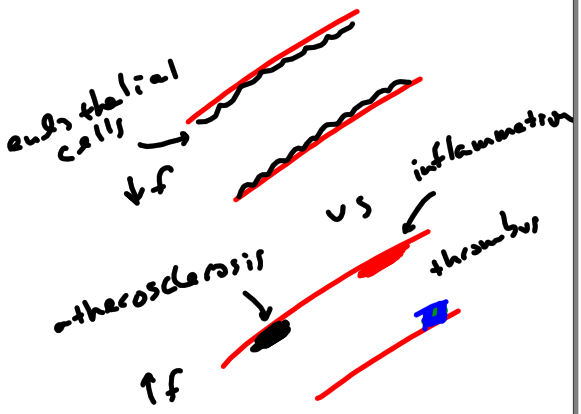
= 110mmHg - 70 mmHg
= 40 mmHg

Feb 24-6:18 PM

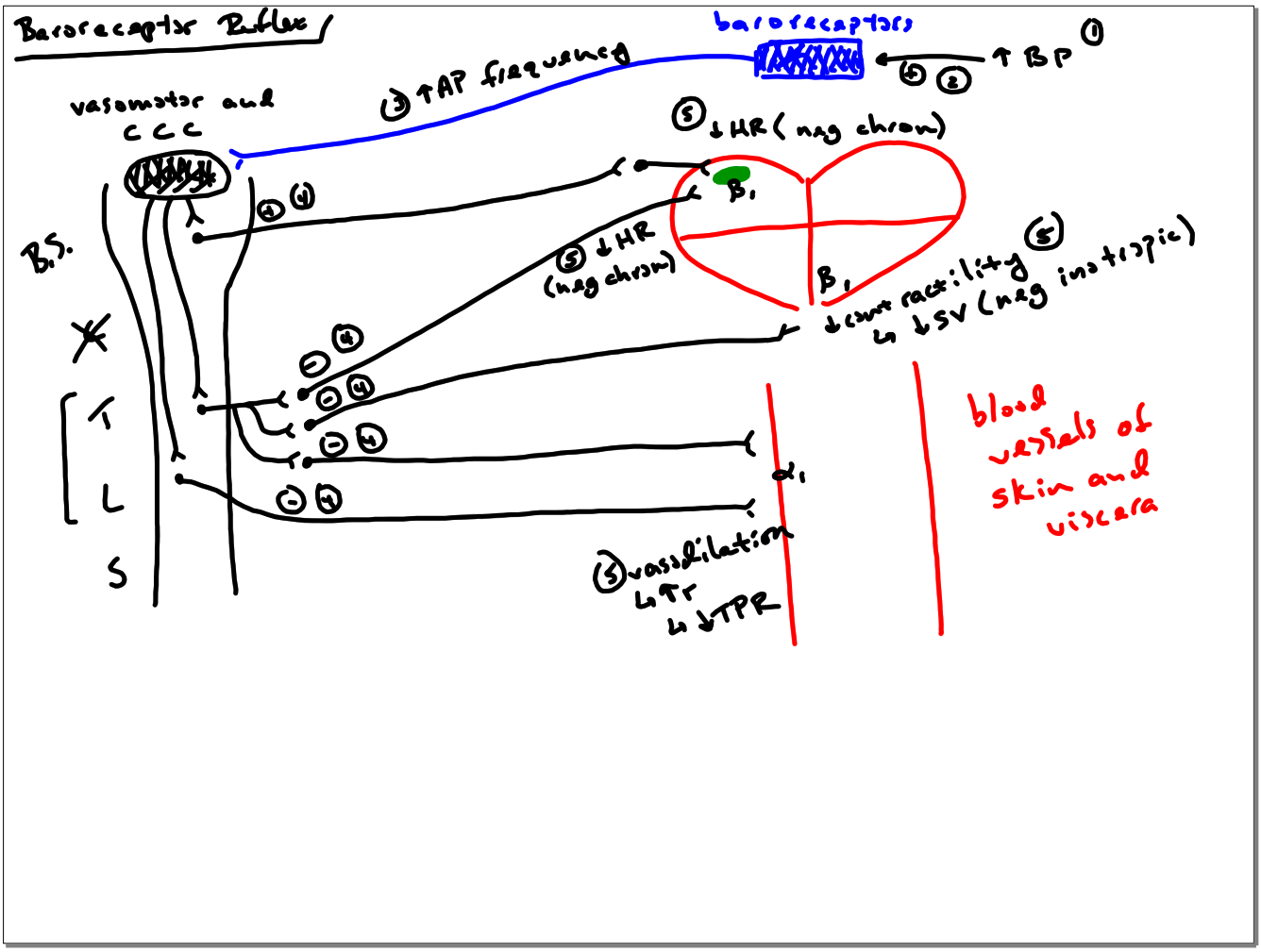
(BP)

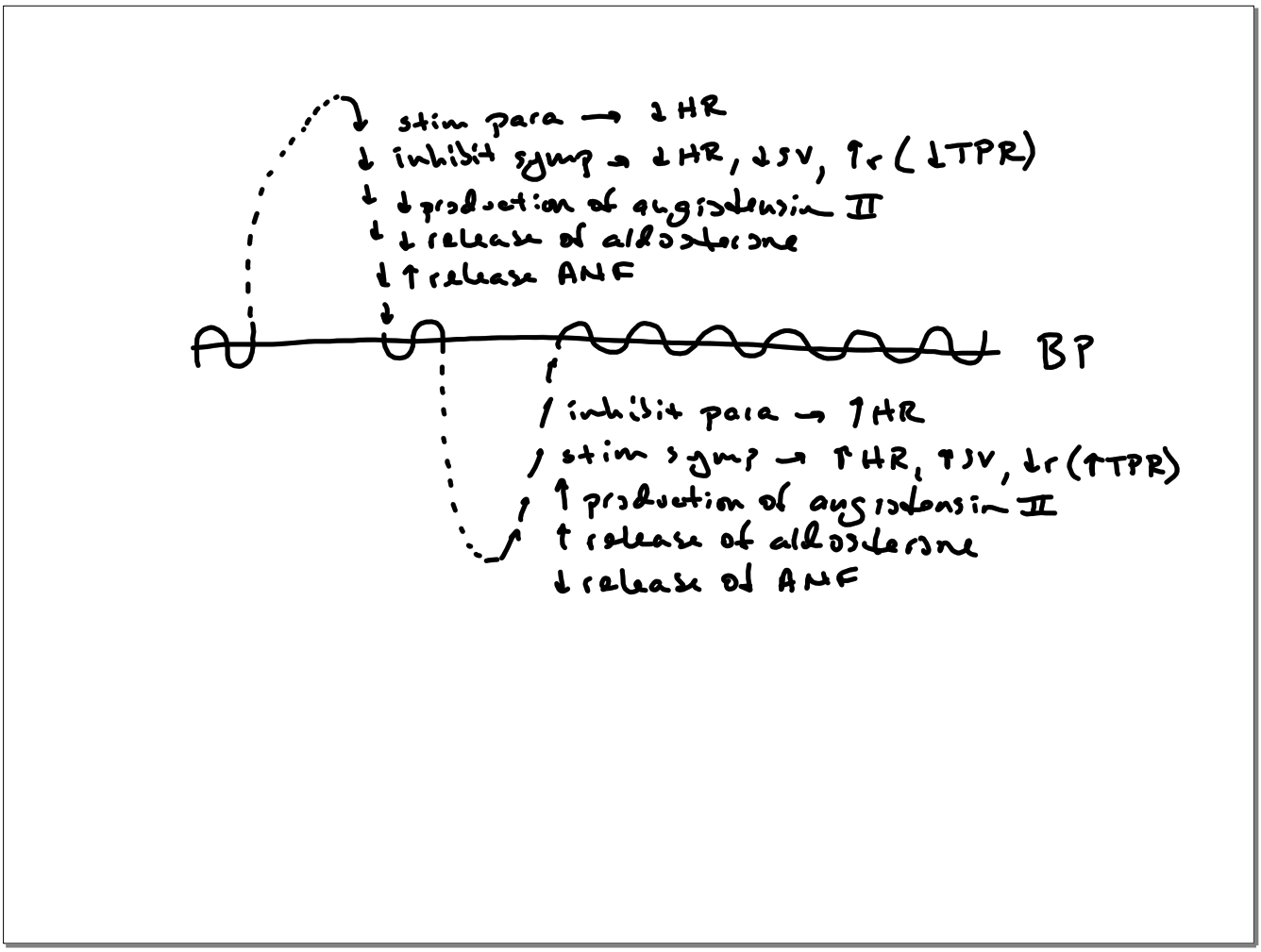
MAP = (CO) × (TPR)

= (HR) × (SV) × (1/r) × (v) × (f)

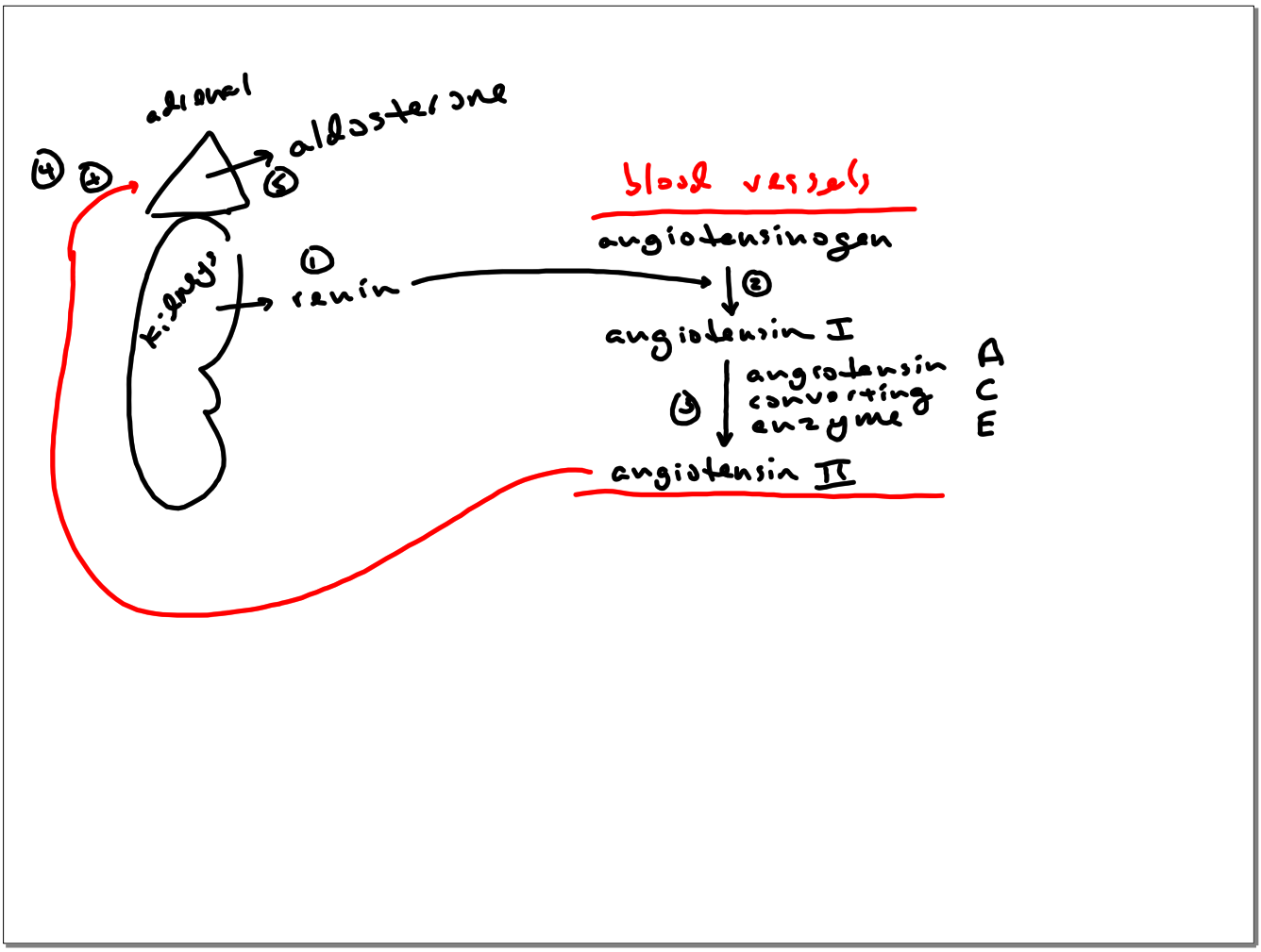


Mar 8-4:59 PM


Baroreceptor Reflex
baroreceptors
↑ BP
③ ↑AP frequency
vasomotor and CCC
⑤ ↓HR (neg chron)
B.S.
⑤ ↓HR (neg chron)
↓ contractility ⑤
↳ ↓SV (neg inotropic)
blood vessels of skin and viscera
⑤ vasodilation
↳ ↑r
↳ ↓TPR


stim para → ↓HR
↓ inhibit symp → ↓HR, ↓SV, ↑r (↓TPR)
↓ ↓production of angiotensin II
↓ ↓release of aldosterone
↓ ↑release ANF
BP
↓ inhibit para → ↑HR
↓ stim symp → ↑HR, ↑SV, ↓r (↑TPR)
↑ ↑production of angiotensin II
↑ ↑release of aldosterone
↓ ↑release of ANF

Mar 8-5:14 PM


adrenal
aldosterone
kidney
renin
blood vessels
angiotensinogen
angiotensin I
angiotensin converting enzyme
ACE
angiotensin II

Mar 8-5:25 PM

* Angiotensin II
  - causes release aldosterone → ↑blood volume → vasoconstricts
  - causes vasoconstriction
  - causes release of ADH → ↑blood volume → vasoconstriction

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* ↑ production of angiotensin II and ↑ release of aldosterone
  - ↑BP
* ↓ production of angiotensin II and ↓ release of aldosterone
  - ↓BP

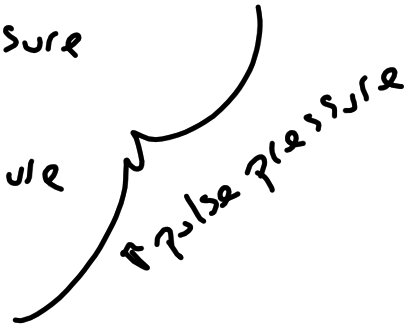
Mar 8-5:28 PM

* Compliance of blood vessels
  - how "stretchy" ... how stiff a blood vessel is

  * with normal compliance:
    - during systole:
      - arteries stretch
        - benefit:
          - decreases force against walls of blood vessel (TBP a little)
    - during diastole:
      - arteries recoil ("snap back")
        - benefit
          - increase force against walls of blood vessels (TBP a little)

Mar 8-5:36 PM

* ↓ compliance → too stiff
  - during systole:
    - ↑ systolic pressure
  - during diastole:
    - ↓ diastolic pressure

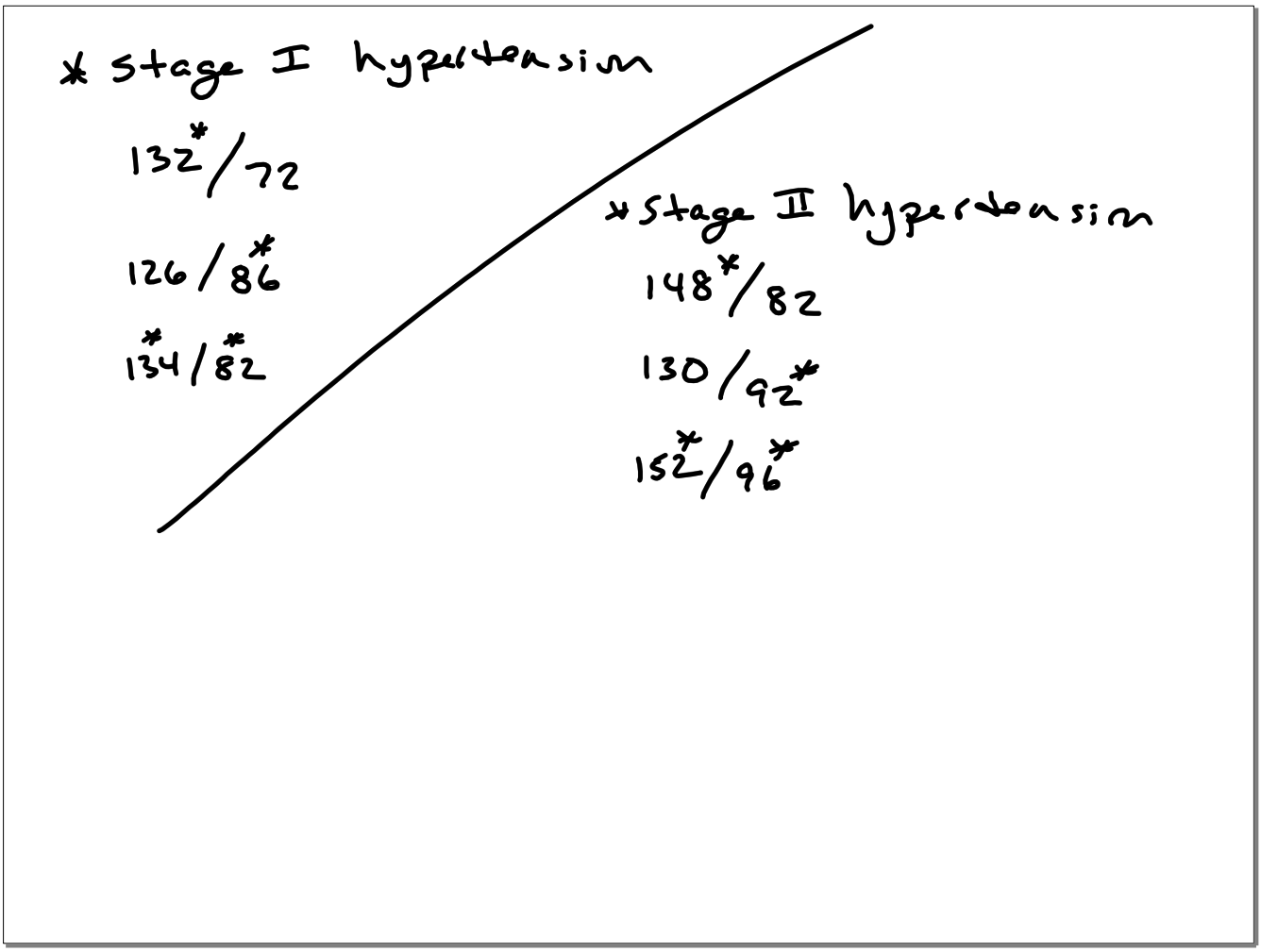


cause:
- arteriosclerosis ← hardening of arteries

Mar 8-5:41 PM

* Weak pulse:
  - ↓compliance (↑s stiff) ← arteriosclerosis
  - ↓↓BP

Mar 8-5:49 PM


* stage I hypertension
132*/72
126/86*
134*/82*
* stage II hypertension
148*/82
130/92*
152*/96*

Mar 8-5:59 PM

* Symptoms of hypertension:

  ↳ There are none

    "The Silent Killer"

Mar 8-6:14 PM