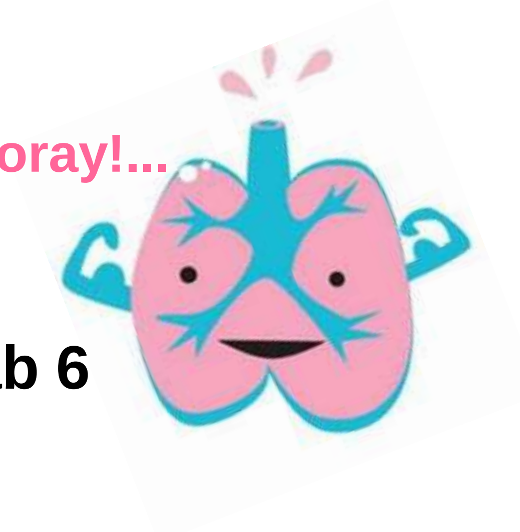
Pulmonary Function Testing today! Hooray!...

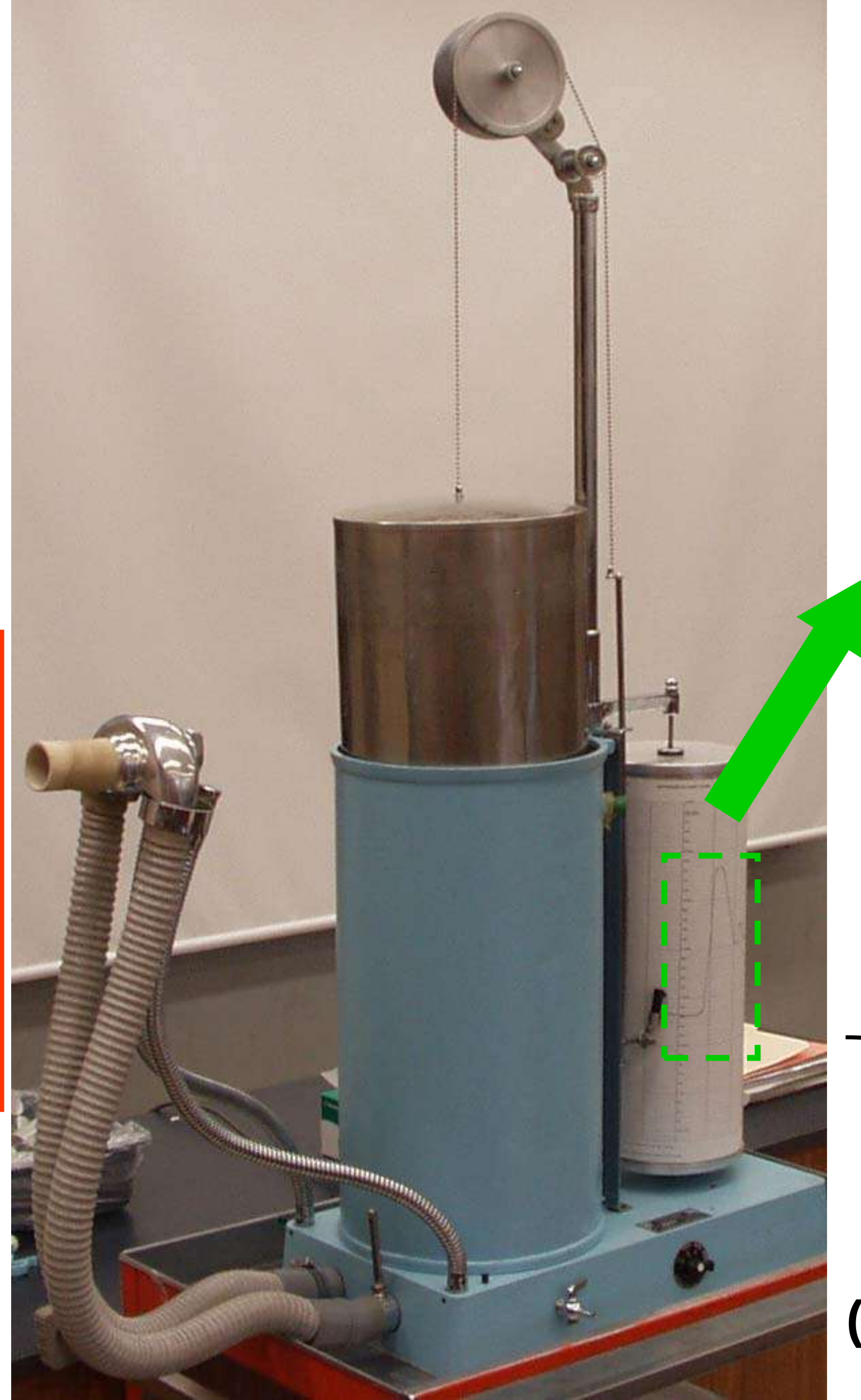


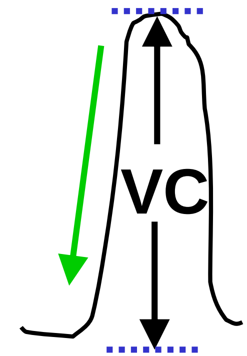
BI 121 Lecture 15

I. ***Announcements*** **Optional notebook ✓ + Lab 6 Pulmonary Function Testing today. Q?**
II. ***Pulmonary Function Lab Overview***
III. ***Muscle Structure & Function*** **LS ch 8, DC Module 12**
   A. **Skeletal muscle organization review** **LS fig 8-2, DC fig 12-2**
   B. **Banding pattern?** **LS fig 8-3, fig 8-7**
   C. **How do muscles contract?** **LS fig 8-6, 8-10**
   D. **What's a cross-bridge cycle?** **LS fig 8-11+…**
   E. **Summary of skeletal muscle contraction**
   F. **Exercise adaptation variables: *mode*, *intensity*, *duration*, *frequency*, *distribution*, *individual* & environmental char...?**
   G. **Endurance *vs.* strength training continuum? fiber types...**

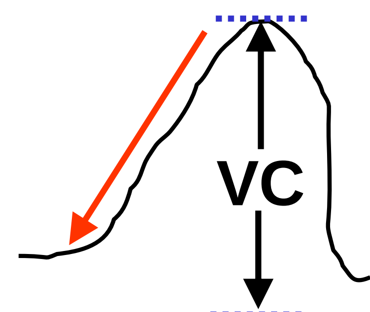
Respirometer → **measures *complete <u>Pulmonary Function Test</u> or PFT*!**

<u>NB</u>: Should be able to blow out $\geq$ 75 - 85% of VC/FVC in 1 second! That's $FEV_{1.0}/FVC \geq$ 0.75 – 0.85. If less, may indicate asthma or other lung disease.



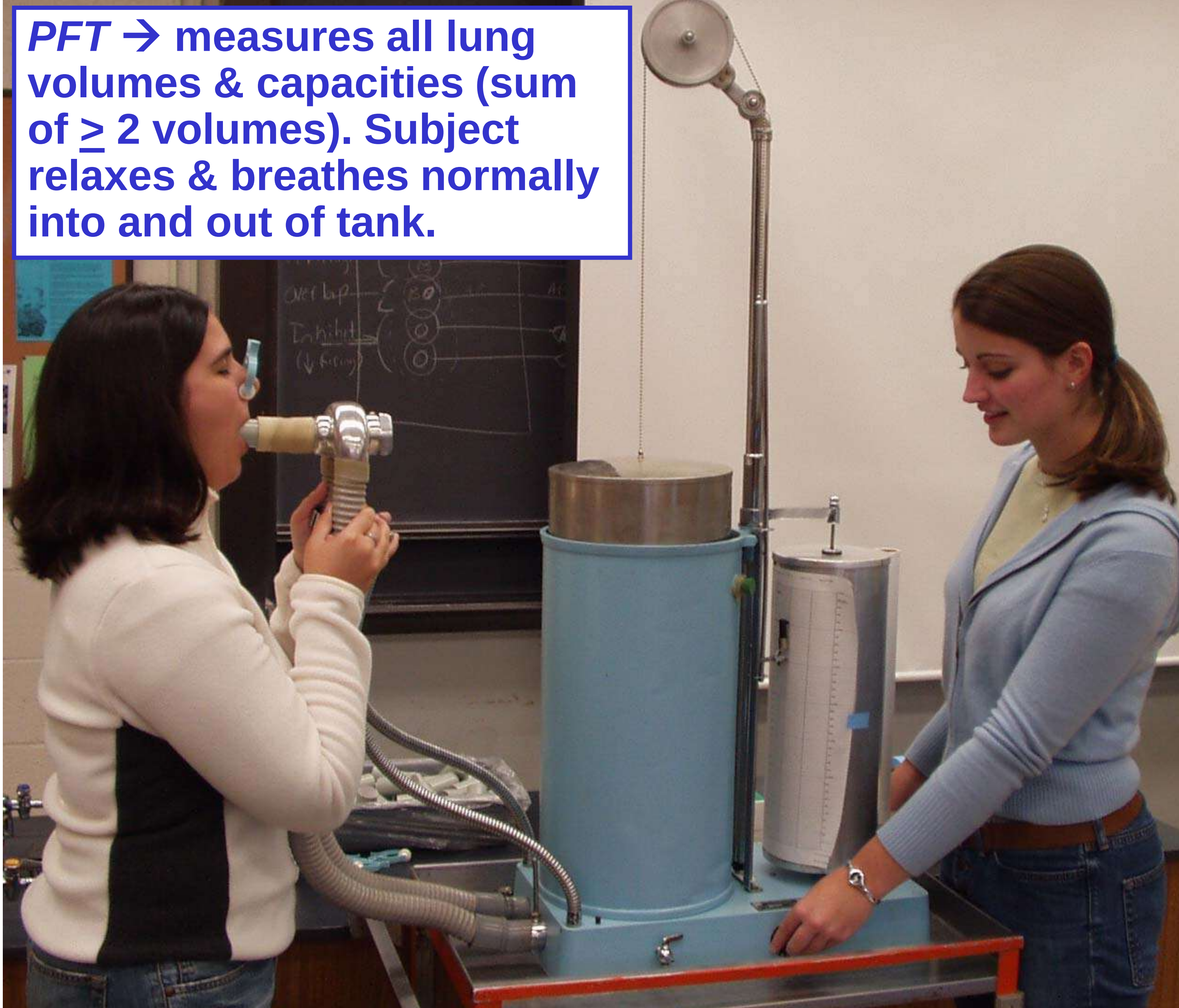


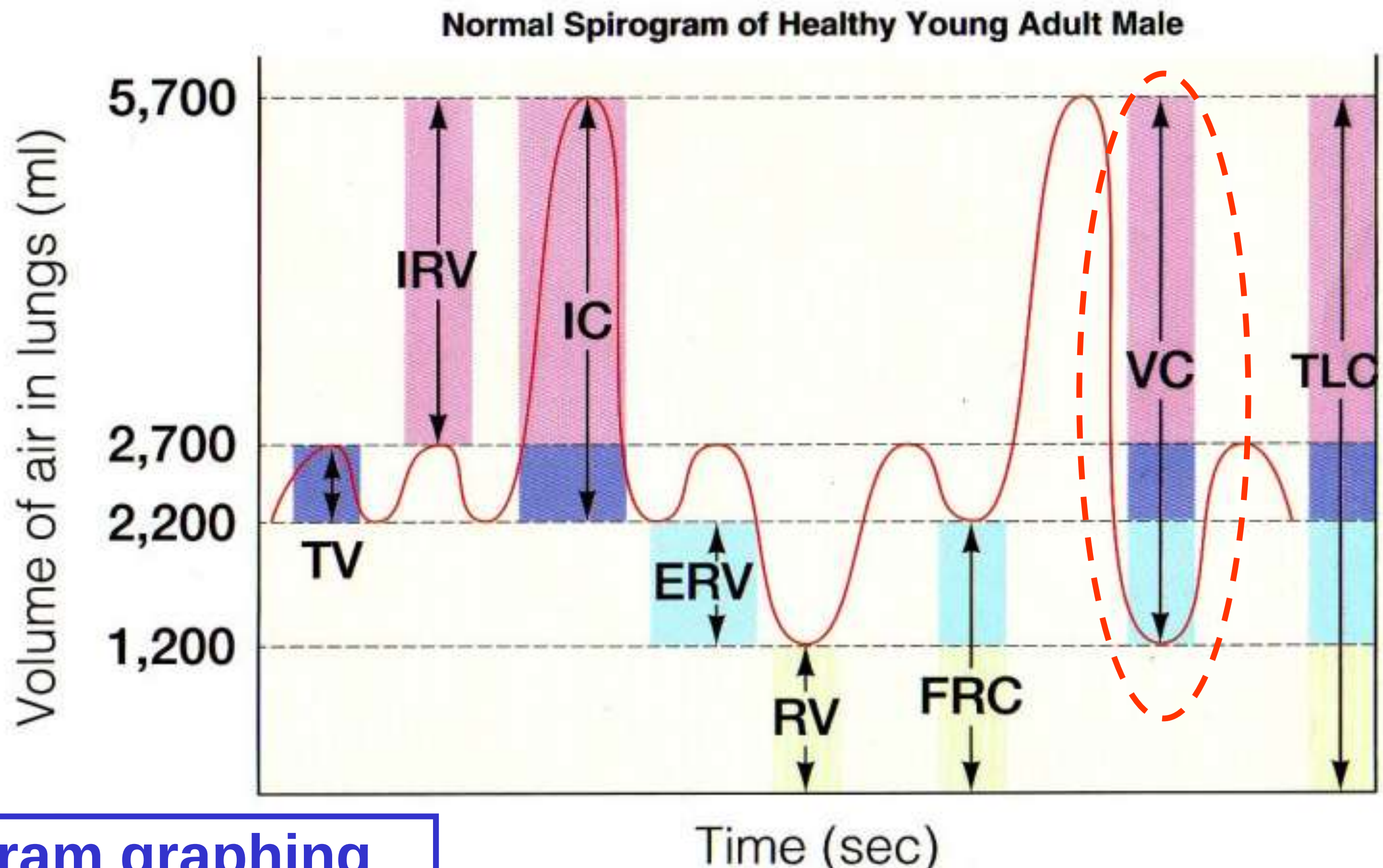
Normal = Steep



Abnormal = Flatter Downslope (eg, Asthma)

PFT **→ measures all lung volumes & capacities (sum of ≥ 2 volumes). Subject relaxes & breathes normally into and out of tank.**





Spirogram graphing complete *PFT* from computer simulation.

TV = Tidal volume (500 ml)
IRV = Inspiratory reserve volume (3,000 ml)
IC = Inspiratory capacity (3,500 ml)
ERV = Expiratory reserve volume (1,000 ml)
RV = Residual volume (1,200 ml)
FRC = Functional residual capacity (2,200 ml)
VC = Vital capacity (4,500 ml)
TLC = Total lung capacity (5,700 ml)

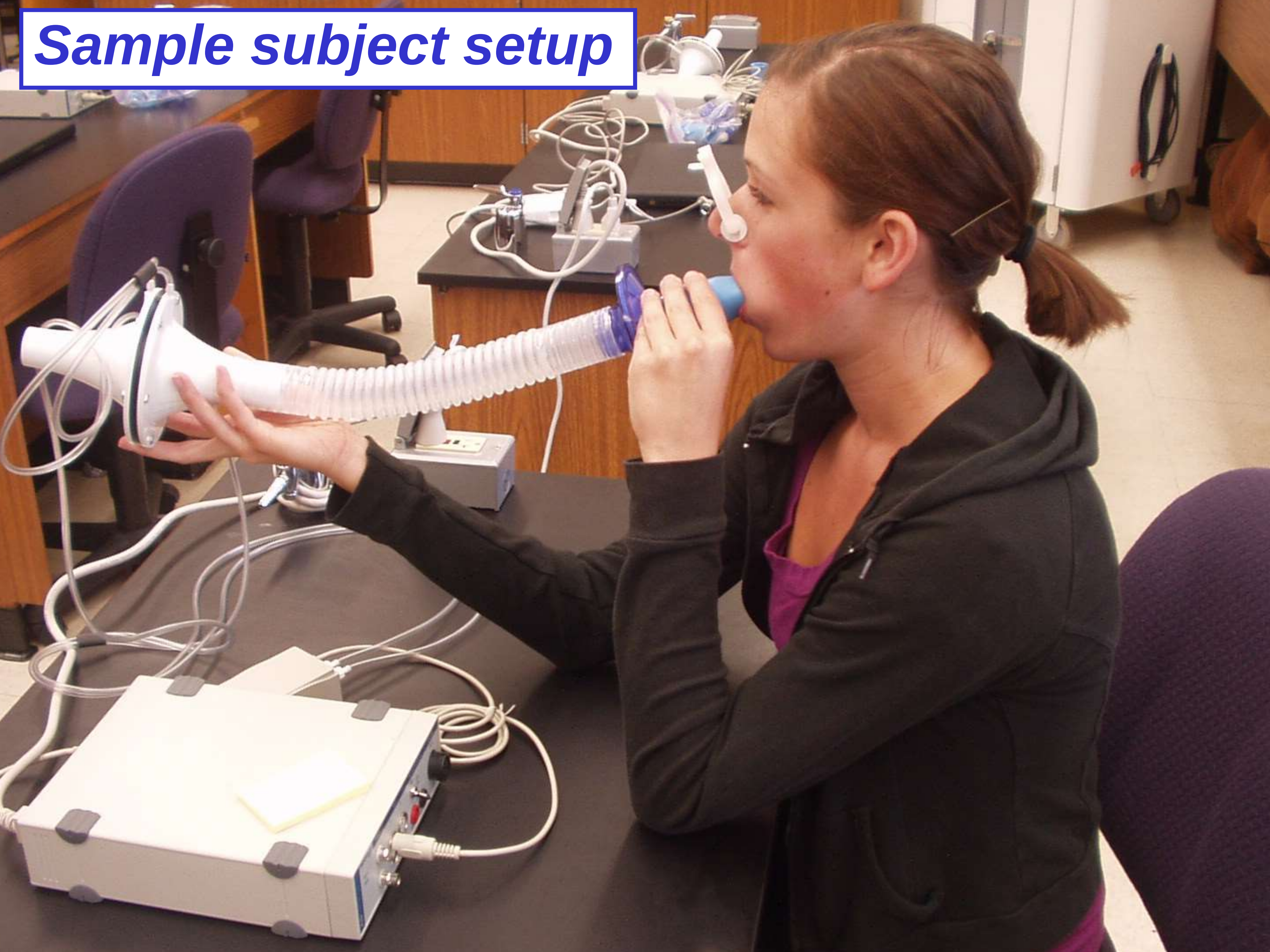
More modern-day computerized Pulmonary Function Testing

Complete with HH!
Happy Helpers!

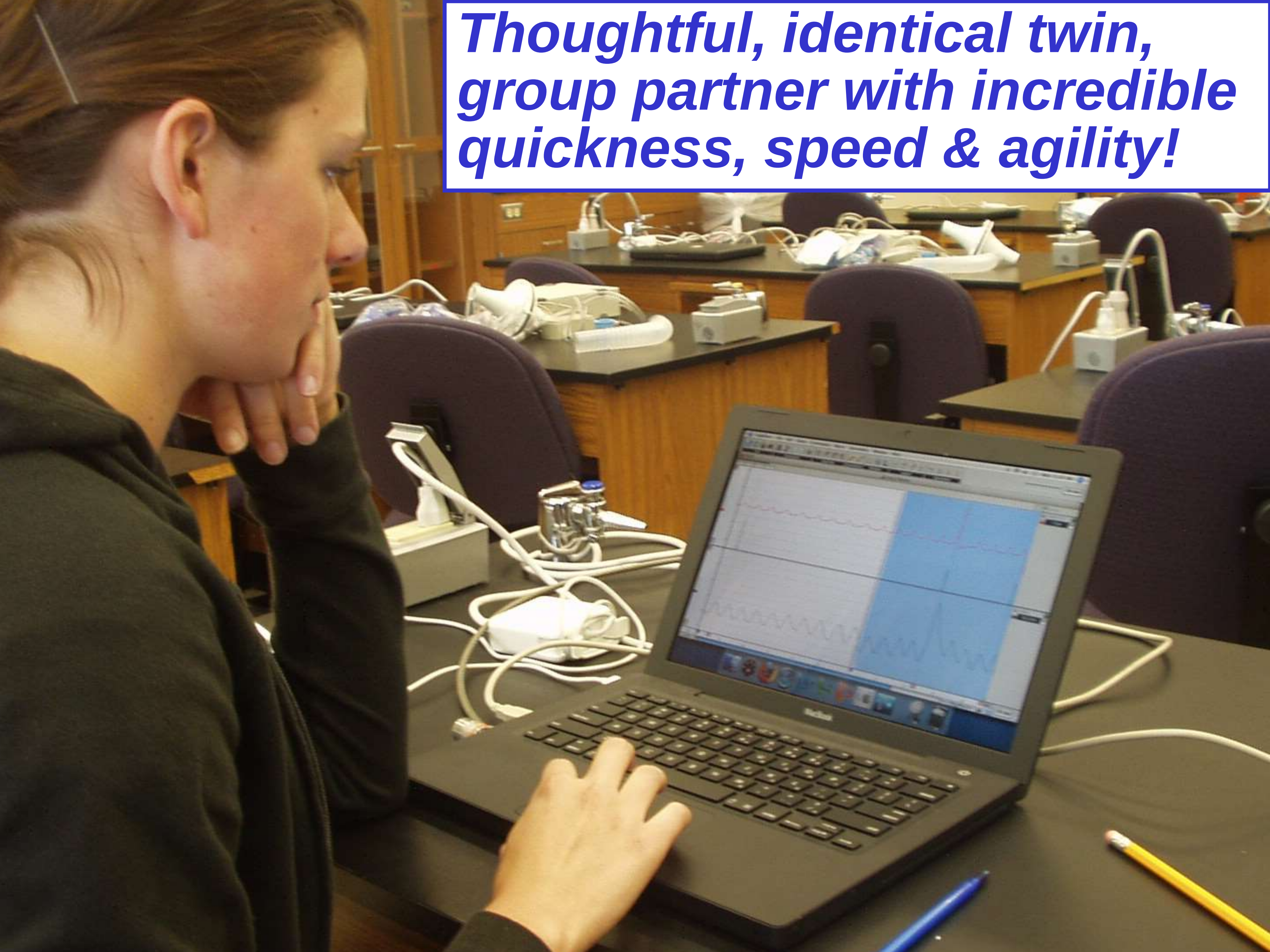

How to put together?

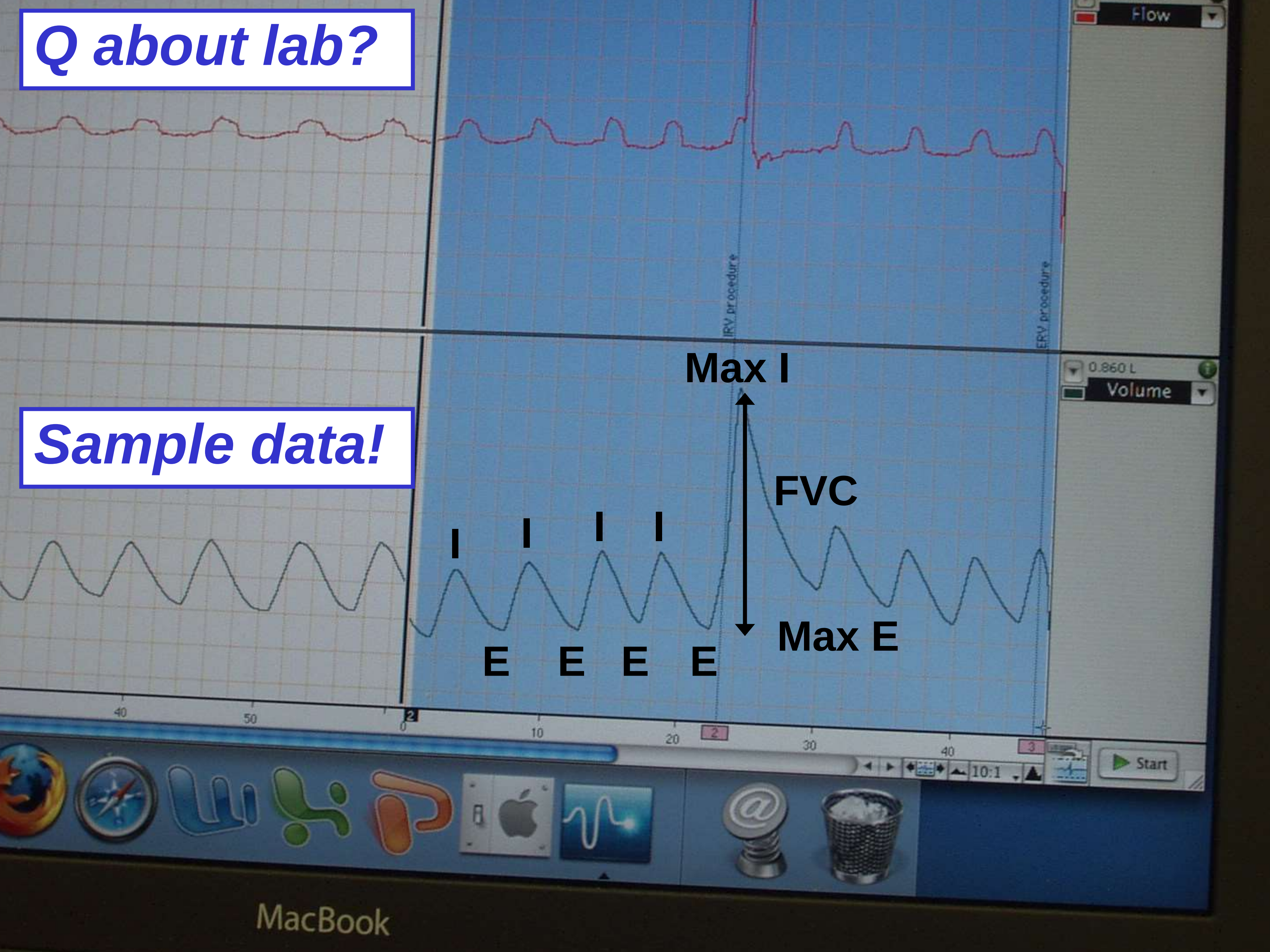

Viola!!

Sample subject setup



Thoughtful, identical twin, group partner with incredible quickness, speed & agility!




Q about lab?
Sample data!
Flow
0.860 L
Volume
IRV procedure
ERV procedure
Max I
FVC
Max E
I I I I
E E E E
40
50
0
10
20
30
40
Start
10:1
MacBook

Whole Muscle

↑

Myocyte or Muscle Fiber

↑

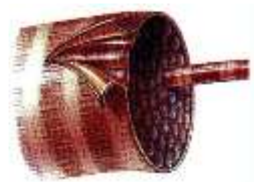
Myofibril

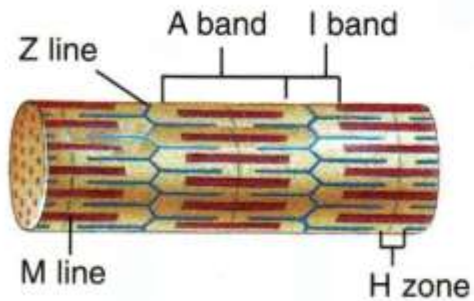
↑

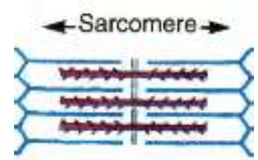
Thick & Thin Filaments

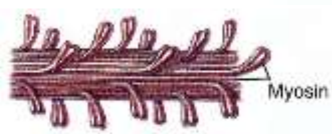
↑

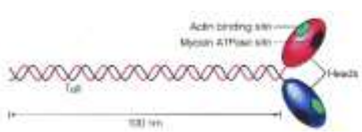
Myosin & Actin











Organ

↑

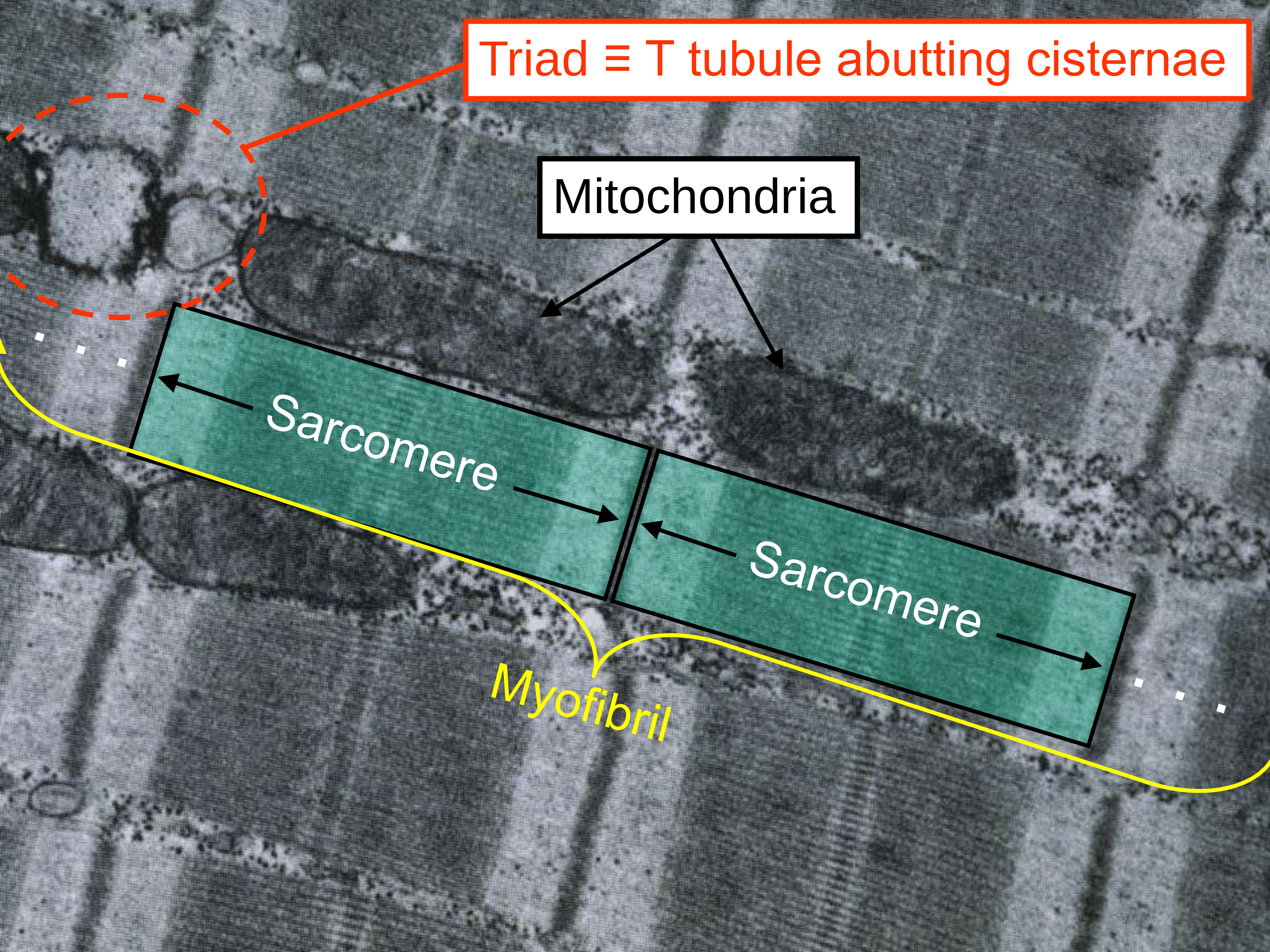
Cell

↑

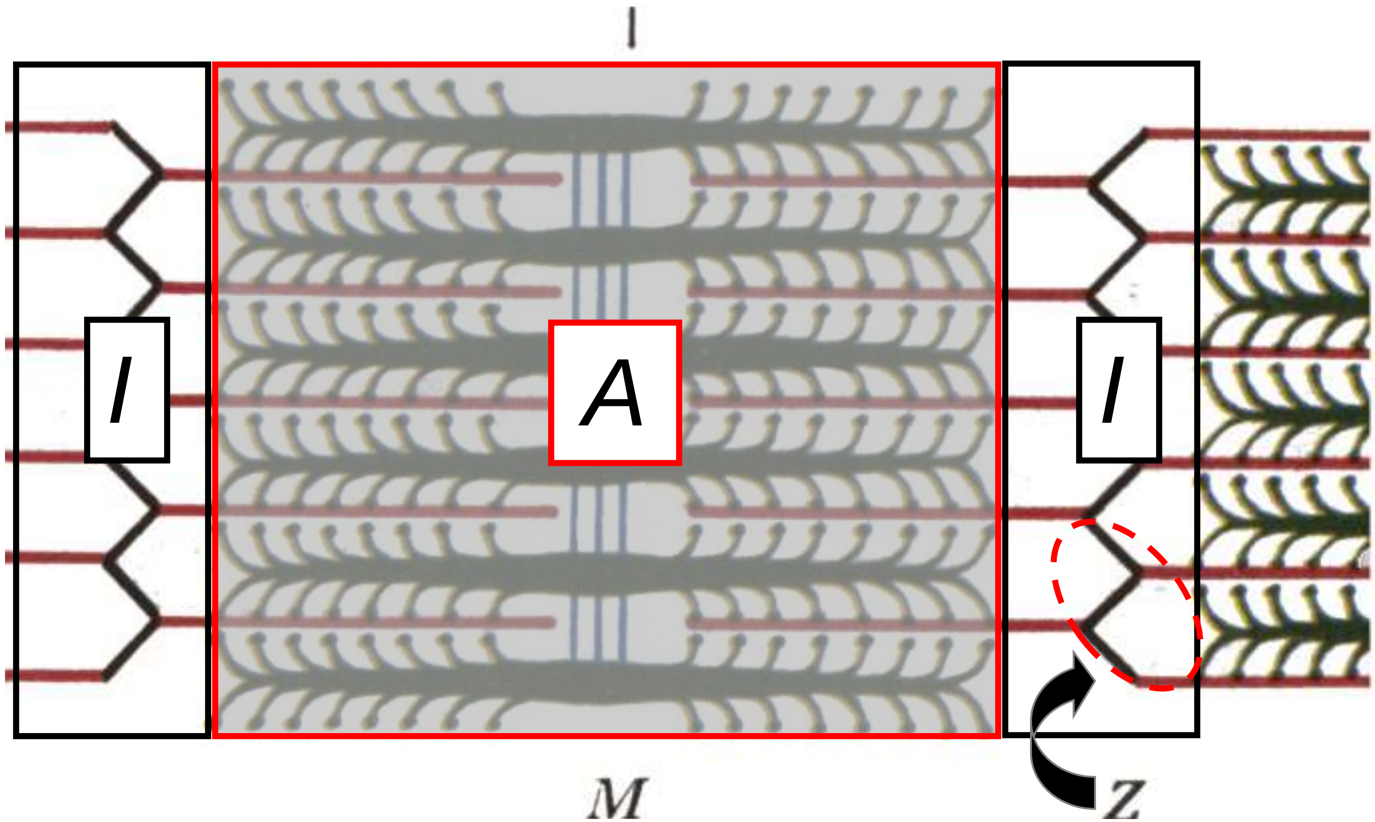
Cytoskeleton

↑

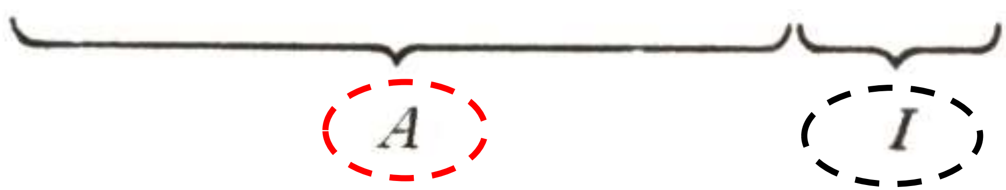
Molecules

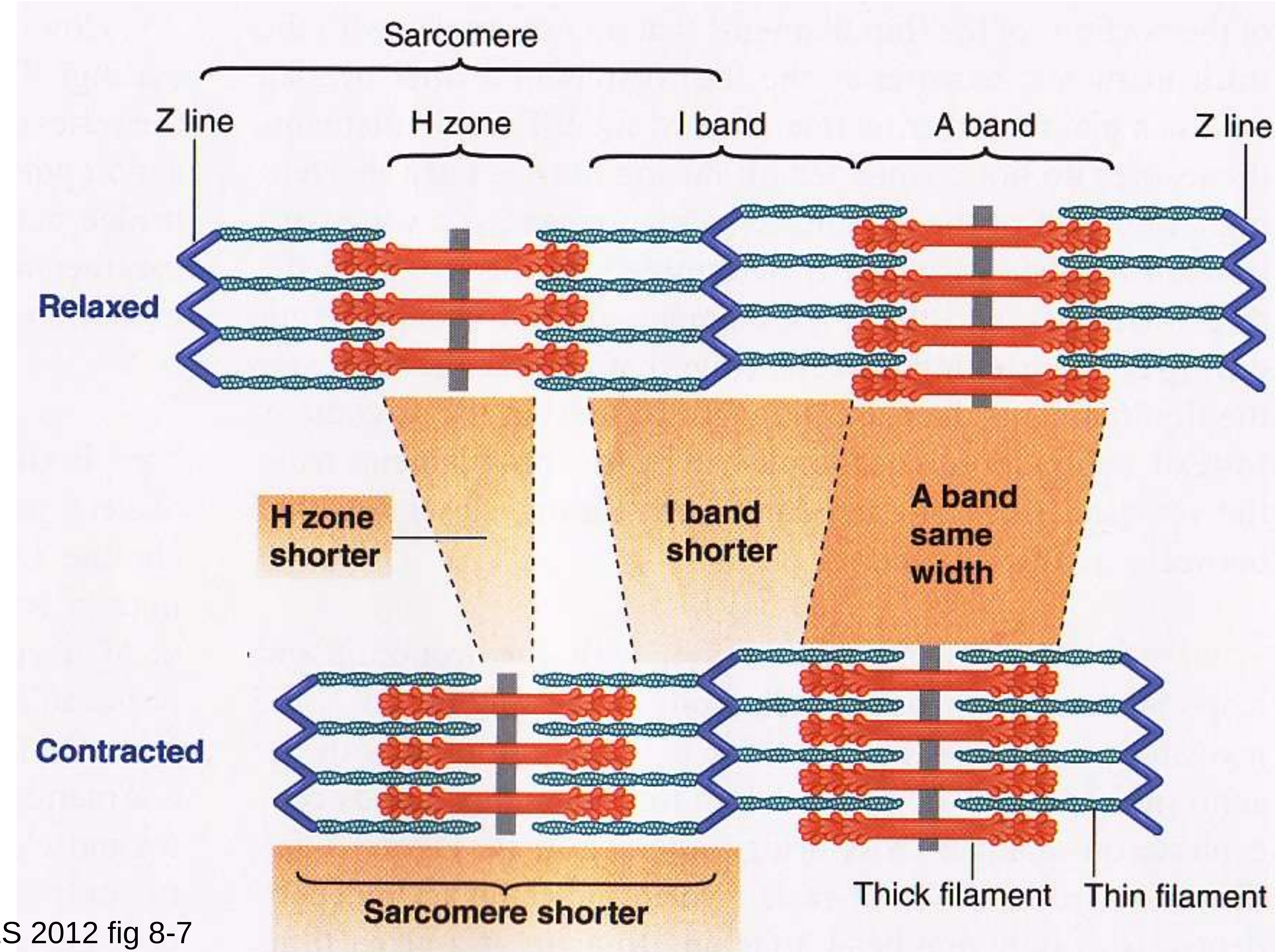

Triad ≡ T tubule abutting cisternae
Mitochondria
Sarcomere
Sarcomere
Myofibril

A Band = Dark Band
*A*nisotropic = Light Can't Shine Through



I Band = Light Band
*I*sotropic = Light Can Shine Through

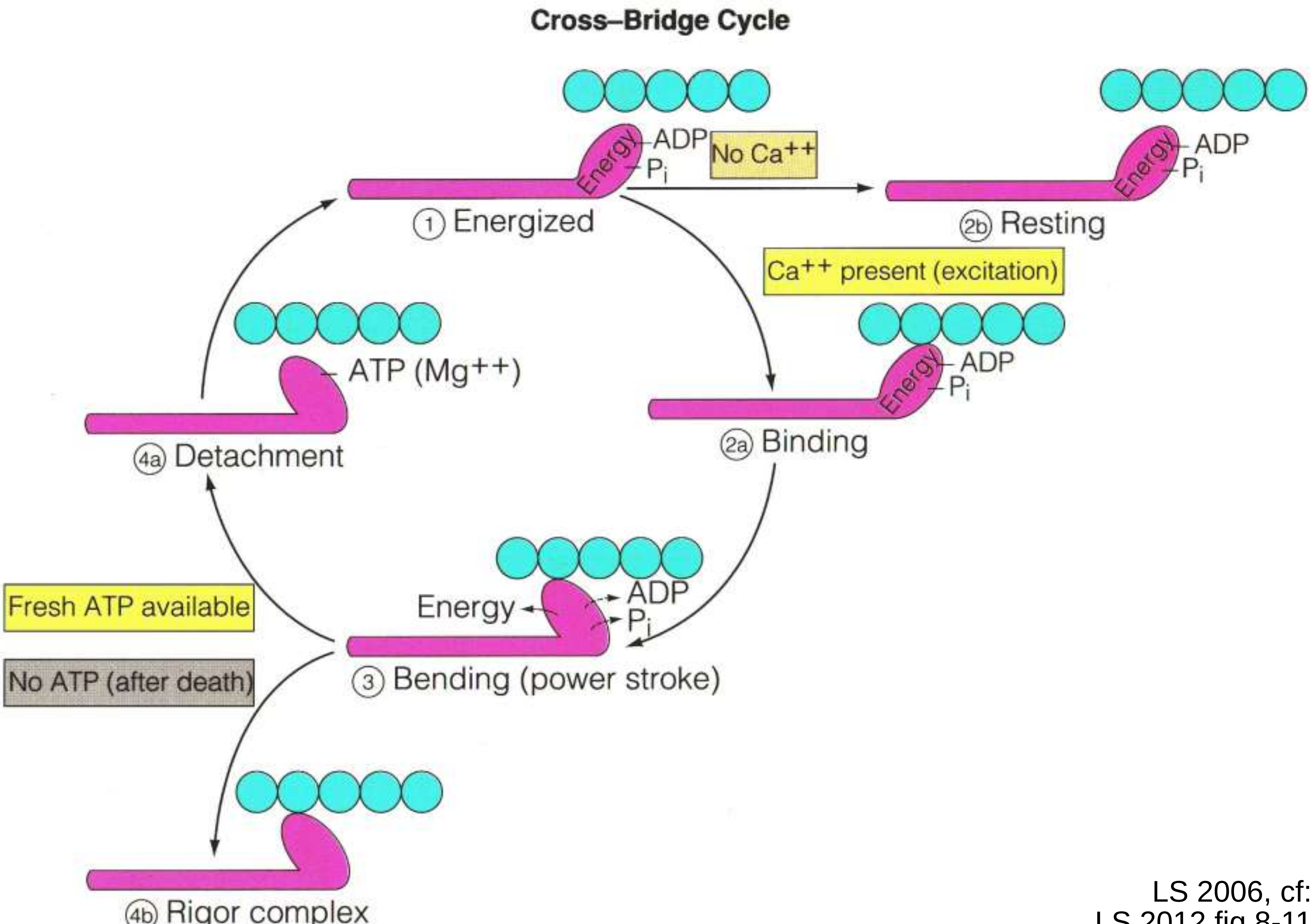




LS 2012 fig 8-7

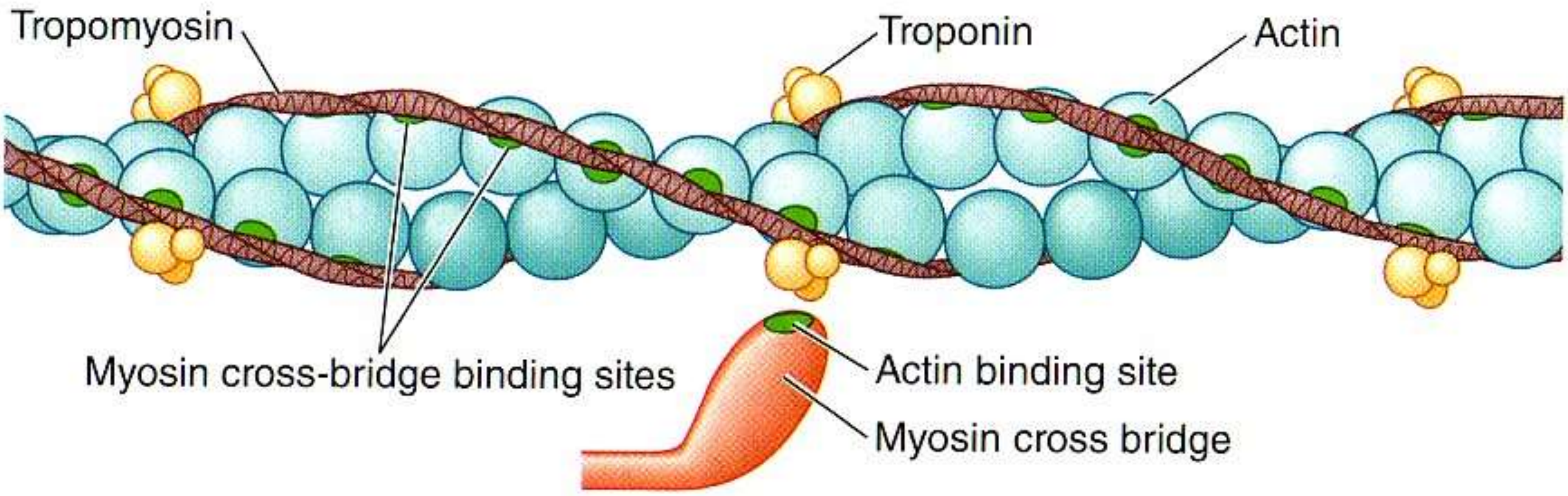
Discussion + Time for Questions!

What do we guess happens at the <u>molecular</u> <u>level</u>?



LS 2006, cf:
LS 2012 fig 8-11

Relaxed: No Cross-Bridge Binding



(a) Relaxed

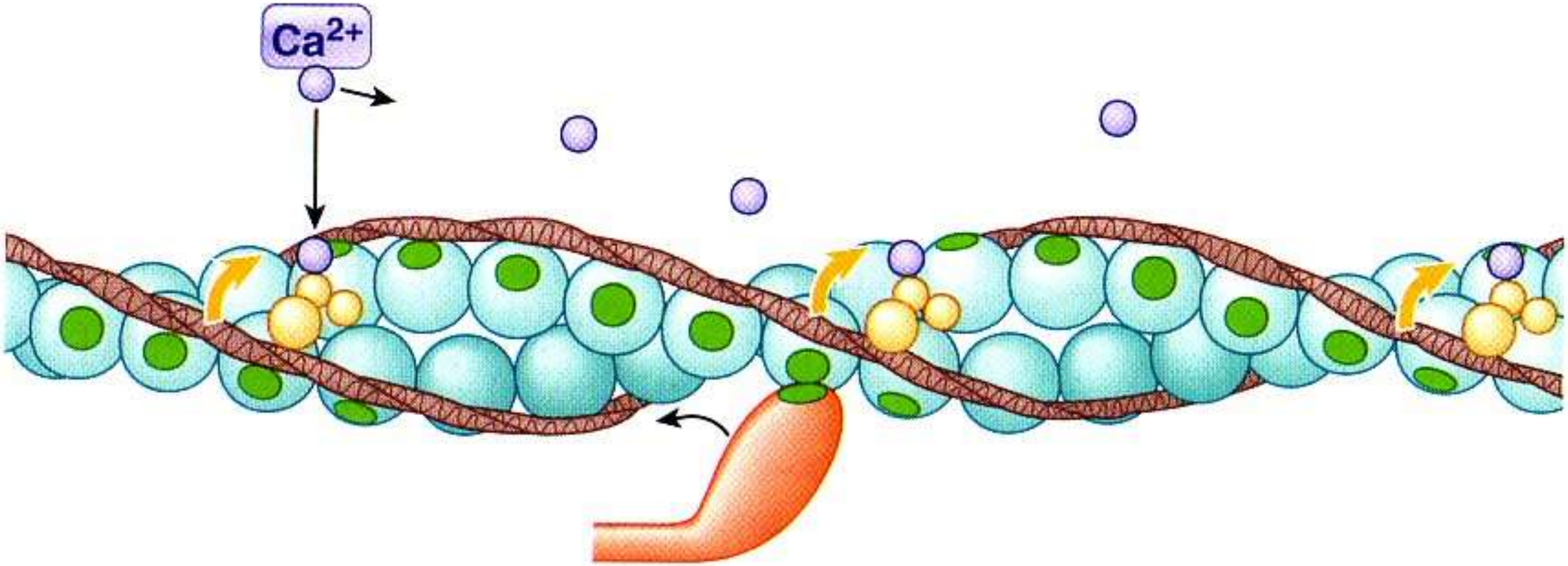
1 No excitation.

2 No cross-bridge binding because cross-bridge binding site on actin is physically covered by troponin–tropomyosin complex.

3 Muscle fiber is relaxed.

LS 2012 fig 8-6a

Excited: Calcium Triggers Cross-Bridge Binding



(b) Excited

1. Muscle fiber is excited and Ca^{2+} is released.

2. Released Ca^{2+} binds with troponin, pulling troponin–tropomyosin complex aside to expose cross-bridge binding site.

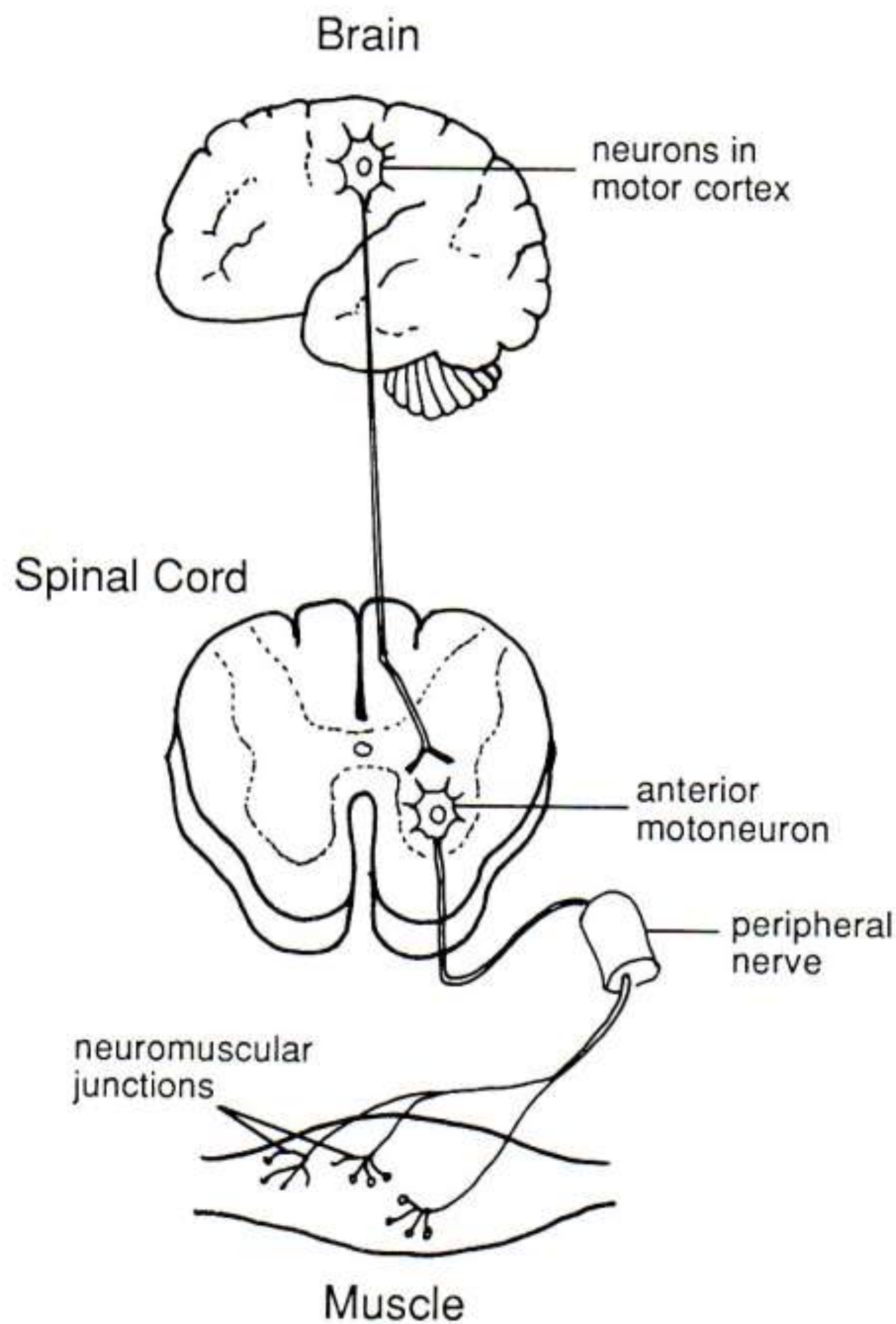
3. Cross-bridge binding occurs.

4. Binding of actin and myosin cross bridge triggers power stroke that pulls thin filament inward during contraction.

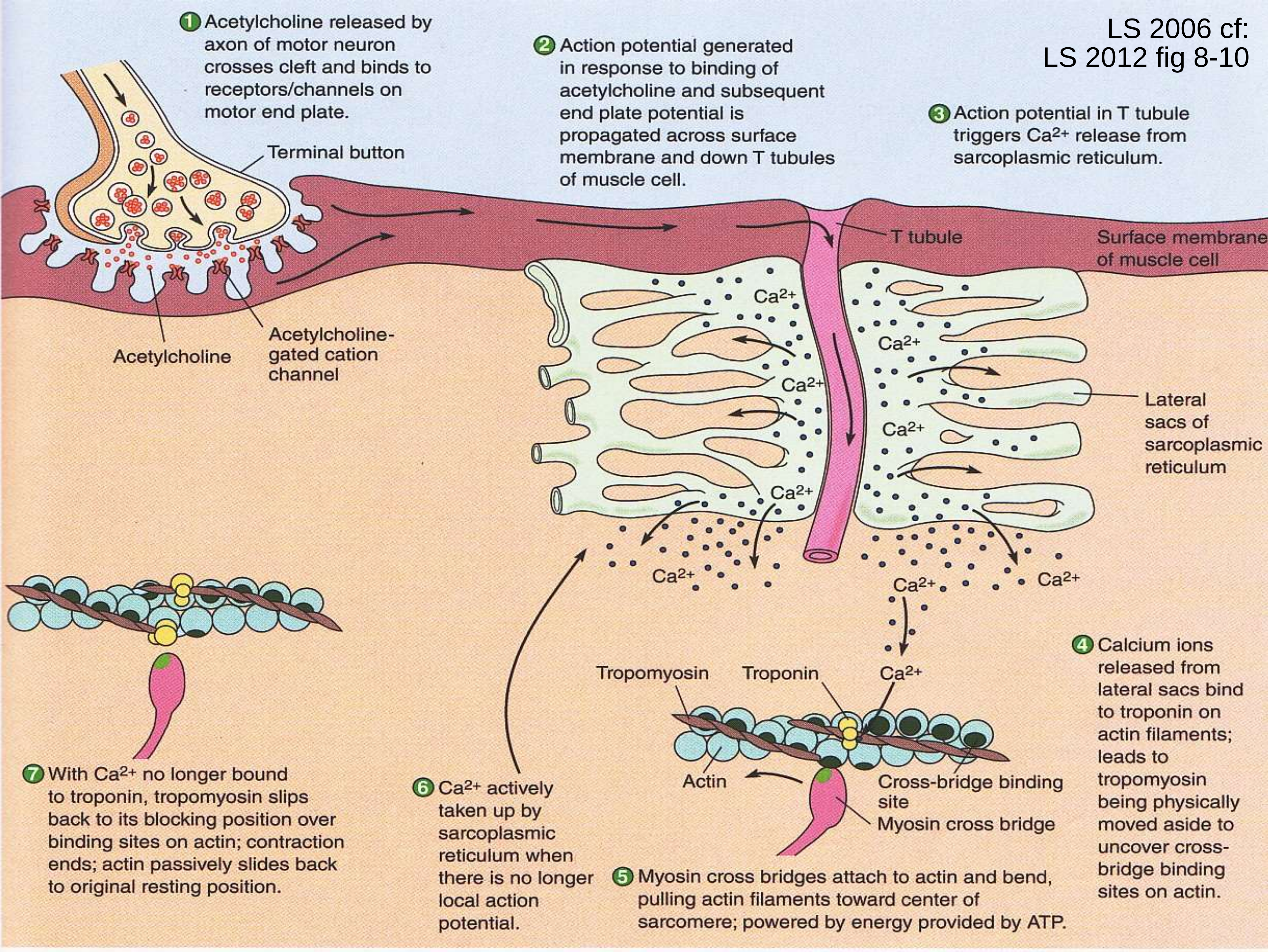
LS 2012 fig 8-6b

Rope Climb or Tug of War
Grasp, then Regrasp!

https://www.youtube.com/watch?v=Ktv-CaOt6UQ



DN Laing & VP Lombardi, 1989


LS 2006 cf:
LS 2012 fig 8-10
1 Acetylcholine released by axon of motor neuron crosses cleft and binds to receptors/channels on motor end plate.
Terminal button
Acetylcholine
Acetylcholine-gated cation channel
2 Action potential generated in response to binding of acetylcholine and subsequent end plate potential is propagated across surface membrane and down T tubules of muscle cell.
3 Action potential in T tubule triggers Ca2+ release from sarcoplasmic reticulum.
T tubule
Surface membrane of muscle cell
Ca2+
Lateral sacs of sarcoplasmic reticulum
Tropomyosin
Troponin
Actin
Cross-bridge binding site
Myosin cross bridge
4 Calcium ions released from lateral sacs bind to troponin on actin filaments; leads to tropomyosin being physically moved aside to uncover cross-bridge binding sites on actin.
5 Myosin cross bridges attach to actin and bend, pulling actin filaments toward center of sarcomere; powered by energy provided by ATP.
6 Ca2+ actively taken up by sarcoplasmic reticulum when there is no longer local action potential.
7 With Ca2+ no longer bound to troponin, tropomyosin slips back to its blocking position over binding sites on actin; contraction ends; actin passively slides back to original resting position.

Muscle Contraction Resources

https://ed.ted.com/lessons/how-your-muscular-system-works-emma-bryce

https://ed.ted.com/on/s3Zzdm8u

https://ed.ted.com/lessons/what-makes-muscles-grow-jeffrey-siegel

https://www.ncbi.nlm.nih.gov/books/NBK9961/

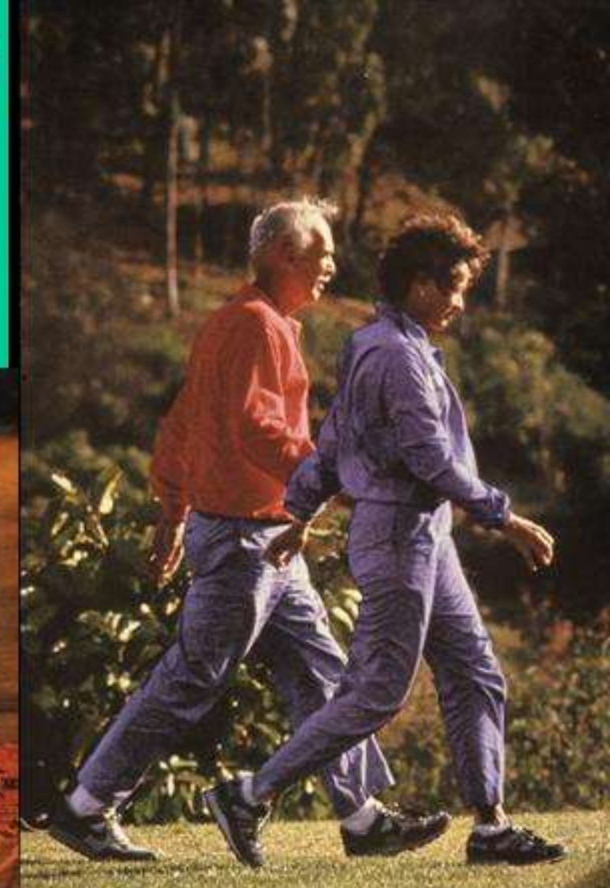
A. Malcolm Campbell
Davidson College, Davidson, NC
www.bio.davidson.edu/courses/movies.html

David Bolinsky, XVIVO
Rocky Hill, CT
http://www.xvivo.net/

Adaptations to Exercise?

Mode, Intensity, Duration, Frequency,
Distribution of Training Sessions?
Conditions of Environment? Individual?

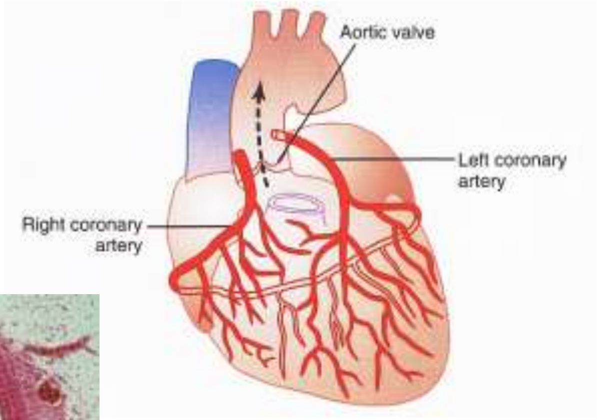


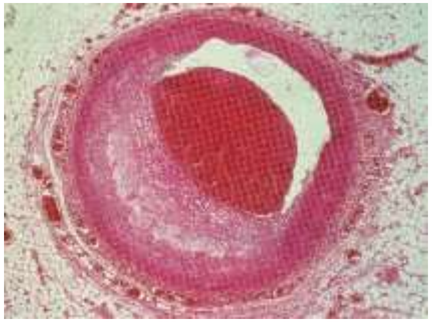

Adaptations to Exercise?

Body Levels of Organization?
Which Body System?

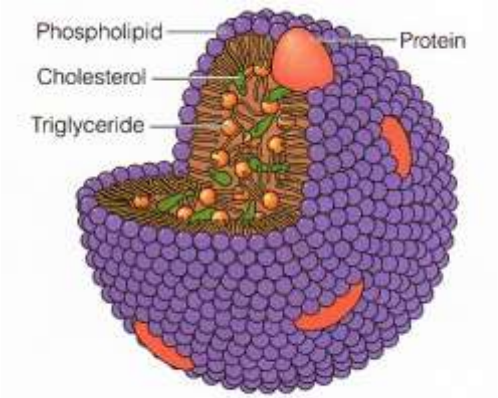
Body System



Organ



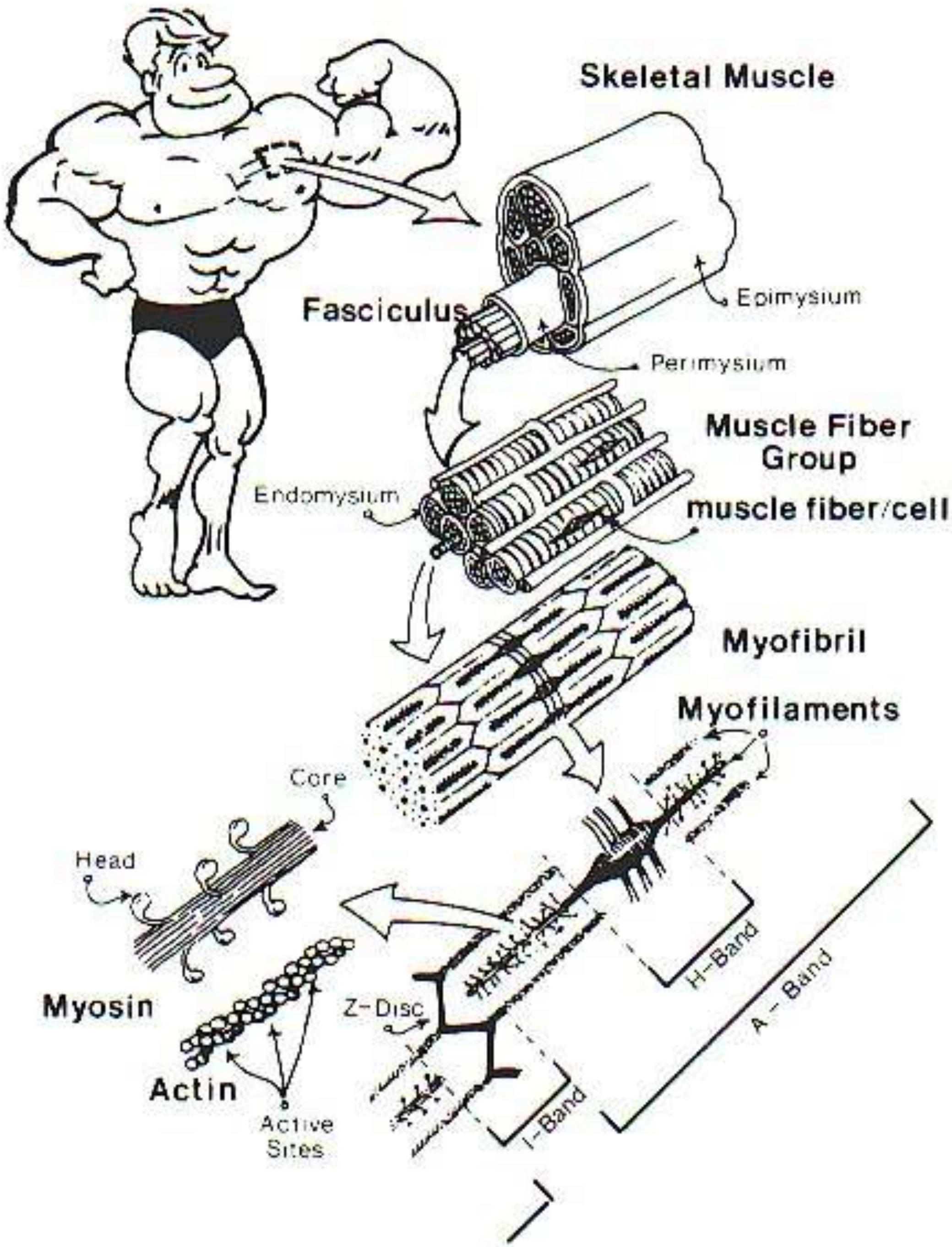
Cell/Tissue



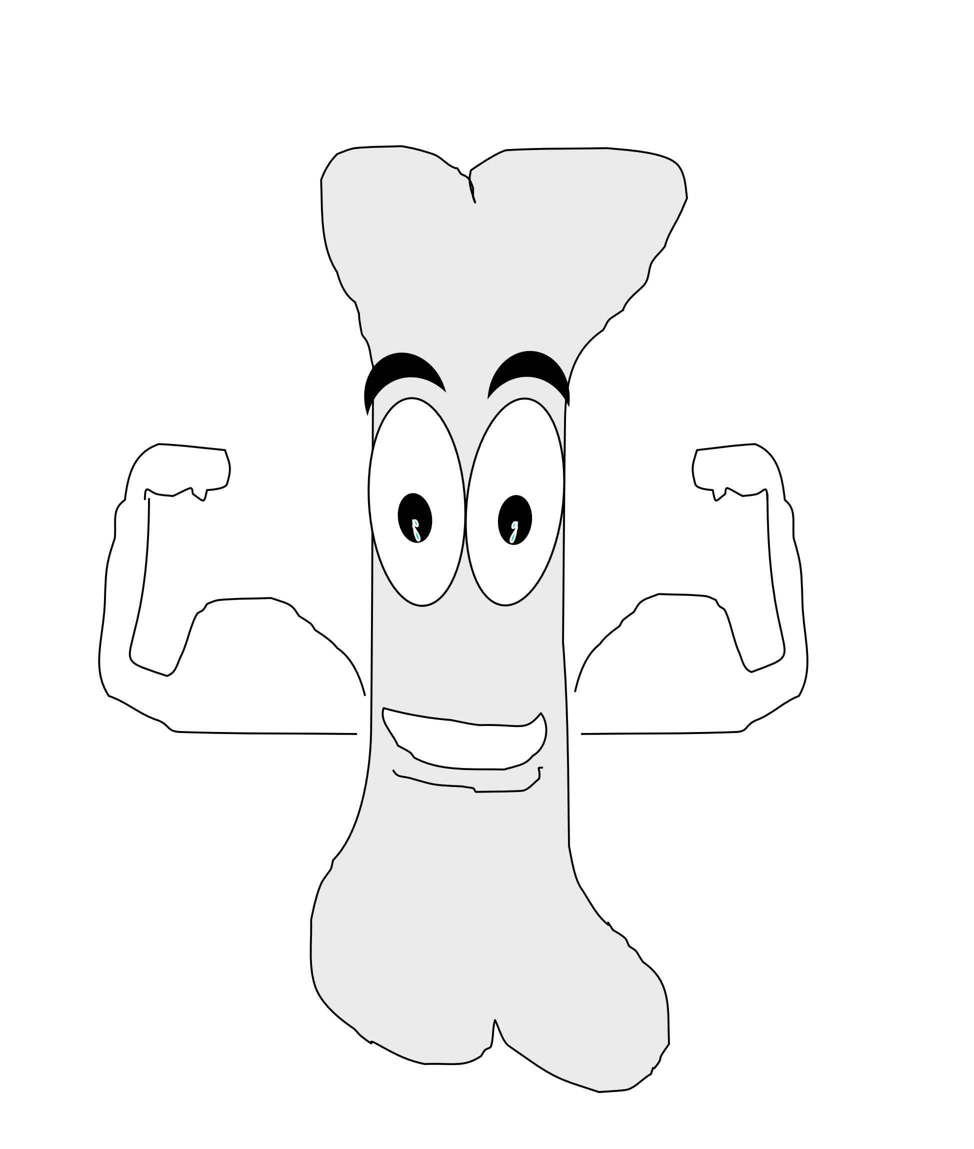
A typical lipoprotein

Molecular

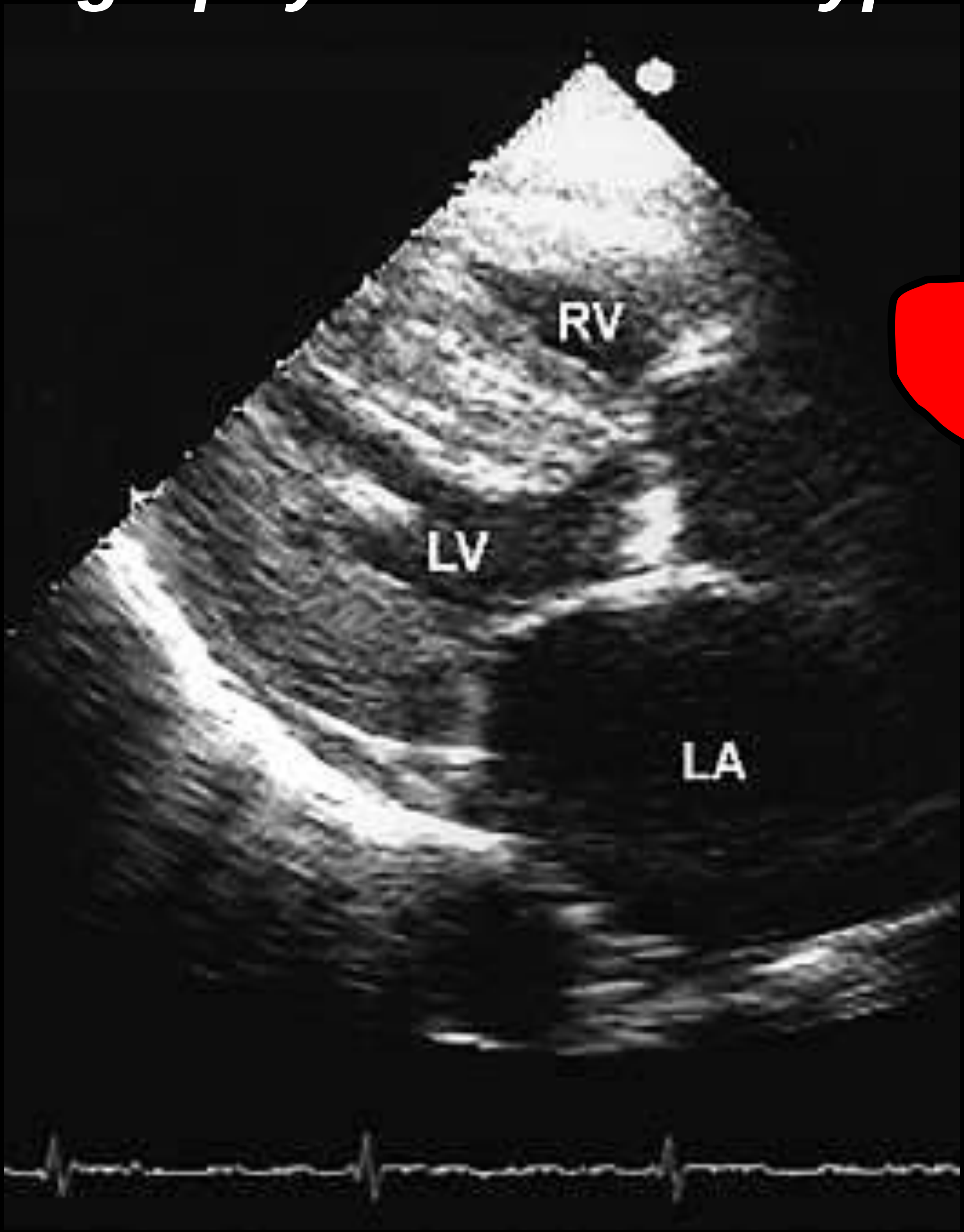
Muscle Adaptations to Exercise



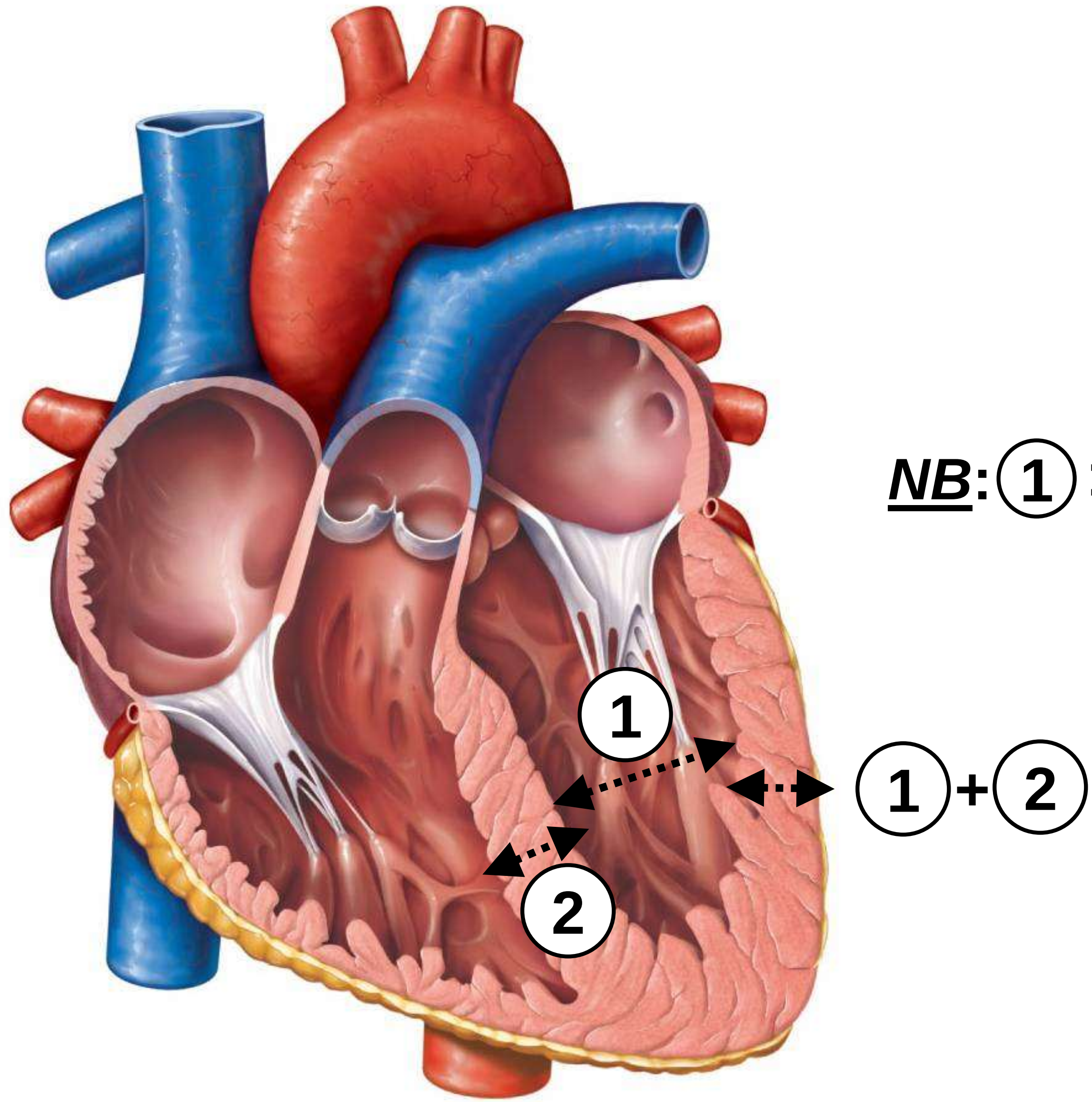
As muscles tug on bones, bones get stronger, too!...many systems adapt!!



Echocardiography documents hypertrophy...



Cardiac Adaptations to Exercise:
① Endurance vs. ② StrengthTraining



NB: ① > ↑ LBM

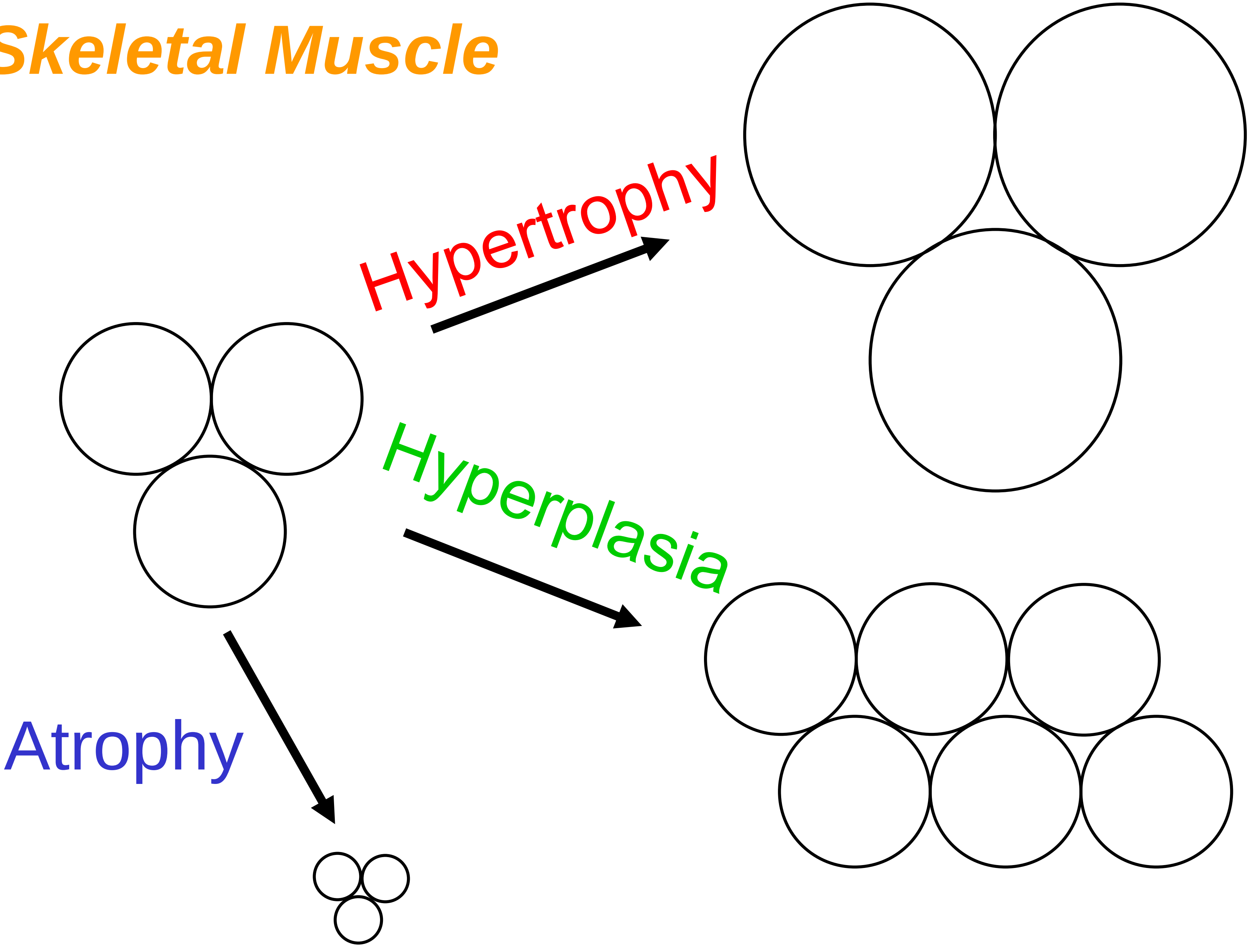
Atrophy

decrease in size & strength

Hypertrophy

increase in size & strength

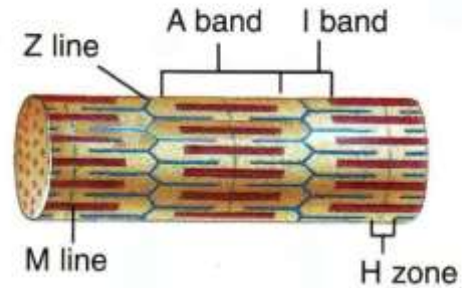
Skeletal Muscle



Women & Hypertrophy?

What happens in muscles at cellular & subcellular levels?

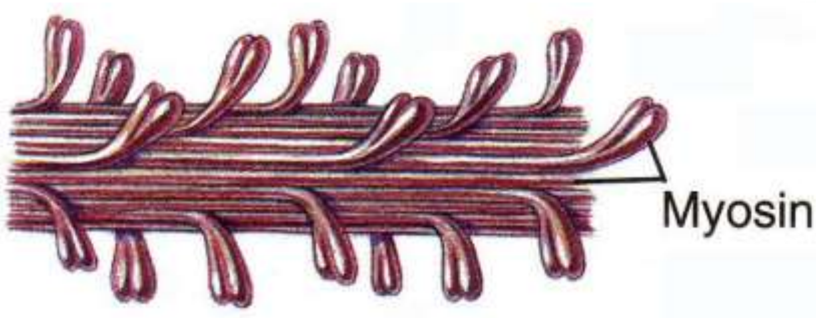
<u>Hypertrophy</u>: *Increased*

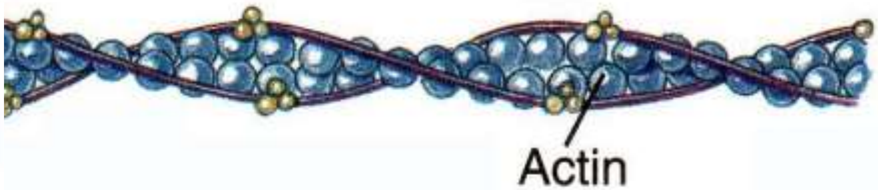


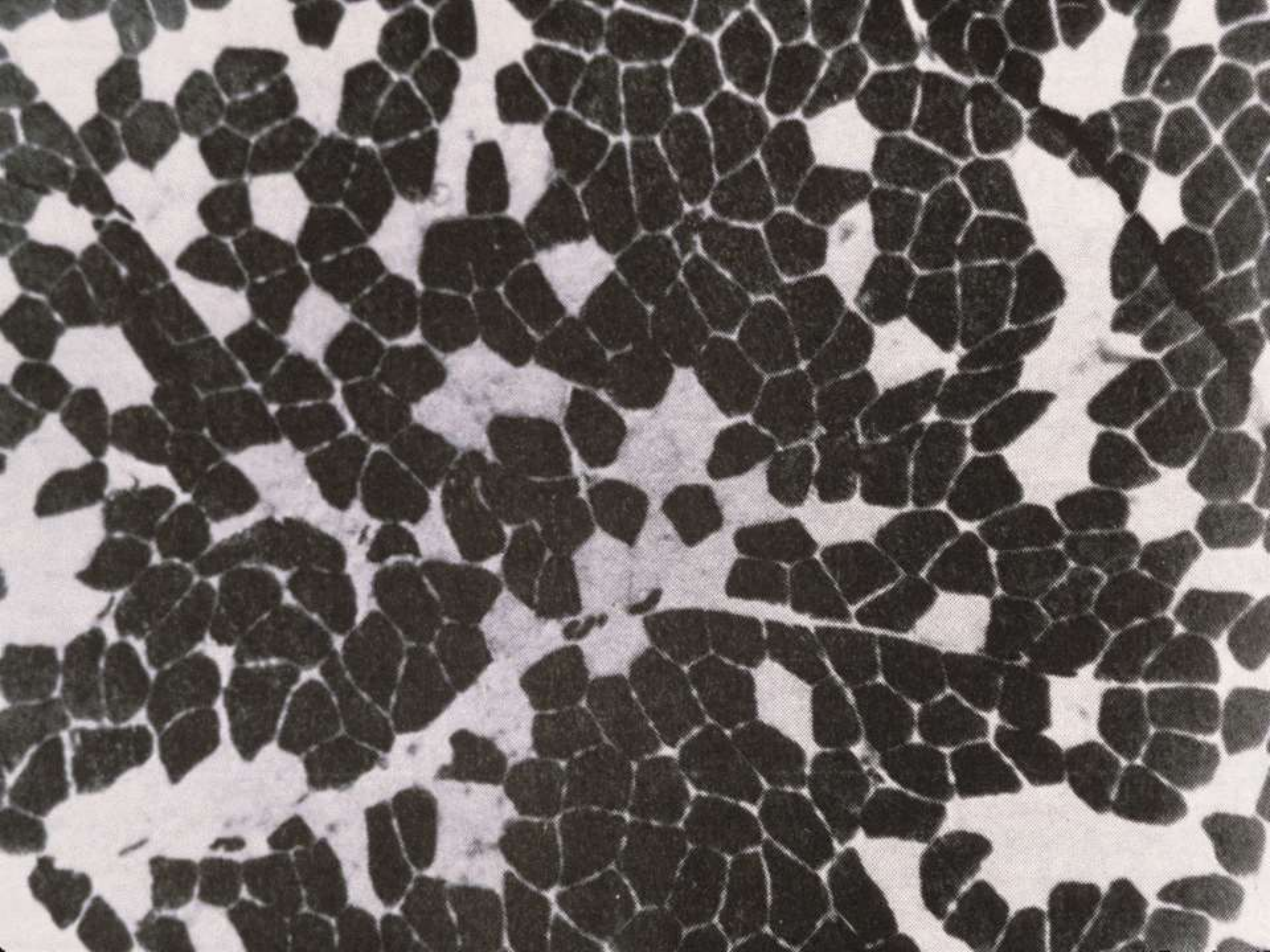
Number of Myofibrils

Thick & Thin Filaments

Myosin & Actin Molecules







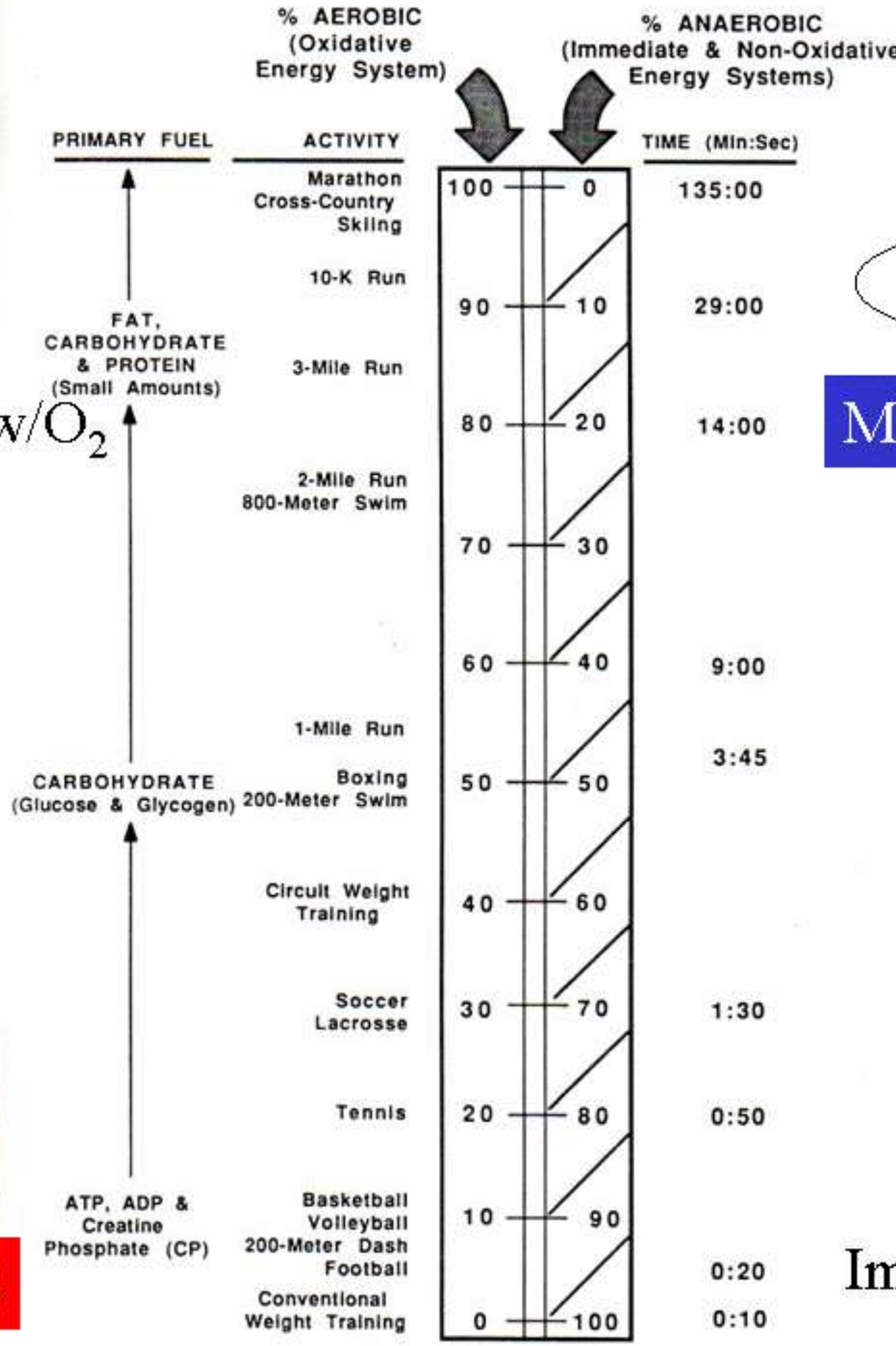
Characteristics of Skeletal Muscle Fibers

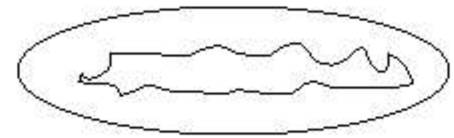
Characteristic	TYPE OF FIBER Slow Oxidative (Type I)	Fast Oxidative (Type IIa)	Fast Glycolytic (Type IIb)
Myosin-ATPase Activity	Low	High	High
Speed of Contraction	Slow	Fast	Fast
Resistance to Fatigue	High	Intermediate	Low
Aerobic Capacity	High	High	Low
Anaerobic Capacity	Low	Intermediate	High
Mitochondria	Many	Many	Few
Capillaries	Many	Many	Few
Myoglobin Content	High	High	Low
Color of Fibers	Red	Red	White
Glycogen Content	Low	Intermediate	High

LS 2012 tab 8-1 modified
> VP Lombardi 1989

AEROBIC w/O_2

ANAEROBIC





MITOCHONDRIA

CYTOSOL

Glycolysis

↑

Immediate/ATP-PC

Extremes of the energy continuum!

Changes in Muscle Due to Strength Training

- ↑ Size of larger fast vs smaller slow fibers
- ↑ CP as well as creatine phosphokinase (CPK) which enhances short-term power output
- ↑ Key enzymes which help store and dissolve sugar including glycogen phosphorylase (GPP) & phosphofructokinase (PFK)
- ↓ Mitochondrial # relative to muscle tissue
- ↓ Vascularization relative to muscle tissue
- ↑ Splitting of fast fibers? Hyperplasia? With growth hormone (GH), androgenic-anabolic steroids (AAS)?

Changes in Muscle Due to <u>Endurance Training</u>

- ↑ Mitochondria, # & size
- ↑ Mitochondrial (aerobic) enzymes including those specific for fat burning
- ↑ Vascularization of muscles (better blood flow)
- ↑ Stores of fat in muscles accompanied by
- ↓ Triglycerides/fats in bloodstream
- ↑ Enzymes: activation, transport, breakdown (β-oxidation) of fatty acids
- ↑ Myoglobin (enhances O_2 transport)
- ↑ Resting energy levels which inhibit sugar breakdown
- ↑ Aerobic capacity of all three fiber types.

Which end of continuum?

+

Which energy nutrient/s?

+ Which specific muscles?

cf:


45





Dancing can be super aerobic exercise, too, & you don't have to be a star!