HESP 147

Review for Exam 2

TOPICS:

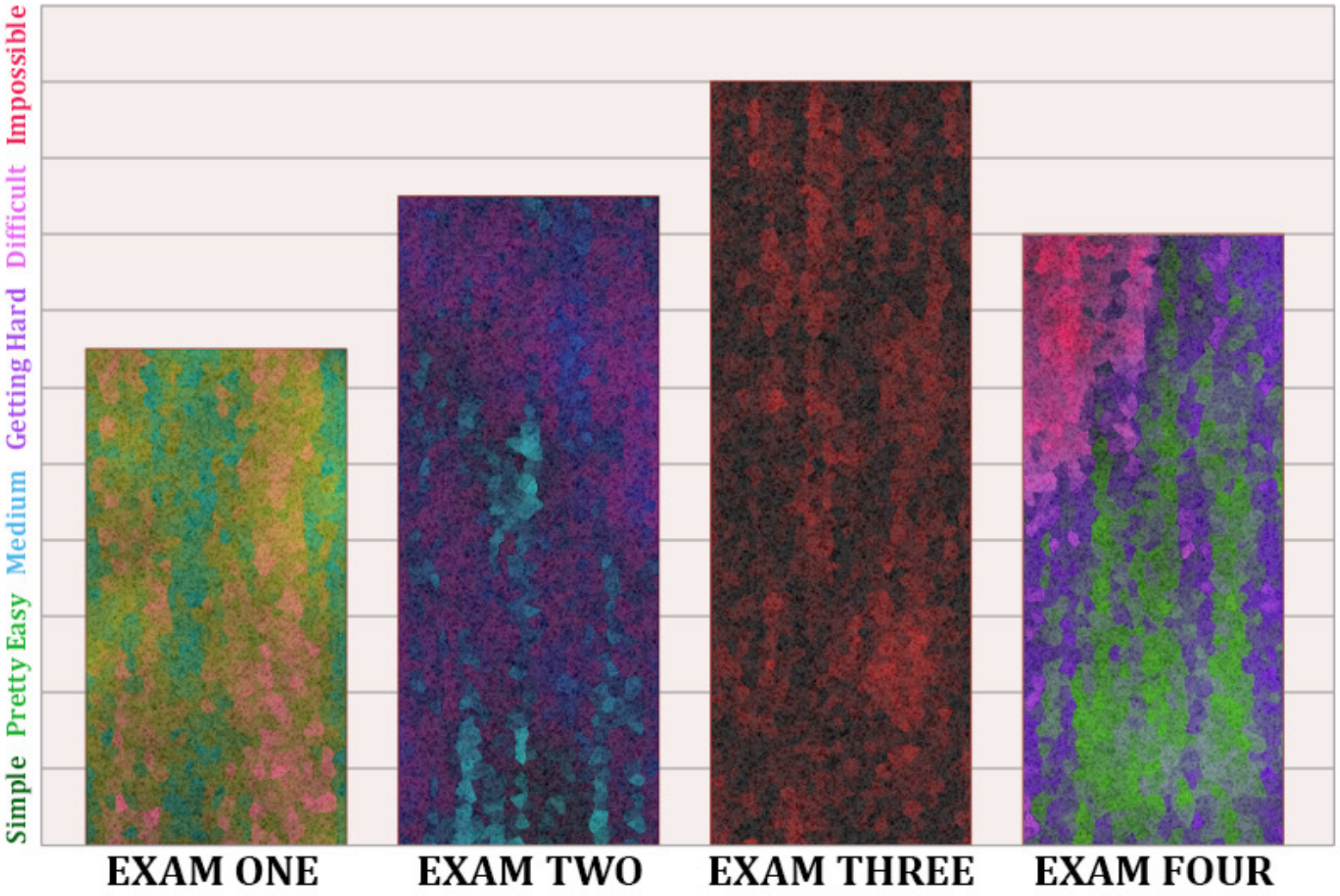
Biomechanics,

Barefoot Running and the Minimalist Movement,

Nociceptor Depolarization

Inflammatory Physiology,

and Tissue Healing


Simple
Pretty Easy
Medium
Getting Hard
Difficult
Impossible
EXAM ONE
EXAM TWO
EXAM THREE
EXAM FOUR

PART ONE

What is Fitness?

University of the Pacific HESP 147

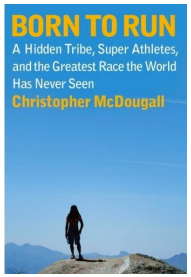
NATURE | VOL 432 | 18 NOVEMBER 2004

Endurance running and the evolution of *Homo*

Dennis M. Bramble[1] & Daniel E. Lieberman[2]

Daniel Lieberman (Harvard anthropologist):

"A lot of foot and knee injuries that are currently plaguing us are actually caused by people running with shoes that actually make our feet weak, cause us to over-pronate, give us knee problems. Until 1972, when the modem athletic shoe was invented by Nike, people ran in very thin-soled shoes, had strong feet, and had much lower incidence of knee injuries."



Christopher McDougall:

"Running shoes may be the most destructive force to ever hit the human foot."

www.runnersworld.com/rt-web-exclusive/daniel-lieberman-10-years-after-born-to-run

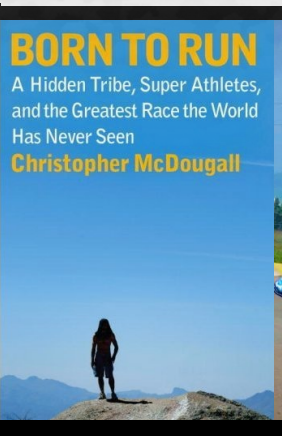
Daniel Lieberman, 10 Years After "Born to Run"

The Harvard evolutionary biologist whose work helped inspire the barefoot running movement talks about his own marathoning and what runners should take from science.

By **Richard A. Lovett** FRIDAY, AUGUST 29, 2014, 1:44 PM

Harvard professor Daniel Lieberman is an evolutionary biologist whose 2004 study on human evolution, "Endurance running and the evolution of *Homo*," caught international attention by arguing that among the animal kingdom, humans are supremely adapted distance runners. The study made the cover of the prestigious journal *Nature* under the title "Born to Run" and subsequently led to Lieberman's being a major figure in Christopher McDougall's bestselling book of the same title. Lieberman, a barefoot running aficionado, has also been strongly associated with the barefoot running boom that followed that book's publication. Writer Richard A. Lovett talked to him in August at an international running science symposium in Calgary, Alberta.

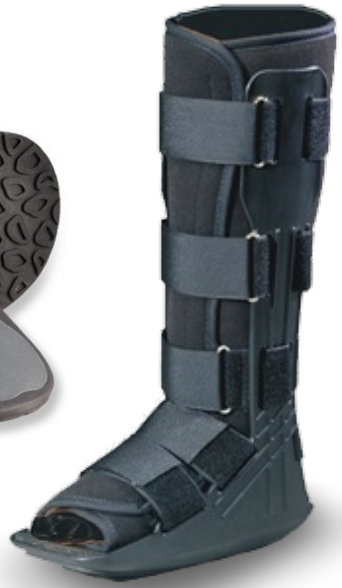
Tarahumara "Indians" (of Mexico's Copper Canyons) are urban legends incarnate.



No warm-ups or cool downs or stretching; just several-hundred-mile runs, feasts of barbecued mice, and excessive drinking (of liquor made from rattlesnake corpses).

Not on the test. Just a modern example of long distance minimalist running. Apparently, after death, they return as ghosts to collect all of the hairs they left behind.

Minimalist *vs* Maximalist



Br J Sports Med 2014;48:1257–1262.

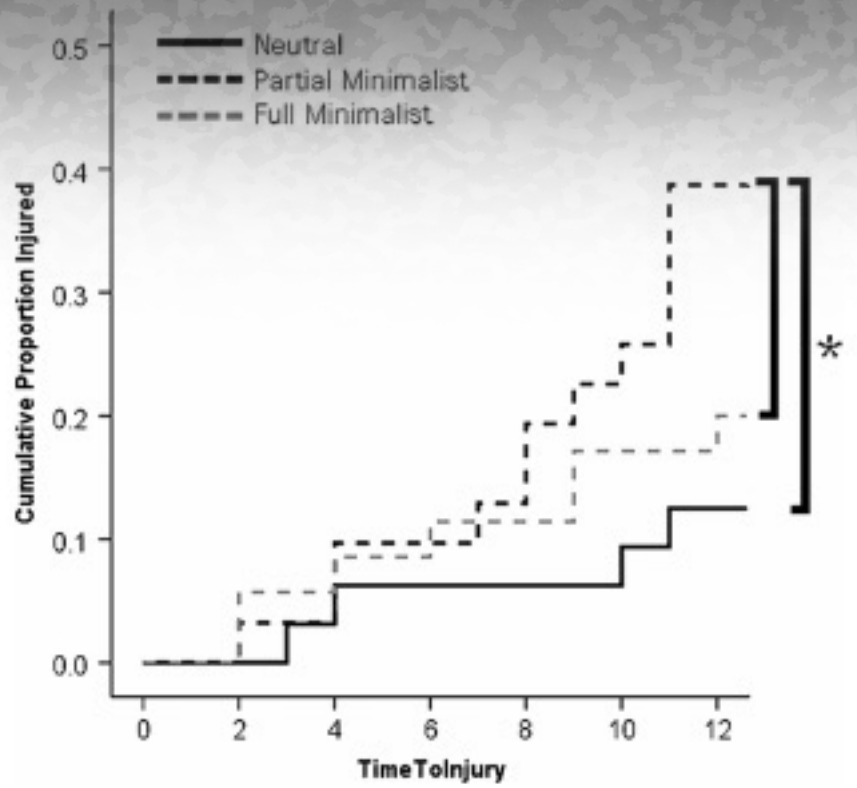


Figure 2 Kaplan-Meier plot of survival analysis illustrating the time sequence of injury for runners in the three footwear groups. *Indicates a significant difference across groups at $p<0.05$.

Examining injury risk and pain perception in runners using minimalist footwear

Michael Ryan,[1] Maha Elashi,[2] Richard Newsham-West,[1] Jack Taunton[2]

103 runners with neutral or mild pronation were randomly assigned a neutral (Nike Pegasus 28), partial minimalist (Nike Free 3.0 V2) or full minimalist (Vibram 5-Finger Bikila) shoe. They spent 12 weeks training for a 10k event.

What are the new findings?

- Both partial and full minimalist footwear designs resulted in a greater risk of injury compared with the neutral footwear group.
- The partial minimalist shoe resulted in a greater overall injury rate.
- Runners in the full minimalist shoe reported greater shin and calf pain than runners in both other footwear groups.

Timeline of the running shoe and the minimalist movement:

1970 — Formation of Nike; beginning of the running boom

1971 — First running-injury survey by *Runner's World*

1973 — Second running-injury survey by *Runner's World*

1977 — Addition of motion control and more cushioning into shoe

1979 — Third running injury survey by Dr Lloyd Smith

2005 — Introduction of the Nike Free

2006 — Introduction of the Vibram Five Fingers shoes; other minimal-footwear companies begin to proliferate

2009 — *Born to Run* published

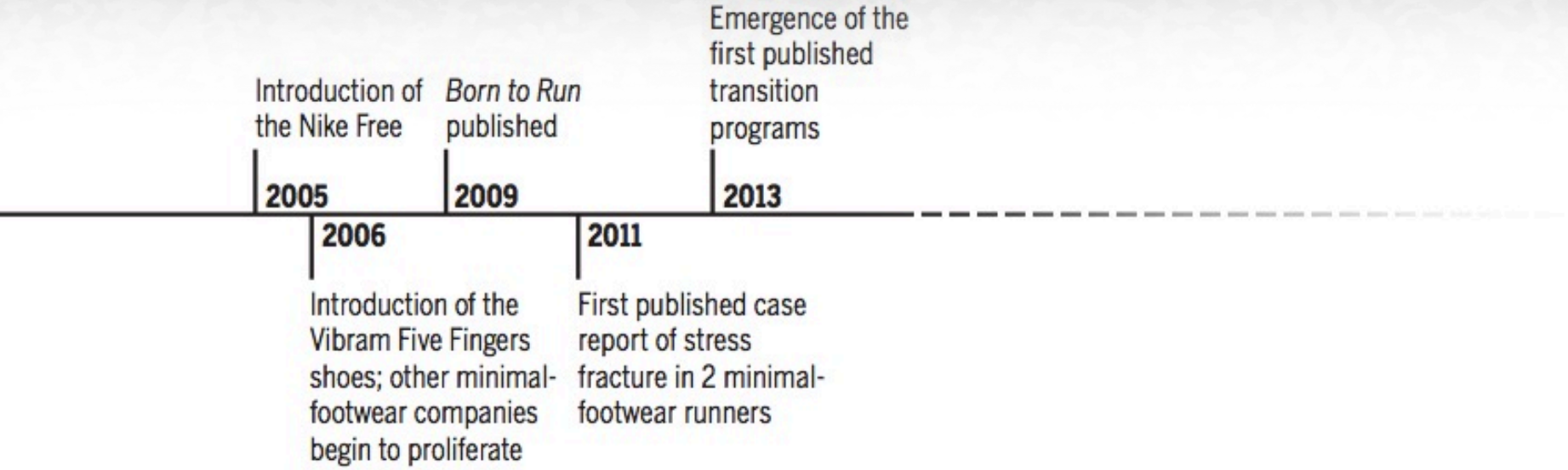
2011 — First published case report of stress fracture in 2 minimal-footwear runners

2013 — Emergence of the first published transition programs

The Re-emergence of the Minimal Running Shoe

VOLUME 44 | NUMBER 10 | OCTOBER 2014

After *Born to Run*…

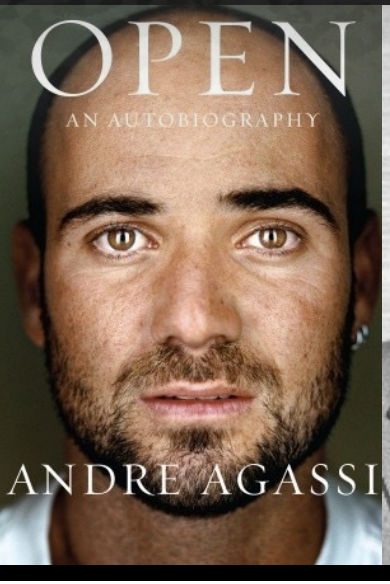


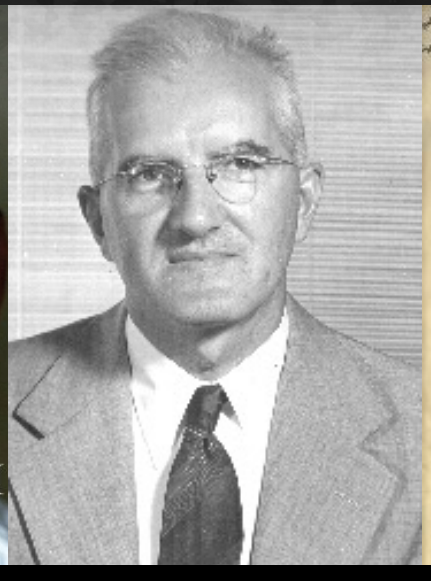
Pronation: When then foot rolls inwardly while walking or jogging. Shin is rotated inward and the leg moves leg toward midline of body. Many modern shoe models are designed to prevent this motion.

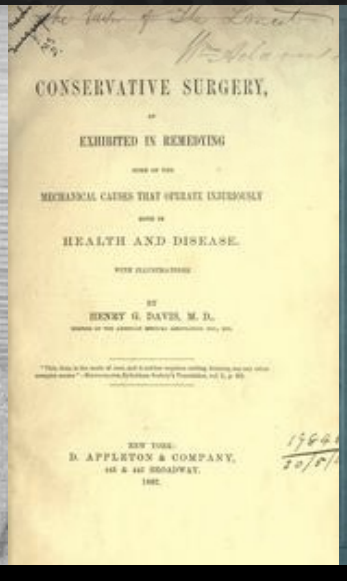
Pronated gait corrected by shoe:

This seems to be a useful thing (in terms of injury risk reduction) to correct with footwear.

If you wear shoes, specificity of adaptation happens.

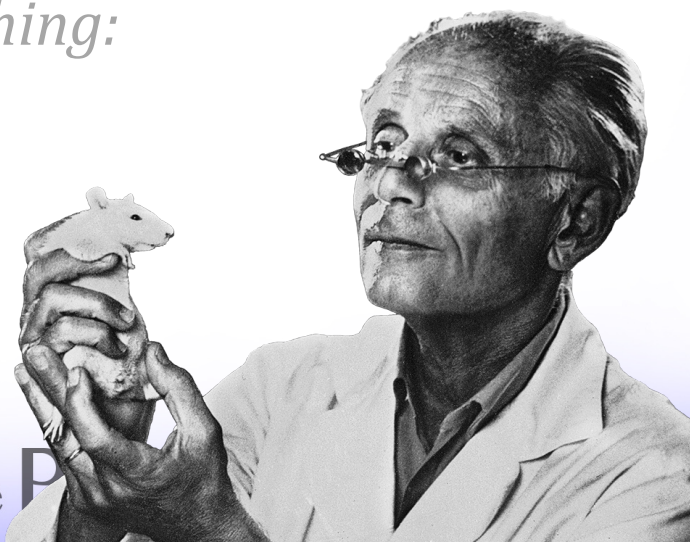






The root/cause/foundation/etc. of the everything:

University of the P



If you wear shoes, specificity of adaptation happens.

The ankle-foot machinery is comprehensive; per foot, it includes 33 joints, 19 muscles, and 26 bones. When you alter how those joints and muscles participate in your movement patterns, you change your movement patterns.

The ankle is not a normal hinge joint, hinging at the malleoli.

The ankle joint *complex* (two joints) is more complex.
Two joints: Talocrural (superior), subtalor (inferior).

Medicine & Science in Sports & Exercise
Issue: Volume 31(11), November 1999, p 1501

Talocrural and subtalar joint instability after lateral ankle sprain

HERTEL, JAY; DENEGAR, CRAIG R.; MONROE, MELANIE M.; STOKES, WAYNE L.

If you wear shoes, specificity of adaptation happens.

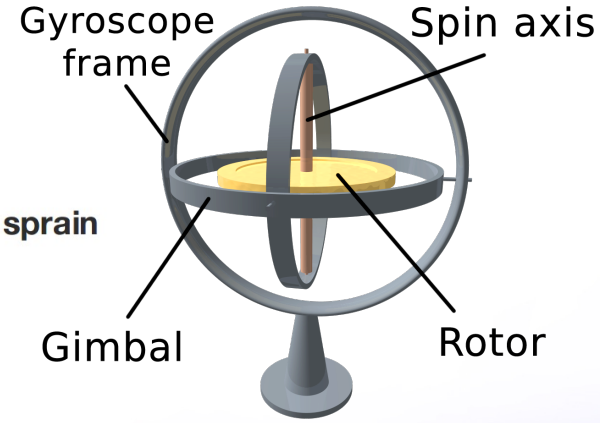
"The talocrural joint is a true hinge joint that allows the motions of plantar flexion and dorsiflexion." (Quotation from Denegar's article.)

The subtalor joint is your gyroscope (coinage belonging to Katy Bowman).
The ability to walk on varied terrain is facilitated by your subtalor joint.

Medicine & Science in Sports & Exercise
Issue: Volume 31(11), November 1999, p 1501

Talocrural and subtalar joint instability after lateral ankle sprain

HERTEL, JAY; DENEGAR, CRAIG R.; MONROE, MELANIE M.; STOKES, WAYNE L.

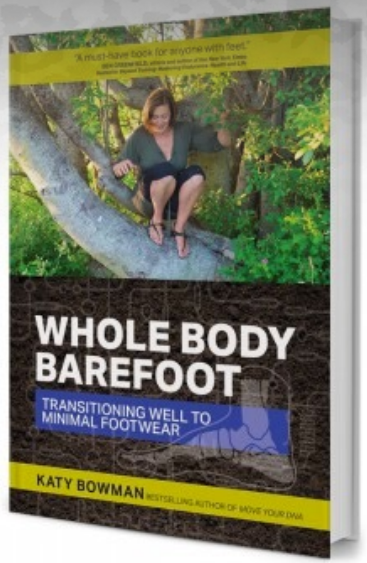


If you wear shoes, specificity of adaptation happens.

It's not just our shoes; we walk on flat stuff everywhere.
This means the talocrural joint is probably fine but the subtalor joint isn't getting worked. So its function is probably poorer than it could (and should) be.

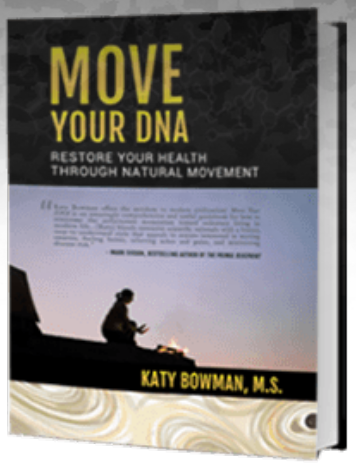
Our understanding of the ankle joint complex is based on unchanging, unchallenging terrain. We study the ankle as it functions in this context (mostly on treadmills).

A healthy ankle is an ankle that is fit for this:



Published April, 2015

"As with most arguments, when you examine this one closely, both sides are right and wrong. Research shows that minimal shoes are not safe for everyone in every situation. But research also shows that conventional shoes wreak their own havoc on the body. The element that seems to be missing from the argument is that shoes don't exist in a vacuum. Shoes and feet are in a relationship with the user and the environment, which means the physical outcome of the body that wears the shoes depends on the state of the wearer's foot, body alignment, gait patterns, frequency of movement, and most frequented terrain. A shoe can't be a problem or a solution in and of itself."



"Over ground walking is flat ground walking. Most anatomical models of the foot and ankle are based on the foot and ankle movements demonstrated by chronically shod flat-walkin' folks. When we apply these models to human movement research without qualification, the therapeutic solutions to many injuries and issues wrought by the diseases of behavior continue to evade us."

Chapter 5:
Transitioning Well

Subsection:
The surfaces you walk upon

"What we know about how humans walk is actually based on how humans walk around in a modern context. If we don't acknowledge that our model of normal walking has nothing to do with nature, our therapeutic options are vastly limited."

SUMMARY: There is no such thing as a good shoe or a bad shoe. There are only *appropriate* and *inappropriate* shoes. Shoes create mechanical environments; they change the loads experienced by your feet. Depending on the context (and your goals) that may be good or it may be bad. Discussions of what is "natural" isn't helpful. Your feet and the surfaces they walk across aren't any more natural.

Applications of biomechanics:

Simulating regular gravity in microgravity.

Go into outer space and see what happens to your movement. Contraptions exist to mimic the mechanics of earth gravity. Force a la carte isn't enough. Wolff's law. Those forces have to replicate earth's mechanics.

Domains of biomechanics.

MEDICINE & SCIENCE IN SPORTS & EXERCISE®

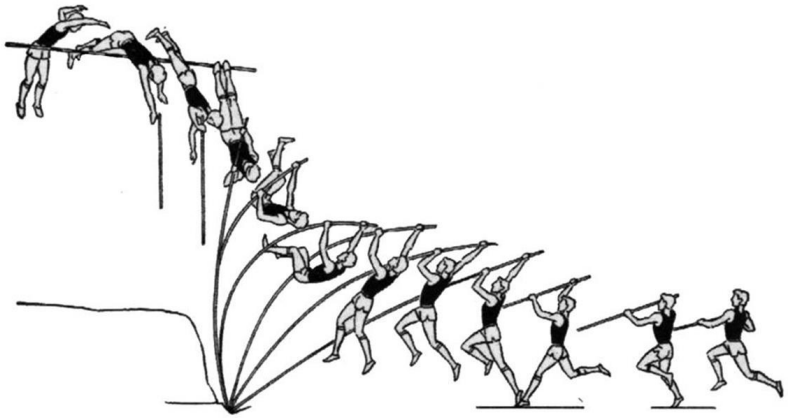
Push-off mechanics in speed skating with conventional skates and klapskates

HAN HOUDIJK, JOS J. DE KONING, GERT DE GROOT, MAARTEN F. BOBBERT, and GERRIT JAN VAN INGEN SCHENAU

The introduction of the klapskate was accompanied by a remarkable improvement of personal records and all world records were shattered during the season of 1997–98. Skaters who switched to klapskates were able to increase their skating speed by as much as 4%. This is equivalent to an increase in mean power output of about 10%.

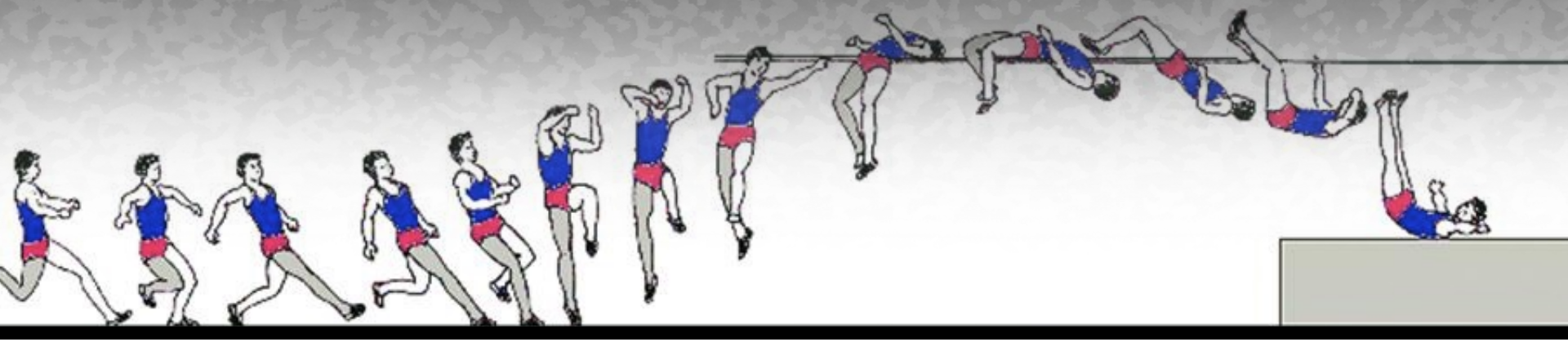
Not on the test. Just illustrating other roles of biomechanics.

Pole-vaulting poles must be appropriately flexible and appropriately stiff; they need to bend sufficiently while storing large amounts of elastic energy. Conflicting requirements also apply to the body that's doing the vaulting. Biomechanical analyses can balance these variables for optimal materials, techniques, and performance.



More roles of a biomechanist. Not on the test.

REVIEW: biomechanics



Journal of Strength and Conditioning Research, 2004, 18(4), 787–791
© 2004 National Strength & Conditioning Association

THE MAXIMAL AND SUBMAXIMAL VERTICAL JUMP: IMPLICATIONS FOR STRENGTH AND CONDITIONING

ADRIAN LEES,[1] JOS VANRENTERGHEM,[2] AND DIRK DE CLERCQ[2]

GEORGIOS PAPAIAKOVOU

Journal of Sports Sciences, 2013
Vol. 31, No. 16, 1789–1796

Kinematic and kinetic differences in the execution of vertical jumps between people with good and poor ankle joint dorsiflexion

Ankles can affect it →

If you have bad ankle flexibility (reduced dorsiflexion), your vertical jump will be lower and your jump mechanics changed.

J Strength Cond Res. 2009 July ; 23(4): 1327–1331.

HIP AND KNEE EXTENSOR MOMENTS PREDICT VERTICAL JUMP HEIGHT IN ADOLESCENT GIRLS

KEVIN R. FORD[1,2], GREGORY D. MYER[1,3], JENSEN L. BRENT[1], and TIMOTHY E. HEWETT[1,4]

Research Quarterly for Exercise and Sport
©2001 by the American Alliance for Health, Physical Education, Recreation and Dance
Vol. 72, No. 1, pp. 63–67

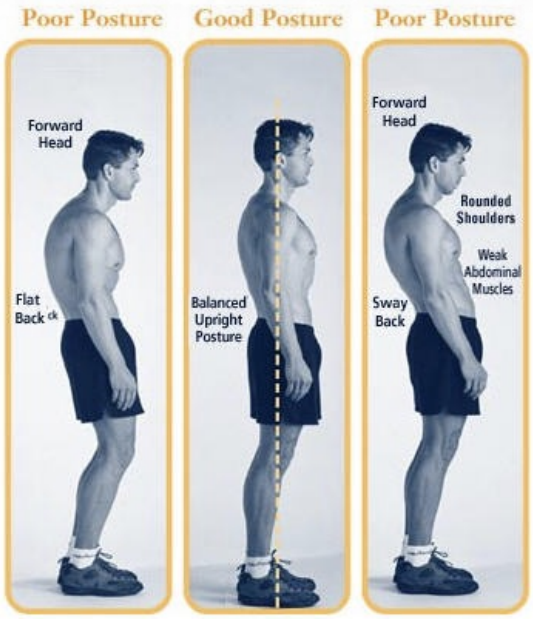
Using Principal Components Analysis to Identify Individual Differences in Vertical Jump Performance

Iraklis Kollias, Vassilia Hatzitaki, George Papaiakovou, and George Giatsis

Athletes, depending on their sport, recruit muscle differently (different time and force parameters).

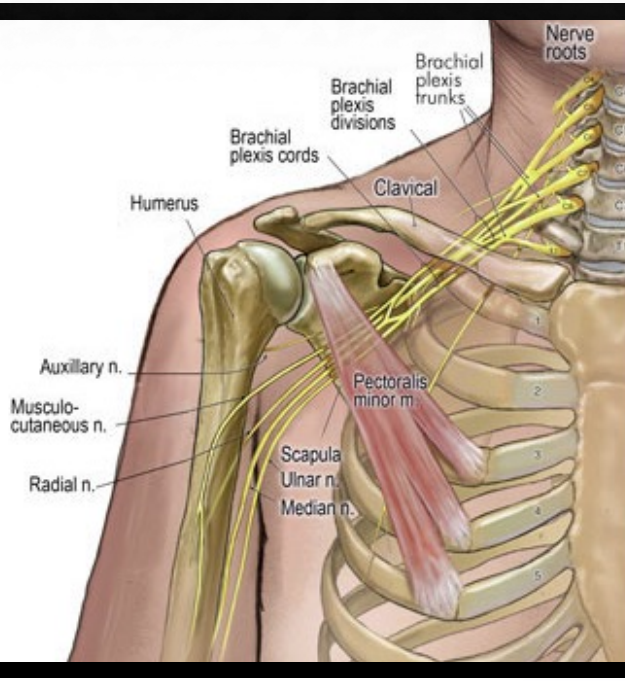
Knees matter in some populations →

Still illustrating roles of biomechanics…



Kyphosis,

The subclavian artery and the brachial plexus:



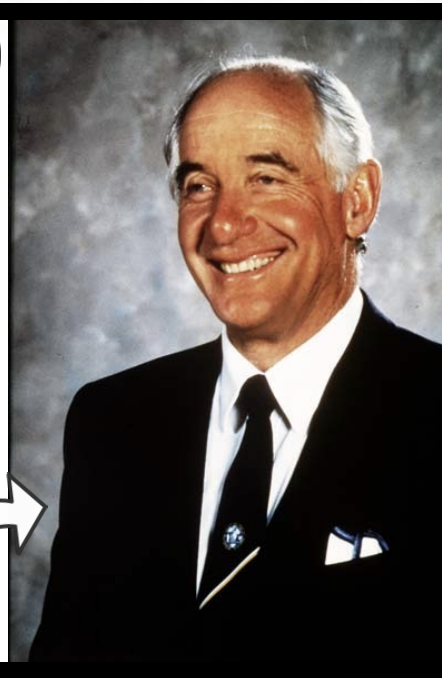
Domains of biomechanics. Might be on the test.

REVIEW: biomechanics

Paul C. Williams (1900–1978)

vs.

Robin McKenzie (1931–2013)



Biomechanics. Not on the test. But useful to know.

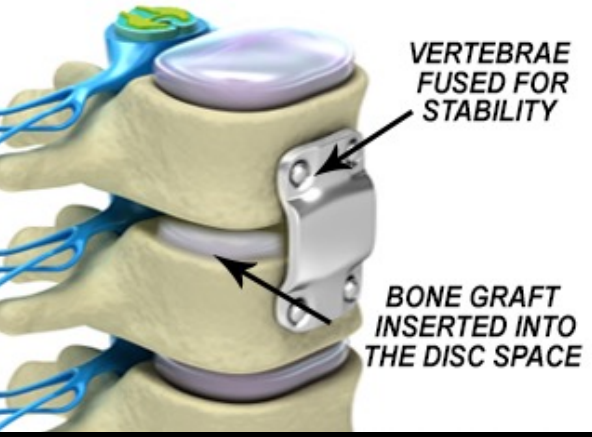
REVIEW: biomechanics

Modern ski boots and bindings. They protect the ankle but all mechanical forces get transferred to the knee.

Three-fold increase in knee injuries in skiing since 1972.

(With snowboarding, injuries to the knees are less likely among people using less rigid, more pliable boots.)

Biomechanics. Might be on the test. Remember: if you cast something, you alter both loading and muscle recruitment characteristics.

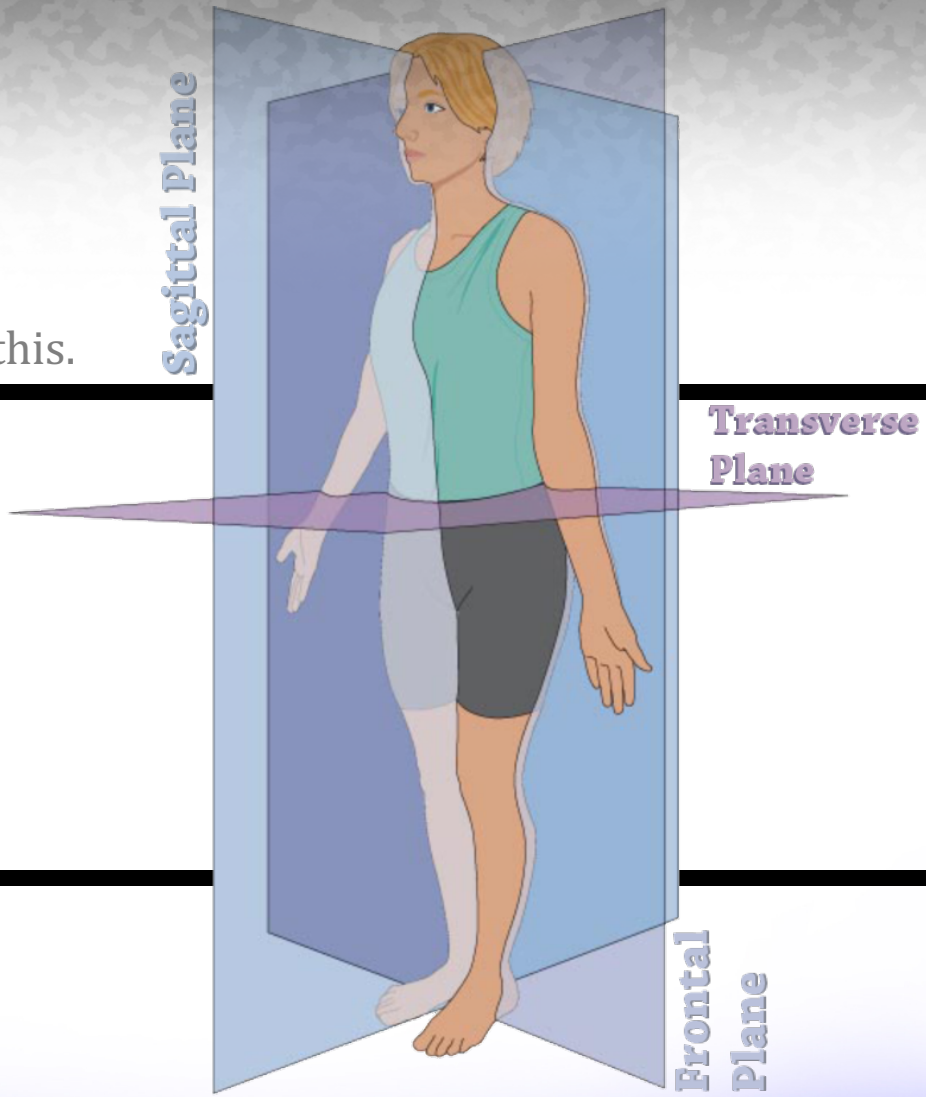


What will happen if you get a vertebral fusion?
Think about ski boots… it should be obvious.

Maybe on the test.

REVIEW: Planes of Motion

There are five (out of fifty) questions about this.



What exercises fall in one plane, two planes, three planes?

REVIEW: Biomechanist's Dictionary

Anatomy: Study of components that make up the musculoskeletal "machine."

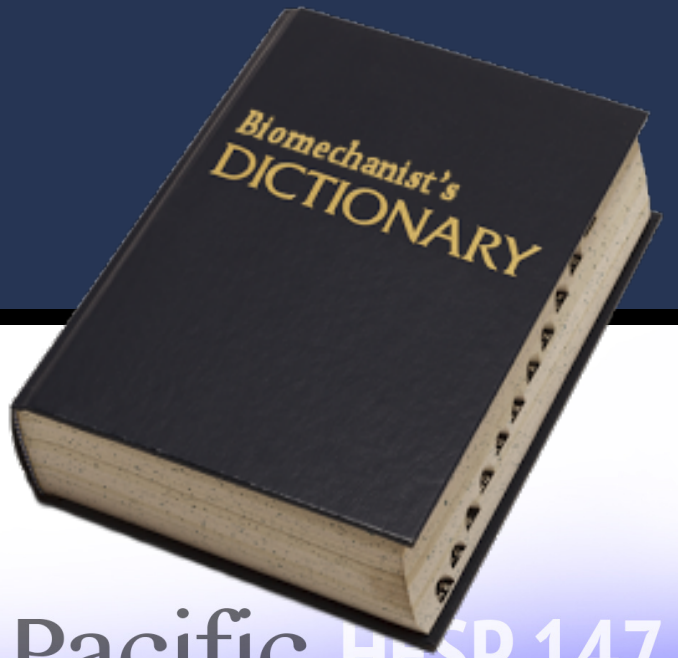
Biomechanics: Mechanisms (mechanical laws) used by those components to create movement (in any living thing… trees, mice, people, whatever).

Statics: Study of systems with motions that are constant (no change in velocity).

Dynamics: Study of systems that involve acceleration.

Kinetics: Causes of a motion; the internal and external forces associated with movement.

Kinematics: The motions themselves (big picture). Don't consider what is causing the motion; just look at the patterns of a biological thing moving around.



REVIEW: Biomechanist's Dictionary

Torque: Tendency for a force to produce rotation.

Work: Force × Displacement × Cosine of Angle.

Rotational Work: Torque × Angular Displacement.

Power: Work ÷ Time.

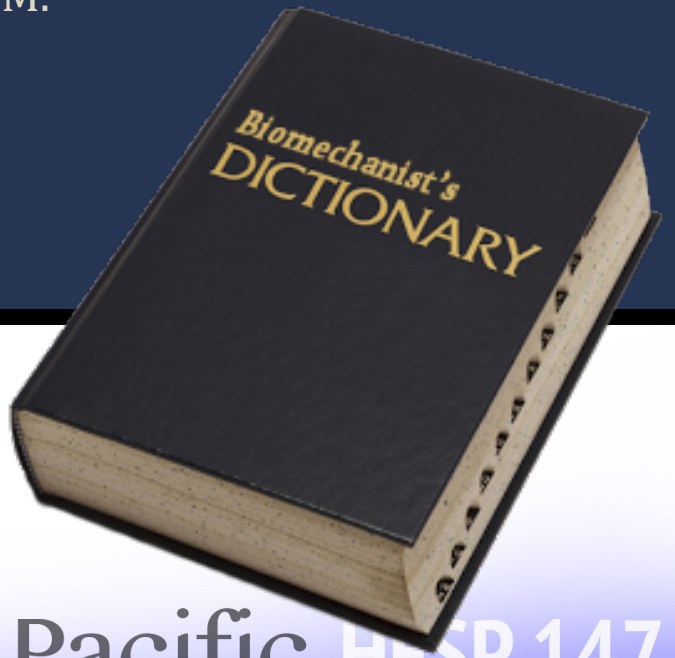
Mechanical advantage/disadvantage: Moment arm length.

Variable Resistance: Variable moment arm through ROM.

First Class Lever: Fulcrum between force and load.

Second Class Lever: Fulcrum, load, force.

Third Class Lever: Fulcrum, force, load.

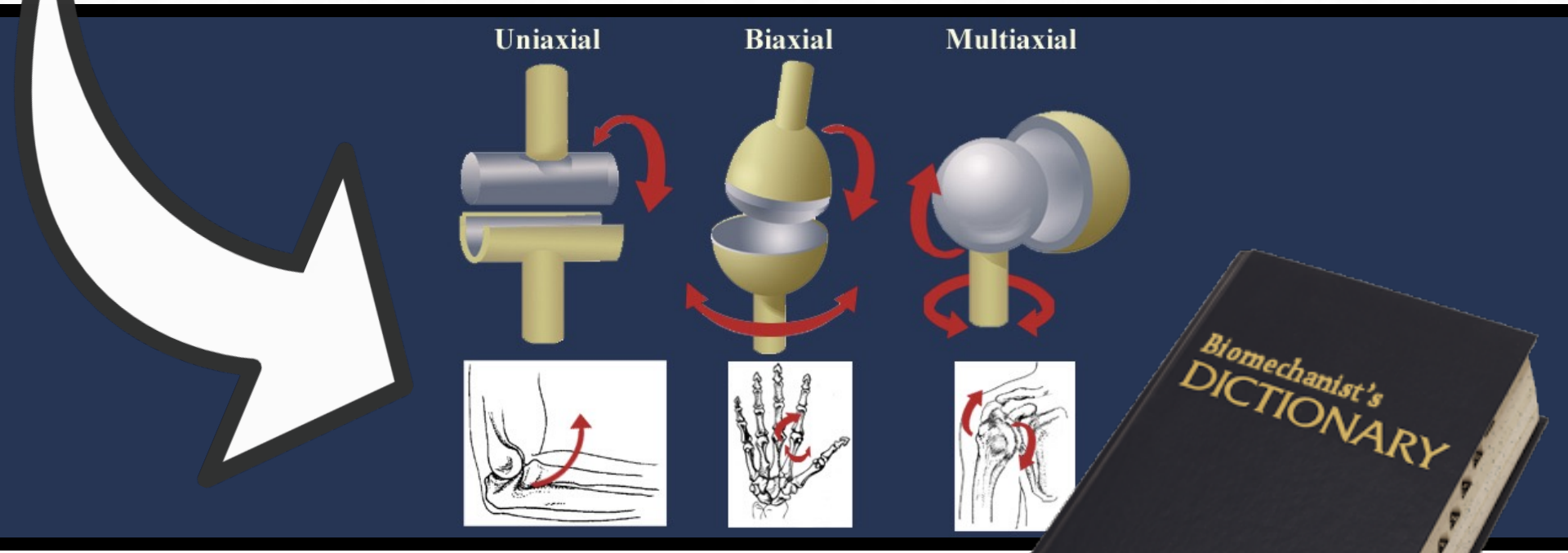


REVIEW: Biomechanist's Dictionary

Uniaxial: You'll want to be able to name a body part or two.

Biaxial: You'll want to be able to name a body part or two.

Multiaxial: You'll want to be able to name a body part or two.



REVIEW: Biomechanist's Dictionary

Work: Force × Displacement × Cosine of Angle

Power: Work ÷ Time

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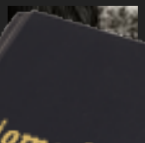
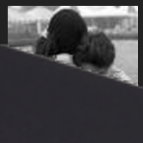
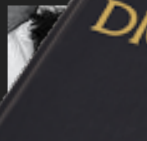





Images may

REVIEW: Biomechanist's Dictionary

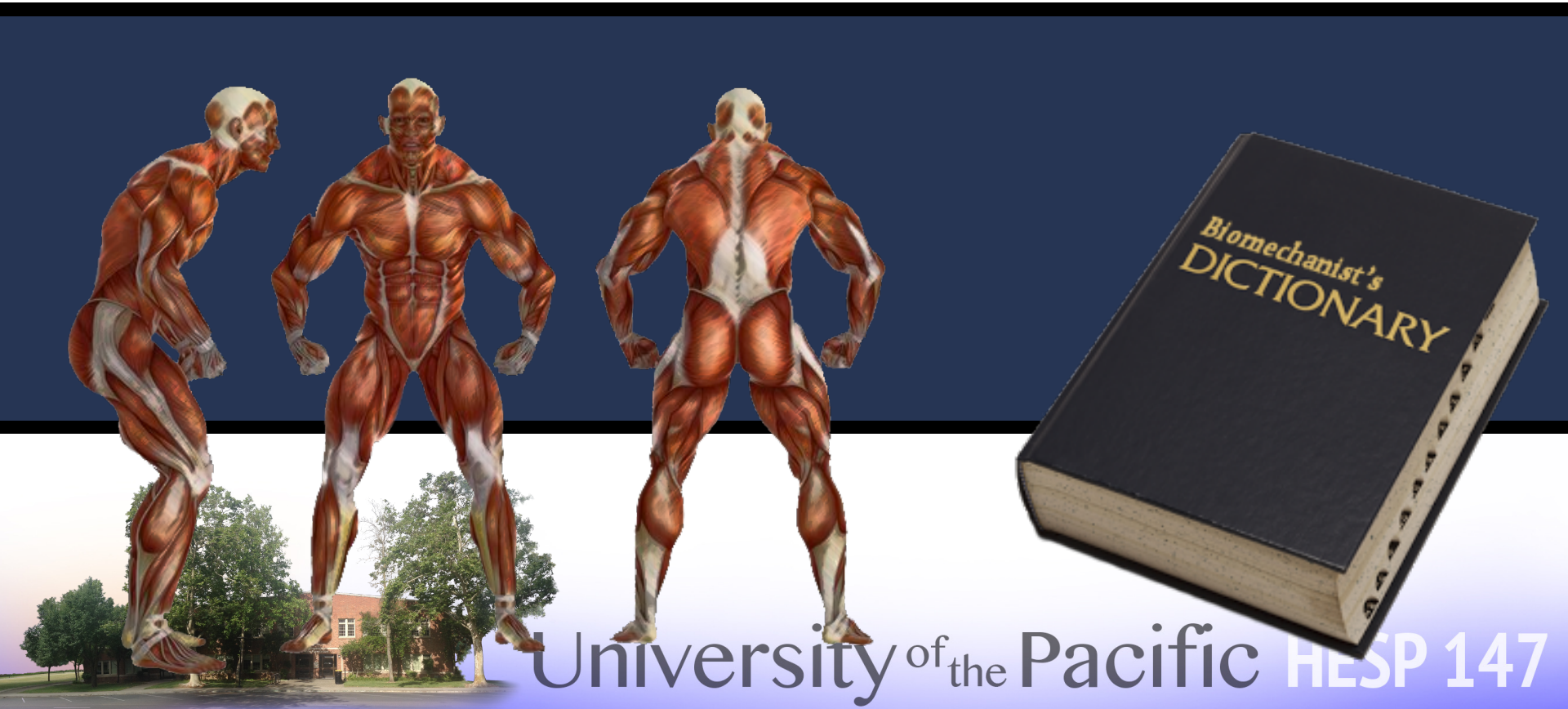
Origin: Proximal insertion.

Insertion: Distal insertion.

Agonist: Prime mover.

Antagonist: Opposite.

Synergist: Indirect assister (stabilizer, etc.).

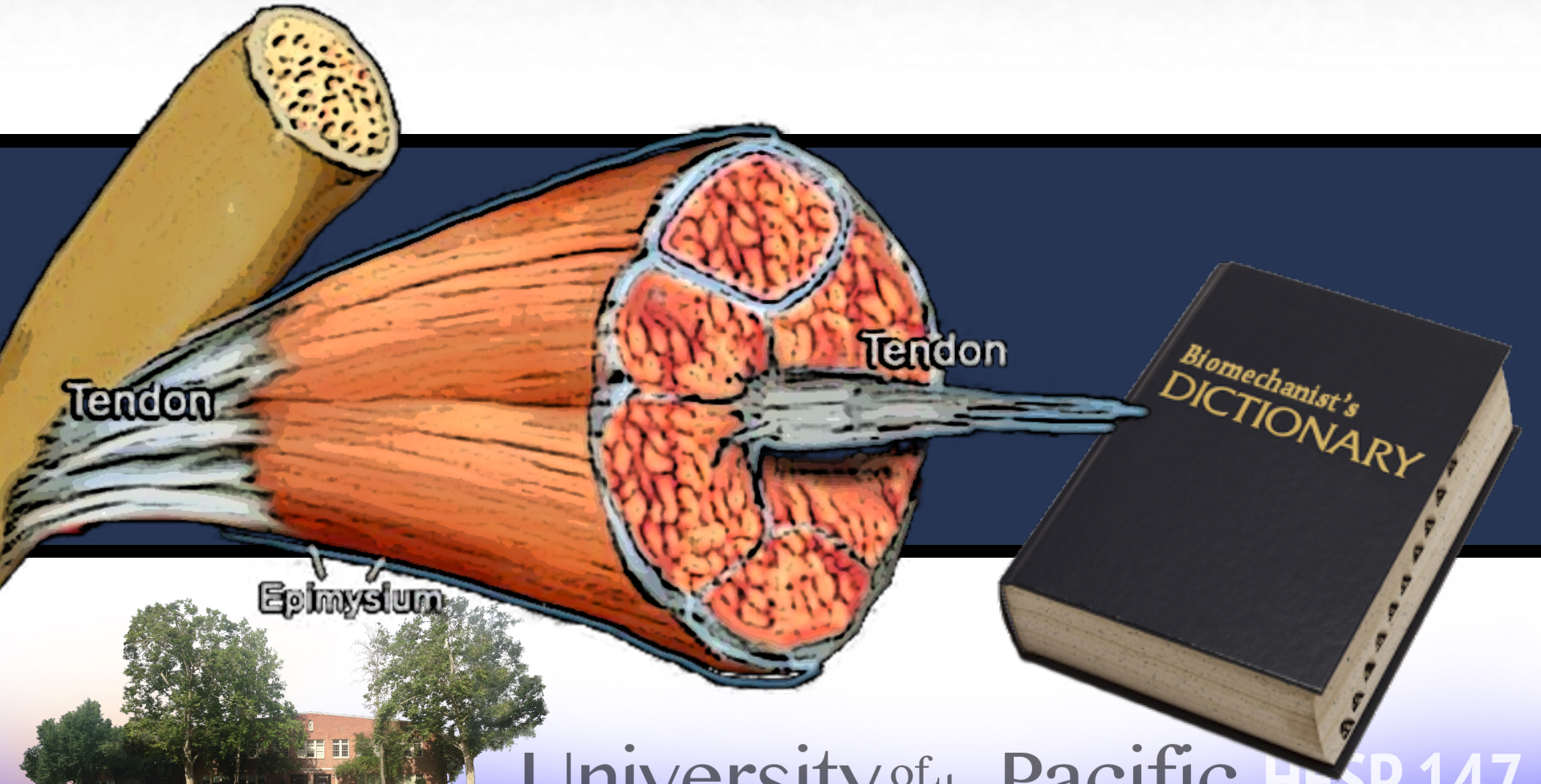


REVIEW: Biomechanist's Dictionary

Fleshy attachment: What is it and what's an example?

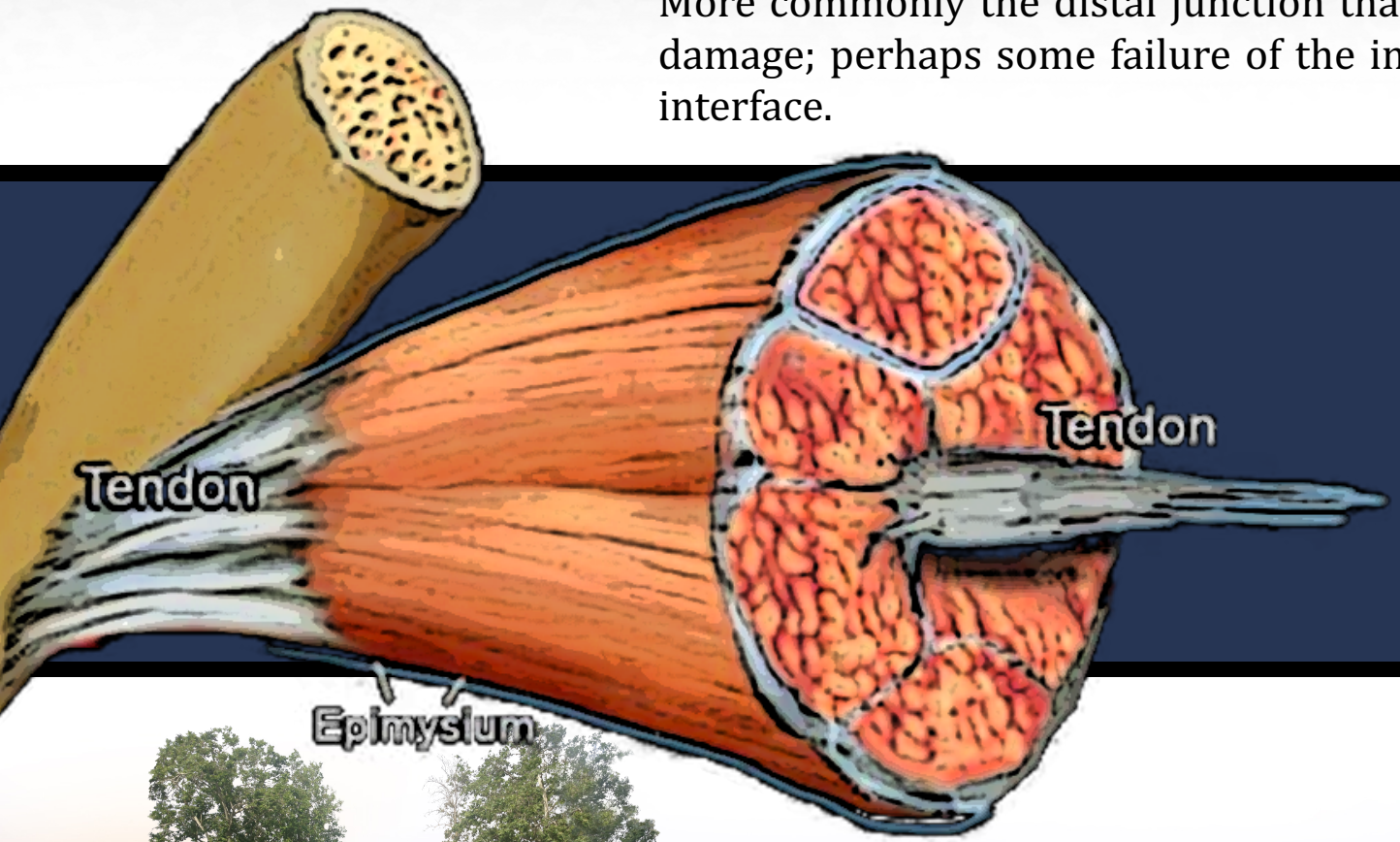
Fibrous attachment: What is it and what's an example?

Myotendinous junction: What is it and what's important about it?

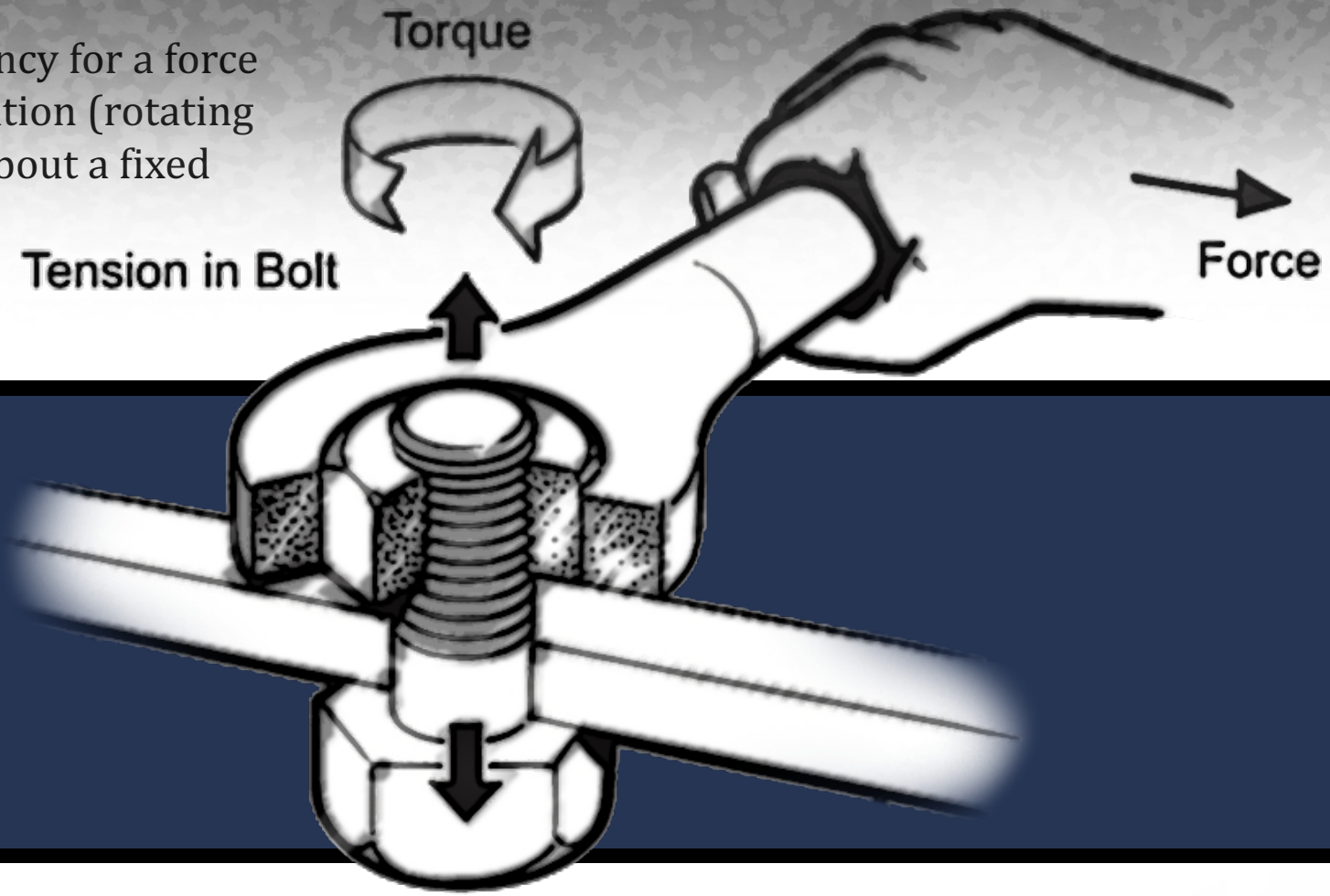


Myotendinous junction:

The sarcomeres in and around the myotendinous junction may be shorter and are likely stiffer and less extensible. With a decreased ability to change length, they're unable to take as much deformity. So they tend to absorb more of the trauma in use-related musculoskeletal injuries. More commonly the distal junction that experiences that damage; perhaps some failure of the integrin-fibronectin interface.



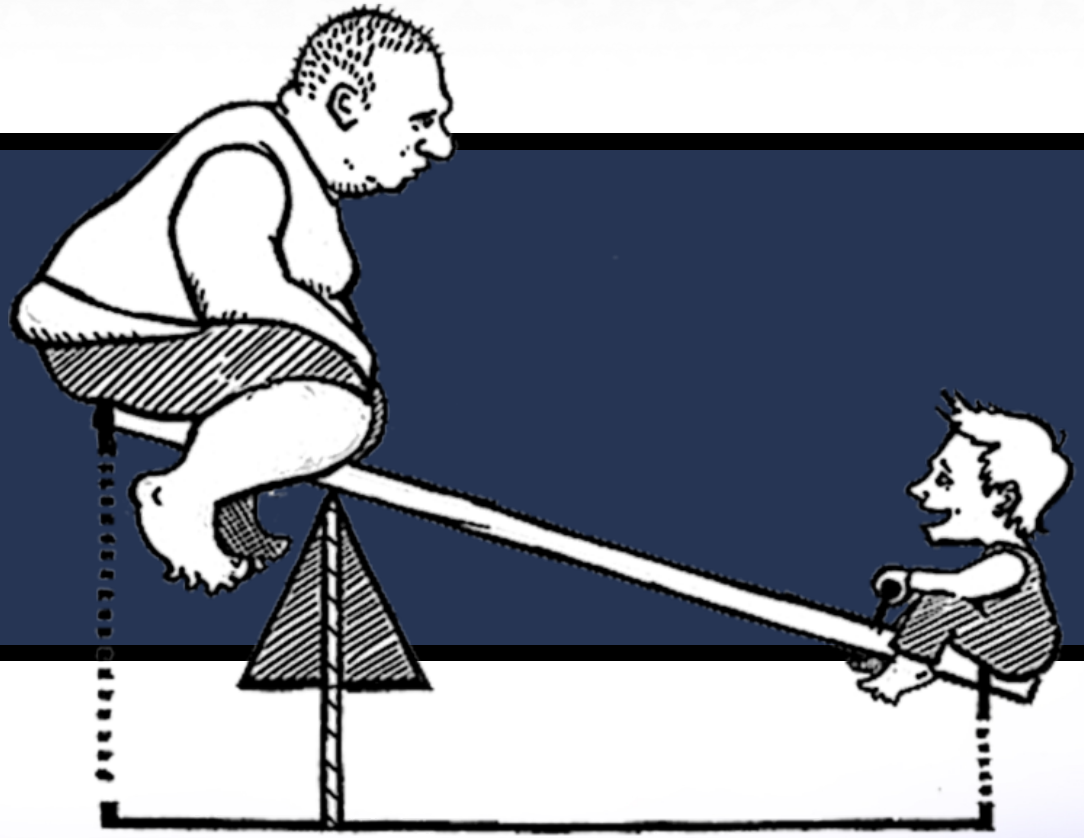
Torque: Tendency for a force to produce rotation (rotating that rigid bar about a fixed point).



Rotational Work:
Torque × Angular Displacement

What is the purpose of levers?
Be able to identify the three classes of levers.

What kind of **lever** is this?



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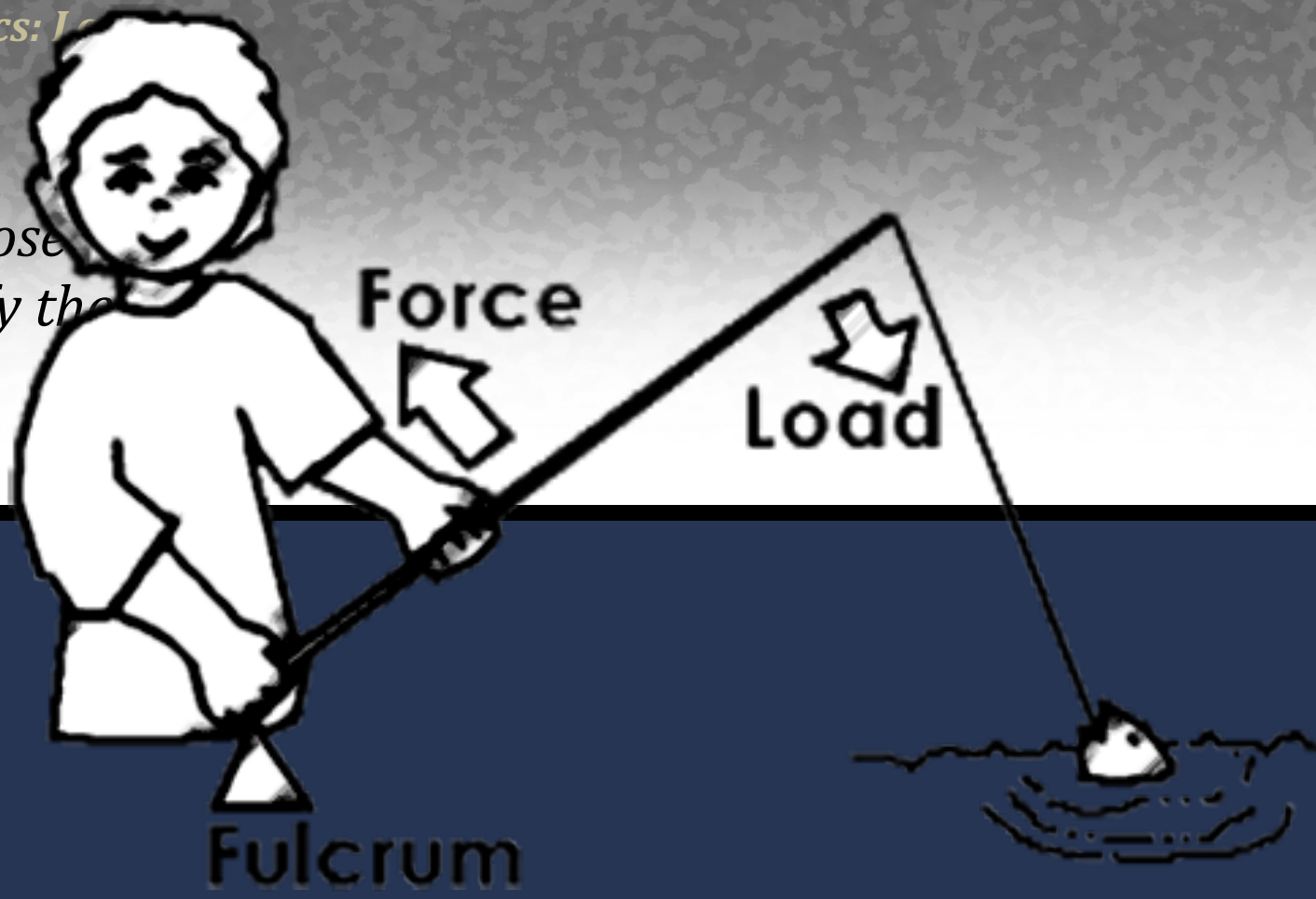

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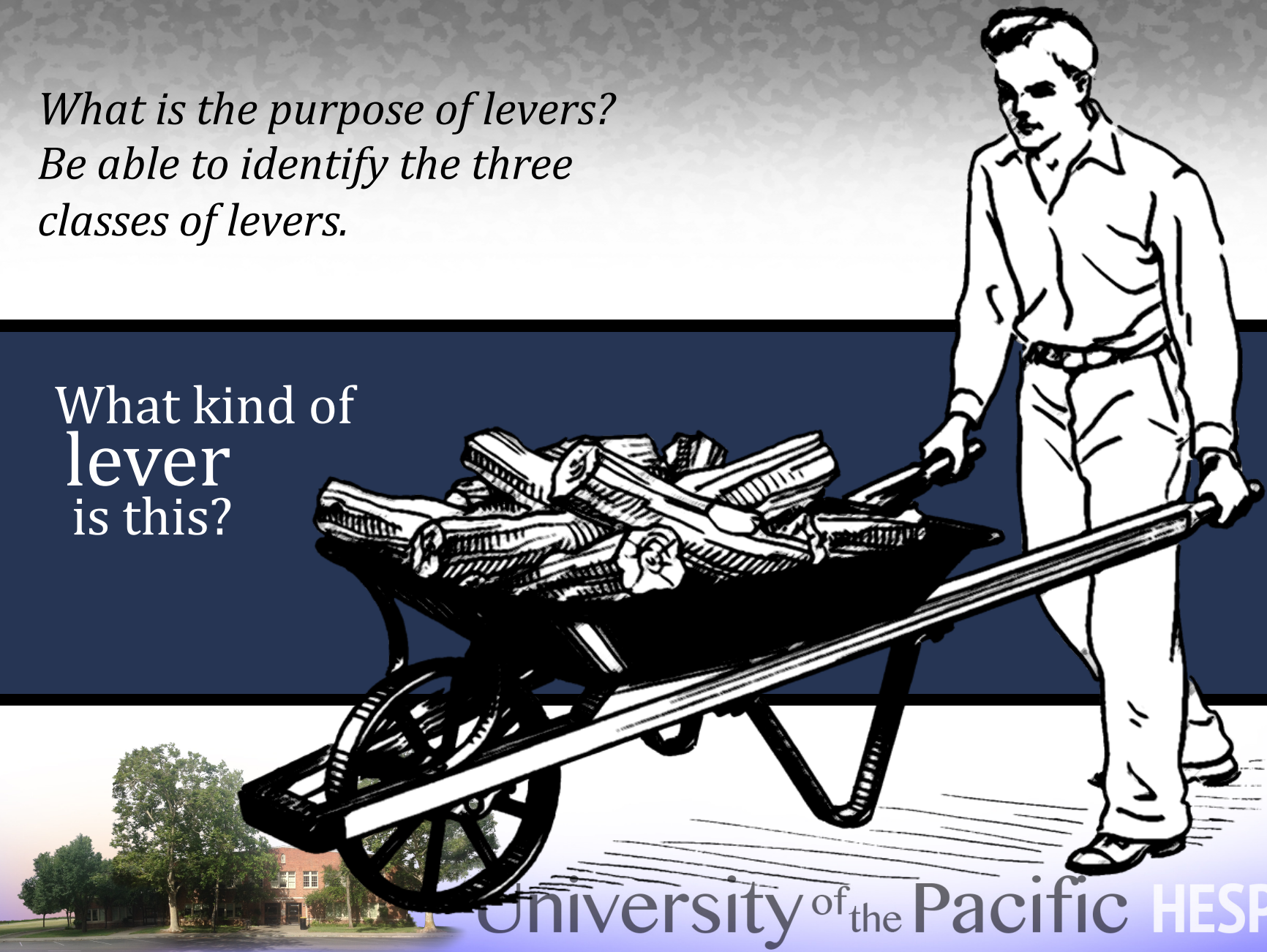
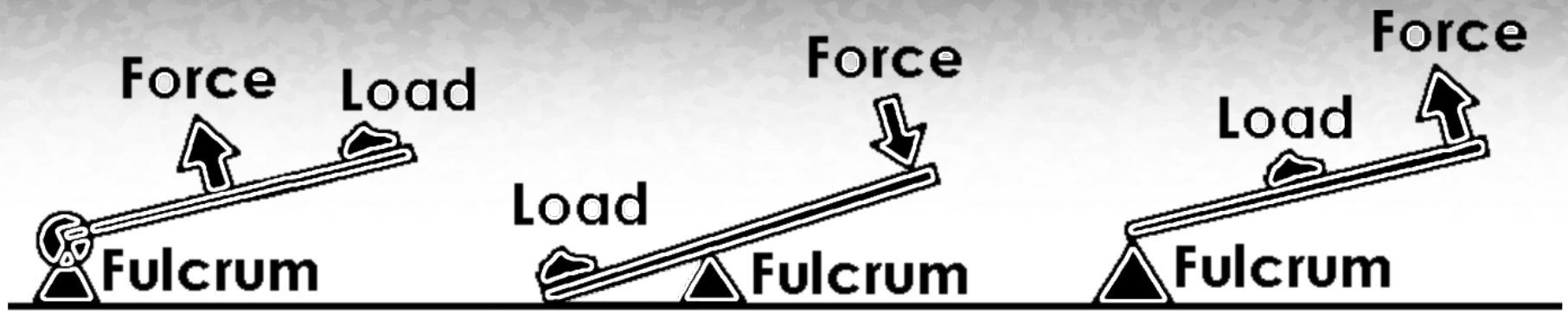
Be able to identify the three classes of levers.

What kind of **lever** is this?

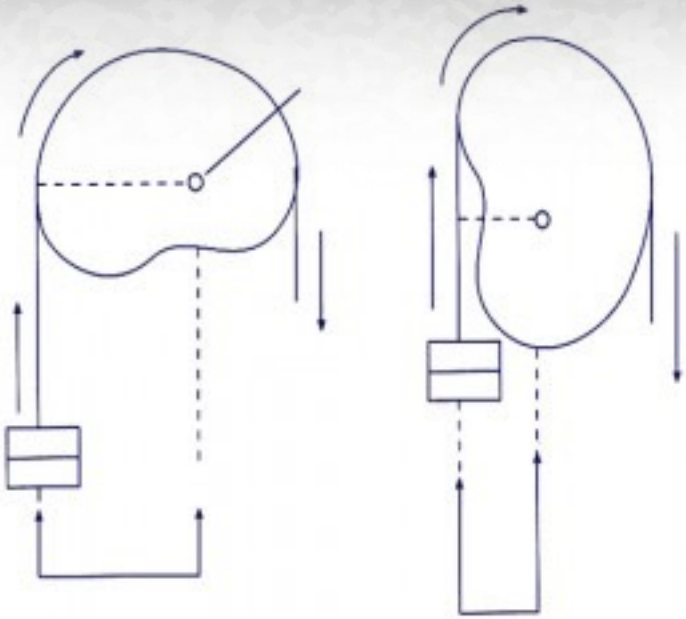




Which kind of lever is which?
Mechanical advantage or disadvantage?

What is the purpose of levers?
Be able to identify the three classes of levers.

What is a
muscle
that *doesn't*
use a lever?



As the cam rotates from position 1 to 2, the distance from the pivot point to the weight plate shortens, which has the effect of reducing the load, thereby resulting in a more uniform muscle effort throughout each repetition.

Variable resistance:

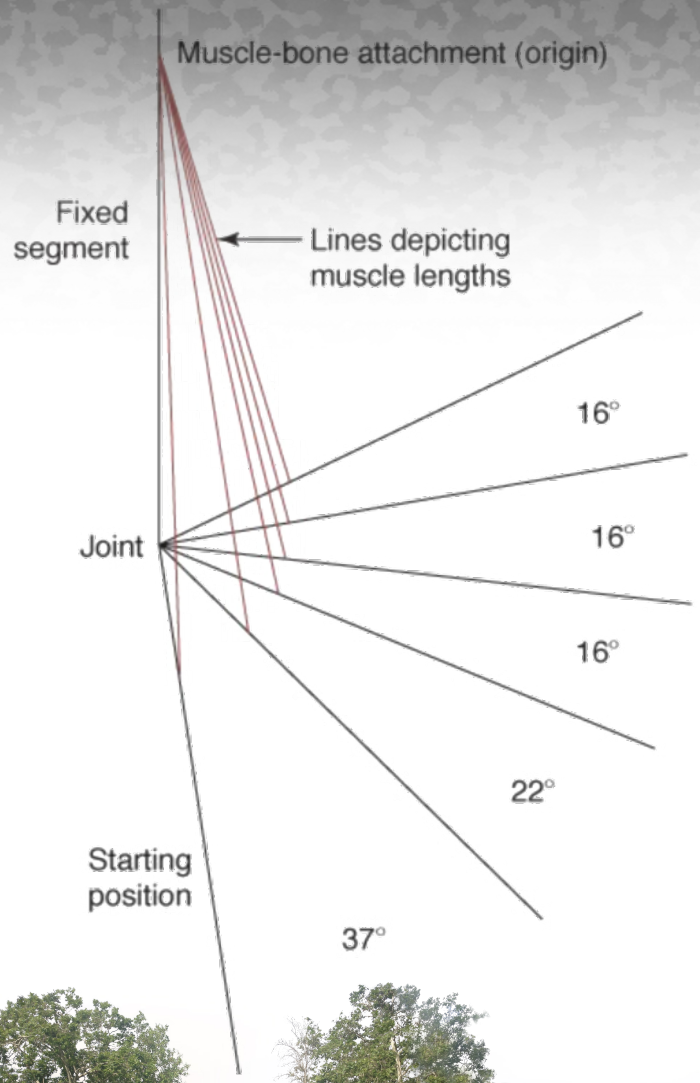
Longer moment arm (distance from the fulcrum to the the load) means a heavier load.

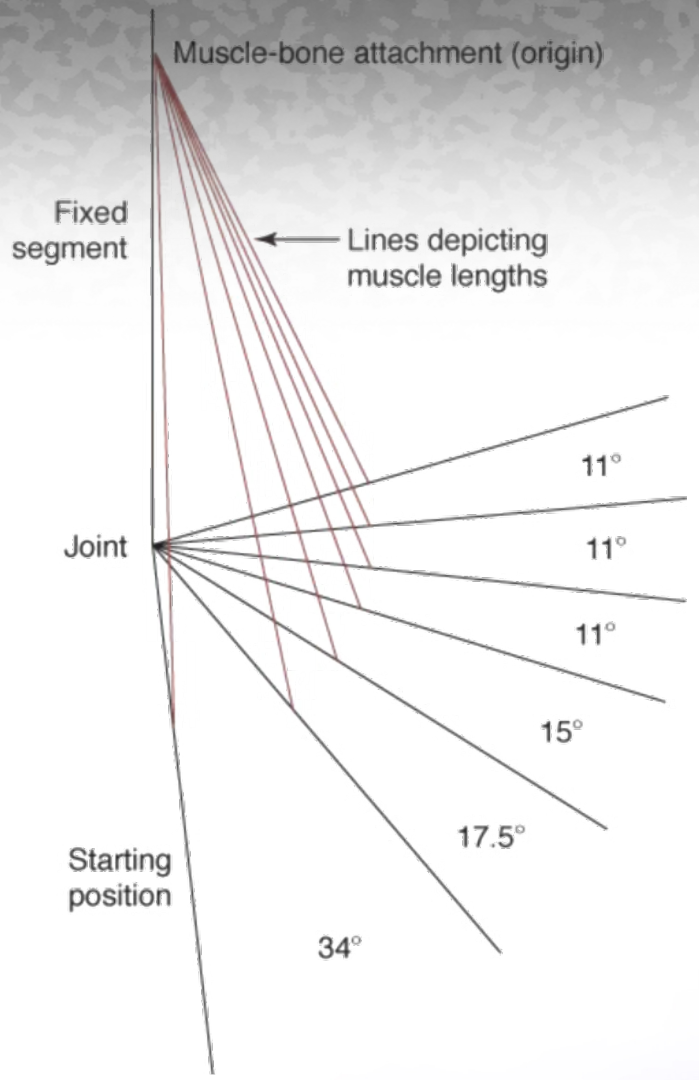
Most muscles function at a large mechanical disadvantage. During physical activity, the forces being generated inside of your muscles are much higher than those being exerted externally.

How much force is generated inside of you when you throw a punch? How much force contacts your opponent?

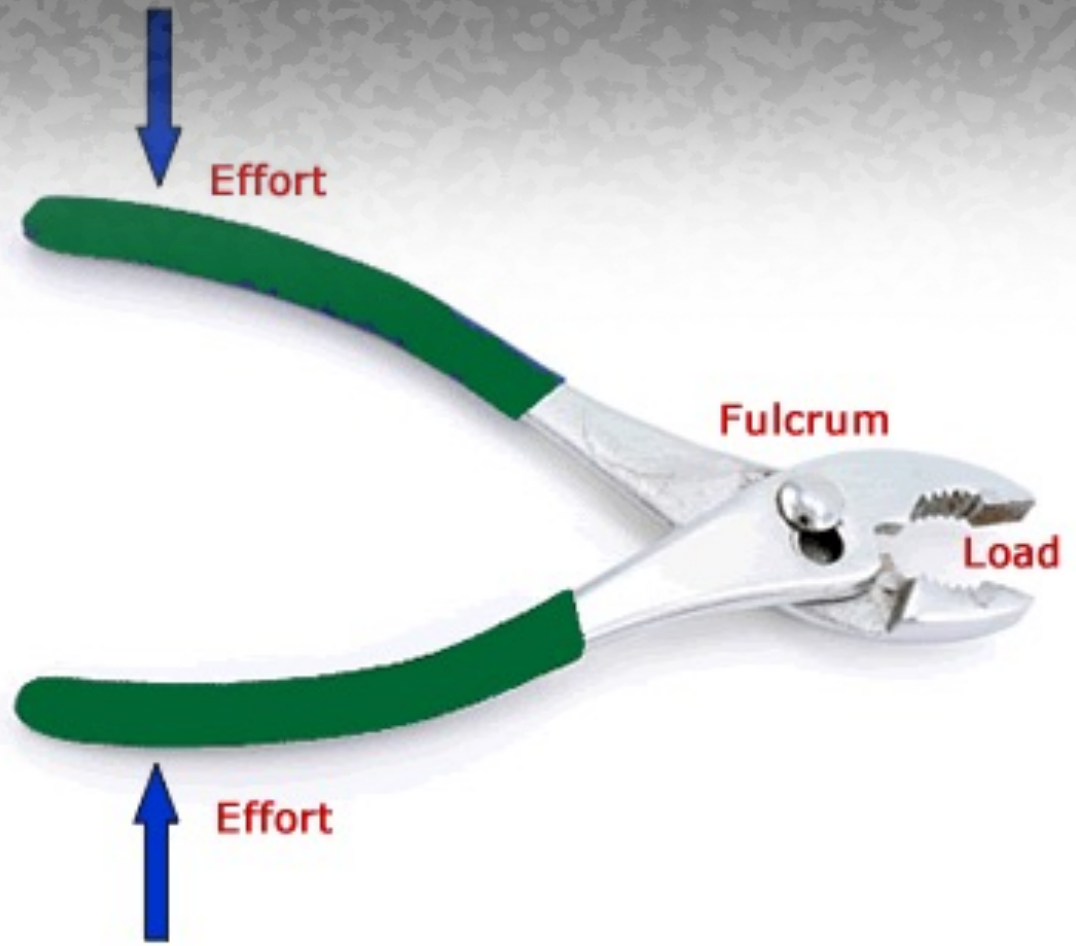
Is a cheetah
going to have
a big bench press?

REVIEW: Location of Insertion and the Force-Speed tradeoff

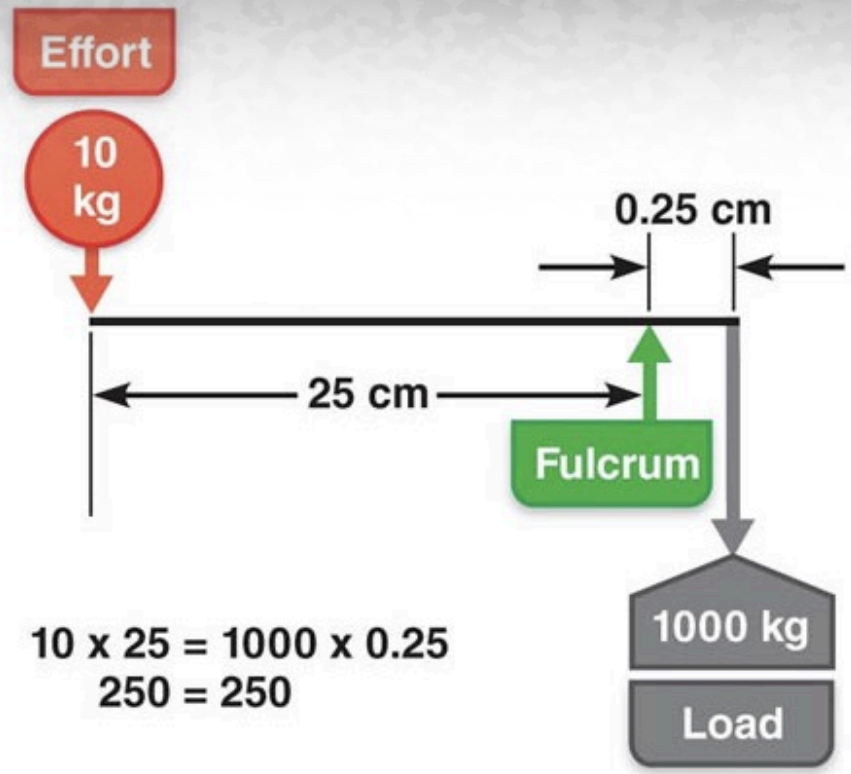




REVIEW: What Class of Lever?

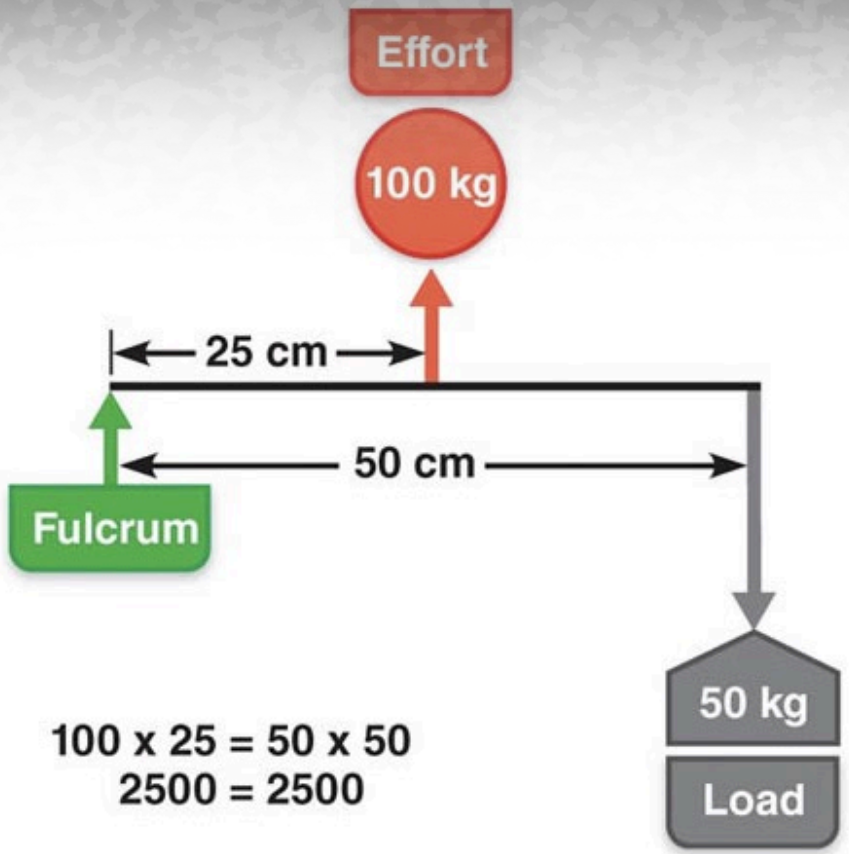


REVIEW: What Class of Lever?

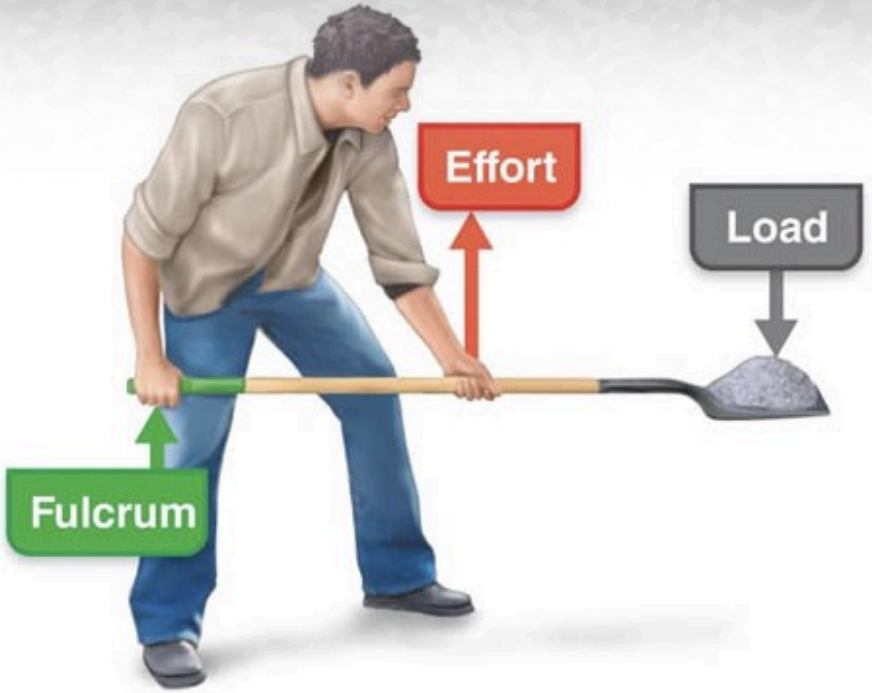


10 x 25 = 1000 x 0.25
250 = 250

REVIEW: What Class of Lever?



100 x 25 = 50 x 50
2500 = 2500



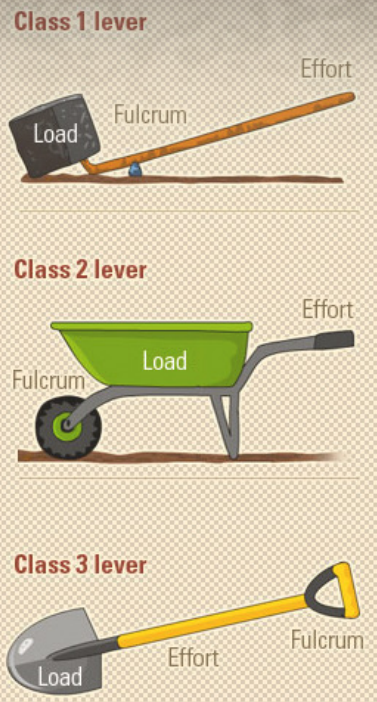
REVIEW: What Class of Lever?

REVIEW: What Class of Lever?

REVIEW: What Class of Lever?

REVIEW: What Class of Lever?

REVIEW: Biomechanical Factors in Human Strength

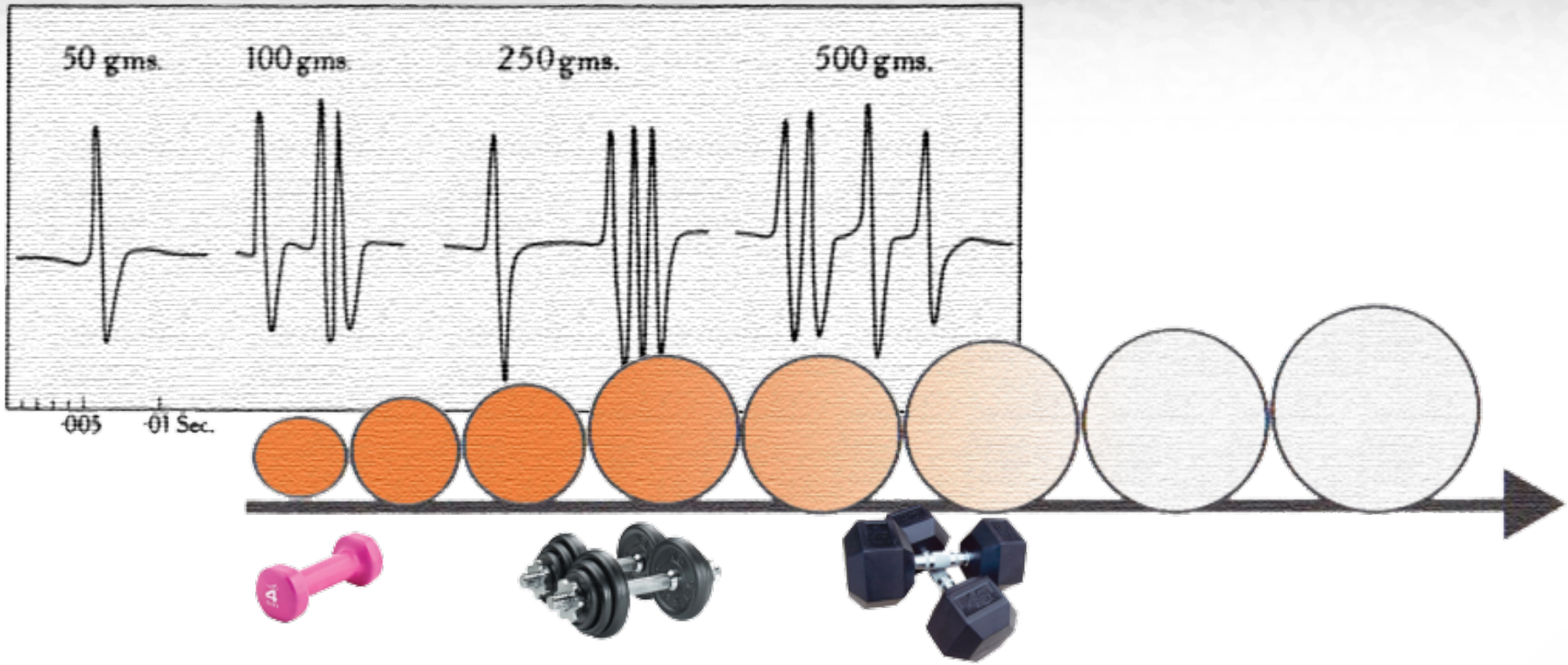


Five (again, out of fifty) questions are about levers.

REVIEW: Biomechanical Factors in Human Strength

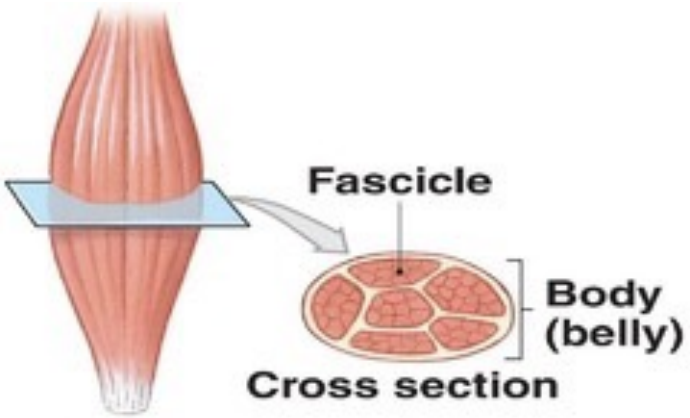
1. Neural Recruitment
2. Muscle CSA
3. Arrangement of Muscle Fibers
4. Muscle Length
5. Joint Angle
6. Muscle Contraction Velocity
7. Strength-to-Mass Ratio
8. Body Size
9. Physiological Explanations

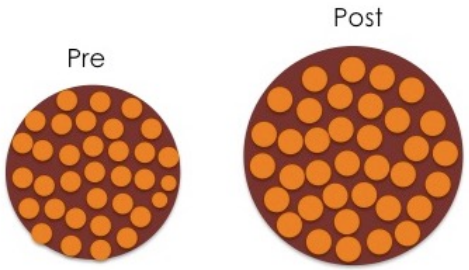
1. Neural recruitment *(What are the variables here?)*



2. Muscle CSA

Cross sectional area, not volume (which includes muscle length) determines strength from the perspective of biological real estate.



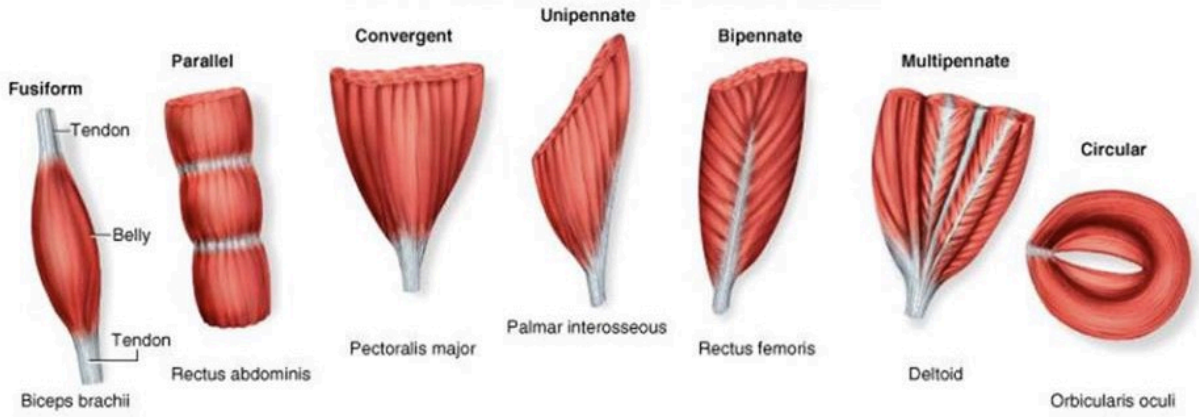


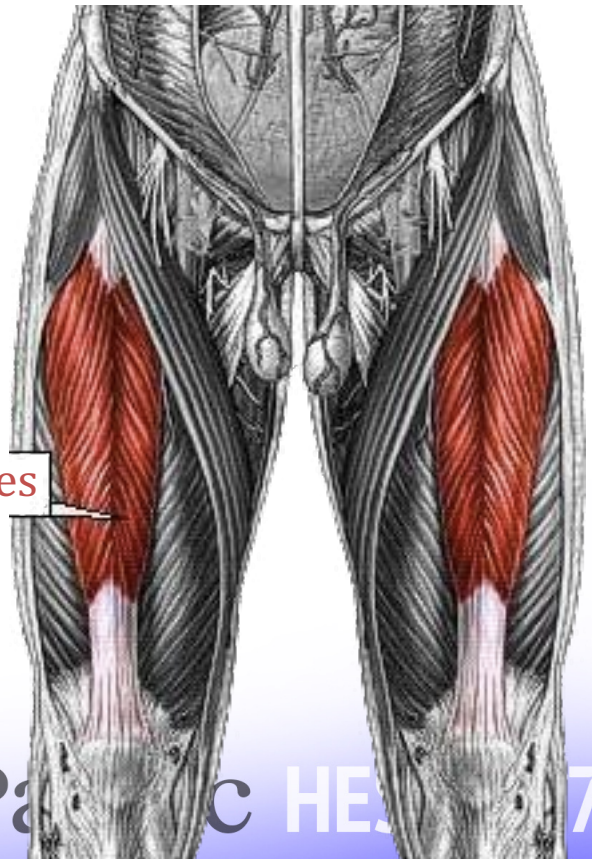
Hypertrophy
Following strength training, the number of muscle fibers remains constant, but the size increases so that there is more surface area to deal with strain inside the muscle belly

3. Arrangement of muscle fibers

Lots of architectural styles of muscle (e.g., parallel, circular, pennate).

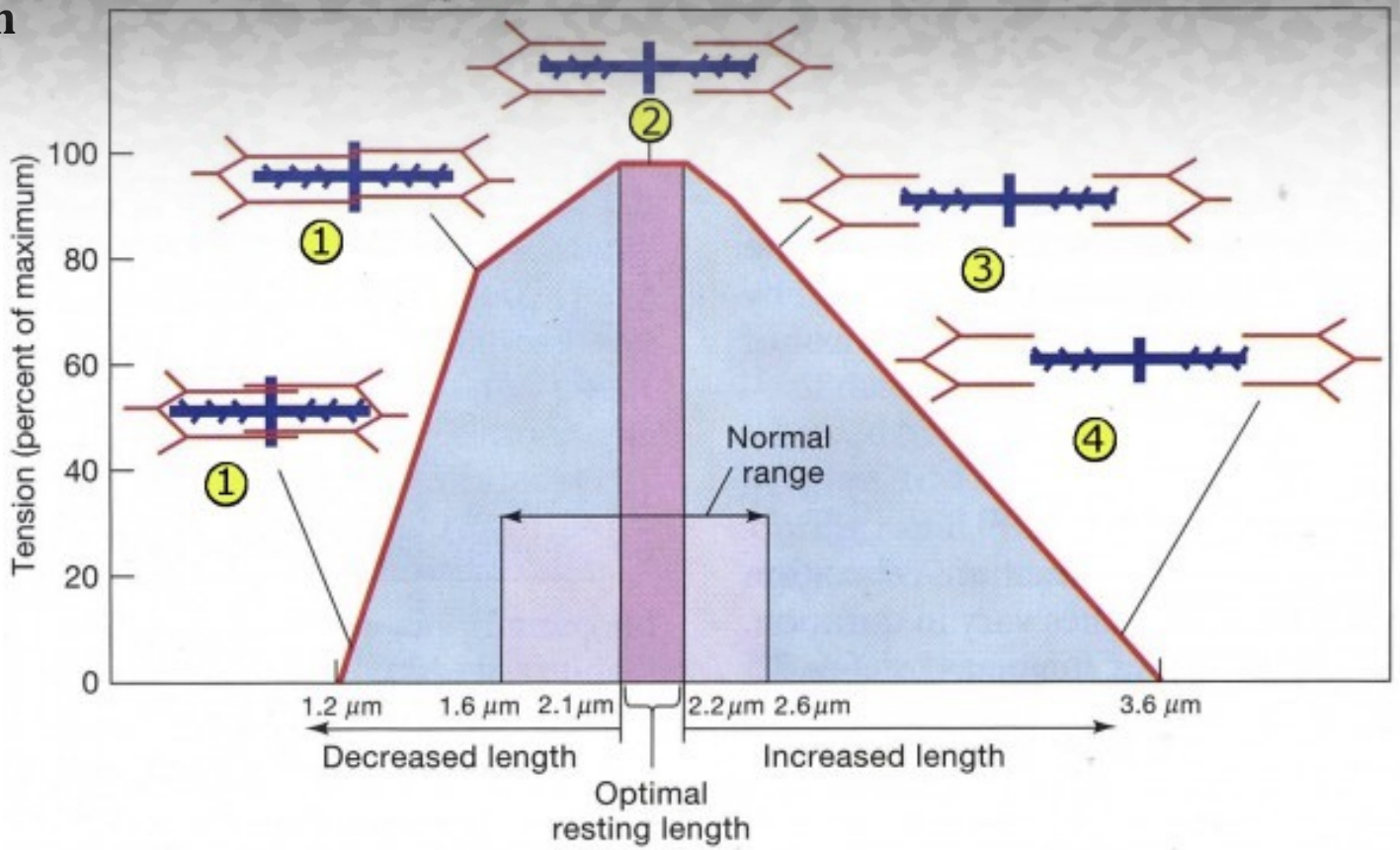
There is variation in the arrangement and the alignment of sarcomeres in a muscle fiber.



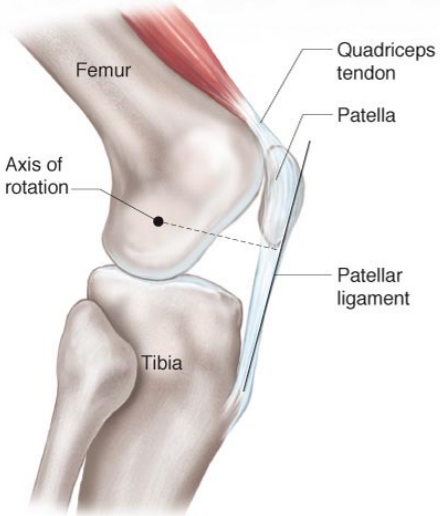


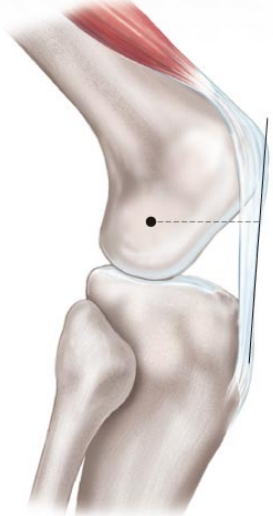
Notice the weird angles of the fasicles

4. Muscle Length

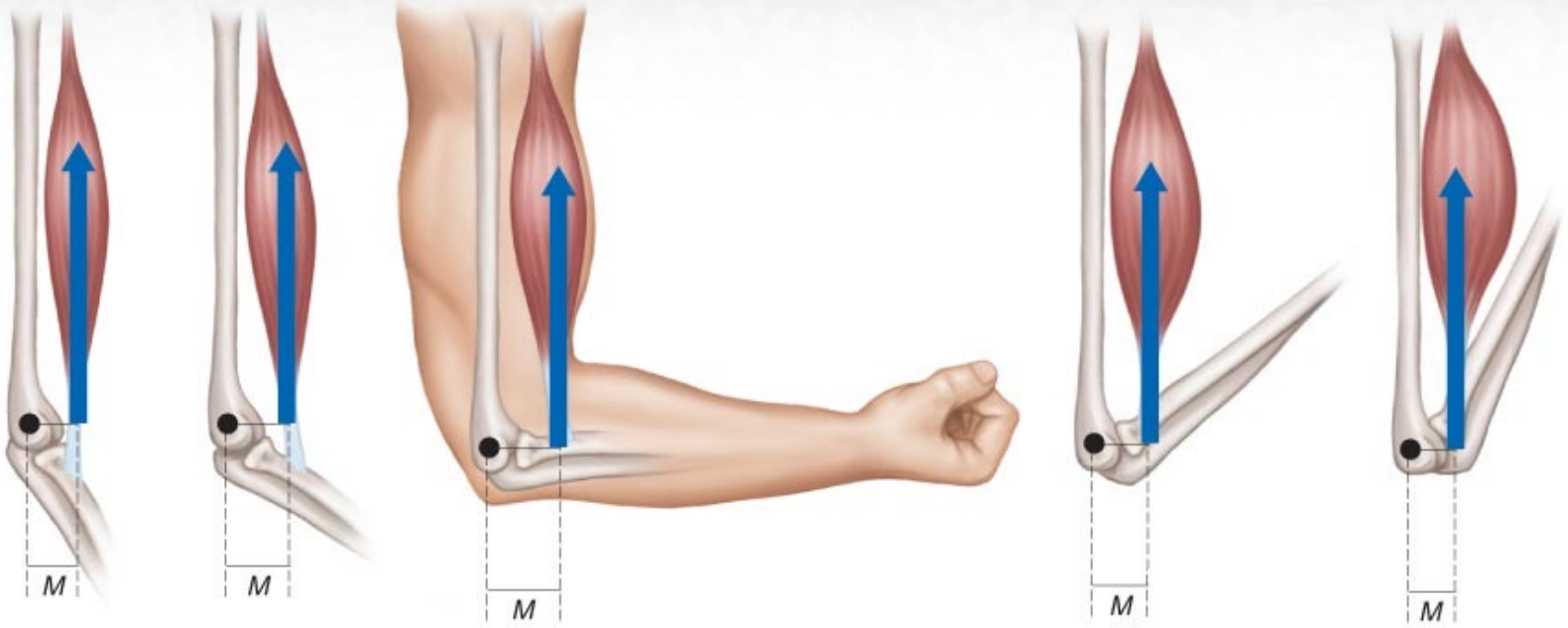


5. Joint angle

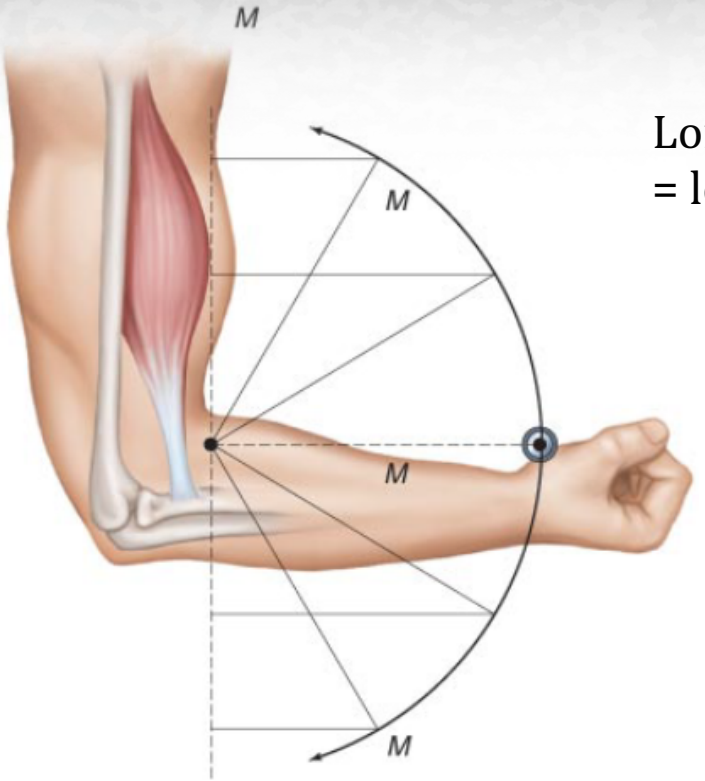




5. Joint angle: Notice the moment arm (joint axis to tendon attachment)



5. Joint angle: Notice the moment arm (joint axis to the ***load***)



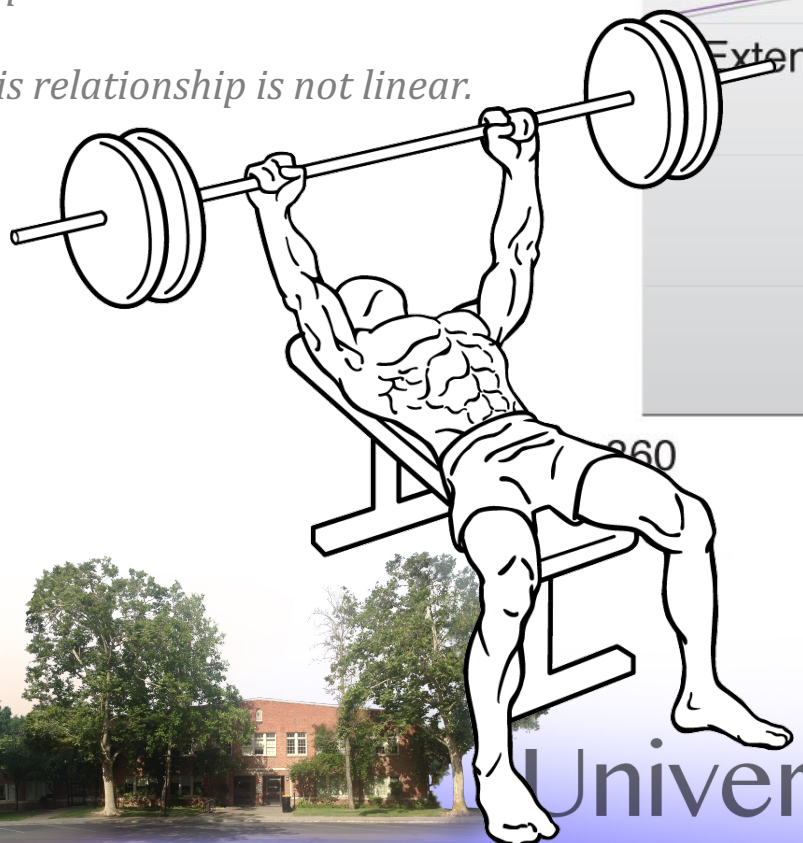
Longer moment arm (joint to the load)
= less mechanical advantage.

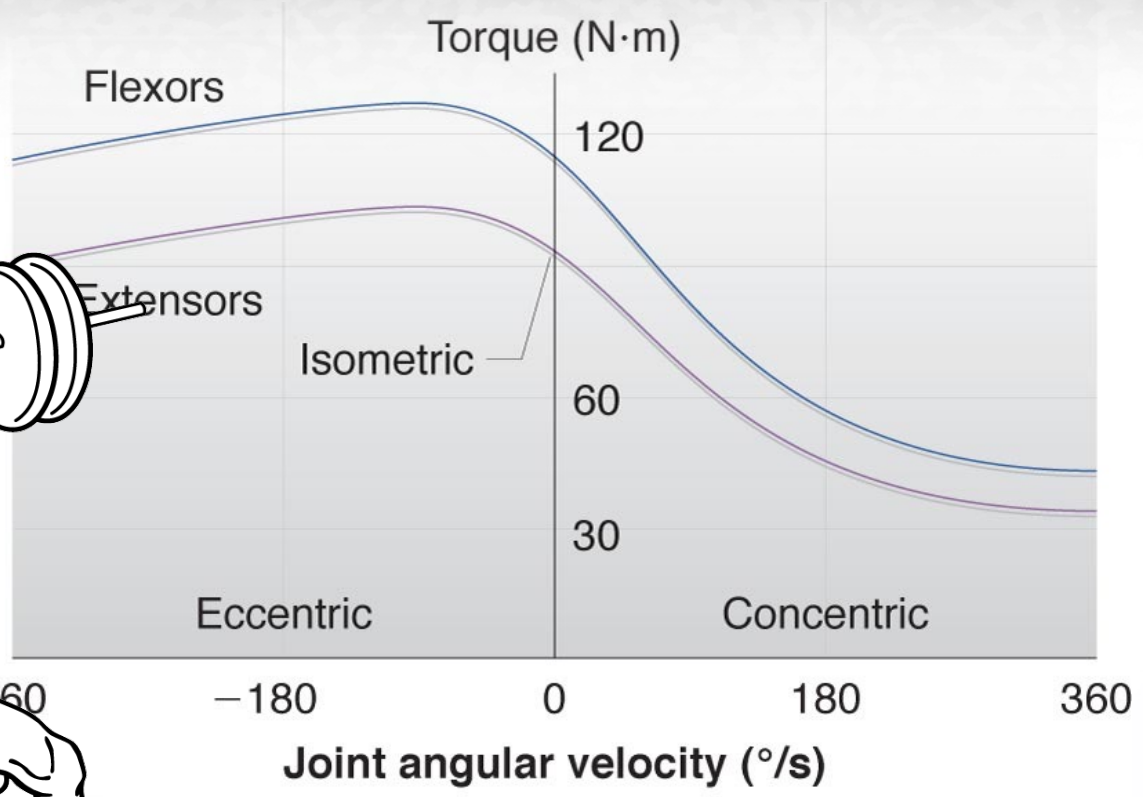
6. Muscle contraction velocity

A fast movement corresponds to a low force output.

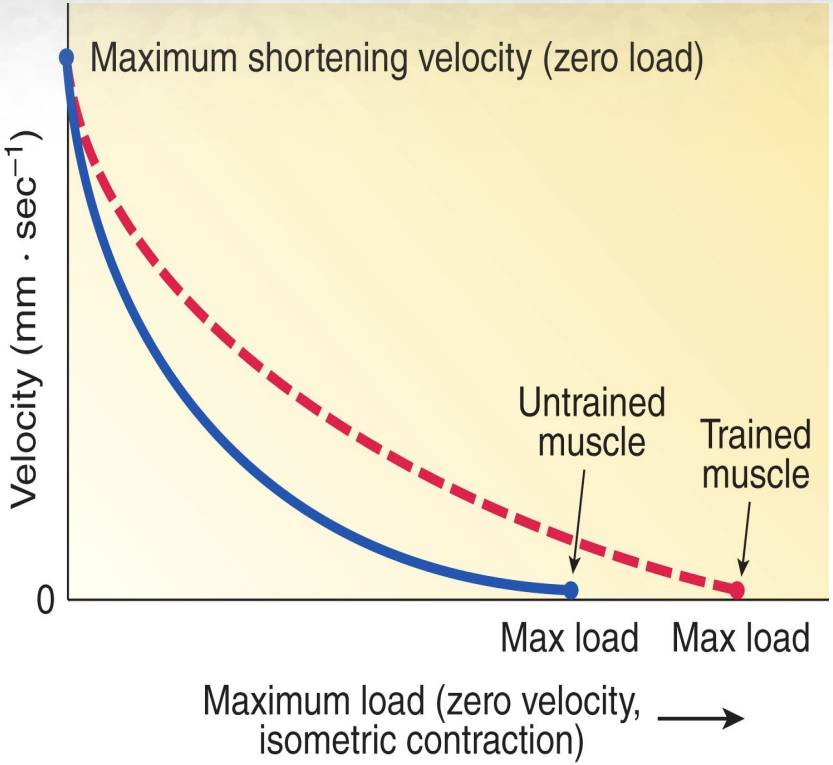
A slow movement has high force output.

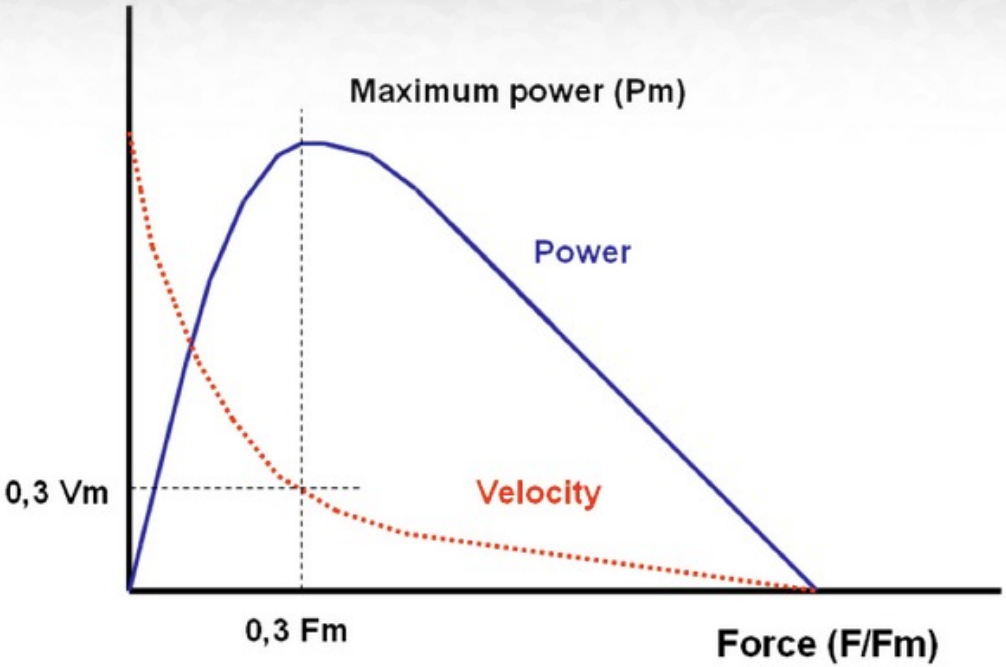
This relationship is not linear.





6. Muscle contraction velocity





As the load on the muscle is increased, the velocity of shortening is decreased. But training helps…

7. Strength-to-mass ratio

Sprinting / jumping:
Ratio affects ability to accelerate body.

Weight class sports:
Ratio helps determine relative success.

8. Body size

Google

Mass reduced, density is the same. At 1/10 your present height, your muscles would be 1/100 as strong, but you would only weigh 1/1,000 as much.

At the size of a nickel, you could jump out.

Can leap 200 times its body length

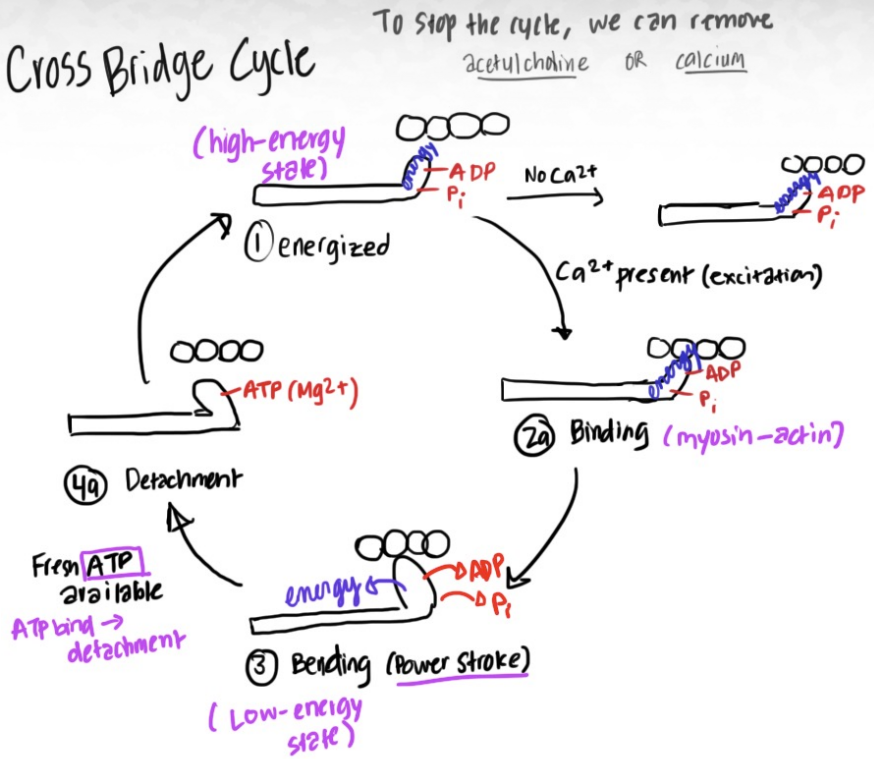
8. Body size

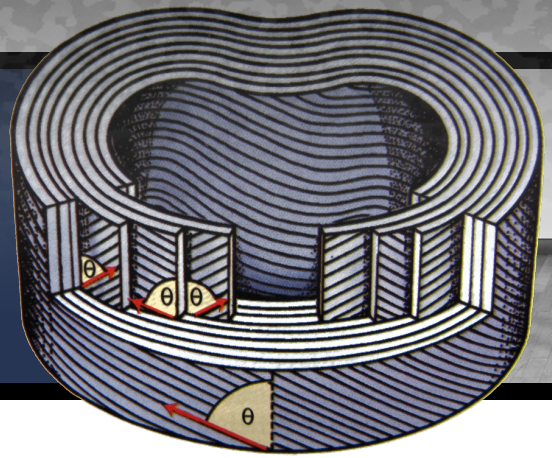
Body mass reflects body size, but body mass increases more rapidly than does functional muscle mass.

It's not *just* the result of bones and organs and blood and carbs and water and fat, etc.

Given constant body proportions, a smaller athlete has a higher strength-to-mass ratio.

9. Physiological explanations (cross-bridge cycling, etc.)





0195-9131/85/1705-0613$2.00/0
Vol. 17, No. 5
Printed in U.S.A.
MEDICINE AND SCIENCE IN SPORTS AND EXERCISE

Lumbar spine loading during half-squat exercises

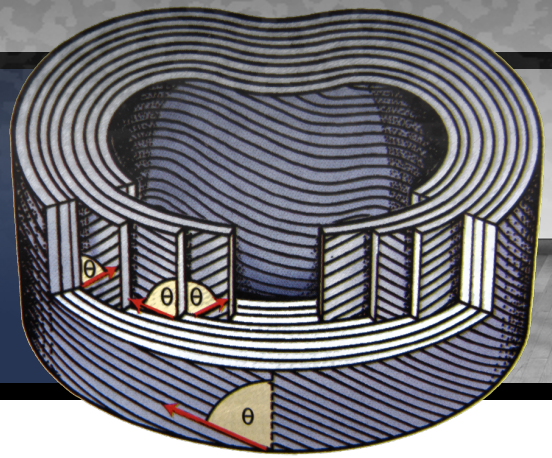
AURELIO CAPPOZZO, FRANCESCO FELICI, FRANCESCO FIGURA, and FABIO GAZZANI

CONCLUSIONS

During half-squat exercises with weights ranging from 8 to 160% of body weight, the trunk extensor muscle contraction forces were predicted to be between 30 and 50% of the relevant maximal isometric force. The compressive load acting on the L3–L4 segment was found to vary from approximately 6 to 10 times body weight.

In the execution of the half-squat exercise, the magnitude of trunk flexion appears to be the variable to which the spinal compressive load is most sensitive.

REVIEW: Compression



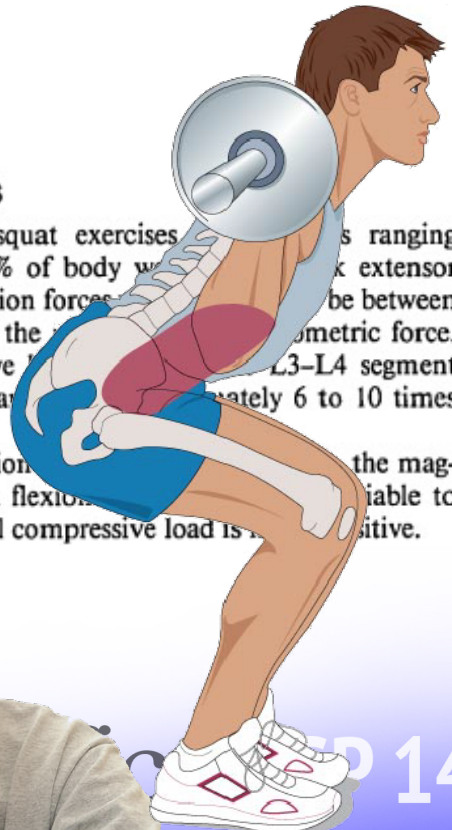
0195-9131/85/1705-0613$2.00/0
Vol. 17, No. 5 MEDICINE AND SCIENCE IN SPORTS AND EXERCISE
Printed in U.S.A. Copyright © 1985 by the American College of Sports Medicine

Lumbar spine loading duri… exercises

AURELIO CAPPOZZO, FRA… FELI…
FRANCESCO FIGURA, and…

CONCLUSIONS

During half-squat exercises … ranging from 8 to 160% of body w… extensor muscle contraction forces … be between 30 and 50% of the … metric force. The compressive … L3–L4 segment … to va… ately 6 to 10 times … the mag- … k flexion … iable to … al compressive load is … itive.

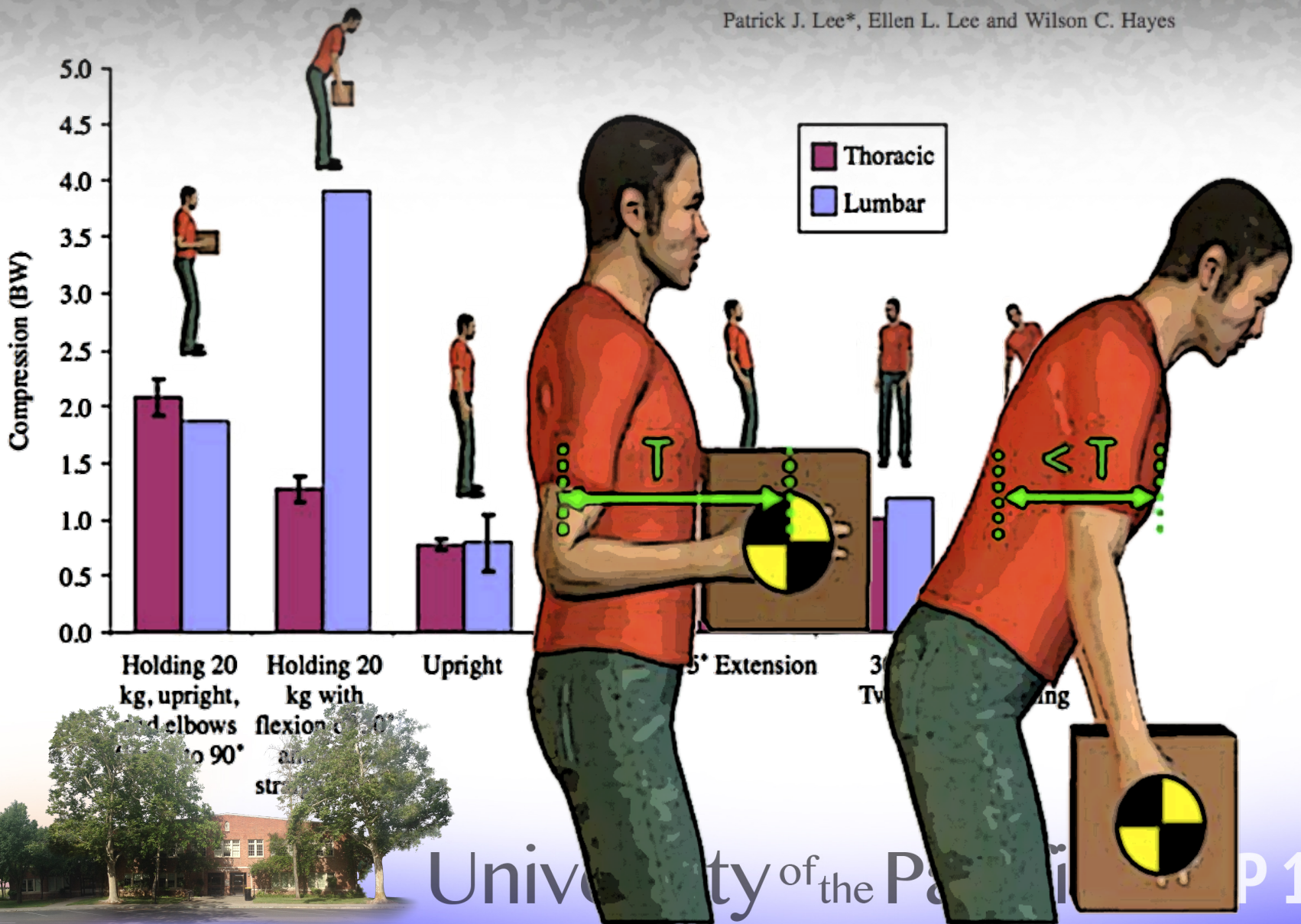


Universit… 147

Ergonomics, 2013
Vol. 56, No. 5, 832–841

The ratio of thoracic to lumbar compression force is posture dependent

Patrick J. Lee*, Ellen L. Lee and Wilson C. Hayes



Ann Acad Med Singapore 2015;44:244-51

A Review[illegible] Injury Cases Notified to the Ministry of Manpower from 2011 to 2012

Shiu Hong Wong, [1] *MBBS, MPH*, Kenneth KY Choy, [2] *MBBS, MMed (Occ Med), FAMS*

Abstract

Introduction: In Singapore, the notification of workplace accidents and occupational diseases is a legal requirement under the Workplace Safety and Health (Incident Reporting) Regulations. This review is to identify back injury cases with physical work stresses and propose recommendations for preventive measures. Materials and Methods: Cases involving back injuries notified to the Ministry of Manpower (MOM) from 1 January 2011 to 31 December 2012 were reviewed. Using predefined criteria, cases with physical work stresses were identified and the associated variables analysed. Results: A total of 1124 notifications involving back injuries were received from 2011 to 2012; 579 cases (52%) were identified to have physical work stresses, of which 447 cases (77%) were males and 375 (64%) of them were between the ages of 21 to 40 years. The commonest physical work stresses were carrying (35%), awkward postures (22%) and lifting (20%). Majority of the cases came from the following sectors: construction (14%), accommodation and food services (12%), transport and storage (12%), manufacturing (10%) and wholesale[illegible] retail trade (10%). Conclusion: We identified the main physical work stre[illegible] injury cases as carrying, awkward postures and lifting. The common indu[illegible] were construction, accommodation and food services, and transport and s[illegible] This is useful for programmes aimed at reducing physical work stresses whi[illegible] improved work and health outcomes for our workers.

You injure yourself somehow

J Appl Physiol 94: 1410–1420, 2003.
First published December 13, 2002; 10.1152/japplphysiol.01183.2001.

Once you have experienced pain…

Activation imbalances in lumbar spine muscles in the presence of chronic low back pain

Lars I. E. Oddsson[1] and Carlo J. De Luca[1,2]
[1]*NeuroMuscular Research Center and* [2]*Department of Biomedical Engineering, Boston University, Boston, Massachusetts 02215*

1: Conscious altering of muscular recruitment characteristics owing to the pain associated with activation of the affected tissue.

2: Unconscious, reflexive alteration in recruitment, which includes a delay in activation of the painful fibers, reduced magnitude of agonist recruitment, altered motor unit firing sequences in individual, whole muscles, and sometimes different muscles being called upon entirely, including simultaneous contributions from different body segments.

J Appl Physiol 94: 1410–1420, 2003.
First published December 13, 2002; 10.1152/japplphysiol.01183.2001.

Activation imbalances in lumbar spine muscles in the presence of chronic low back pain

Lars I. E. Oddsson[1] and Carlo J. De Luca[1,2]

In the shoulder too: After the athlete is injured, kinetics will change.

Shoulder-specific reprogramming

Rajaratnam *et al. BMC Sports Science, Medicine, and Rehabilitation* 2013, **5**:26
http://www.biomedcentral.com/2052-1847/5/2/26

BMC Sports Science, Medicine & Rehabilitation

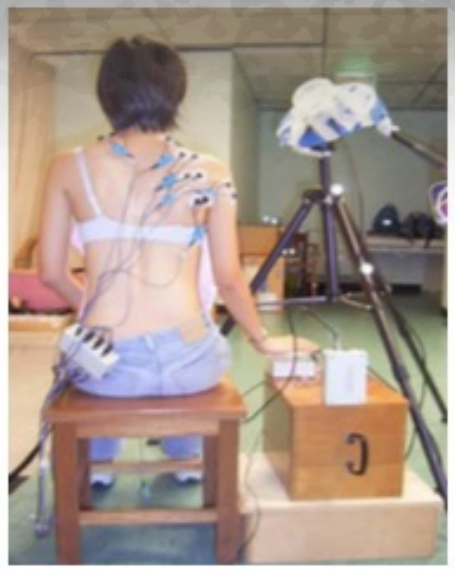
Control strategies to re-establish glenohumeral stability after shoulder injury

Bala S Rajaratnam[1*], James CH Goh[2] and Prem V Kumar[2]

0363-5465/103/3131-0542$02.00/0
THE AMERICAN JOURNAL OF SPORTS MEDICINE, Vol. 31, No. 4

Scapular Muscle Recruitment Patterns: Trapezius Muscle Latency with and without Impingement Symptoms

Ann M. Cools,*† PT, Erik E. Witvrouw,* PT, PhD, G… …clercq,‡ MD,
Lieven A. Danneels,* PT, PhD, and Dirk … …ier,* PT, PhD



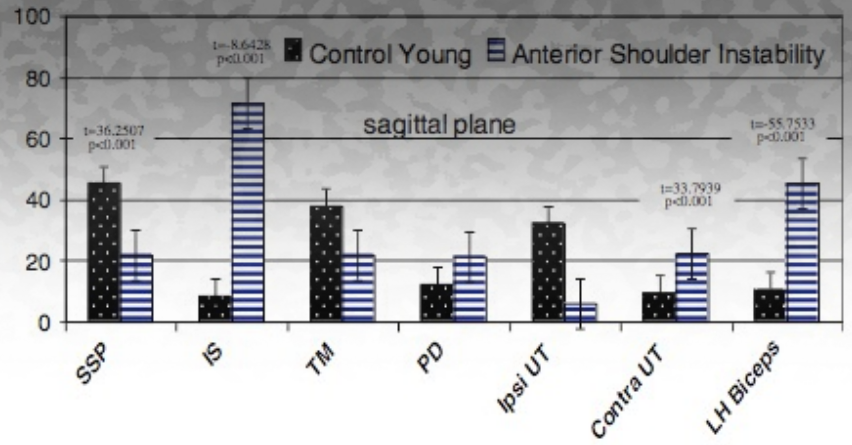
Fine Wire electrodes
(sampling rate 1800Hz)

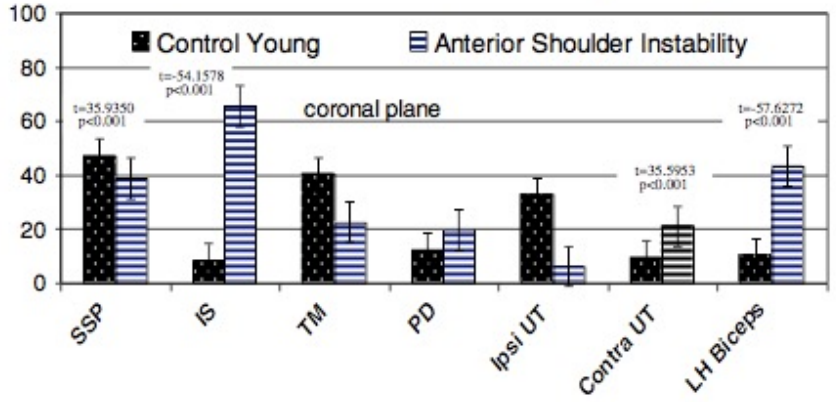
- Infraspinatus
- Supraspinatus
- Teres Major

Surface electrodes
(sampling rate 1800Hz)

- Posterior Deltoid
- Bilateral Upper Trapezius
- Biceps brachii

Figure 1 Experimental set-up.





Control strategies to re-establish glenohumera stability after shoulder injury

Bala S Rajaratnam[1*], James CH Goh[2] and Prem V Kumar[2]

Pain 86 (2000) 151–162

Experimental muscle pain produces central modulation of proprioceptive signals arising from jaw muscle spindles

Norman F. Capra, Jin Y. Ro*

Not just voluntarily:

Even in your muscle spindles.

These are centrally modulated in the presence of pain.

PHYSIOLOGICAL REVIEWS
Vol. 80, No. 2, April 2000
Printed in U.S.A.

Synaptic Control of Motoneuronal Excitability

JENS C. REKLING,* GREGORY D. FUNK,* DOUGLAS A. BAYLISS, XIAO-WEI DONG, AND JACK L. FELDMAN

Substances that are released during central sensitization (e.g., substance P) can modify motor neuron excitability.

This can alter muscle recruitment patterns.

IJSPT

ORIGINAL RESEARCH

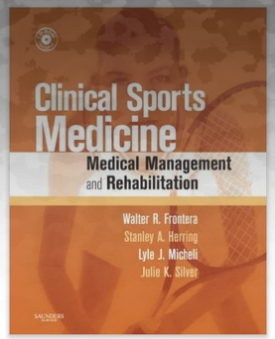
INJURY RISK IS ALTERED BY PREVIOUS INJURY: A SYSTEMATIC REVIEW OF THE LITERATURE AND PRESENTATION OF CAUSATIVE NEUROMUSCULAR FACTORS

Jessica Fulton, PT, DPT, HFS[1]
Kathryn Wright, PT, DPT[1]
Margaret Kelly, PT, DPT, CSCS[1]
Britanee Zebrosky, PT, DPT, CSCS[1]
Matthew Zanis, PT, DPT, ATC, CSCS[1]
Corey Drvol, PT, DPT[1]
Robert Butler, PT, PhD[1]

Altered neural recruitment changes gross kinematics.

Altered firing predisposes you to re-injury.

REVIEW: Rewiring After Injury



CHAPTER 26

Shoulder Injuries

Jason Nielson and Peter Gerbino

FIG. 153

J Appl Physiol 94: 1410–1420, 2003.
First published December 13, 2002; 10.1152/japplphysiol.01183.2001.

Activation imbalances in lumbar spine muscles in the presence of chronic low back pain

Lars I. E. Oddsson[1] and Carlo J. De Luca[1,2]
[1]*NeuroMuscular Research Center and* [2]*Department of Biomedical Engineering, Boston University, Boston, Massachusetts 02215*

These phenomena change the underlying biomechanics of a serve, a strike, a throw, a swing, a pass, a kick, typically increase the risk of re-injury, and likely escape detection by the standard return-to-play testing battery, which evaluates only pain, strength, and range of motion (and maybe function/coordination, depending on the joint).

REMEMBER

10820 • The Journal of Neuroscience, September 2, 2009 • 29(35):10820–10826

Behavioral/Systems/Cognitive

Motor Unit Recruitment Strategies Are Altered during Deep-Tissue Pain

Kylie Tucker,[1] Jane Butler,[3] Thomas Graven-Nielsen,[4] Stephan Riek,[2] and Paul Hodges[1]

PART TWO

Congenital Insensitivity to Pain and Anhydrosis: Diagnostic and Therapeutic Dilemmas revisited

[1]KS Ravichandra, [2]Chaitanya Ram Kandregula, [3]Srikanth Koya, [4]Disha Lakhotia

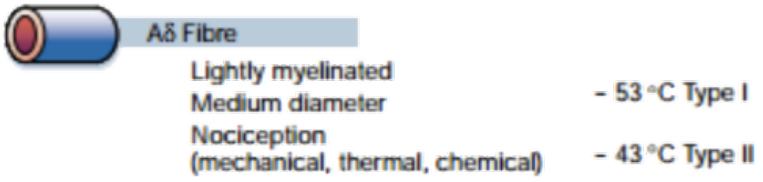
International Journal of Clinical Pediatric Dentistry, January-April 2015;8(1):75-81

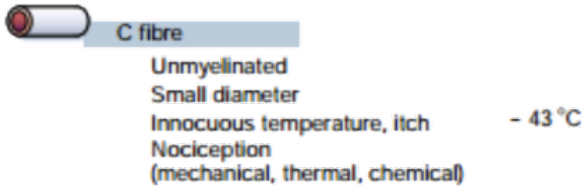
Gene mutations.

Depending on the gene, a variety of consequences.

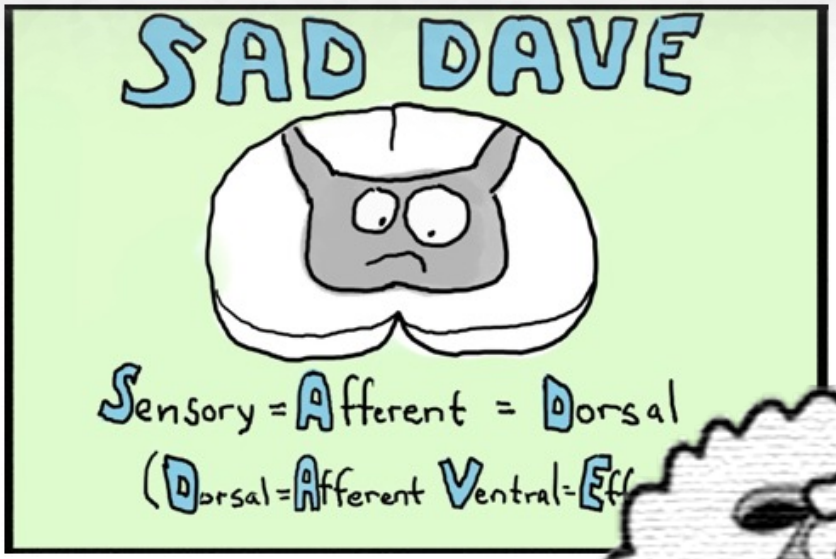
Afferents fail to transmit nociceptor information.

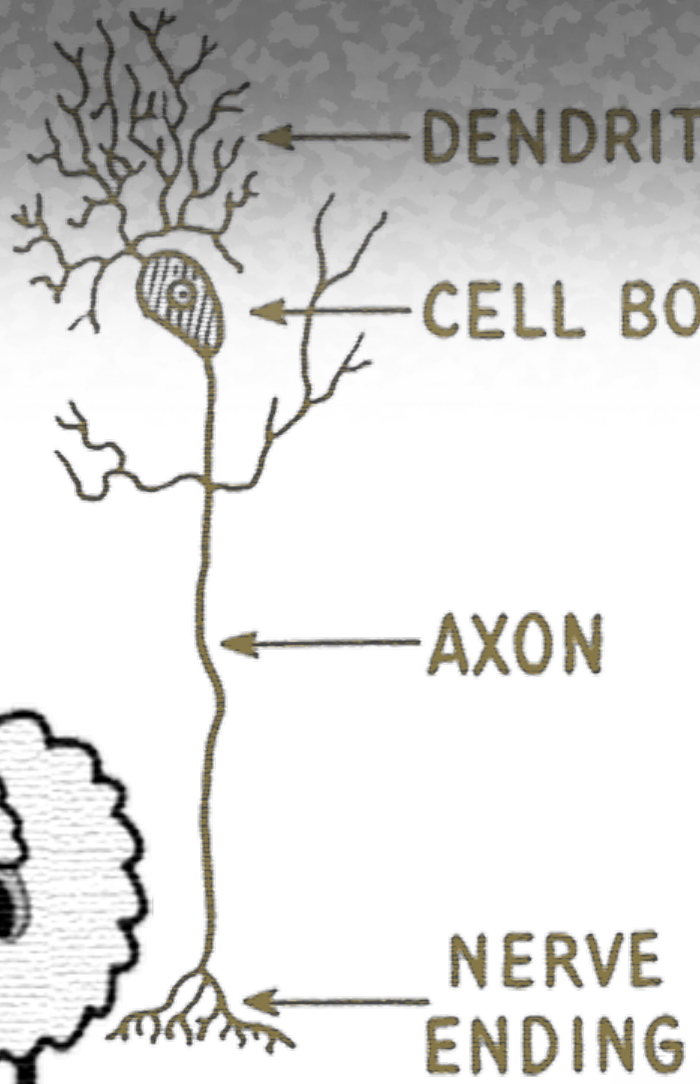
Primarily caused by a mutation of the tropomyosin receptor kinase A (TrkA) gene, which codes for TrkA, which mediates nerve growth factor, which is critical to the formation of autonomic and sensory neurons. Without it, there will be an absence of C fibers and a reduction of Aδ fibers. Thus an inability to sense heat and pain.





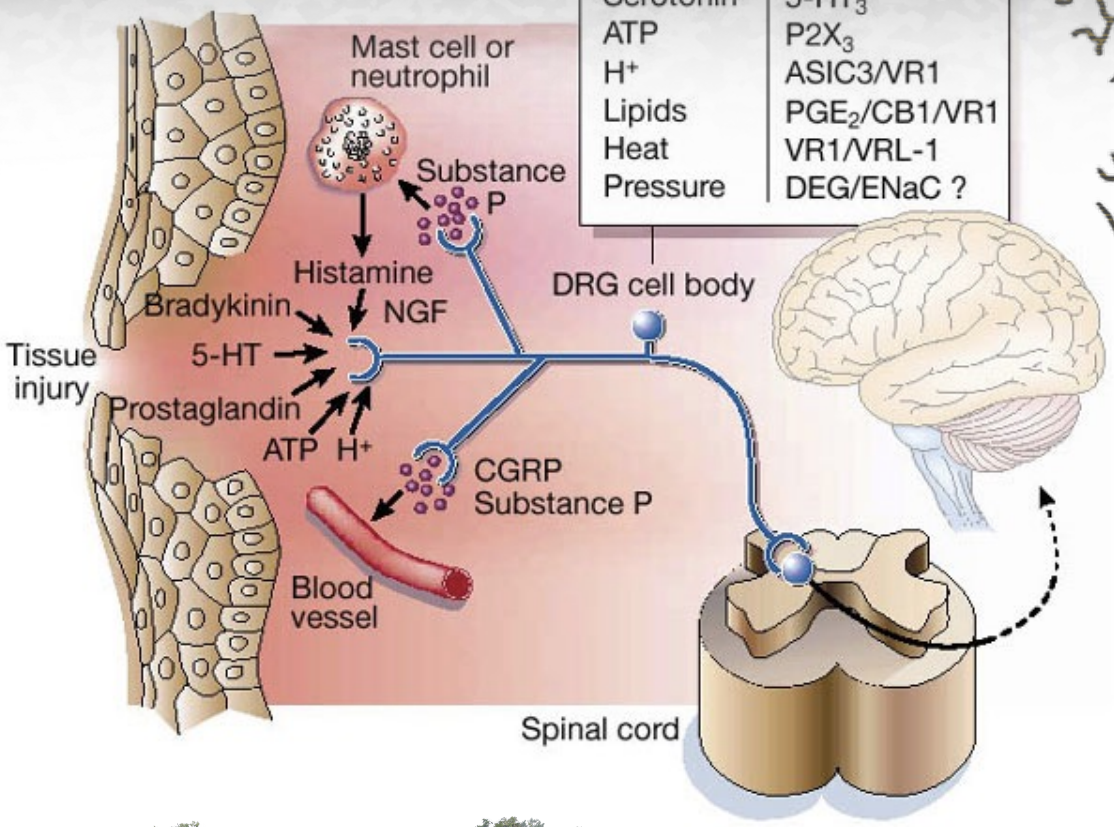
REVIEW: Molecular Mechanisms of Nociception

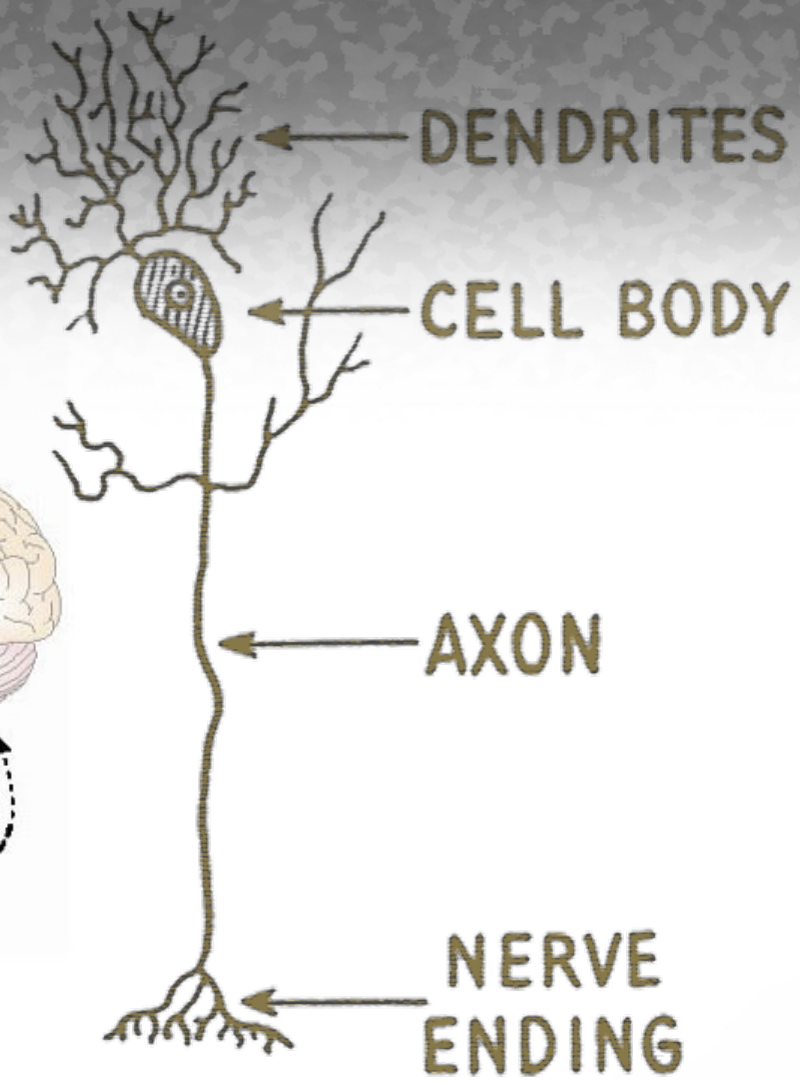




REVIEW: Molecular Mechanisms of Nociception

Stimulus	Representative receptor
NGF	TrkA
Bradykinin	BK_2
Serotonin	$5\text{-}HT_3$
ATP	$P2X_3$
H^+	ASIC3/VR1
Lipids	PGE_2/CB1/VR1
Heat	VR1/VRL-1
Pressure	DEG/ENaC ?





Neurobiology of Pain 6 (2019) 100028

Cyclic nucleotide signaling in sensory neuron hyperexcitability and chronic pain after nerve injury

Ze-Hua Li[a,b,1], Dong Cui[a,b,1], Cheng-Jie Qiu[a], Xue-Jun Song[a,b,*]

The cyclic nucleotide signaling, including cAMP-PKA and cGMP-PKG pathways, has been well known to play critical roles in regulating cellular growth, metabolism and many other intracellular processes. In recent years, more and more studies have uncovered the roles of cAMP and cGMP in the nervous system. The cAMP and cGMP signaling mediates chronic pain induced by different forms of injury and stress. Here we summarize the roles of cAMP-PKA and cGMP-PKG signaling pathways in the pathogenesis of chronic pain after nerve injury. In addition, acute dissociation and chronic compression of the dorsal root ganglion (DRG) neurons, respectively, leads to neural hyperexcitability possibly through PAR2 activation-dependent activation of cAMP-PKA pathway. Clinically, radiotherapy can effectively alleviate bone cancer pain at least partly through inhibiting the cancer cell-induced activation of cAMP-PKA pathway. Roles of cyclic nucleotide signaling in neuropathic and inflammatory pain are also seen in many other animal models and are involved in many pro-nociceptive mechanisms including the activation of hyperpolarization-activated cyclic nucleotide (HCN)-modulated ion channels and the exchange proteins directly activated by cAMP (EPAC). Further understanding the roles of cAMP and cGMP signaling in the pathogenesis of chronic pain is theoretically significant and clinically valuable for treatment of chronic pain.

PAIN 158 (2017) 543–559

The role of calcitonin gene–related peptide in peripheral and central pain mechanisms including migraine

Smriti Iyengar[a,*], Michael H. Ossipov[b], Kirk W. Johnson[a]

Numerous behavioral and electrophysiologic studies provide strong evidence that CGRP contributes to central sensitization in animal models of inflammation or nerve injury.[13,14,159,175,200]

Cell and Tissue Research
https://doi.org/10.1007/s00441-018-2922-y

Substance P and pain chronicity

W. Zieglgänsberger [1]

Substance P (SP) is a highly conserved member of the tachykinin peptide family that is widely expressed throughout the animal kingdom. The numerous members of the tachykinin peptide family are involved in a multitude of neuronal signaling pathways, mediating sensations and emotional responses (Steinhoff et al. in Physiol Rev 94:265–301, 2014). In contrast to receptors for classical transmitters, such as glutamate (Parsons et al. in Handb Exp Pharmacol 249–303, 2005), only a minority of neurons in certain brain areas express neurokinin receptors (NKRs) (Mantyh in J Clin Psychiatry 63:6–10, 2002). SP is also expressed by a variety of non-neuronal cell types such as microglia, as well as immune cells (Mashaghi et al. in Cell Mol Life Sci 73:4249–4264, 2016). SP is an 11-amino acid neuropeptide that preferentially activates the neurokinin-1 receptor (NK1R). It transmits nociceptive signals via primary afferent fibers to spinal and brainstem second-order neurons (Cao et al. in Nature 392:390–394, 1998). Compounds that inhibit SP's action are being investigated as potential drugs to relieve pain. More recently, SP and NKR have gained attention for their role in complex psychiatric processes. It is a key goal in the field of pain research to understand mechanisms involved in the transition between acute pain and chronic pain. The influence of emotional and cognitive inputs and feedbacks from different brain areas makes pain not only a perception but an experience (Zieglgänsberger et al. in CNS Spectr 10:298–308, 2005; Trenkwaldner et al. Sleep Med 31:78–85, 2017). This review focuses on functional neuronal plasticity in spinal dorsal horn neurons as a major relay for nociceptive information.

REVIEW: Molecular Mechanisms of Nociception

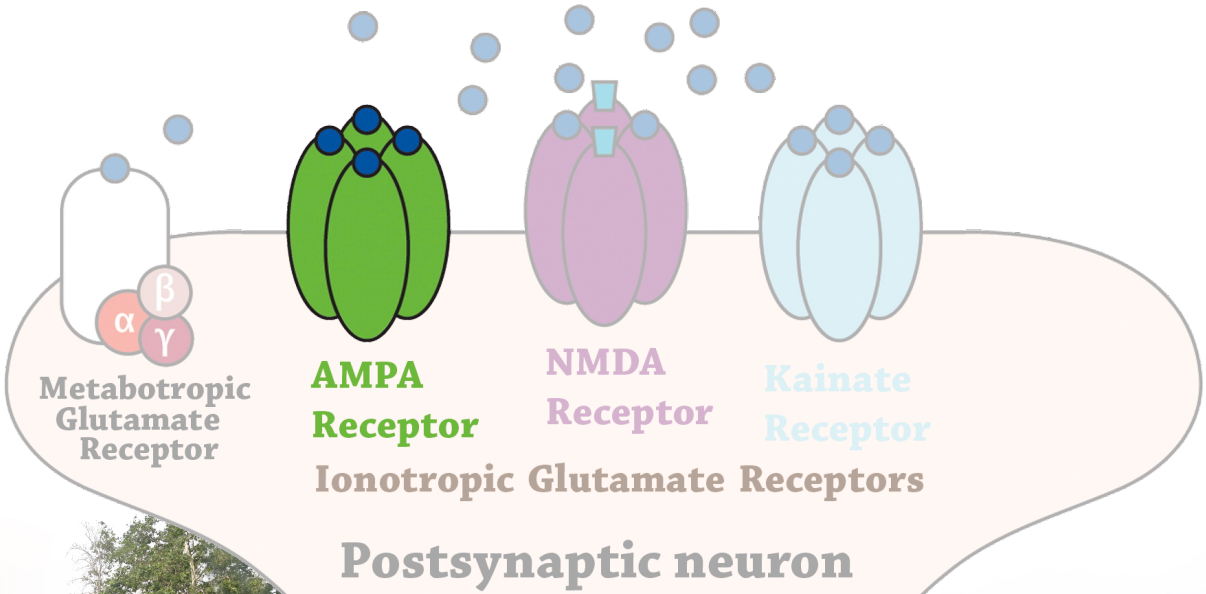
Neurosci Res. 2008 October ; 62(2): 97–104.

Peripheral AMPA receptors contribute to muscle nociception and *c-fos* activation

Yang-Hyun Chun[2], Dorie Frank[1], Jong-Seok Lee[1], Youping Zhang[1], Q-Schick Auh[2], and Jin Y. Ro[1,*]

α-amino-3-hydroxy-5 methylisoxazole-4-proprionic acid receptors

AMPA Receptor permits the influx of sodium, which depolarizes the post-synaptic membrane.



REVIEW: Molecular Mechanisms of Nociception

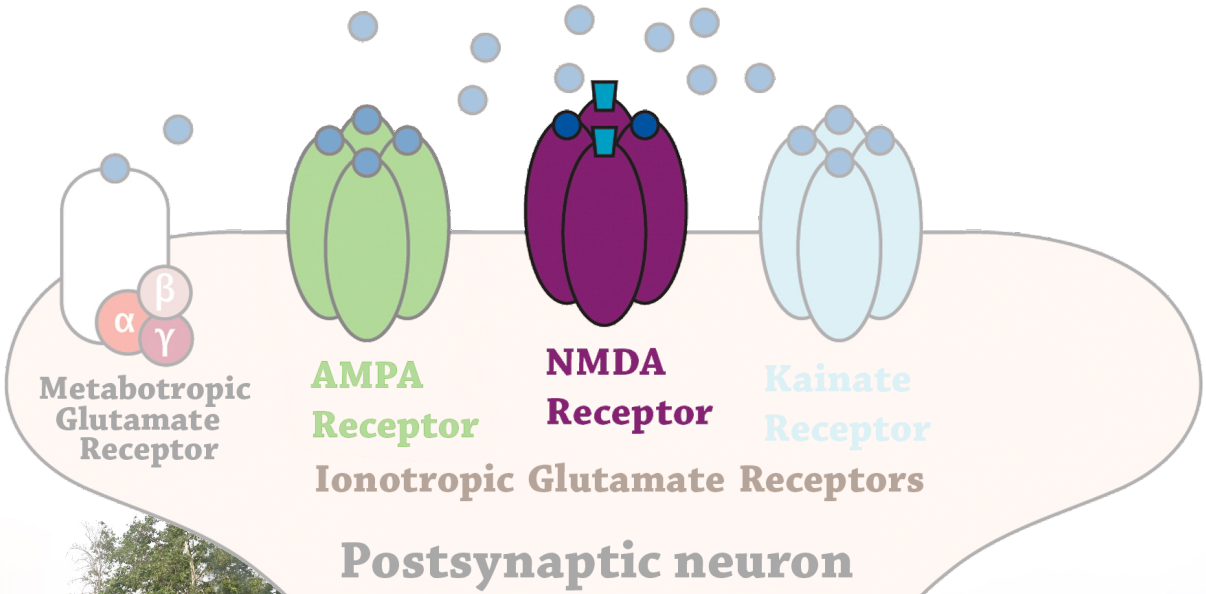
Anesth Analg 2003;97:1108–16

The Role of *N*-Methyl-D-Aspartate (NMDA) Receptors in Pain: A Review

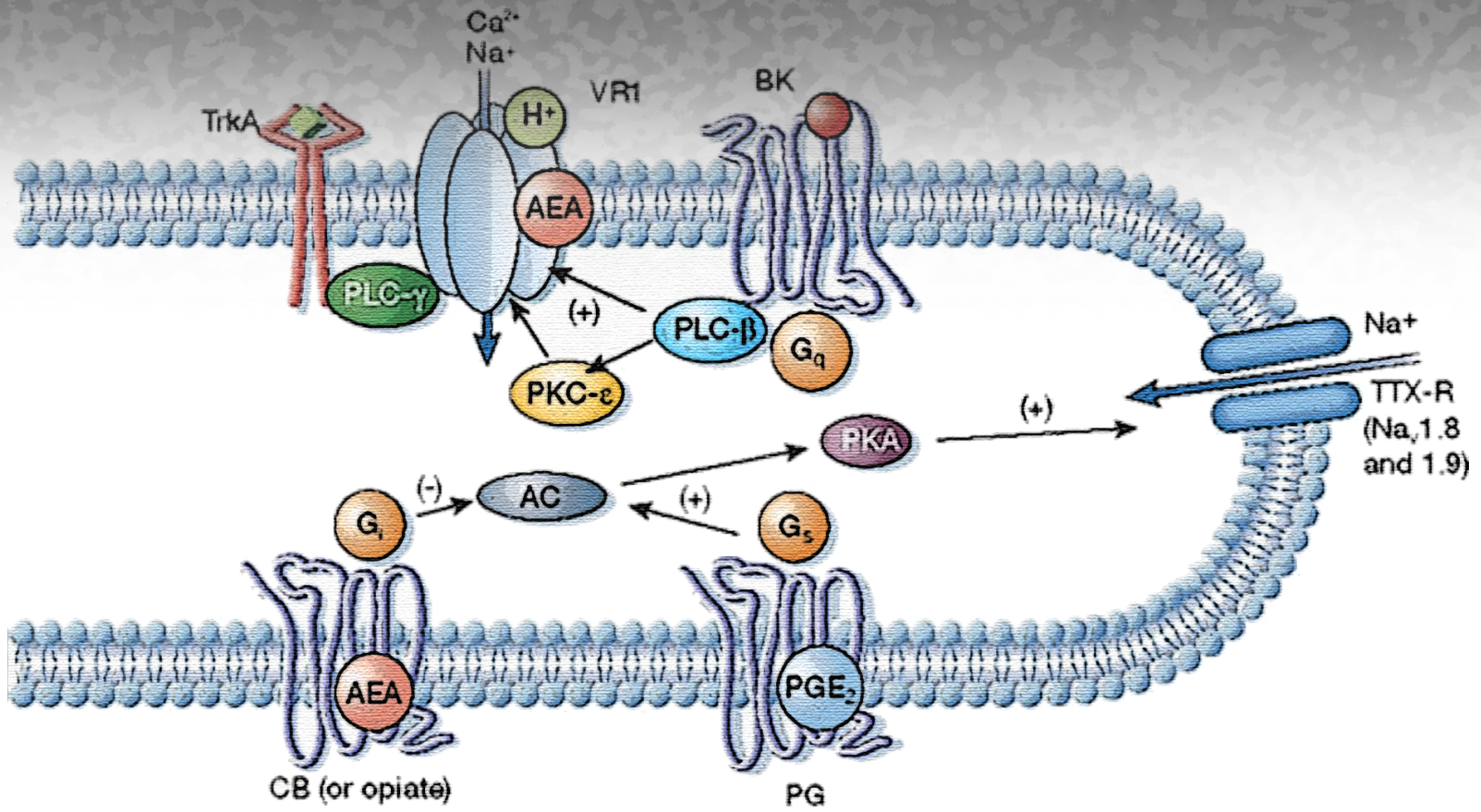
Andrei B. Petrenko, MD, Tomohiro Yamakura, MD, PhD, Hiroshi Baba, MD, PhD, and Koki Shimoji, MD, PhD, FRCA

N-methyl-d-aspartate receptors

NMDA Receptor permits the influx of sodium *and* calcium; the calcium can affect gene expression (and interact with other proteins and receptors) related to long-term changes.



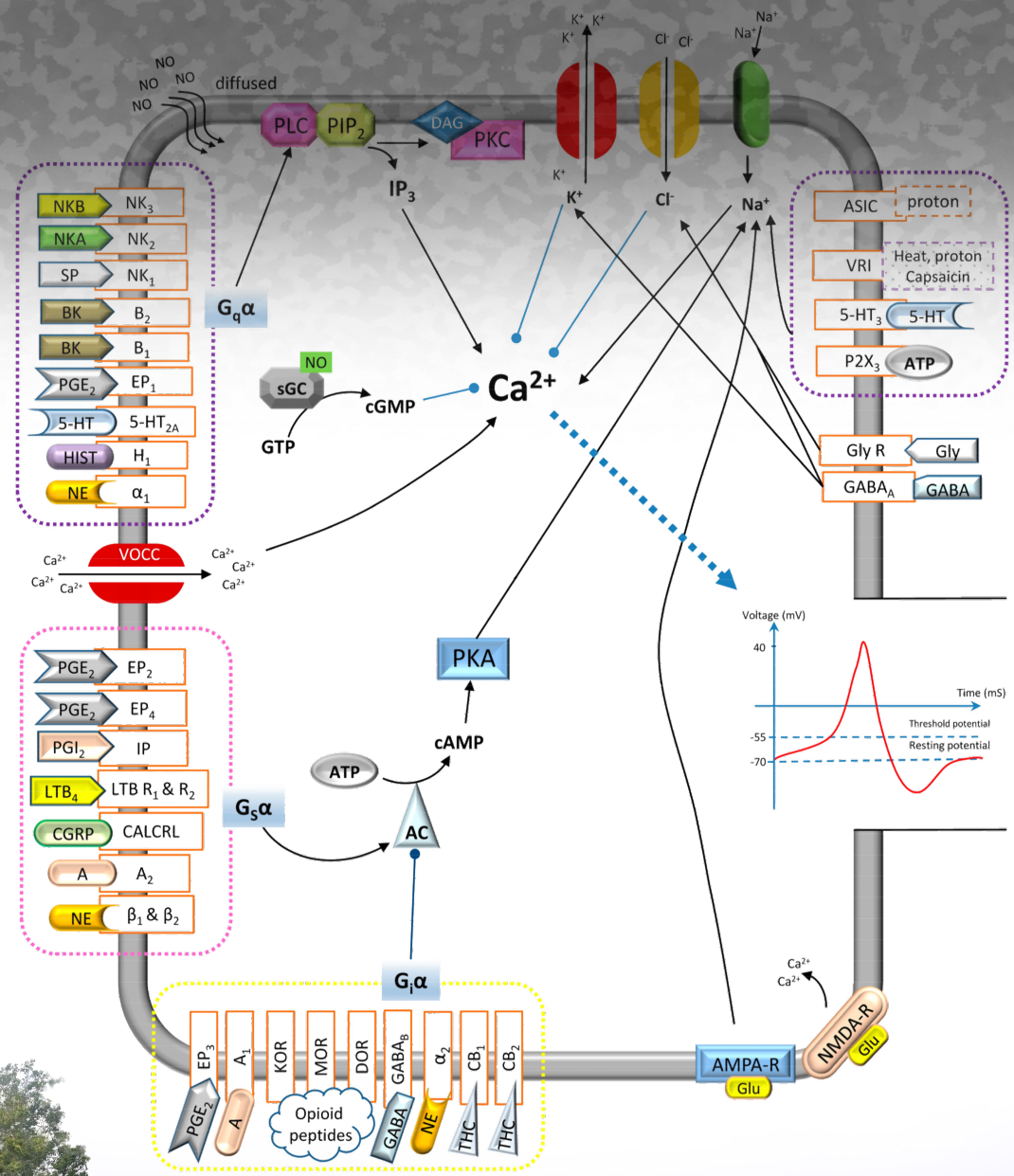
REVIEW: Molecular Mechanisms of Nociception



You should know
all *of this by now.*

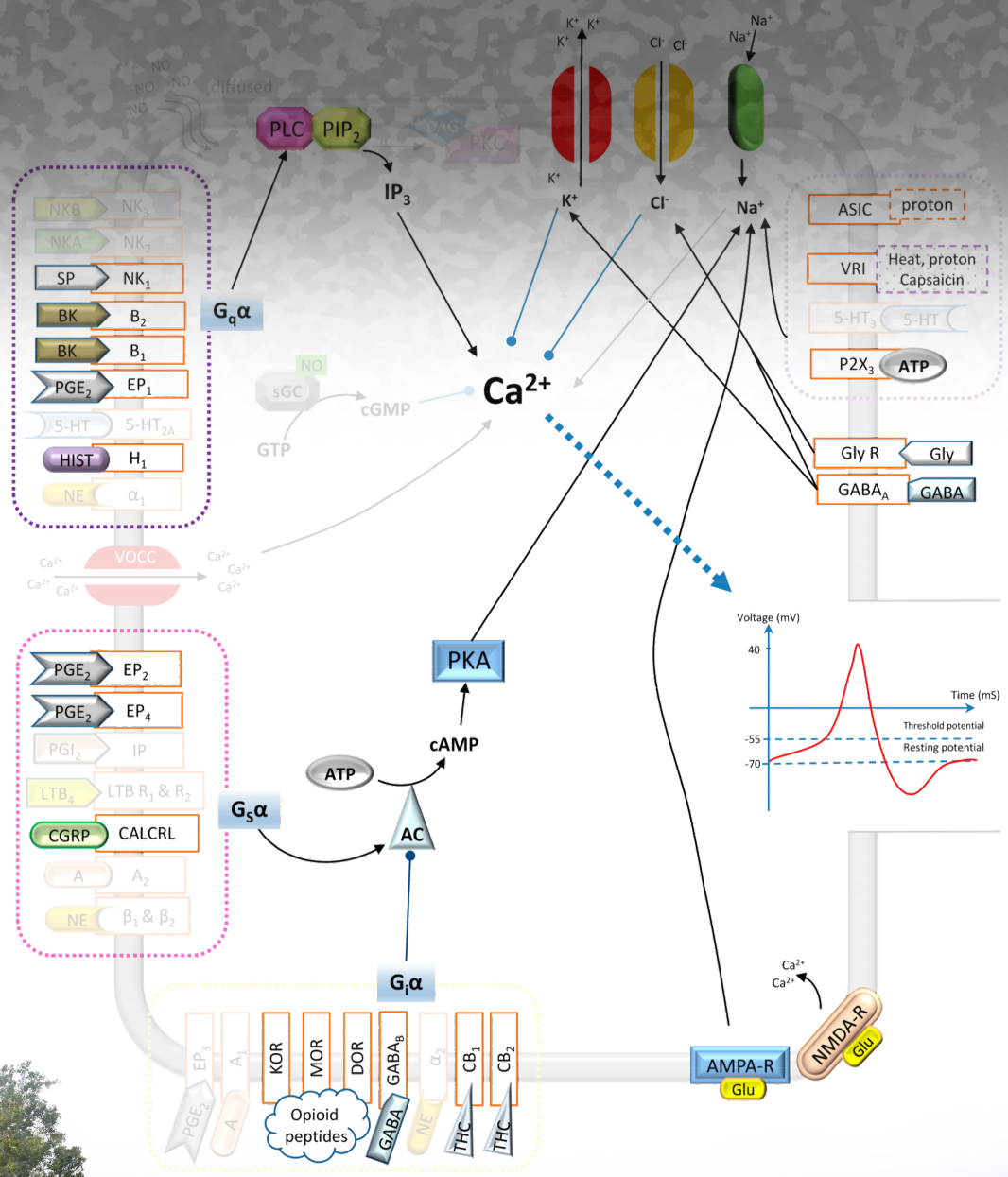
REVIEW: Molecular Mechanisms of Nociception

A nociceptor's cell-surface receptors:



REVIEW: Molecular Mechanisms of Nociception

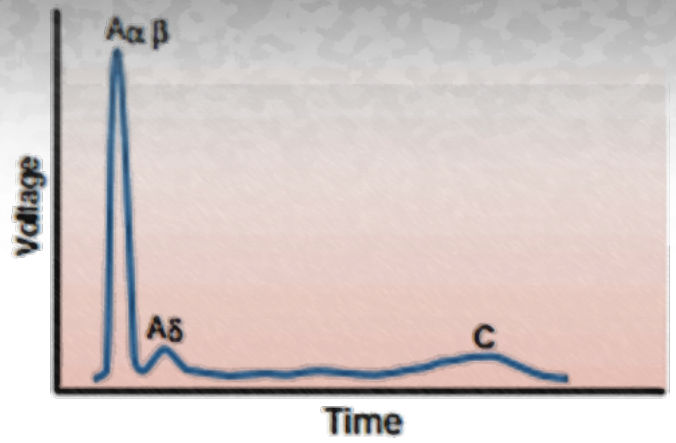
A nociceptor's cell-surface receptors:

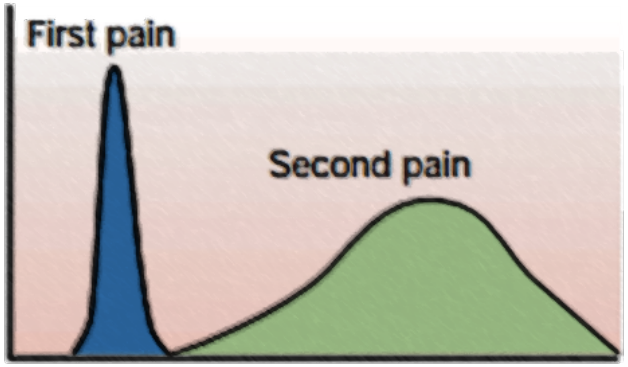


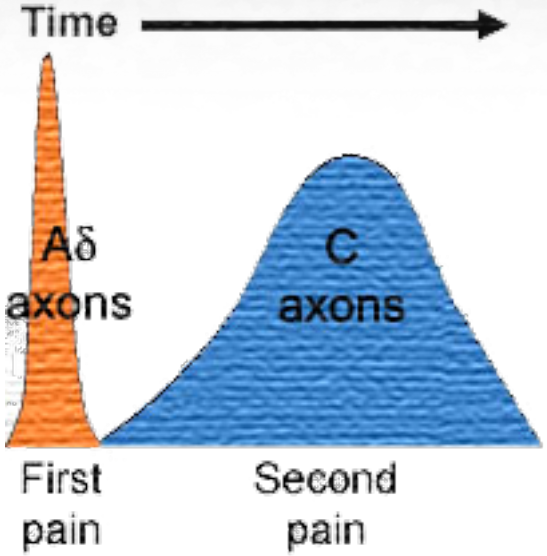
REVIEW: Molecular Mechanisms of Nociception

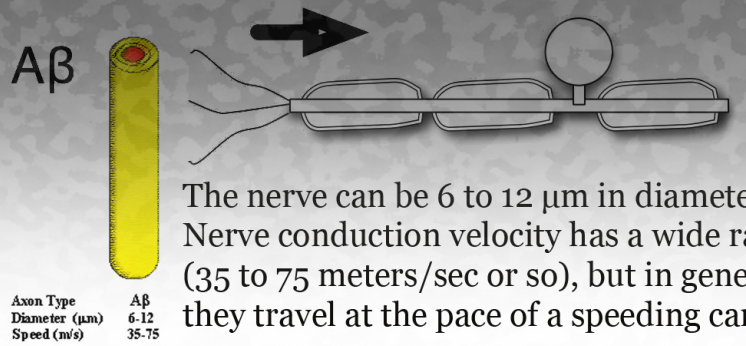
Primary afferent axons

		Thermal threshold
Aα and Aβ fibres	Myelinated Large diameter Proprioception, light touch	None
Aδ Fibre	Lightly myelinated Medium diameter Nociception (mechanical, thermal, chemical)	– 53 °C Type I – 43 °C Type II
C fibre	Unmyelinated Small diameter Innocuous temperature, itch Nociception (mechanical, thermal, chemical)	– 43 °C

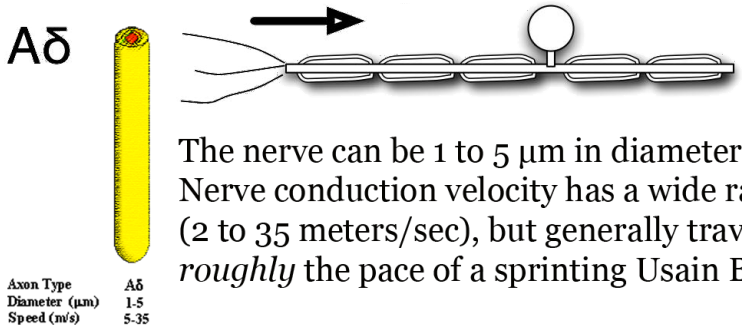




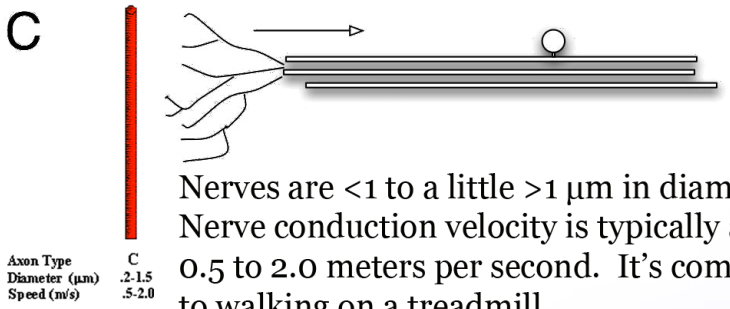




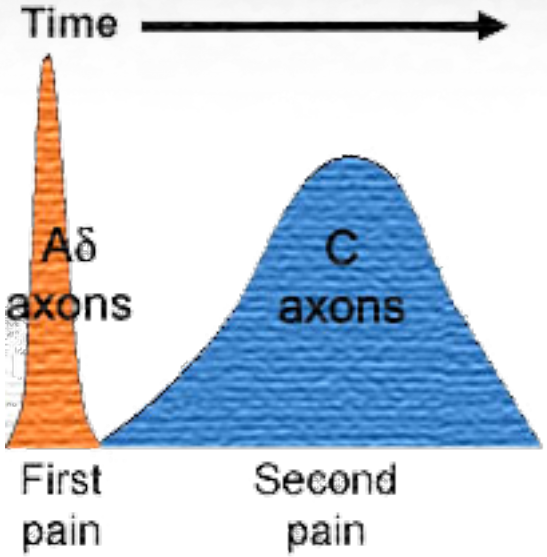
The nerve can be 6 to 12 µm in diameter. Nerve conduction velocity has a wide range (35 to 75 meters/sec or so), but in general, they travel at the pace of a speeding car.

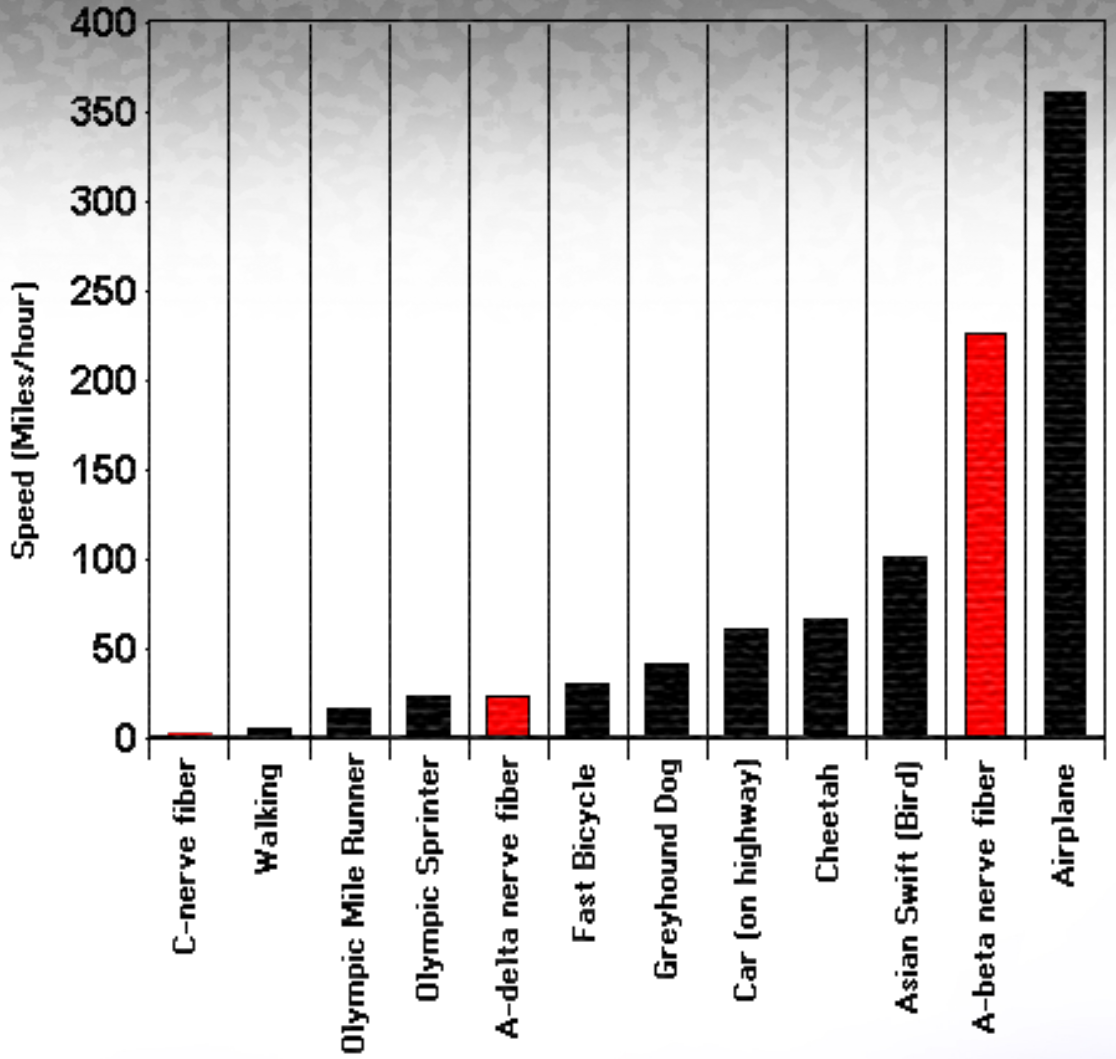


The nerve can be 1 to 5 µm in diameter. Nerve conduction velocity has a wide range (2 to 35 meters/sec), but generally travels at *roughly* the pace of a sprinting Usain Bolt.



Nerves are <1 to a little >1 µm in diameter. Nerve conduction velocity is typically about 0.5 to 2.0 meters per second. It's comparable to walking on a treadmill.


Time
Aδ axons
C axons
First pain
Second pain

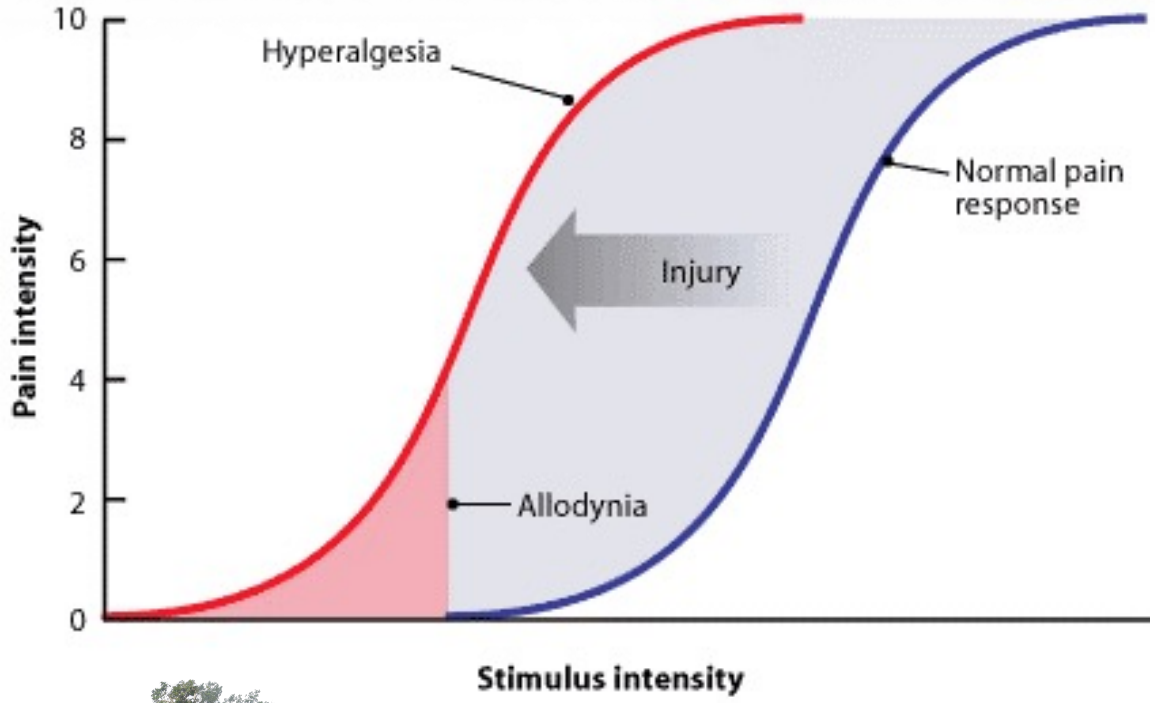

Speed (Miles/hour)
400
350
300
250
200
150
100
50
0
C-nerve fiber
Walking
Olympic Mile Runner
Olympic Sprinter
A-delta nerve fiber
Fast Bicycle
Greyhound Dog
Car (on highway)
Cheetah
Asian Swift (Bird)
A-beta nerve fiber
Airplane

If you were to touch Will Ferrell's skin, it would cause him pain.

Nociceptor depolarization thresholds are probably lowered, silent nociceptors may have been activated, and it's possible that other nerves have been rewired, such as touch-sensitive fibers rerouting synaptic connections in the spine to areas that are supposed to receive input from nociceptors.

What are all of these changes called?

Hyperalgesia *vs.* allodynia



REVIEW: Injuries, Rehabilitation, and Reconditioning

Macrotrauma

Microtrauma

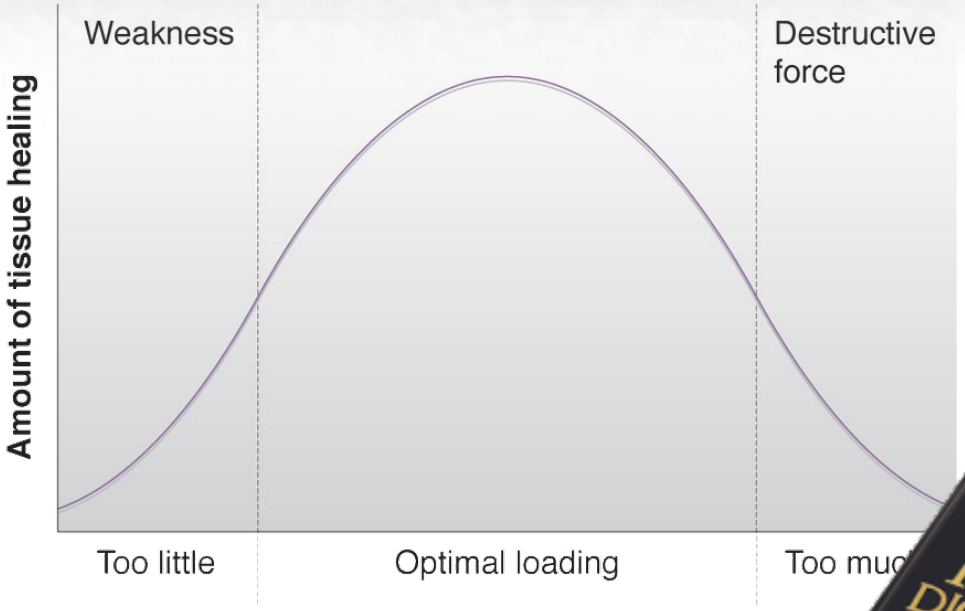
Indication

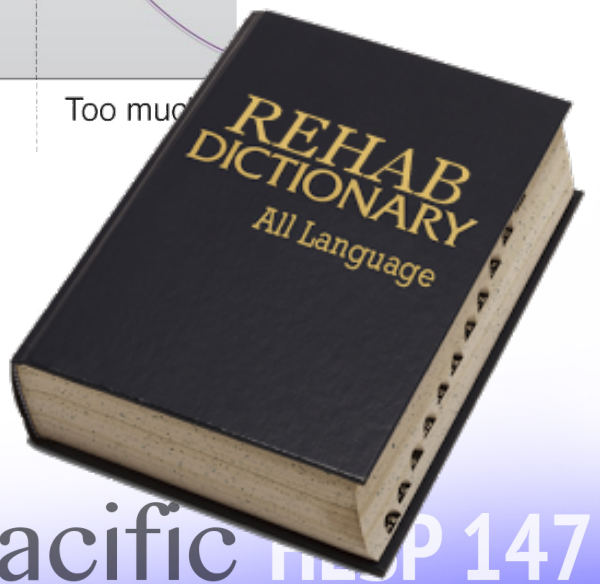
Contraindication

Dislocation

Subluxation

Sprain vs. strain





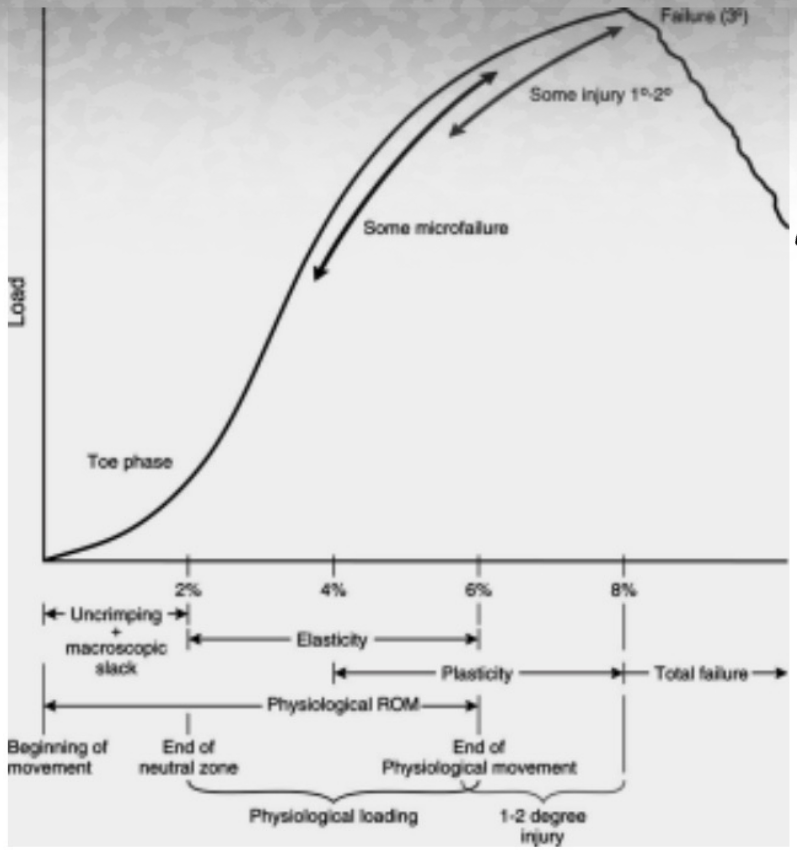
Macrotrauma

Overwhelming force (more than the tissue can tolerate). Force vector and body position determine which tissues are involved.

Microtrauma

There is a gradual accumulation of micro-insults.
Anabolism vs. catabolism: Catabolic signal predominates.

Microtrauma ..... Macrotrauma.



Example of how your tendon tolerates (or doesn't tolerate) stress / load.

Macrotrauma: Overwhelming force (more than the tissue can tolerate). It's probably bone, ligament, and/or muscle-tendon.

WHICH IS WHICH?

You hardly ever injure just one thing; completely isolated injuries are scarce. If you injure one thing, chances are you've blown through some other tissues in the process.

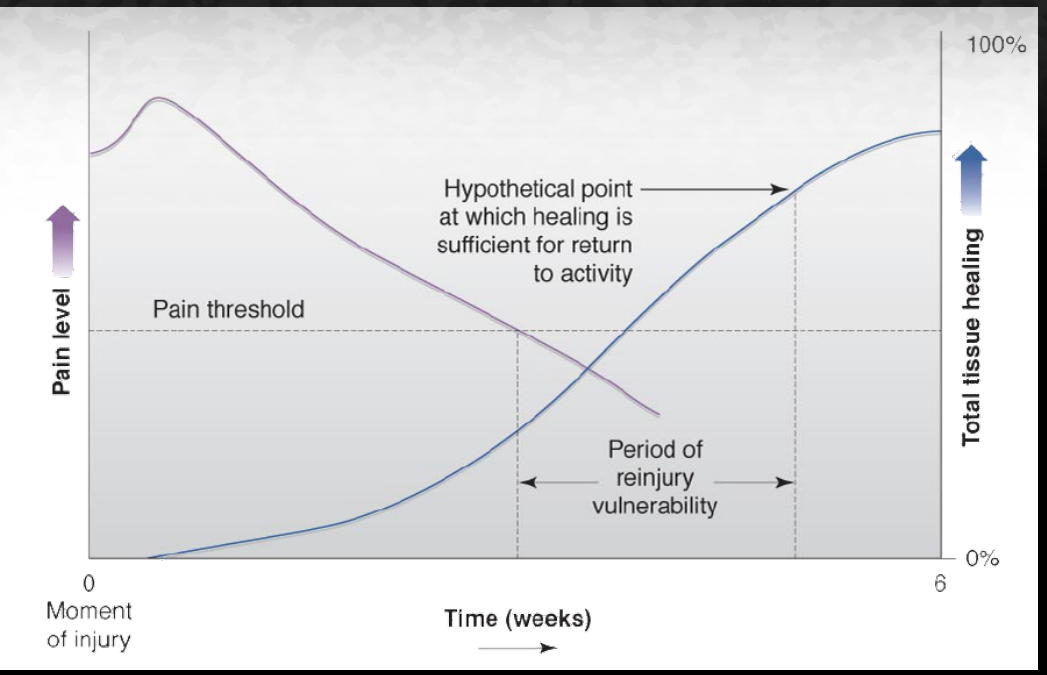
Macrotrauma: Overwhelming force (more than the tissue can tolerate). It's probably bone, ligament, and/or muscle-tendon.

WHICH IS WHICH?

Directionality and the magnitude of the force determine which tissue it is that gets injured and how much tissue the injury blows through.

Macrotrauma: Overwhelming force (more than the tissue can tolerate). It's probably bone, ligament, and/or muscle-tendon.

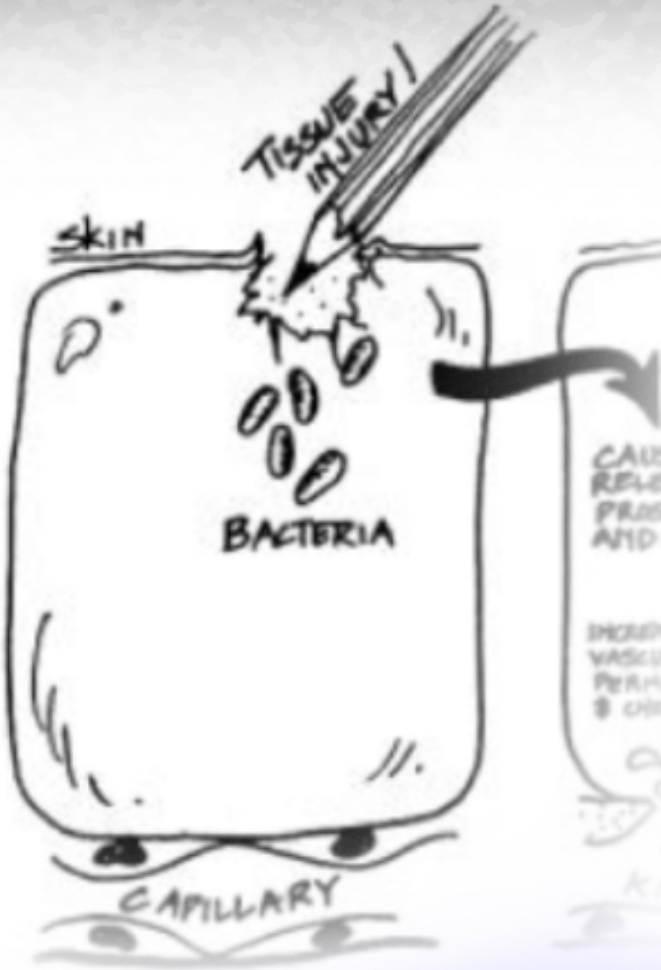
Body position during the trauma also matters. Depending on position, some tissues will be in states of flexion and others will be relaxed. The loose tissues are probably fine; the taut tissues probably popped.



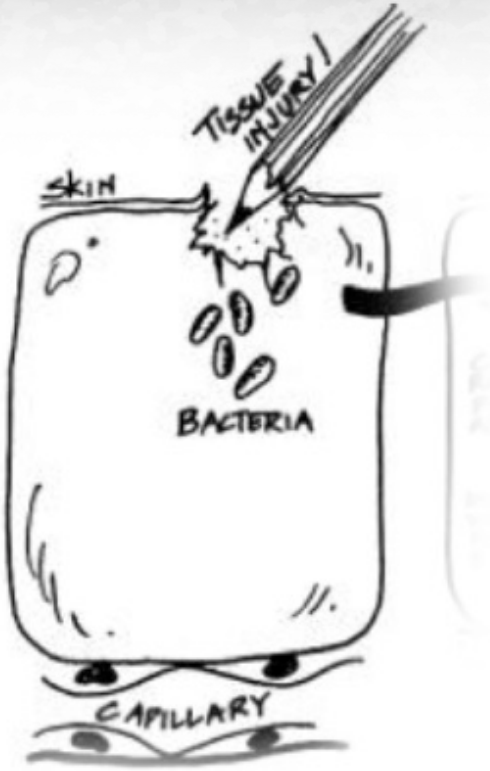
Classifying Injuries

- **Acute**
- **Subacute**
- **Chronic**
- **Acute *on* chronic**

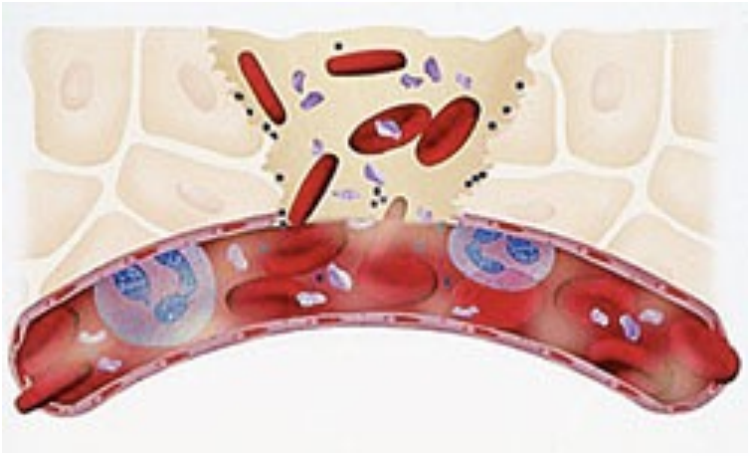
STEP ONE: You get injured



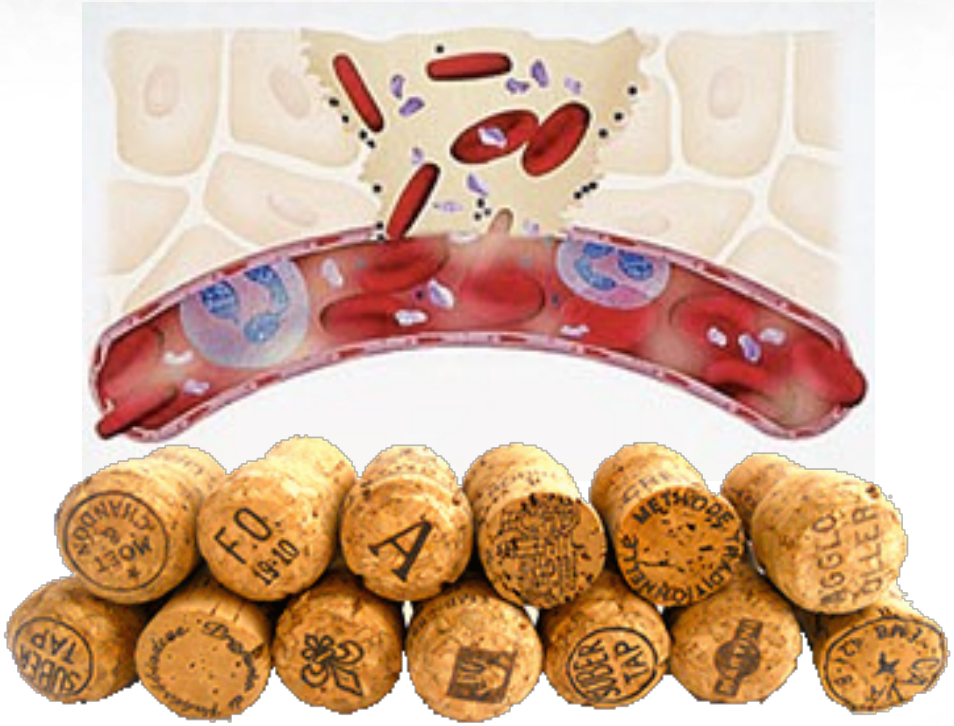
STEP ONE: You get injured



Blood vessels and lymphatic vessels are disrupted.

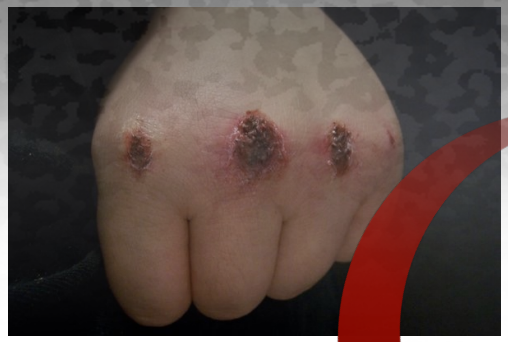


STEP TWO: Plug up the wound

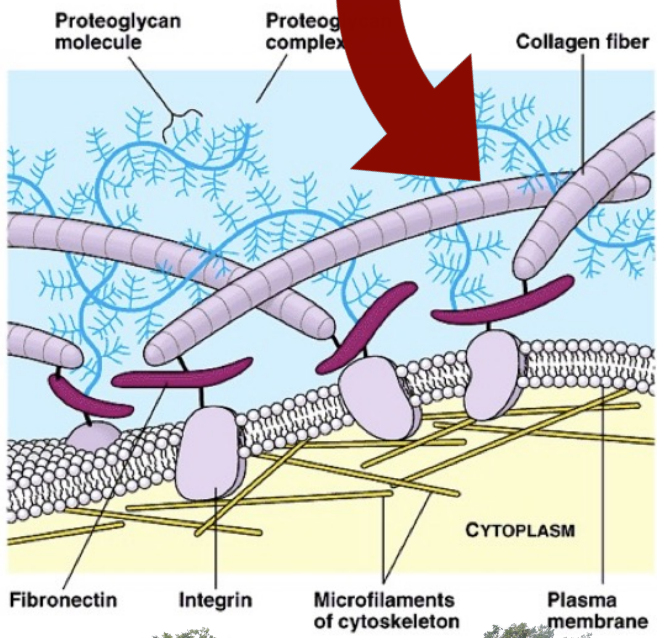


REVIEW: Cellular Stages of Healing

On the surface:



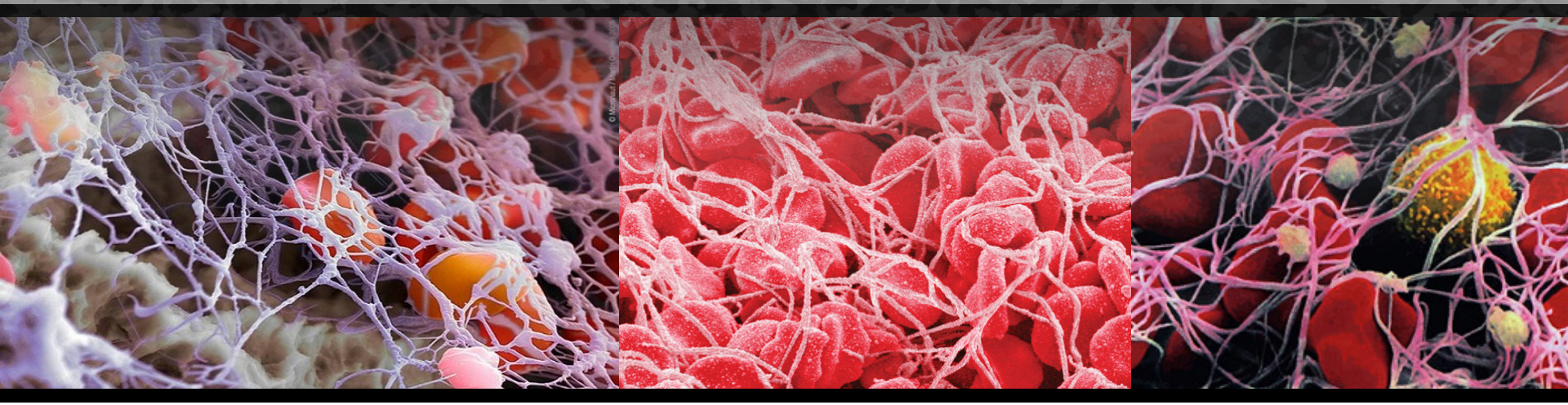
Within the tissue



A weak fibrin-fibronectin mesh.

Fibrin is a fibrous protein involved in blood clots.

Fibronectin is a glycoprotein that binds to stuff.



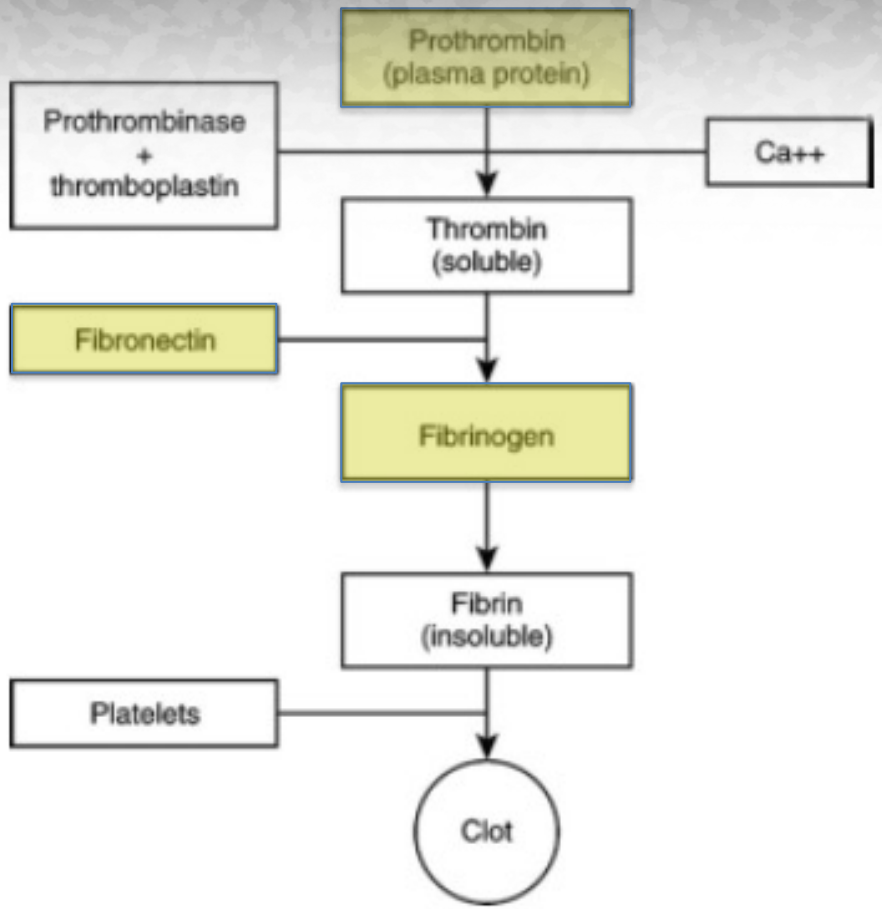
A weak fibrin-fibronectin mesh.

Fibrin is a fibrous protein involved in blood clots.

Fibronectin is a glycoprotein that binds to stuff.

In the blood and lymph:

- Fibrinogen
- Fibronectin
- Prothrombin



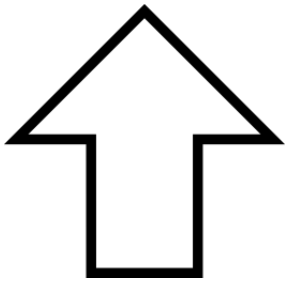
Whenever there's bleeding, platelets (a.k.a. "thrombocytes") show up.

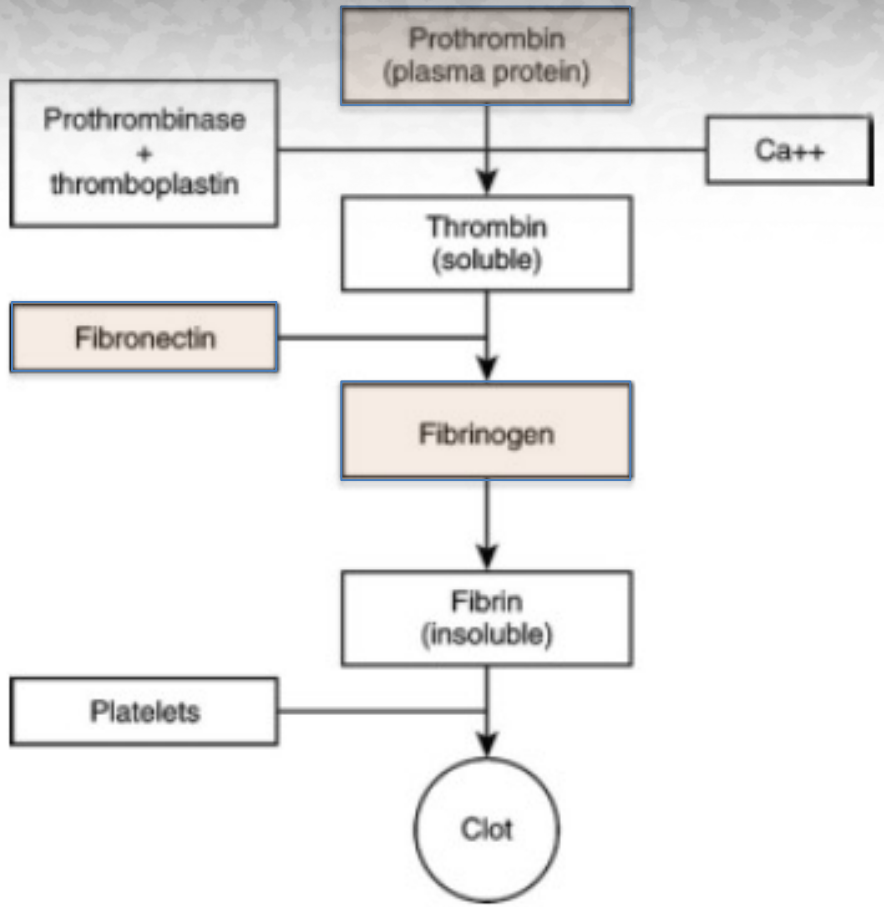
Platelets produce *more* thrombin.

Platelets and Thrombin Generation

Dougald M. Monroe, Maureane Hoffman, Harold R. Roberts

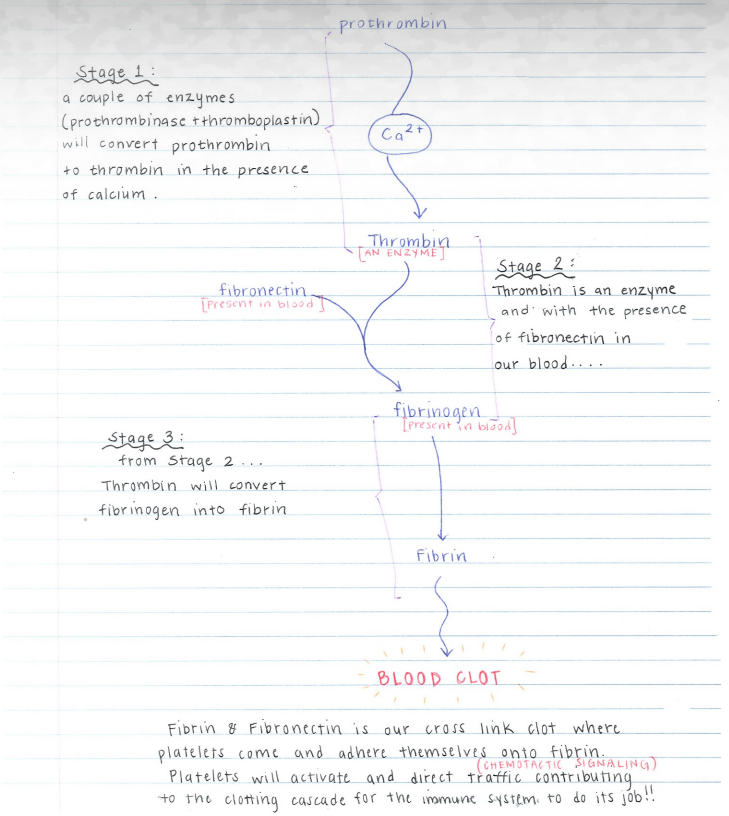
Arterioscler Thromb Vasc Biol. **September 2002;22:1381-1389.**

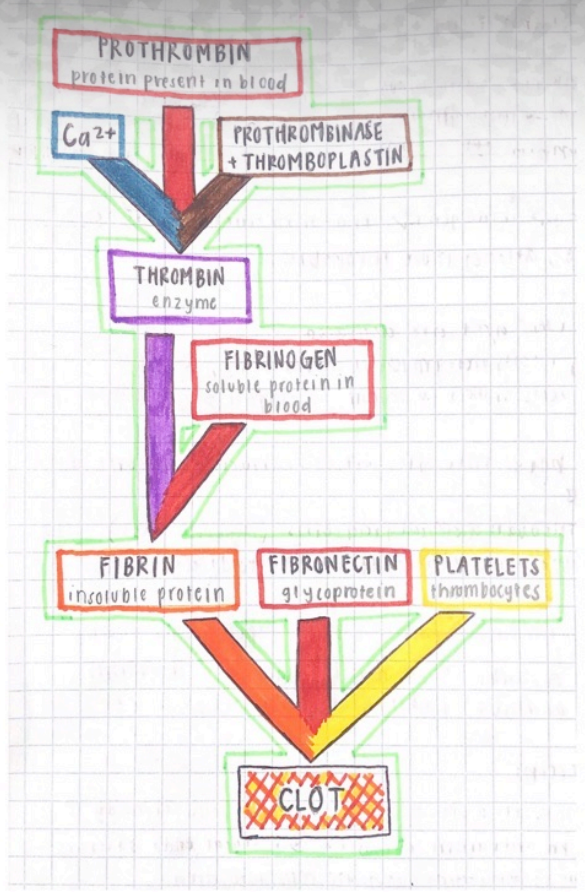




So now you have more thrombin converting fibrinogen into fibrin. No shortage of fibrin at the site of injury.

REVIEW: Cellular Stages of Healing



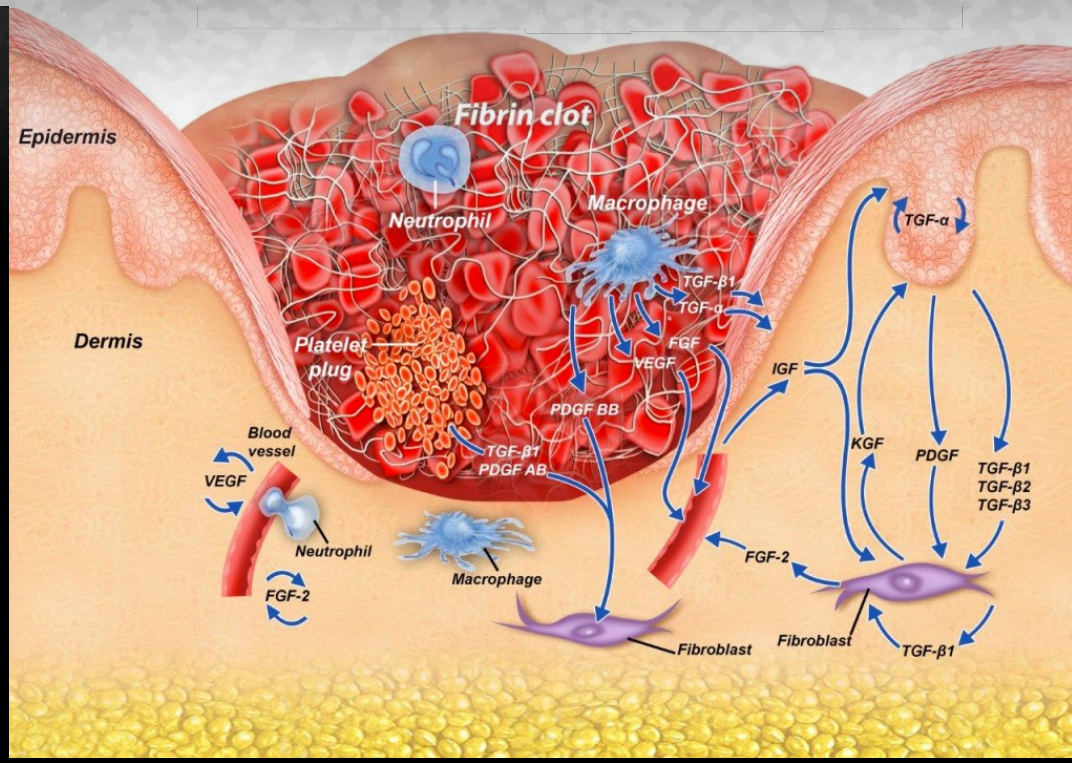


REVIEW: Cellular Stages of Healing

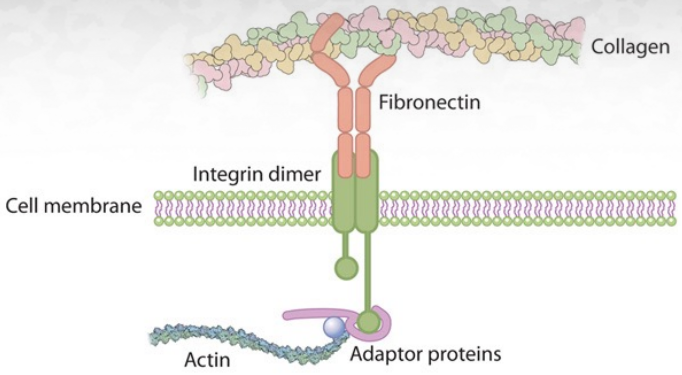
Insoluble fibronectin is already hanging out in the extracellular matrix.

Soluble fibronectin is a major component of blood plasma.

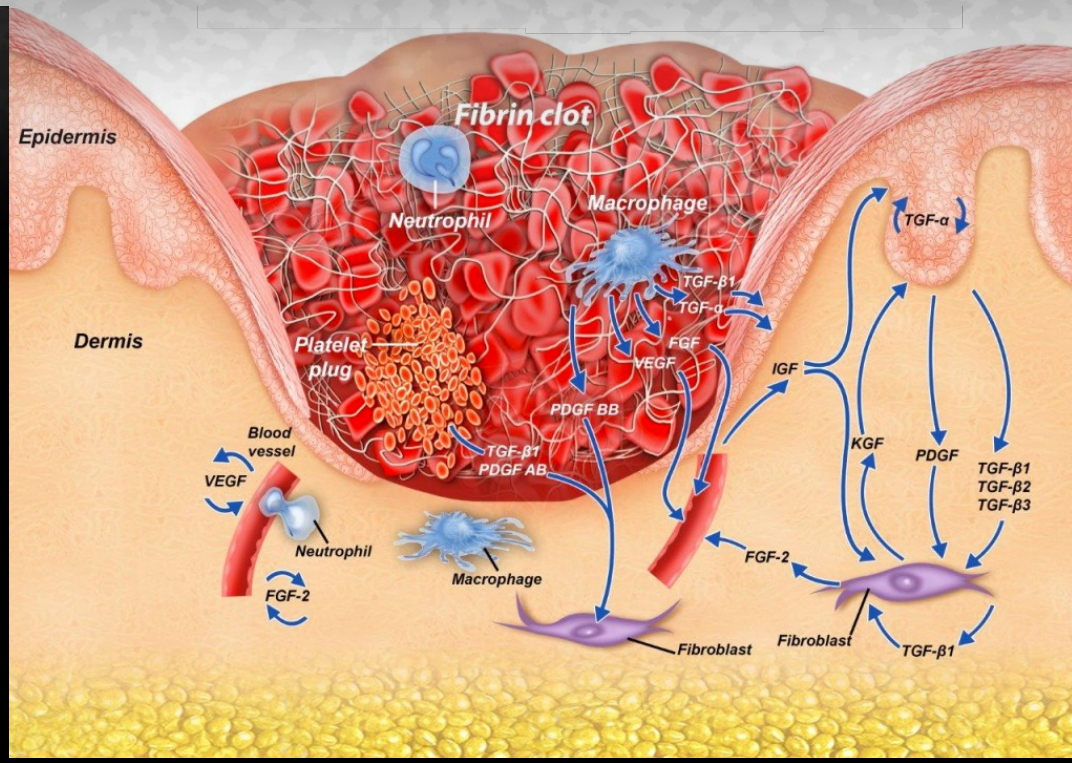
There's a hemorrhage into the tissue. The extravasated fibronectin cross-links to fibrin, collagen, and other ECM stuff.



REVIEW: Cellular Stages of Healing



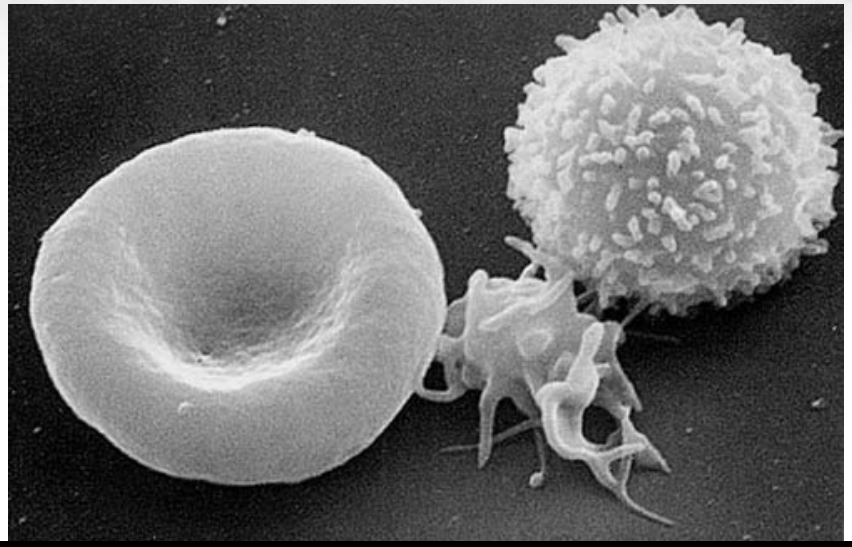
This cross-linking provides a provisional mechanical stabilization of the wound.



What is von Willebrand factor (vWF)?

vWF: Blood glycoprotein (not an enzyme) that binds to other proteins. Platelets are among the proteins it binds to; that's how they adhere to a wound site. If you have a vWF deficiency or dysfunction (i.e., von Willebrand disease), you'll probably have a pretty bleedy constitution. Internally: gastrointestinal bleeding. Externally, a bunch of nose bleeds.

The platelets adhere to exposed ECM via von Willebrand factor (vWF) and are activated.

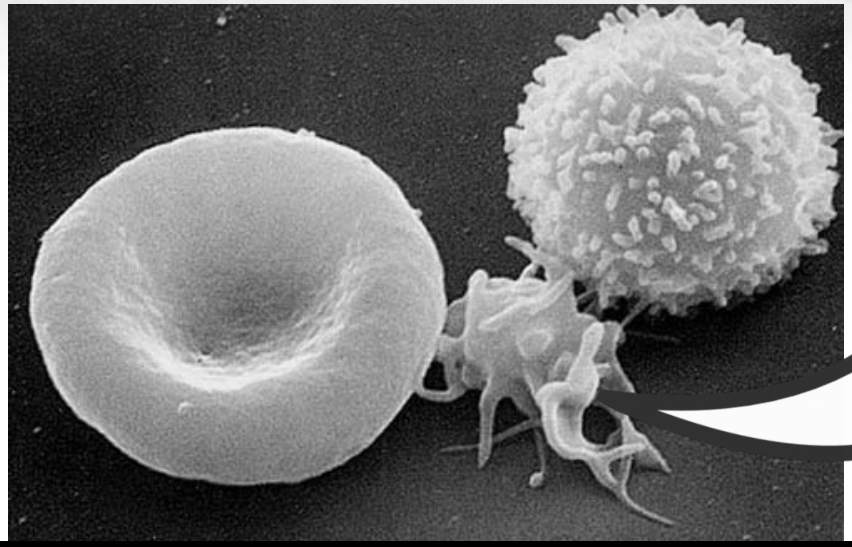


When activated, platelets change shape and begin degranulating.

They come from granulocytes and thus have preformed granules, so they secrete stuff like growth factors, cytokines, bradykinins, serotonin, histamine, TNF-alpha, etc.

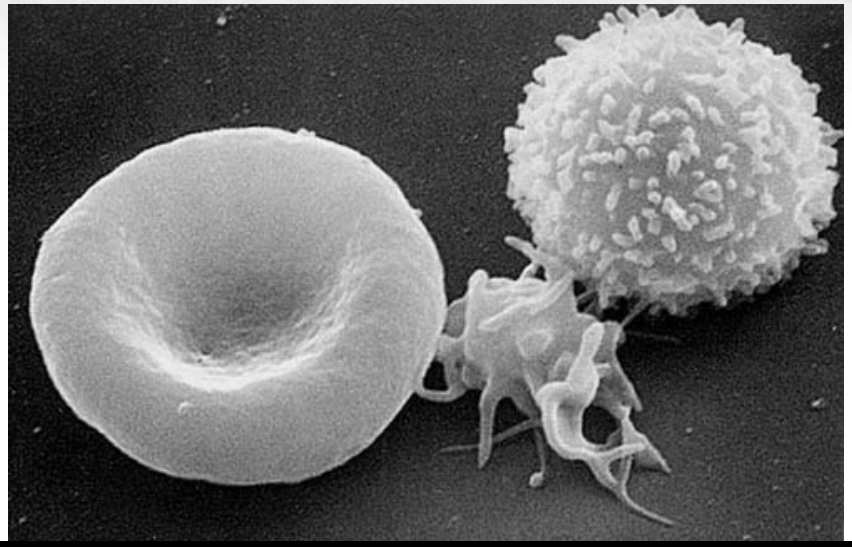
The matrix itself becomes a hub for chemotactic signaling.

The platelets adhere to exposed ECM via von Willebrand factor (vWF) and are activated.



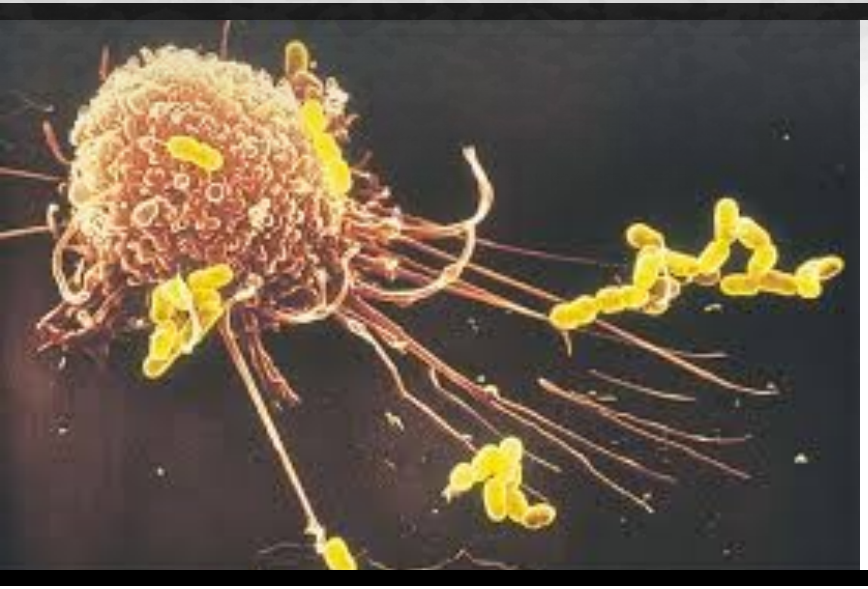
The matrix itself becomes a hub for chemotactic signaling.

What are these?



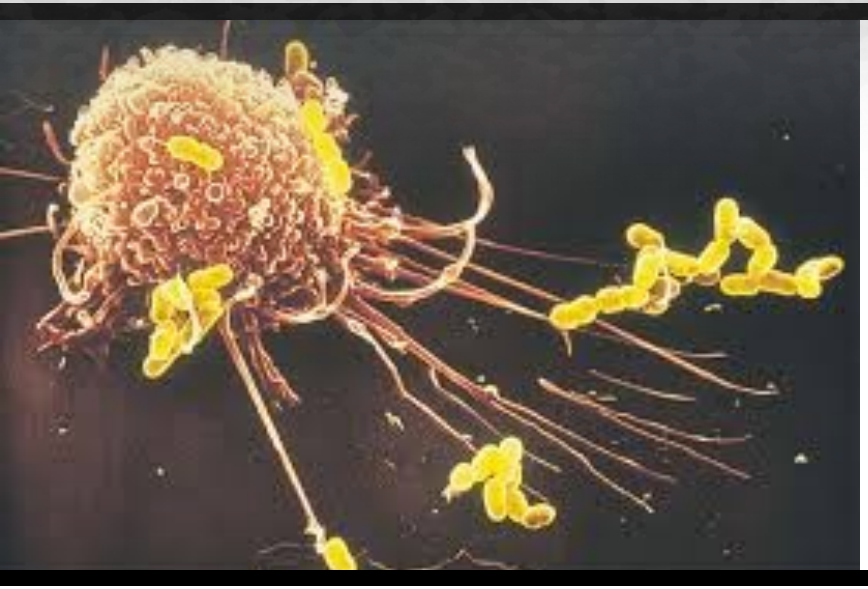
Pretend this man is a platelet

What is Mr. Platelet doing?



Some macrophages will also (already) be in the area ("resident macrophages").

What is their first duty after you sustain an injury?



Some macrophages will also (already) be in the area ("resident macrophages").

They put out chemotactic signals too, helping to recruit a neutrophil army (and more macrophages).

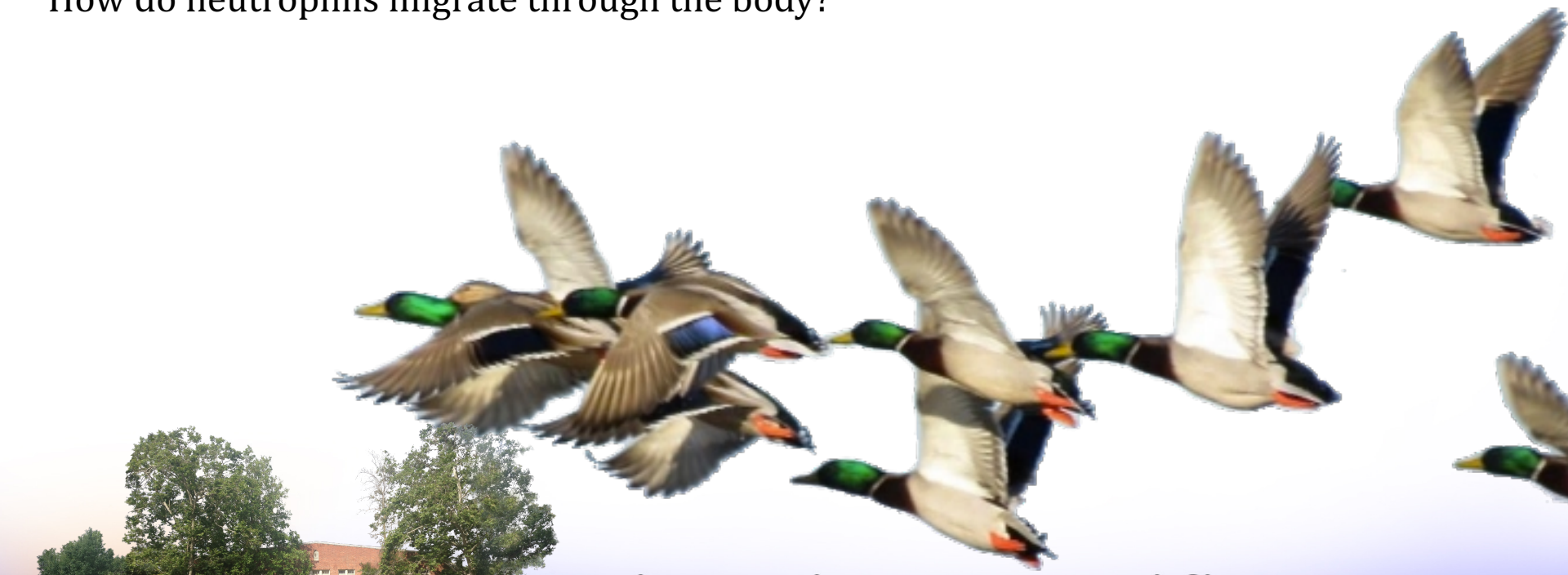
REVIEW: Cellular Stages of Healing

How do ions move in the body?

How do gases move in the body?

How do the migrating cells do their migrating?

How do neutrophils migrate through the body?



Chemotaxis helps direct a bunch of cells to the site of the injury.

How do ions move in the body?

Concentration (or "electrochemical") gradients.

How do gases move in the body?

Pressure gradients.

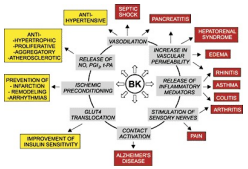
How do incoming immune cells migrate through the body?

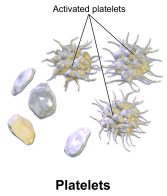
Chemical gradients.

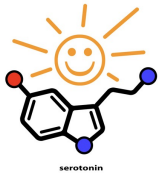
The migration is chemotaxis. To "chemotax" is to direct them based on chemicals – attractants and repellents.

REVIEW: Cellular Stages of Healing

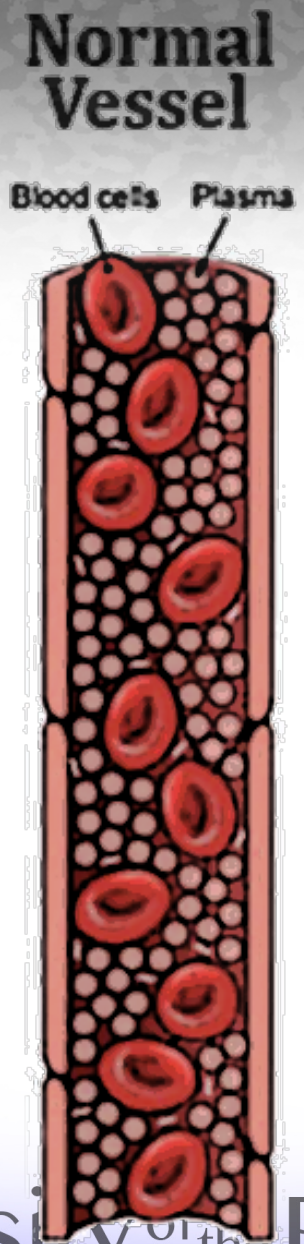
What causes this?
And why is it helpful?

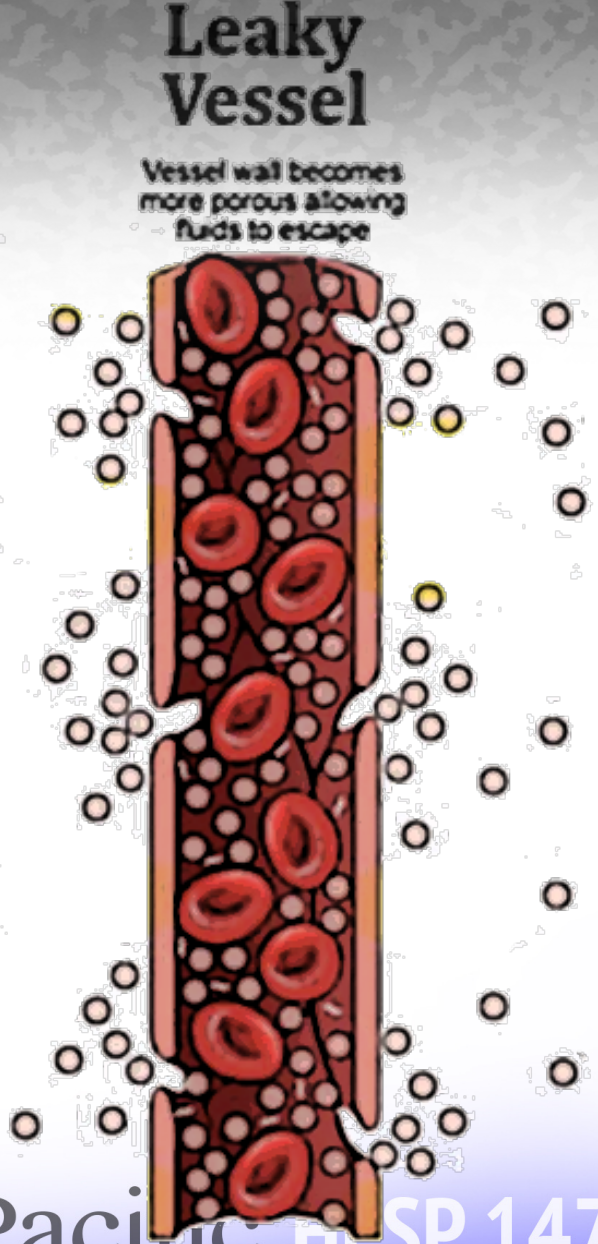




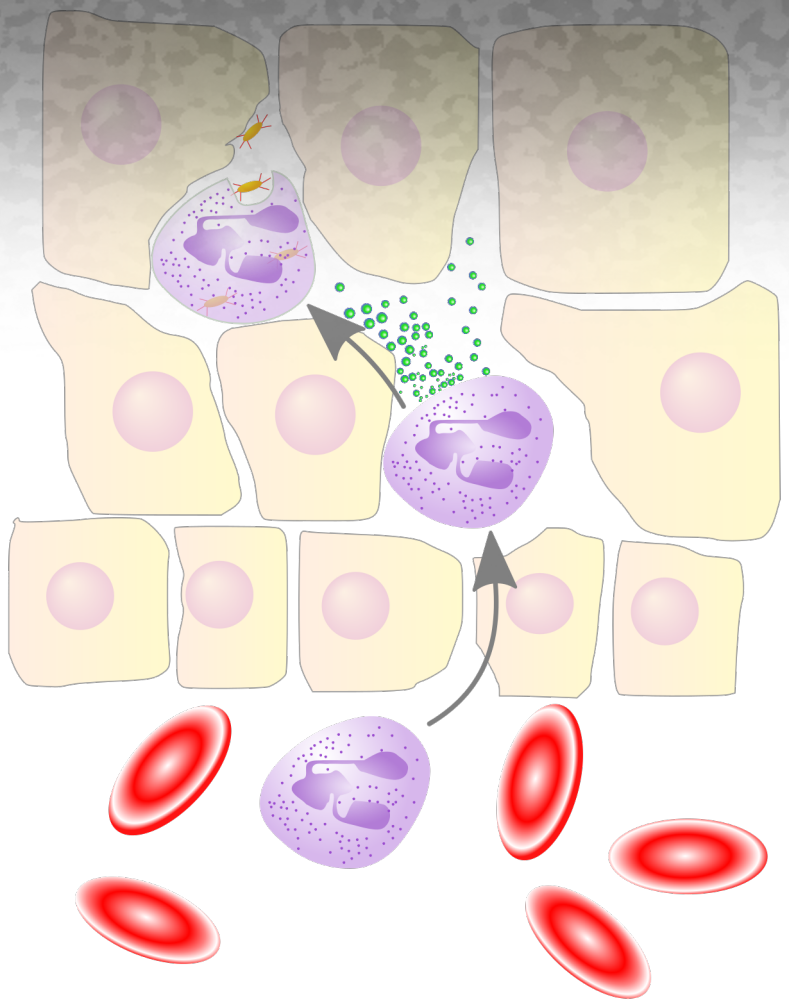


Name 6 things that can make vessels leaky.





REVIEW: Cellular Stages of Healing



Stuff that can induce vascular permeability: vascular endothelial growth factor (produced by numerous cell types, including platelets and macrophages; generally upregulated in response to hypoxia), prostaglandins (E2, but D2 can reduce permeability; from arachidonic acid), complement proteins (mostly synthesized by hepatocytes, but also by endothelial, epithelial, and immune cells), histamine (mostly mast cells and basophils), bradykinin (kallikrein is an enzyme in plasma and tissue that converts kininogens to bradykinin), thrombin (made from prothrombin, which is produced in the liver), platelet activating factor (once endothelial cells are activated by histamine or thrombin, they express it; but it's also synthesized by platelets, neutrophils, and macrophages), serotonin (released by platelets, neurons, and enterochromaffin cells in the lumen of the digestive tract), nitric oxide (made from the enzyme endothelial nitric oxide synthase, stimulated in part by platelets and cytokines), reactive oxygen species (superoxide, hydrogen peroxide), tumor necrosis factor (largely from macrophages, but neutrophils and mast cells are among the contributors), and vasodilation itself...

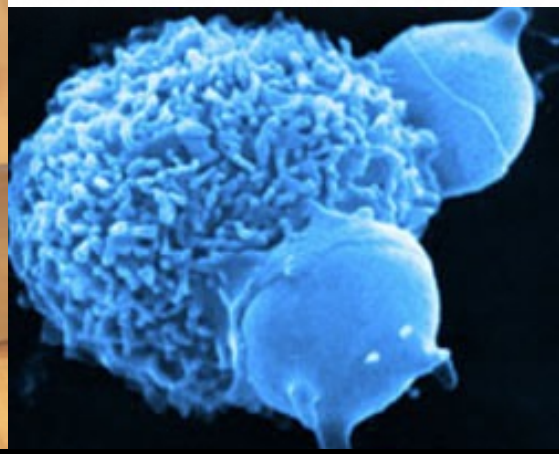
Leakiness partly caused by:

Complement proteins.

Your complement system is part of the innate immune system, which means that it doesn't change over the course of your lifetime.

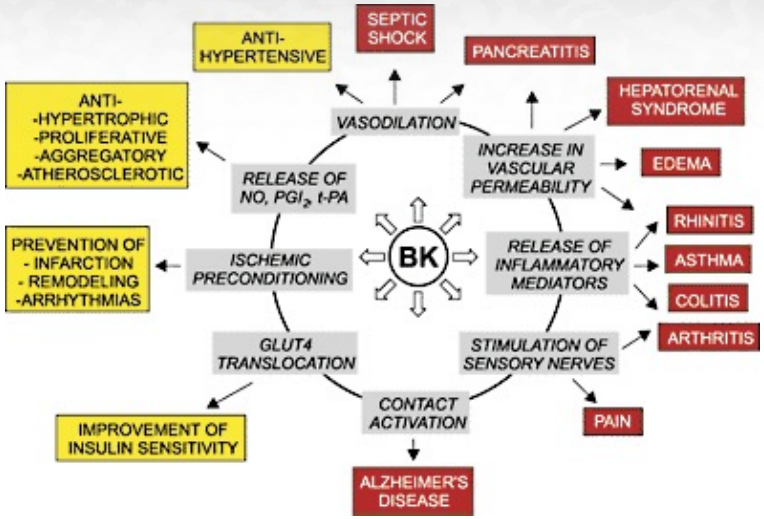
Complement proteins complement your phagocytic cells, which helps them to clear pathogens.

These make it easier for your phagocytic cells to dine on stuff.



> 30 different proteins and protein fragments, which circulate around until activated; then they attract macrophages and neutrophils, rupture membranes of foreign cells, and make all the stuff phagocytes will be eating more easily eaten.

Leakiness partly caused by:
The kinin system

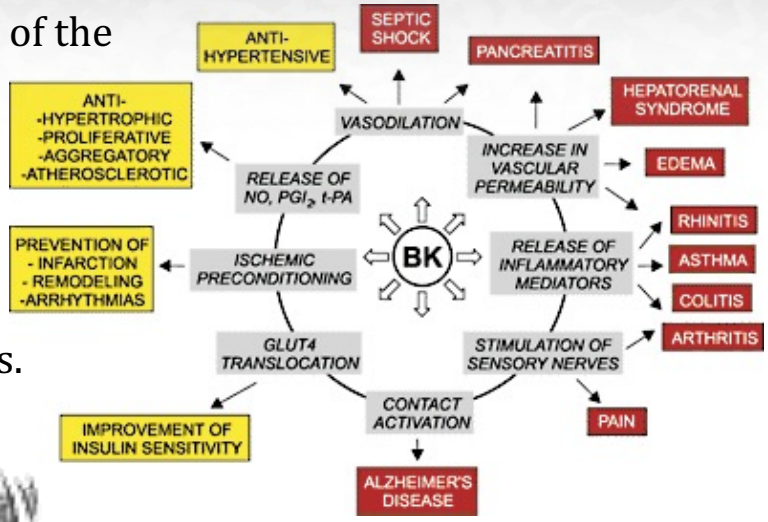


Mostly just bradykinin and kallidin (which is just bradykinin with a lysine connected to it.) Kinin system is initiated by Hageman factor activation (which also initiates the fibrinolytic clotting system and complement proteins). Kinins are mediator molecules that induce vasodilation and vascular permeability *and* stimulate local cells to generate cytokines, nitric oxide, and tachykinins (e.g., substance P).

Leakiness partly caused by:
The kinin system

Kinins are amplifiers of the **inflammatory response.**

They stimulate the local tissues and inflammatory cells to generate additional mediators.



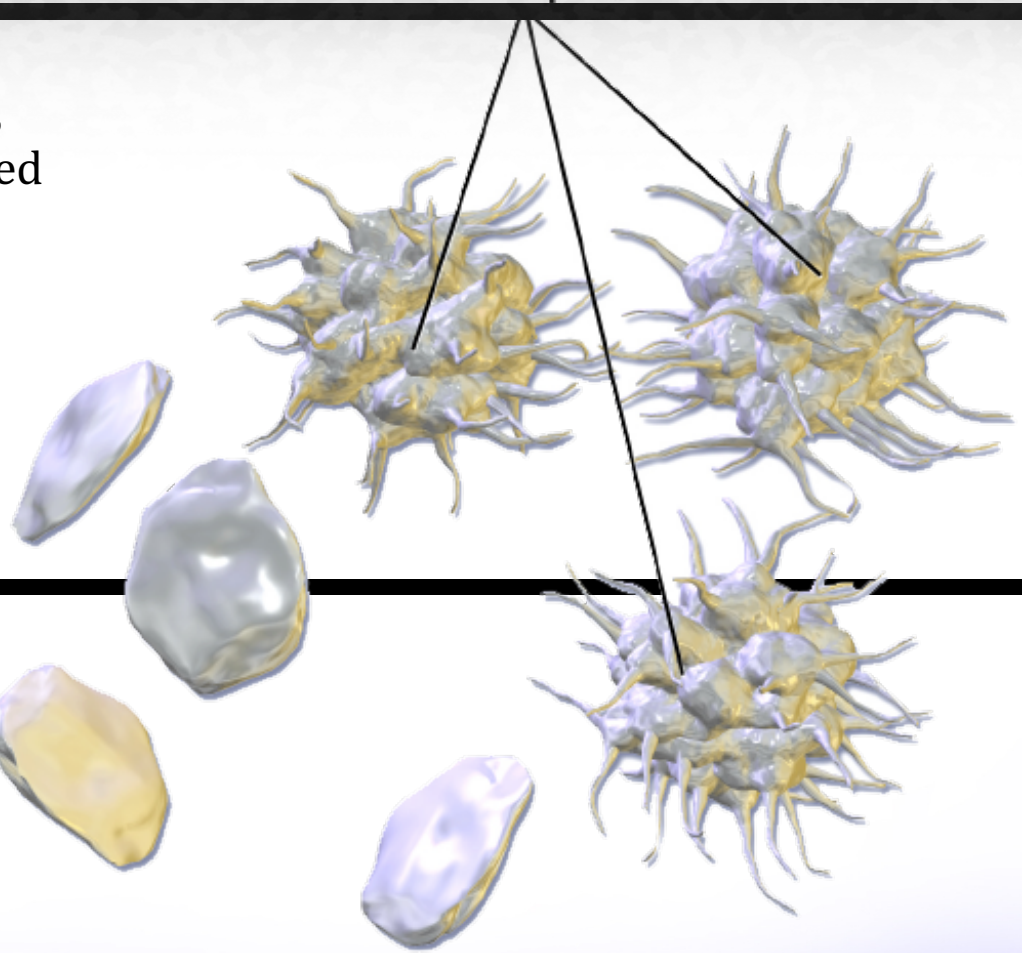
Mostly just brady[illegible]d kalli[illegible]ich is [illegible]t bradykinin with a lysine connected to it.)
Kinin system is i[illegible]t[illegible]d by Hageman factor a[illegible]on (which also initiates the fibrinolytic clotting system a[illegible] complement p[illegible]). [illegible]re mediator molecules that induce vasodilation a[illegible]cular permeability *and* [illegible]te [illegible]al cells to generate cytokines, nitric oxide, a[illegible]kinins (e.g., substance P).

Leakiness partly caused by:

Platelet-activating factor

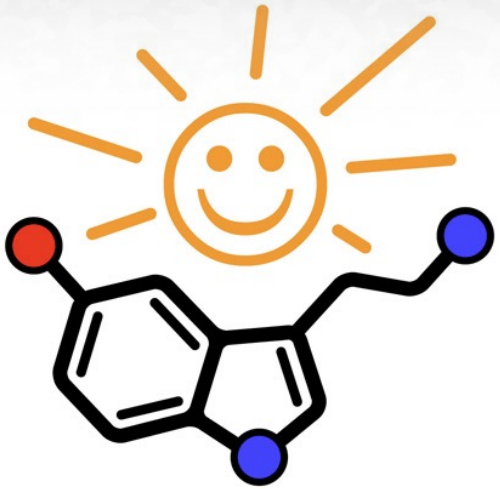
When there's a stimulus (e.g., injury), platelet-activating factor gets produced by lots of stuff (e.g., endothelial cells, platelets, neutrophils, macrophages).

It causes the aggregation of platelets and their degranulation as well as leukocyte chemotaxis and increases to vascular permeability.



Leakiness partly caused by:
Serotonin

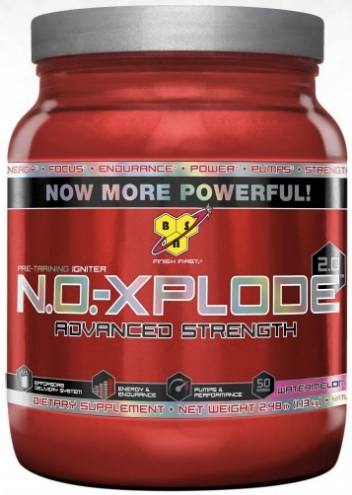
Serotonin is *one* of the things that gets released by the platelets.



Leakiness partly caused by:

Nitric oxide

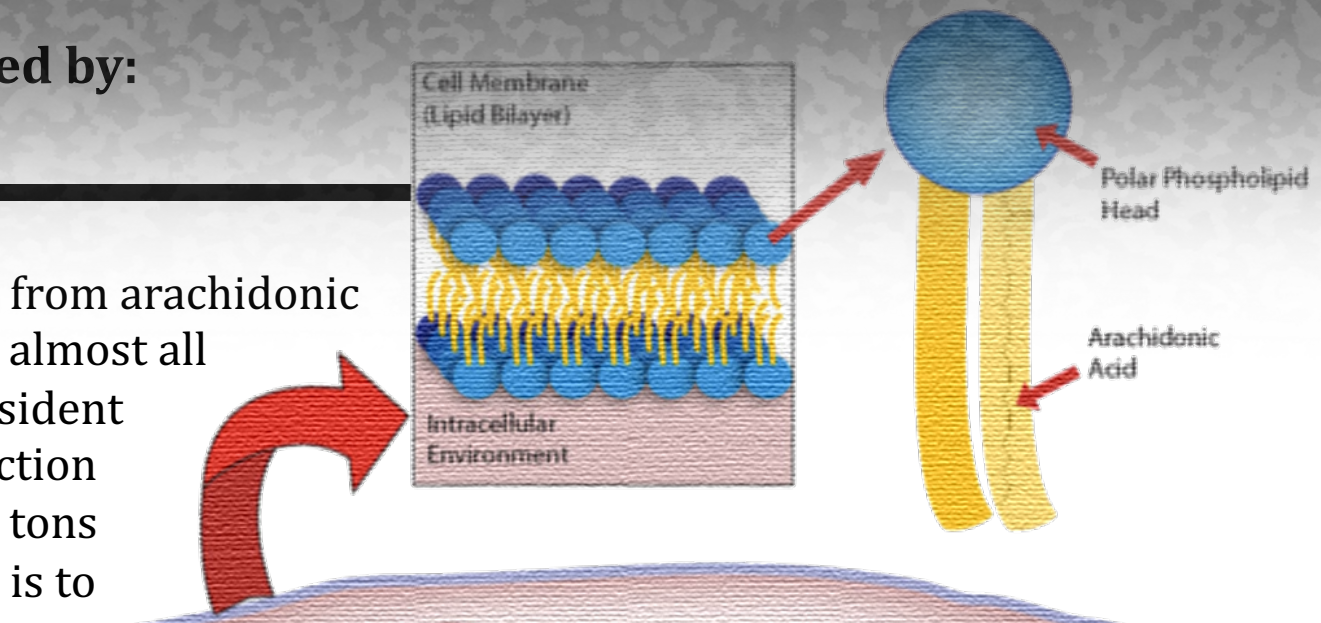
Powerful vasodilator generated by phagocytes (i.e., monocytes, macrophages, and neutrophils). These cells have nitric oxide synthase which can be activated by interferon-gamma or TNF-alpha.

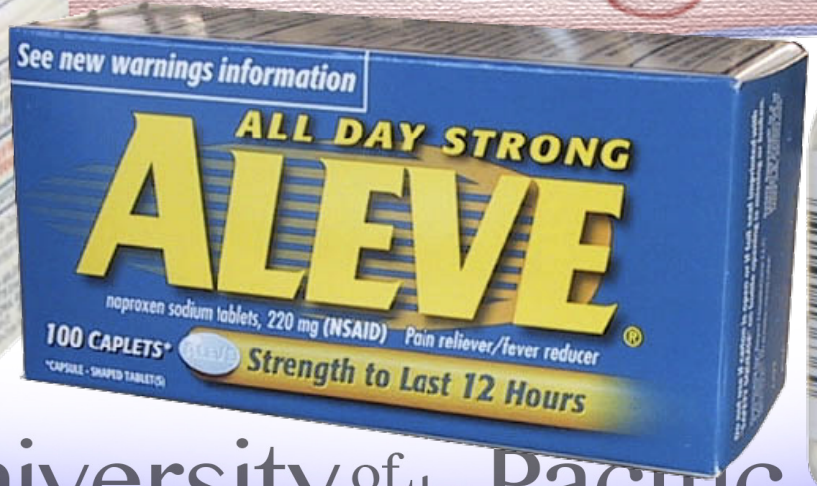


Leakiness partly caused by:

Prostaglandins

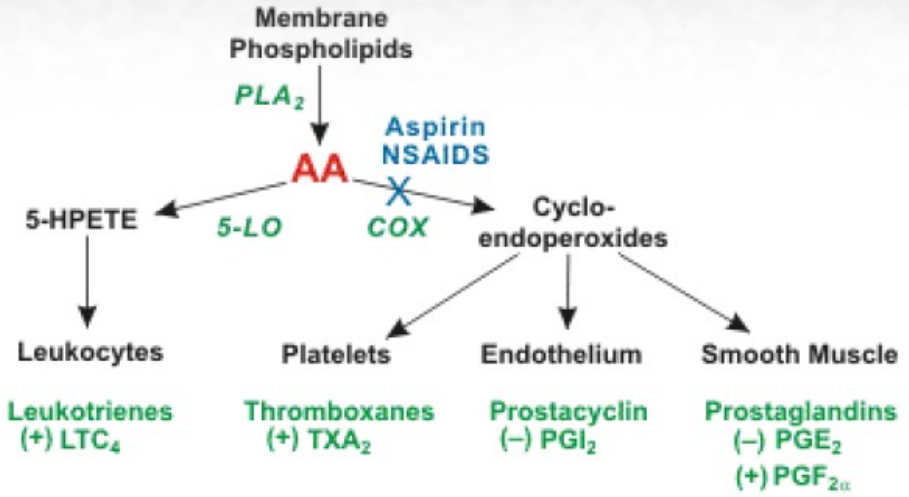
Lipid compounds derived from arachidonic acid that are produced by almost all nucleated cells and are resident in most tissues. They function as messengers that signal tons of functions; one function is to sensitize nerves to pain.





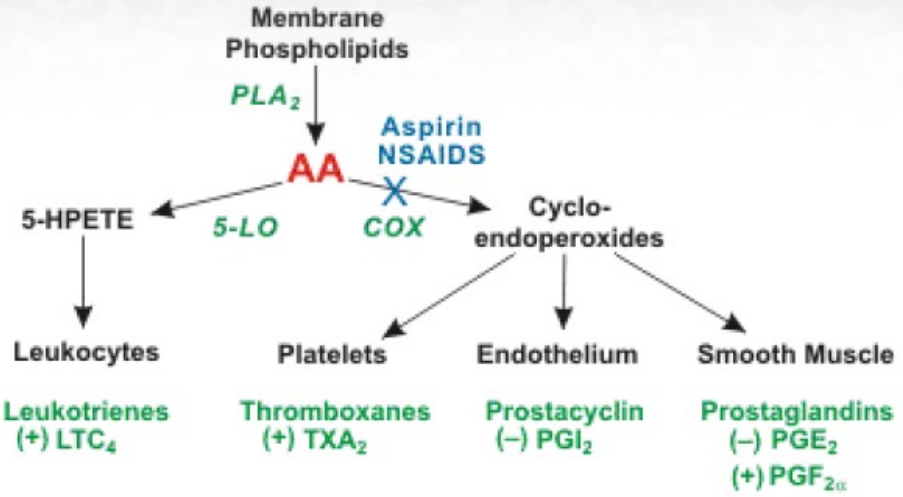
Leakiness partly caused by:
Prostaglandins

AA can be liberated by mechanical injury and by the stretching of cell membranes. Liberation by PLA_2 is where most of your prostaglandins will come from, but you can also get some from diglycerides by the enzyme diacylglycerol lipase (nobody cares about this though).



Leakiness partly caused by:
Prostaglandins

If COX is blocked, you'll increase activity of lipoxygenase and wind up with more leukotrienes. These serve important chemotactic effects and are associated with the inflammation in asthma.



Upsala Journal of Medical Sciences. 2015; 120: 135–143

Vascular permeability—the essentials

LENA CLAESSON-WELSH

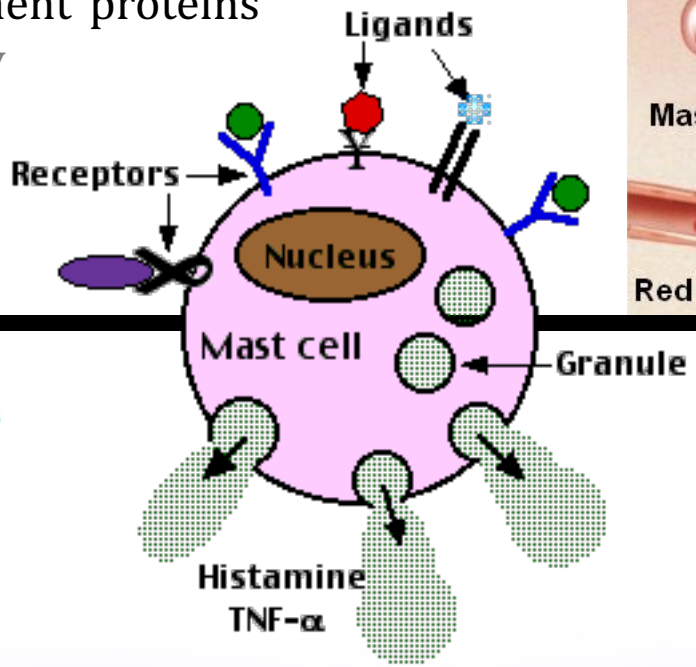
Vascular permeability to solute, molecules, and cells

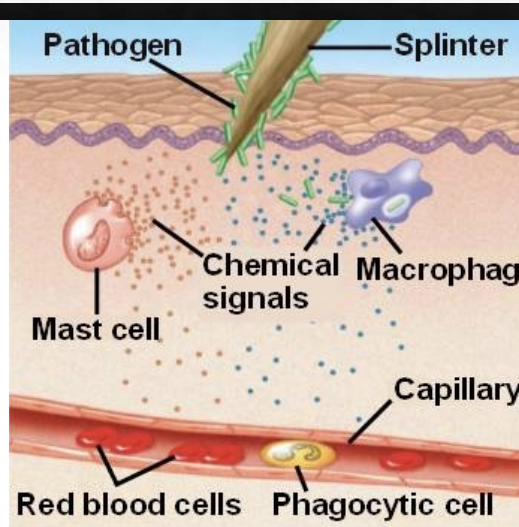
Plasma contains three main molecular constituents: albumin, globulins, and fibrinogen (27). Extravasation of macromolecules serves diverse purposes, for example to maintain the balanced blood and interstitial pressures, to act in immune surveillance, and to carry other molecules, such as hormones and lipids, across the vessel wall. Extravasated fibrinogen, processed to fibrin, may form a provisional matrix on which new blood vessels extend (28).

Extravasation of inflammatory and immune cells serves specific purposes in different pathologies. These cells are a prerequisite for healing of an acute disease process but may also propagate a chronic disease and interfere with recovery.

These cells (and other stuff) also interact with each other.

Example: Mast cells can degranulate (releasing granules of histamine) in response to physical injury *or* substance P *or* complement proteins *or* macrophage factors (released by macrophages). Histamine causes further vasodilation and even more leakiness.





PLoS PATHOGENS April 2012 | Volume 8 | Issue 4

The Role of Mast Cells in the Defence against Pathogens

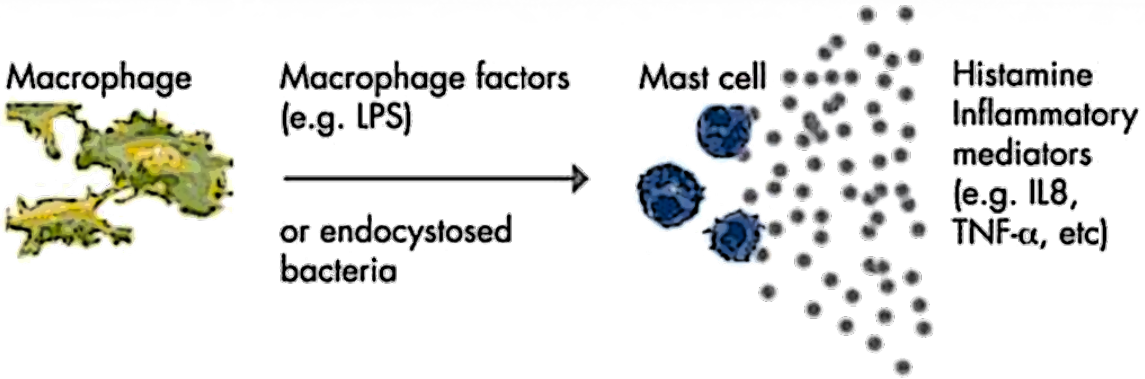
Mirjam Urb[1], Donald C. Sheppard[1,2]*

These cells (and other stuff) also interact with each other.

Endocr Rev. 33(1): 71-108

Emerging Role of Mast Cells and Macrophages in Cardiovascular and Metabolic Diseases

Jia-Ming Xu, Guo-Ping Shi



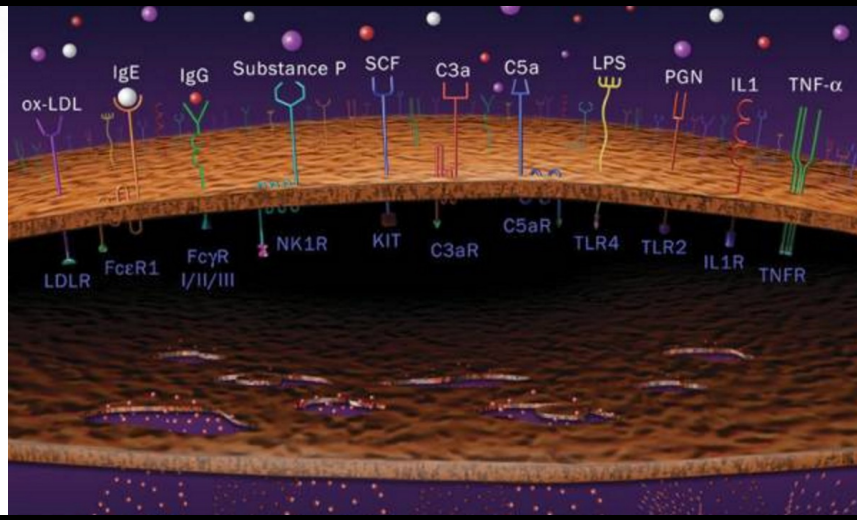
These cells (and other stuff) also interact with each other.

Endocr Rev. 33(1): 71-108

Emerging Role of Mast Cells and Macrophages in Cardiovascular and Metabolic Diseases

Jia-Ming Xu, Guo-Ping Shi

Plenty of stuff (extracellular ligands binding to their receptors on the cell surface) can cause the mast cells to degranulate.



Just know damage, substance P, complement proteins, and marophages.

You're stung by a bee…

You take some Benadryl, the brand name of diphenhydramine (an antihistamine).

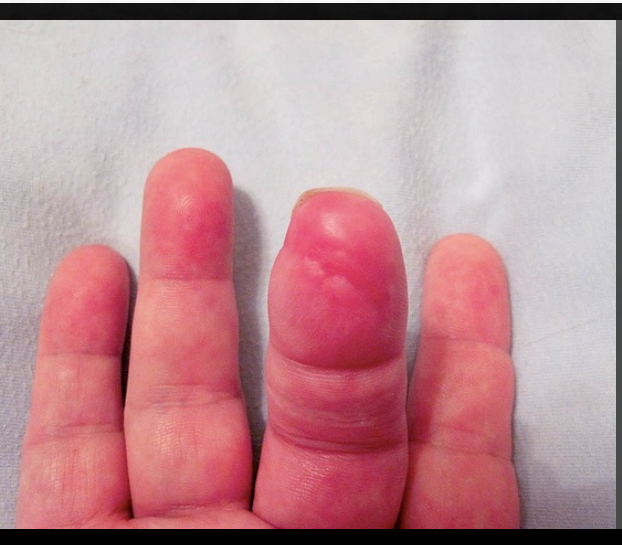
Why?

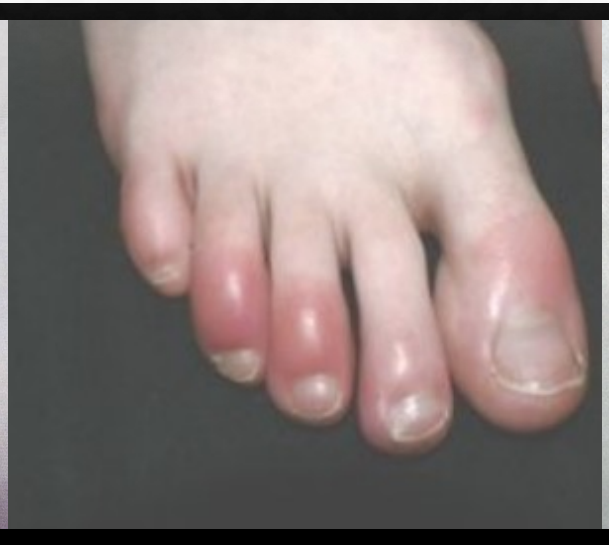
You're stung by a bee…

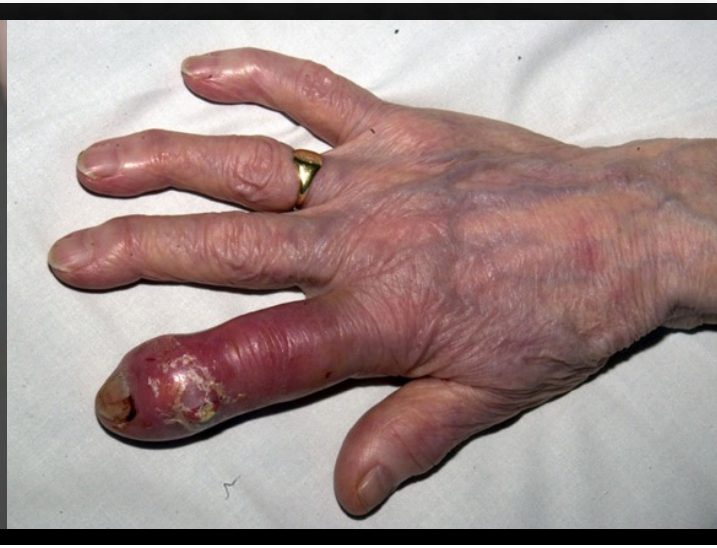
Overview of diphenhydramine targets and effects

Biological target	Mode of action	Effect
H_1 receptor (Peripheral)	Inverse agonist	Allergy reduction
H_1 receptor (Central)	Inverse agonist	Sedation

Why is this stuff swollen?



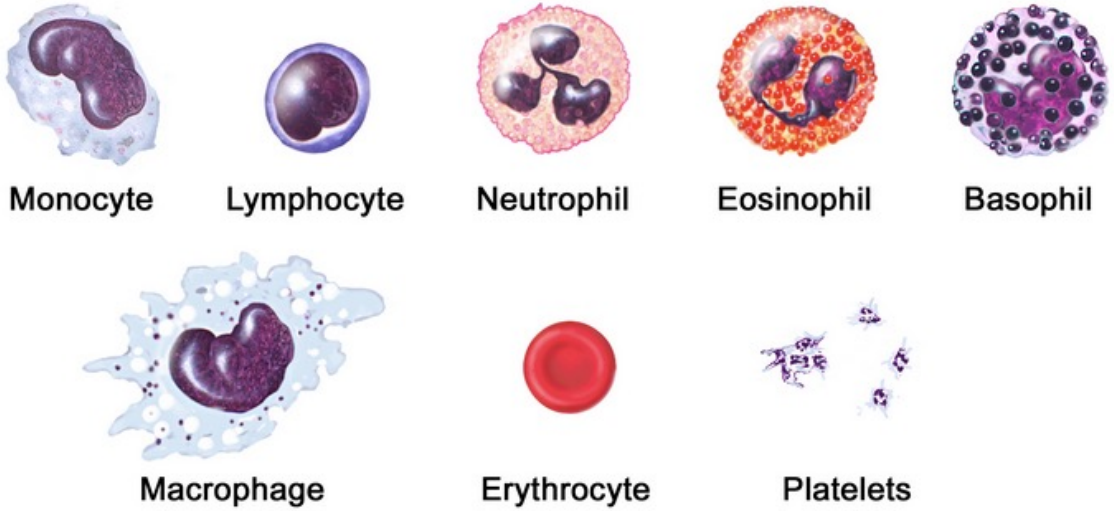




And what explains the redness, pain, and heat?

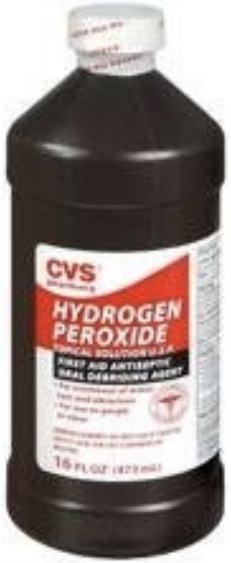
Now it's all leaky and dilated; lots of cells are going to the site of injury.

What's the first white blood cell to arrive in the area?

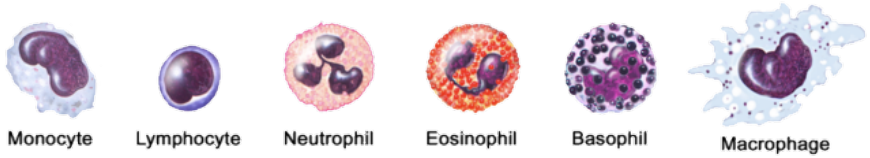


How do neutrophils do their janitoring?

Neutrophils: The first immune cells to show up. They're bactericidal agents; they go in and squirt superoxide and hydrogen peroxide around, killing, everything.



And they secrete proteases (enzymes that perform proteolysis). They only live for a couple days.



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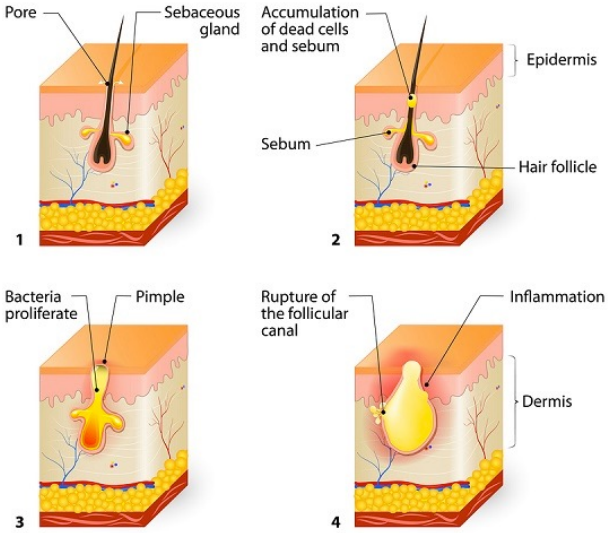


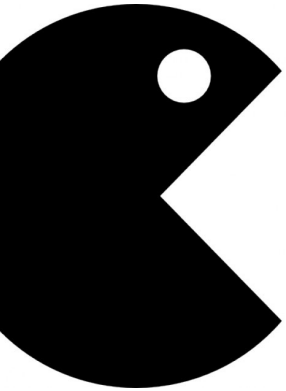
NATURE REVIEWS | IMMUNOLOGY VOLUME 6 | MARCH 2006

Neutrophils and immunity: challenges and opportunities

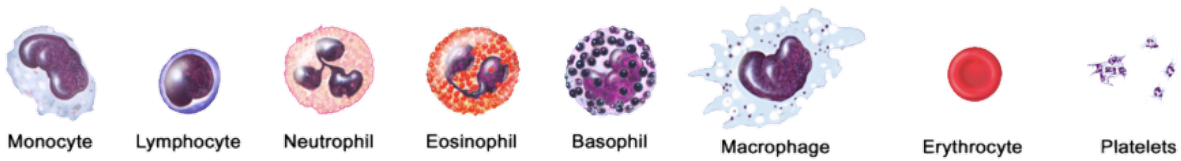
Carl Nathan

It is not realistic to expect neutrophils to find and eat each bacterium in a wound before any bacteria have had time to escape into the lymph or blood. Neutrophils that sense tissue damage plus infection but fail to encounter a bacterium within a short time, fire off their arsenal into the extracellular space[28]. *In vitro*, this period of restraint ranges from ~15 to ~45 minutes, which is thought to be the approximate time it takes for a neutrophil to emigrate from the blood into the extravascular tissues. When restraint is abandoned, what ensues is the liquefaction of tissue — that is, the formation of pus — through neutrophils' release of proteases, their activation of proteases that are expressed in a latent form by cells resident in the tissues, and their oxidative inactivation of anti-proteases (proteins that specifically bind and inactivate proteases)[3,4].

Macrophages: Phage is a digestive term; they arrive after the neutrophils and engulf (and digest) the dead cells (e.g., neutrophils), pathogens, and cellular debris (phagocytosis).



In exercise-induced stress, macrophages can lysis muscle membranes, enter the cell, and degrade the innards of damaged fibers.



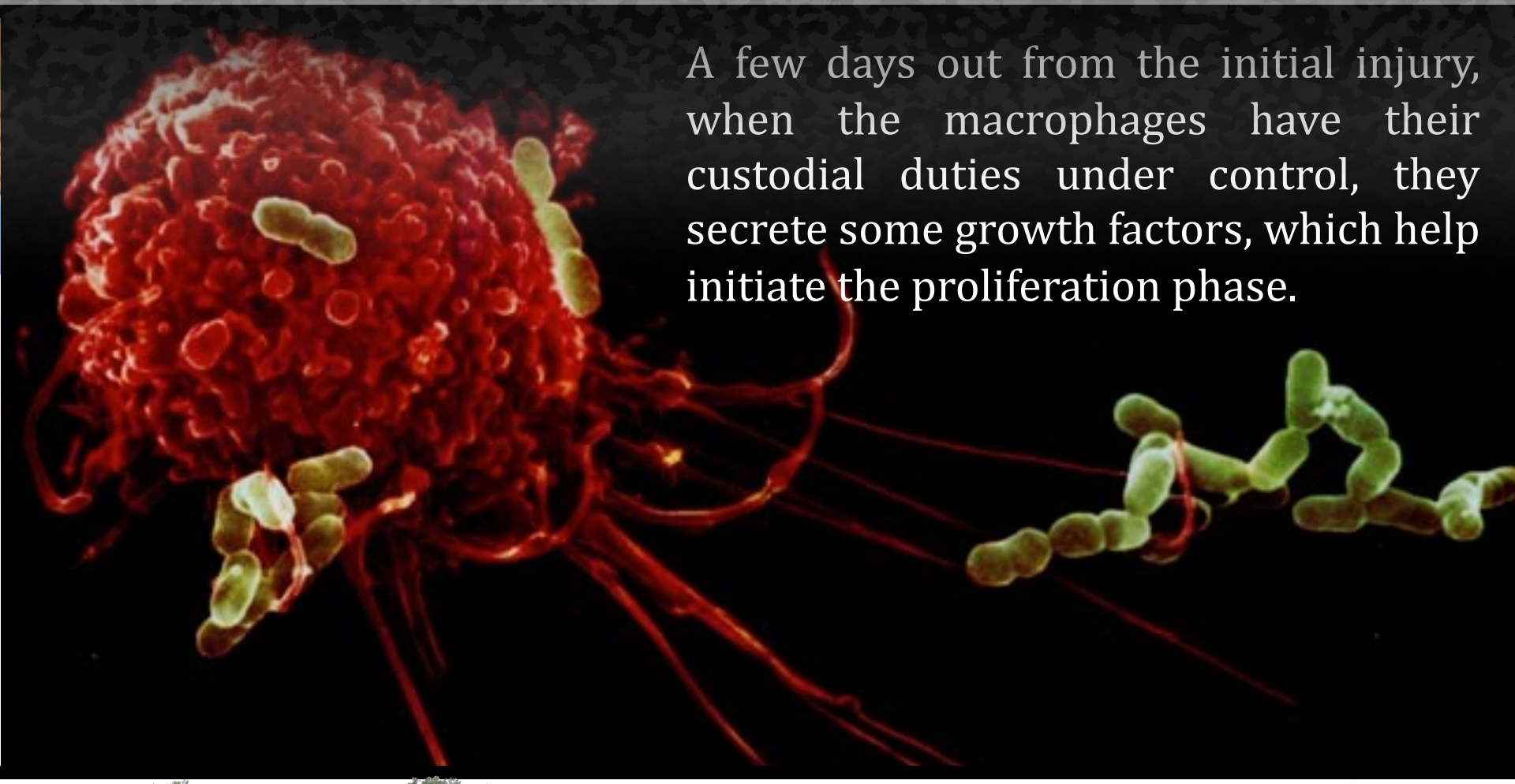
Macrophages: Phage is a digestive term; they arrive after the neutrophils and engulf (and digest) the dead cells (e.g., neutrophils), pathogens, and cellular debris (phagocytosis).

Semin Liver Dis. 2010 August ; 30(3): 245–257.

Macrophages: Master Regulators of Inflammation and Fibrosis

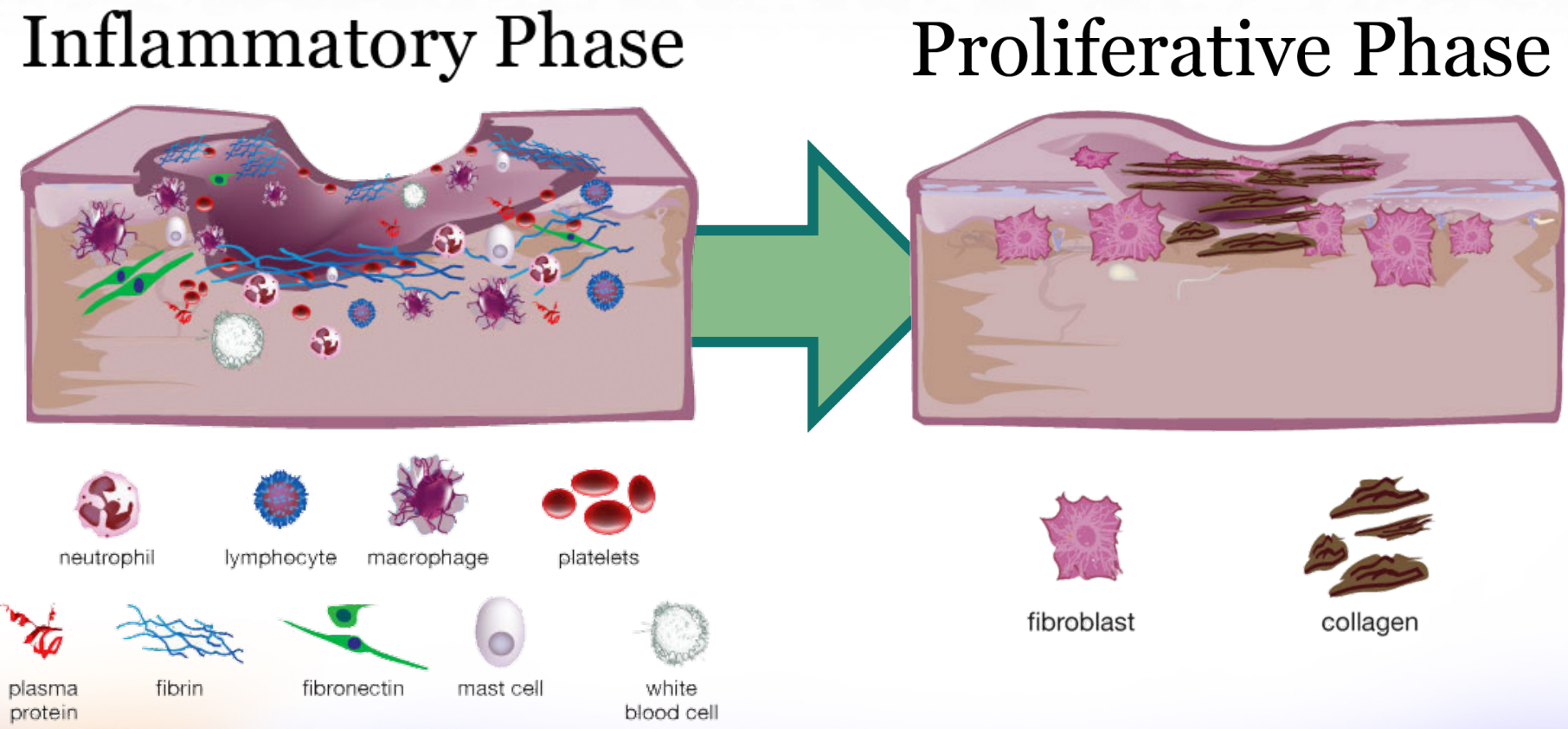
Thomas A. Wynn, Ph.D.[1] and **Luke Barron, Ph.D.**[1]

Macrophages are found in close proximity with collagen-producing myofibroblasts and indisputably play a key role in fibrosis. They produce profibrotic mediators that directly activate fibroblasts, including transforming growth factor-β1 and platelet-derived growth factor, and control extracellular matrix turnover by regulating the balance of various matrix metalloproteinases and tissue inhibitors of matrix metalloproteinases. Macrophages also regulate fibrogenesis by secreting chemokines that recruit fibroblasts and other inflammatory cells. With their potential to act in both a pro- and antifibrotic capacity, as well as their ability to regulate the activation of resident and recruited myofibroblasts, macrophages and the factors they express are integrated into all stages of the fibrotic process. These various, and sometimes opposing, functions may be performed by distinct macrophage subpopulations, the identification of which is a growing focus of fibrosis research. Although collagen-secreting myofibroblasts once were thought of as the master "producers" of fibrosis, this review will illustrate how macrophages function as the master "regulators" of fibrosis.



A few days out from the initial injury, when the macrophages have their custodial duties under control, they secrete some growth factors, which help initiate the proliferation phase.

Proliferation phase = growth of new tissue. Angiogenesis (new blood vessels), some collagen starts being deposited, wound contraction begins to occur.



Proliferation phase = growth of new tissue. Angiogenesis (new blood vessels), some collagen starts being deposited, wound contraction begins to occur.

Who is this and what's the analogy?

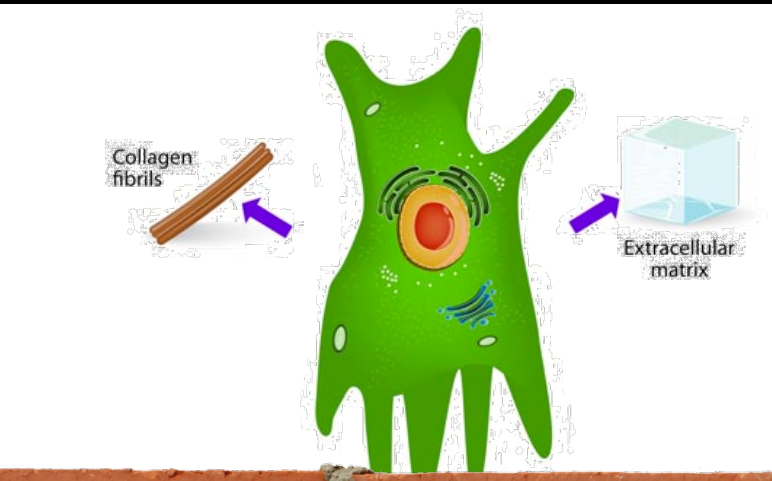
Proliferative Phase

fibroblast

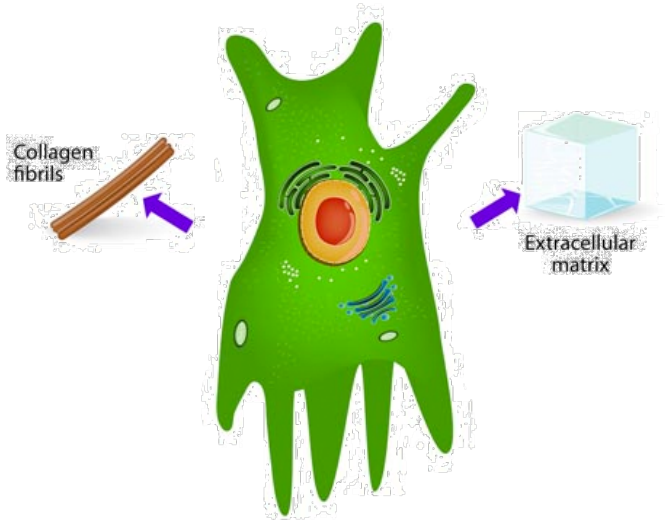
collagen

REVIEW: Cellular Stages of Healing

Fibroblasts are what do your remodeling; they're cells that synthesize stuff (stuff = collagen and fibronectin, i.e., ECM).



Fibroblasts are what do your remodeling; they're cells that synthesize stuff (stuff = collagen and fibronectin, i.e., ECM).



Osteoblasts are fibroblasts. Myoblasts, osteoblasts, fibroblasts, anything-blasts; they all do the same stuff.

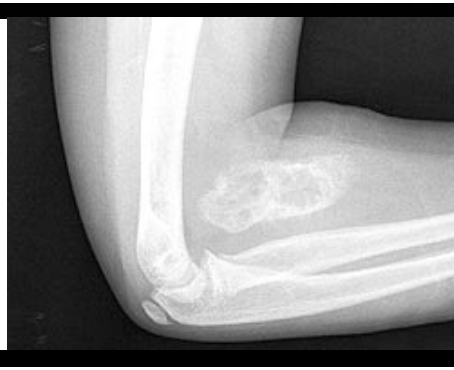
Progenitor cells: A little bit less freedom than stem cells, but they can still differentiate into the wrong stuff.

Fibroblasts are what do your remodeling; they're cells that synthesize stuff (stuff = collagen and fibronectin, i.e., ECM).

ORTHOPEDICS December 2008 - Volume 31 · Issue 12

Myositis Ossificans Mimicking Compartment Syndrome of the Forearm

Eitan Melamed, MD; David Angel, MD



Joint Bone Spine 83 (2016) 416–420

Myositis ossificans traumatica (circumscripta) and return to sport: A retrospective series of 19 cases

Thomas Simon[a,b], Yannick Guillodo[a,b], Gwenaelle Madouas[a], Alain Saraux[b,c,*]

Transcription factors do the determining.
But we can influence those factors…

Experimental & Molecular Medicine (2019) 51:54

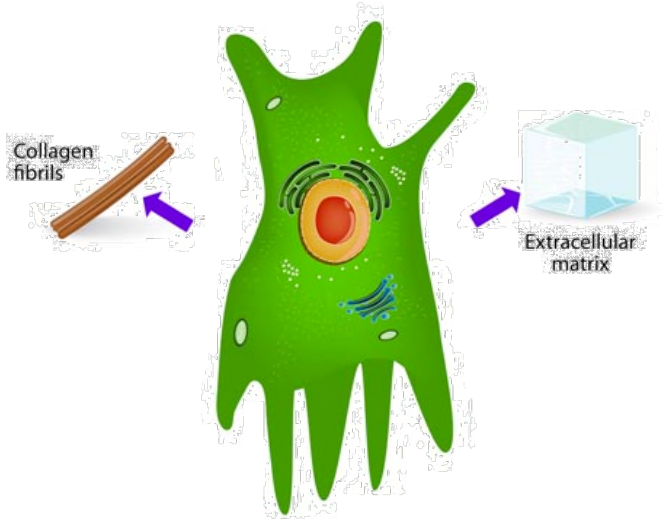
Direct conversion of fibroblasts to osteoblasts as a novel strategy for bone regeneration in elderly individuals

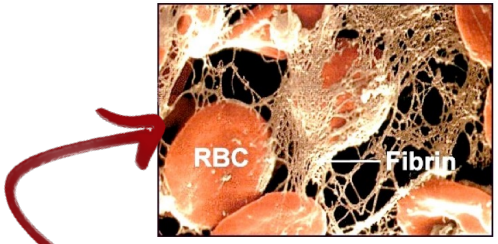
Yujung Chang[1], Byounggook Cho[1], Siyoung Kim[1] and Jongpil Kim[1,2]

Direct reprogramming

The conversion of somatic cells into specific cell types without passing through an intermediate stage by the introduction of combinations of lineage-specific factors is called direct reprogramming. Direct reprogramming was first introduced by Davis et al.[68], who demonstrated that fibroblasts can be converted into myoblasts by the ectopic expression of the muscle-specific transcription factor *MyoD*. Recent studies have reported that specific transcription factors can induce somatic cells to form several cell types, including cardiomyocytes[7,8], neurons[69], hematopoietic progenitor cells[70], and pancreatic beta cells[71], without a transient pluripotent stage. For instance, three transcription factors, Ascl1, Brn2, and Mty1l, can efficiently induce the formation of functional neurons from fibroblasts, resulting in the expression of neuronal proteins and the generation of action potentials[72]. In addition, three transcription factors, namely, Gata4, Hnf1a, and Foxa3, have been reported to induce the formation of functional hepatocyte-like cells (iHep) from mouse fibroblasts. The resulting iHep cells express hepatic genes and show an epithelial morphology[73].

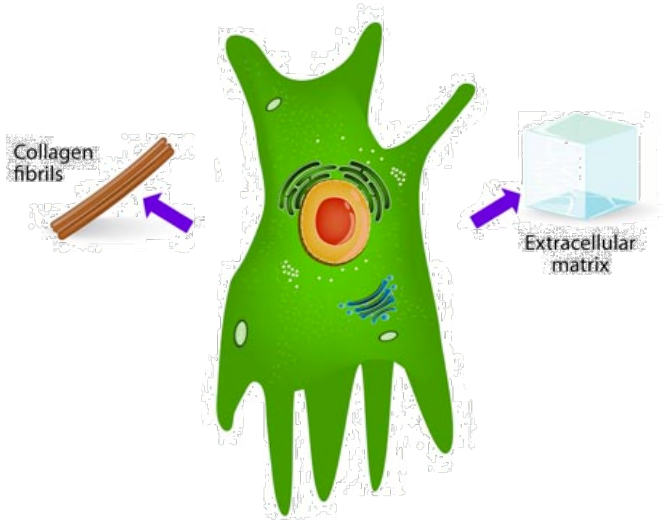
Fibroblasts are what do your remodeling; they're cells that synthesize stuff (stuff = collagen and fibronectin, i.e., ECM).





Fibroblasts adhere to fibrin, but require the presence of fibronectin to do so. The cross-linked provisional matrix is the perfect hub for fibroblasts to adhere and get to work (produce collagen).

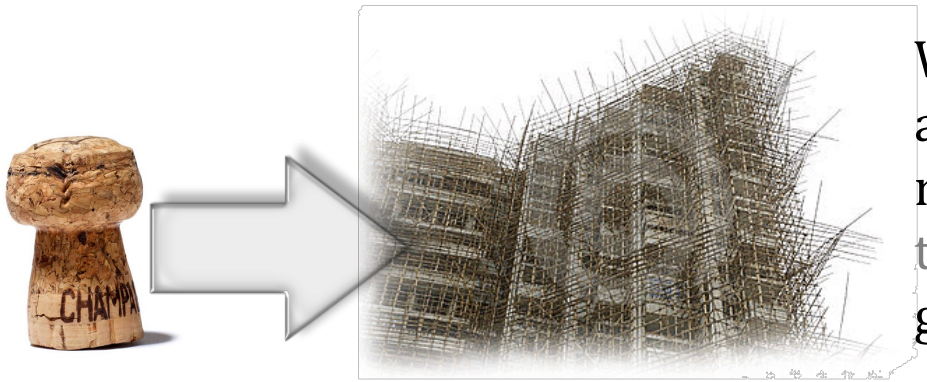
Fibroblasts are what do your remodeling; they're cells that synthesize stuff (stuff = collagen and fibronectin, i.e., ECM).



Collagen is the most abundant protein in your body. Your bones, muscles, ligaments, tendons, teeth, lips, whatever. At least a quarter of your protein.

When fibroblasts arrive (after a few days, up to five), they attach to the provisional matrix (fibrin). They continue to build in numbers; within a week of the initial injury, they're the most common cell in the area.

When fibroblasts arrive (after a few days, up to five), they attach to the provisional matrix (fibrin).



When fibroblasts arrive at the wound, as fibrin is being removed, a better matrix is being formed (better than the clot site) by proteoglycans, glycoproteins, and type III collagen.

Fibroblasts need appropriate architecture to do their job; this temporary scaffolding enables good cellular adhesion.

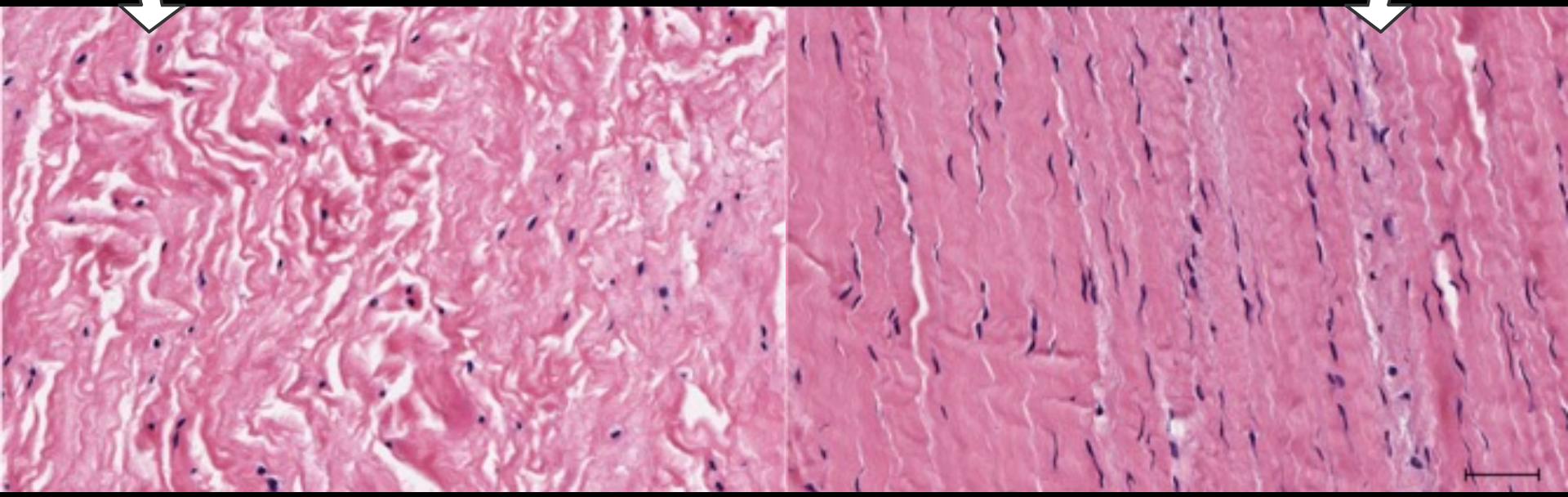
Univ

Necessary stage of tissue healing;
broken down with collagenases

Most of the collagen in your body

Collagen III

Collagen I



ADVANCES IN WOUND CARE, VOLUME 4, NUMBER 3

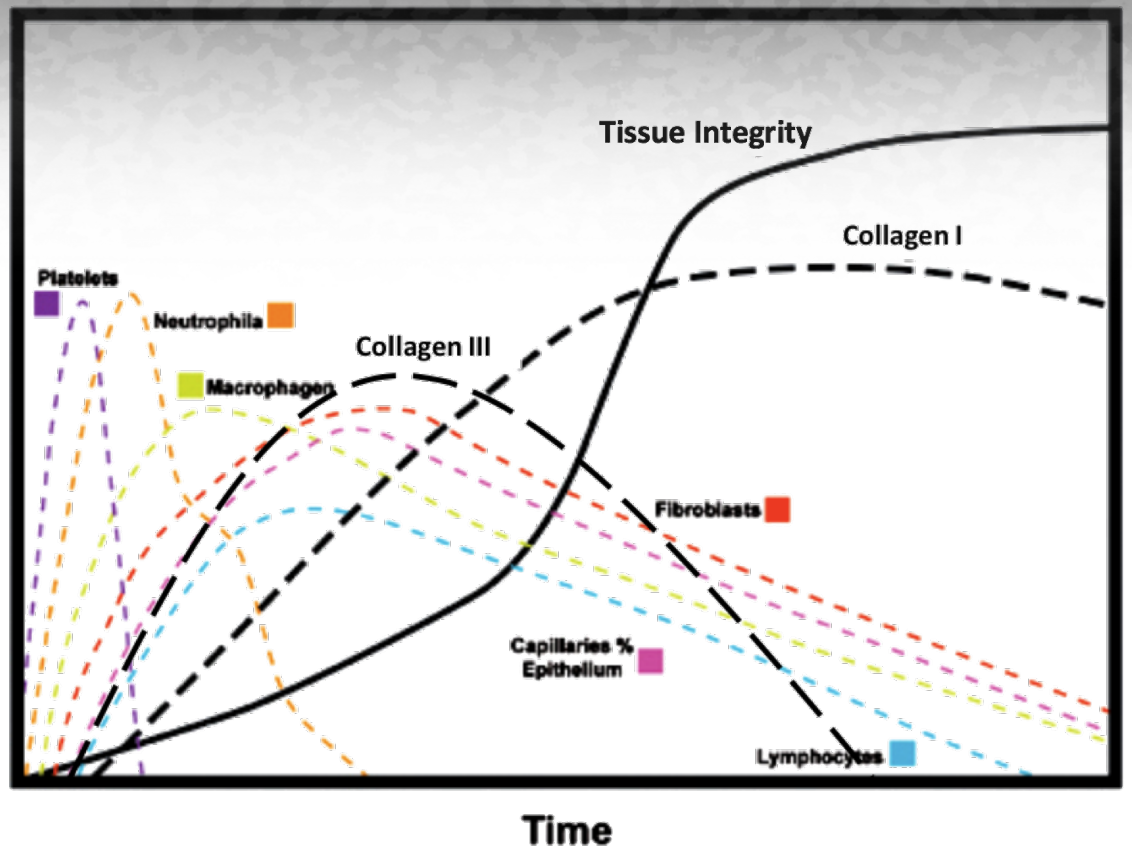
Extracellular Matrix Reorganization During Wound Healing and Its Impact on Abnormal Scarring

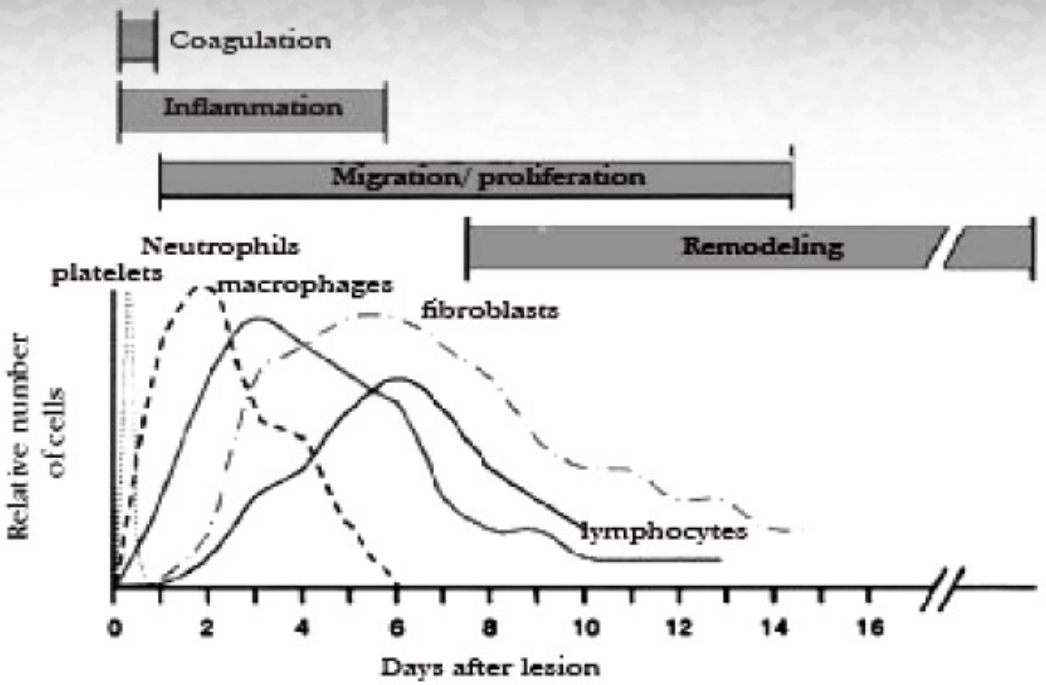
Meilang Xue and Christopher J. Jackson*

The collagens comprise the main structural component and comprise the highest protein concentration in the ECM, with 85% of the dermis being collagen. The collagens form a relaxed network of cross-linked long-chain fibers to give the strength and the elasticity of healthy skin and scar tissue. The two dominant types of collagen in wound repair are collagen I and III. In normal skin, collagen fibrils are composed of both collagen I and III with collagen III comprising ~20% of the total.

During the early stages of granulation tissue formation, myofibroblasts lay down collagen III, which plays a role in fibrillogenesis and determines the fibril diameter of collagen I. Collagen III expression increases more than the collagen I expression in the early stages of healing, resulting in increased ratio between the two collagen subtypes from 20% up to 50% collagen III.[79] During maturation of the scar, the ratio decreases again to normal levels. Thus, increased amounts of collagen III relative to collagen I identifies an immature scar. Table 4 shows main histological differences in collagen between normal, hypertrophic, and keloid scars.

REVIEW: Cellular Stages of Healing



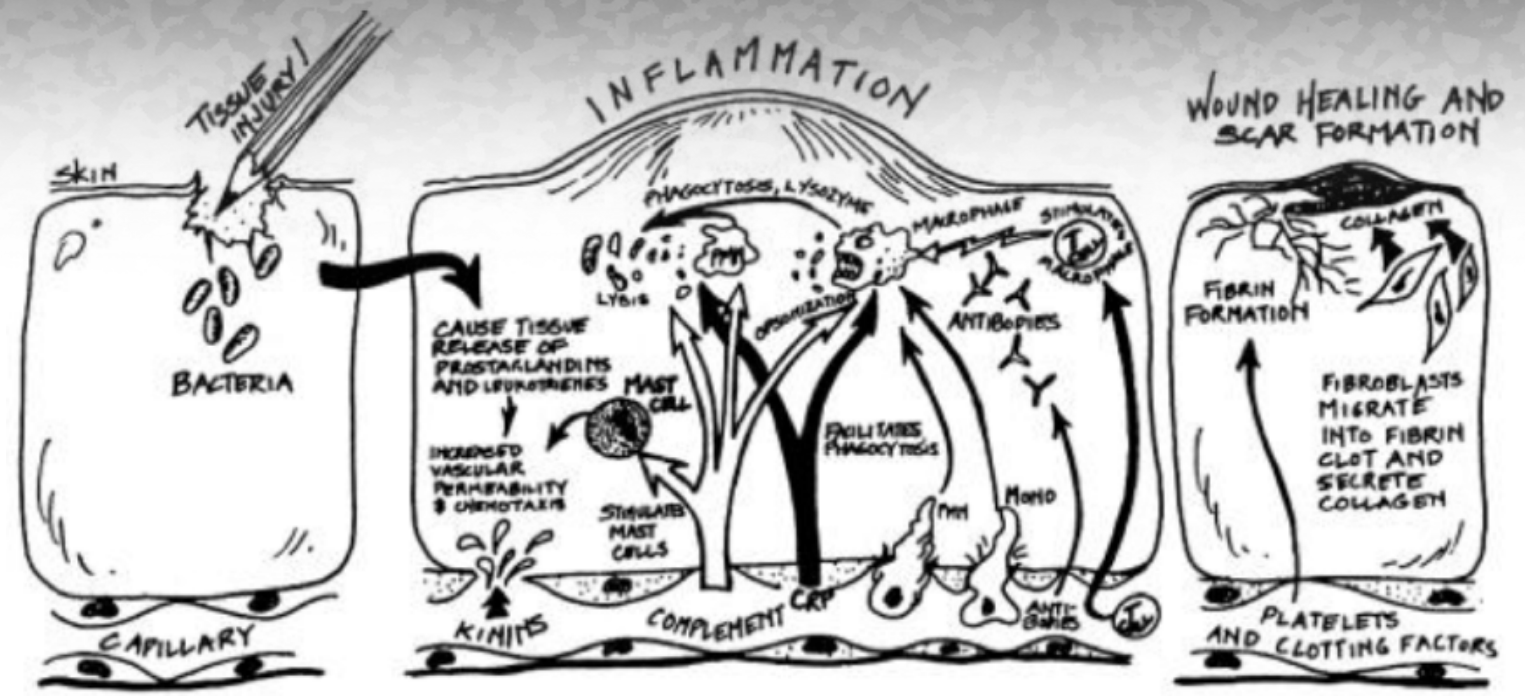


The key for fast, quality healing is a robust but brief inflammatory reaction. Clean the house quickly, then get the builders in.

Leaving inflammation in a tissue forever impairs the action of the fibroblasts. The timeframe should look something like this:

Healing can be likened to a kitchen fire

REVIEW: Cellular Stages of Healing



---Time---

Injury → Bleed → Clot → Clean → Repair → Heal →

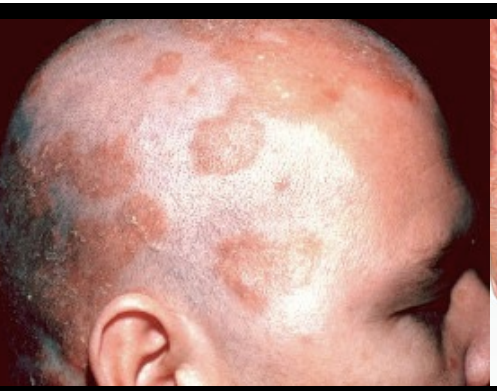
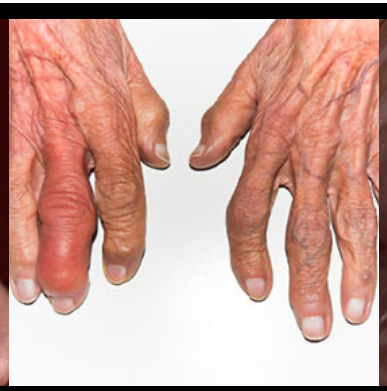
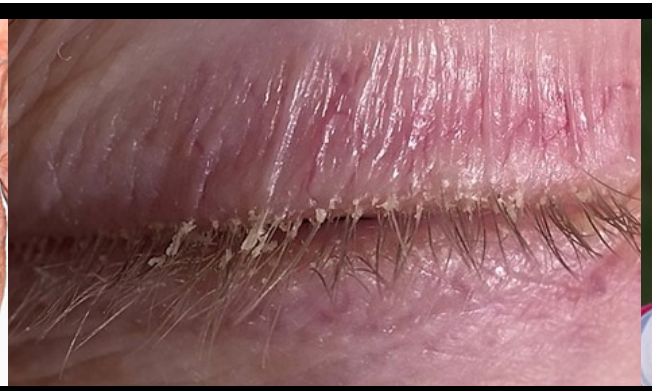
Function or Dysfunction

Exercise stress and injury stress result in the same stuff, the only difference is scale. Getting hit in the hip with a rock and performing twelve sets of pelvic Alfredsons produce the same *initial* series of responses (at the cellular level). The more damage, the more robust the response.

Good and bad healing environments:

Good = Brief, robust inflammatory response, then a robust fibroblast presence with proper mechanical signaling so that the collagen aligns properly.

Bad = Inflammation hanging around too long, impairing the arrival and action of the fibroblasts.

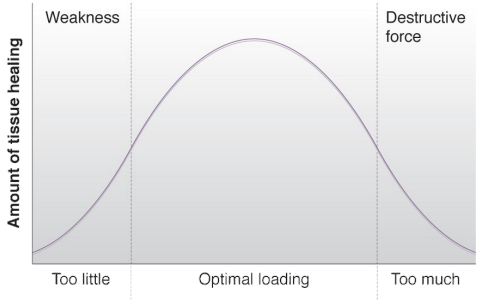





The stuff you need in order to heal:

Cells. They do the work.

Blood. It brings the cells to the site of tissue damage.

Tissue articulation. The tissues have to be touching.

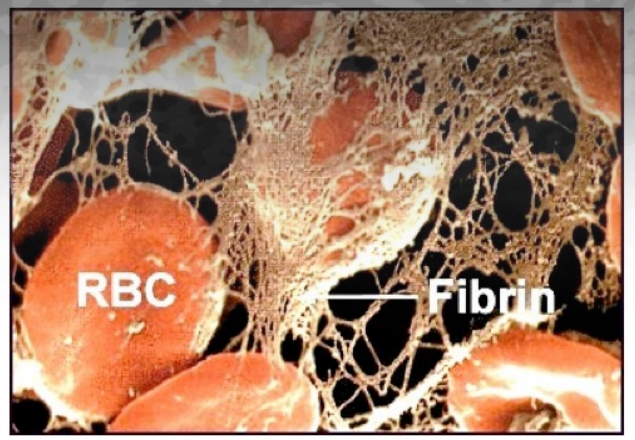


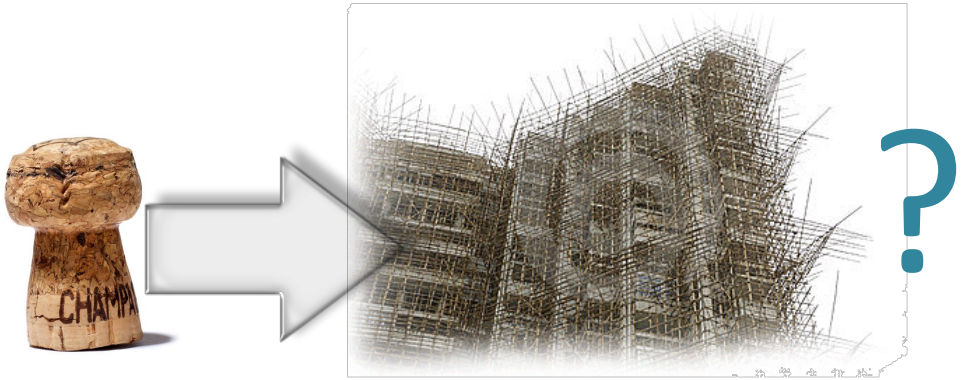
Once you've gotten all the cells there, they need to adhere to the wound (to the provisional matrix) in order to do their remodeling. Then you need mechanical stress.

Just to quiz yourself…

I'm not saying any of this will or won't be on the exam.

What's the first immune cell to show up?
What comes next?
What are their jobs?
How and when do fibroblasts come?
What are fibroblasts?
Where do they attach?
What do they do?



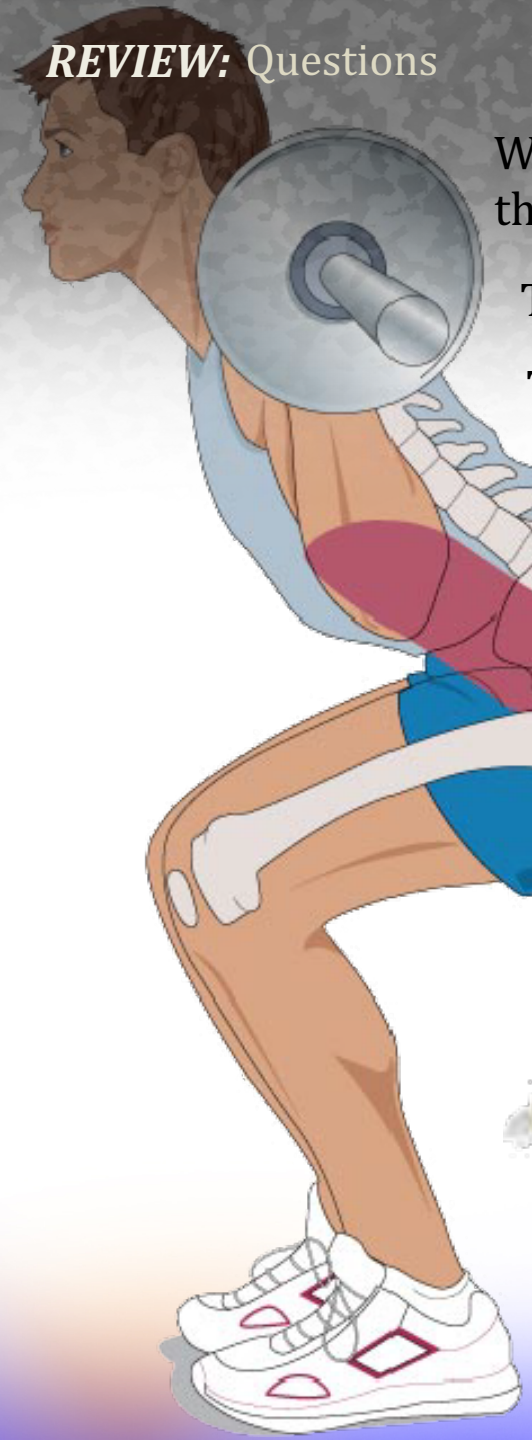


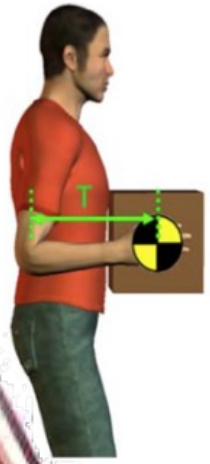
REVIEW: Questions

What causes compressive forces on the lumbar spine? Why are the erector spinae important in squatting?

Talk about intraabdominal pressure… Valsalva maneuver?

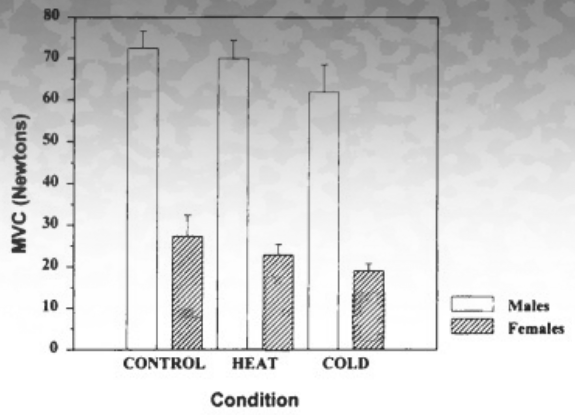
Talk about lumbar vs. thoracic compressive forces. How do the different lifting/carrying postures affect compression?

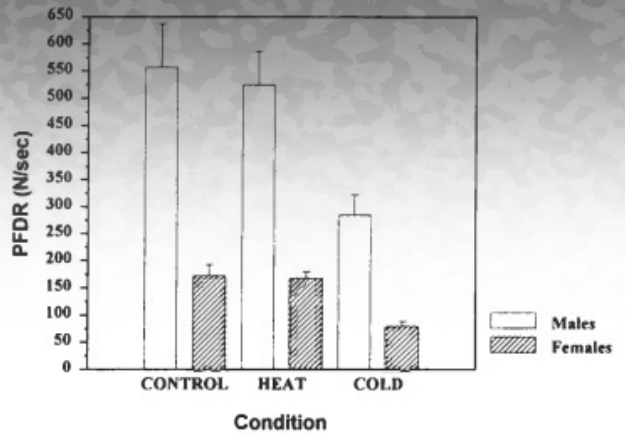




REVIEW: Questions

FIG. 153





What is the effect of pain, temperature, illness on voluntary force output?

What about the *rate* of achievement of maximal voluntary contraction?

What's nerve conduction velocity? What is a variable that slows it down?

Which nerve fibers have the fastest conduction velocity? Do they ordinarily transmit messages of pain?

Which nerve fibers are the slowest? What accounts for the speed difference?

J Appl Physiol 94: 1410–1420, 2003.
First published December 13, 2002; 10.1152/japplphysiol.01183.2001.

What did these guys find:

Activation imbalances in lumbar spine muscles in the presence of chronic low back pain

Lars I. E. Oddsson[1] and Carlo J. De Luca[1,2]
[1]NeuroMuscular Research Center and [2]Department of Biomedical Engineering, Boston University, Boston, Massachusetts 02215

1: Conscious altering of muscular recruitment characteristics owing to the pain associated with activation of the affected tissue.

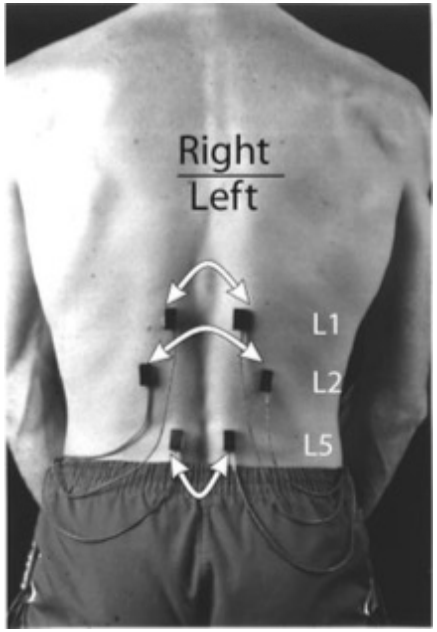
2: Unconscious, reflexive alteration in recruitment, which includes a delay in activation of the painful fibers, reduced magnitude of agonist recruitment, altered motor unit firing sequences in individual, whole muscles, and sometimes different muscles being called upon entirely, including simultaneous contributions from different body segments.

How do we know this?

J Appl Physiol 94: 1410–1420, 2003.
First published December 13, 2002; 10.1152/japplphysiol.01183.2001.

Activation imbalances in lumbar spine muscles in the presence of chronic low back pain

Lars I. E. Oddsson[1] and Carlo J. De Luca[1,2]
[1]*NeuroMuscular Research Center and* [2]*Department of Biomedical Engineering, Boston University, Boston, Massachusetts 02215*



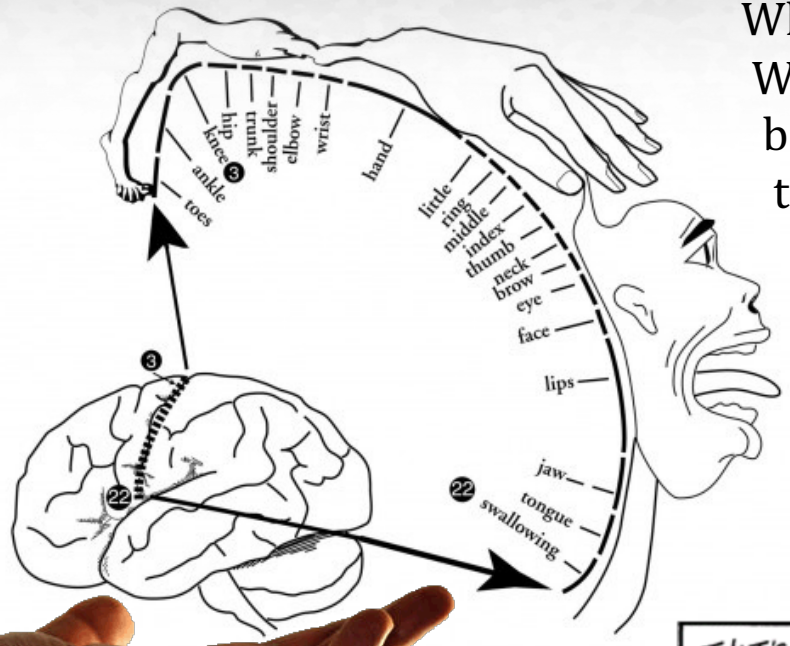
Surface EMG.

Is your back injured or healthy?

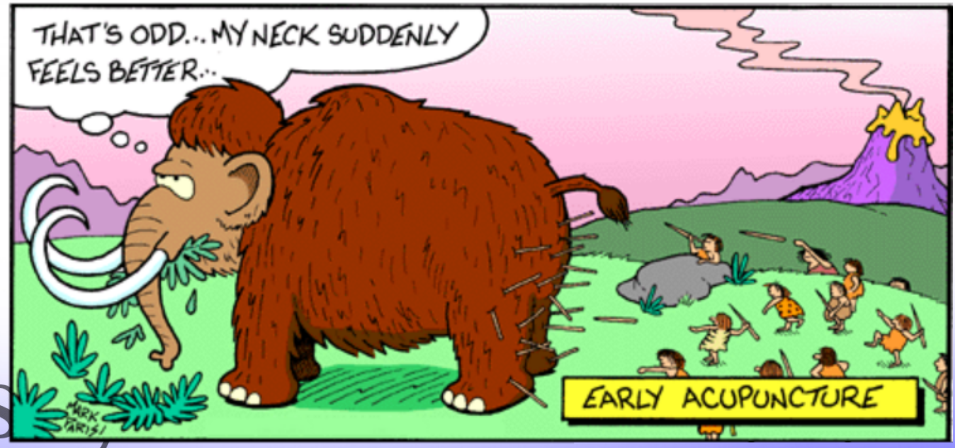
The firing patterns of injured muscle produce weird EMG signals. Some of it is bilateral, some of it is contribution from other body segments.

REVIEW: Questions

Think about Aα and Aβ fibers. You injure your hand in a game of ping pong (your opponent loses, gets angry and throws his paddle at you; it hits you on the wrist). You begin to either shake or rub that wrist. Why? What's the point of rubbing it?



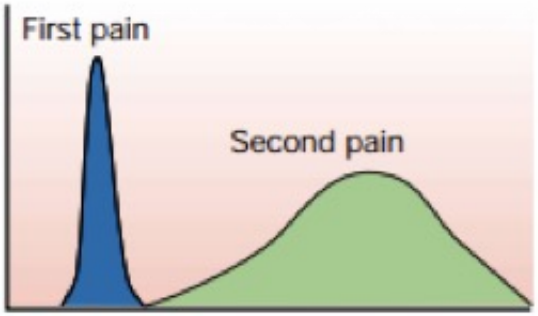
What about Icy Hot and acupuncture? What's a context in which those could be effective? And what would explain their effects?



REVIEW: Questions

What's the difference between peripheral and central plasticity of the neuronal system?

What's the difference between allodynia and hyperalgesia?



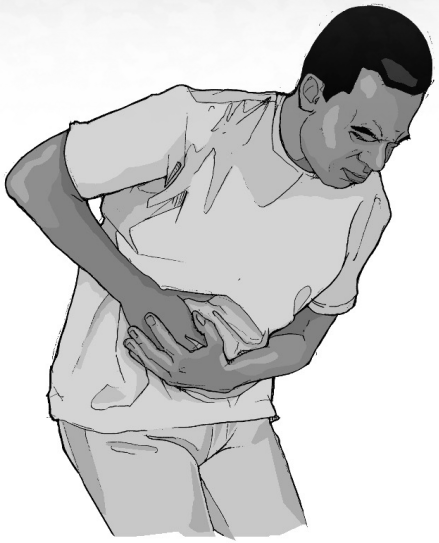
Which fibers are your "first responders" for pain; when they depolarize, you feel acute, sharp pain.

Which fibers produce dull, diffuse, delayed pain?

In consideration of these nerve differences, why would you pinch your face's bits to relieve a headache?

What else (other than mechanical stress and heat) do **Aδ fibers** and **C fibers** respond to?

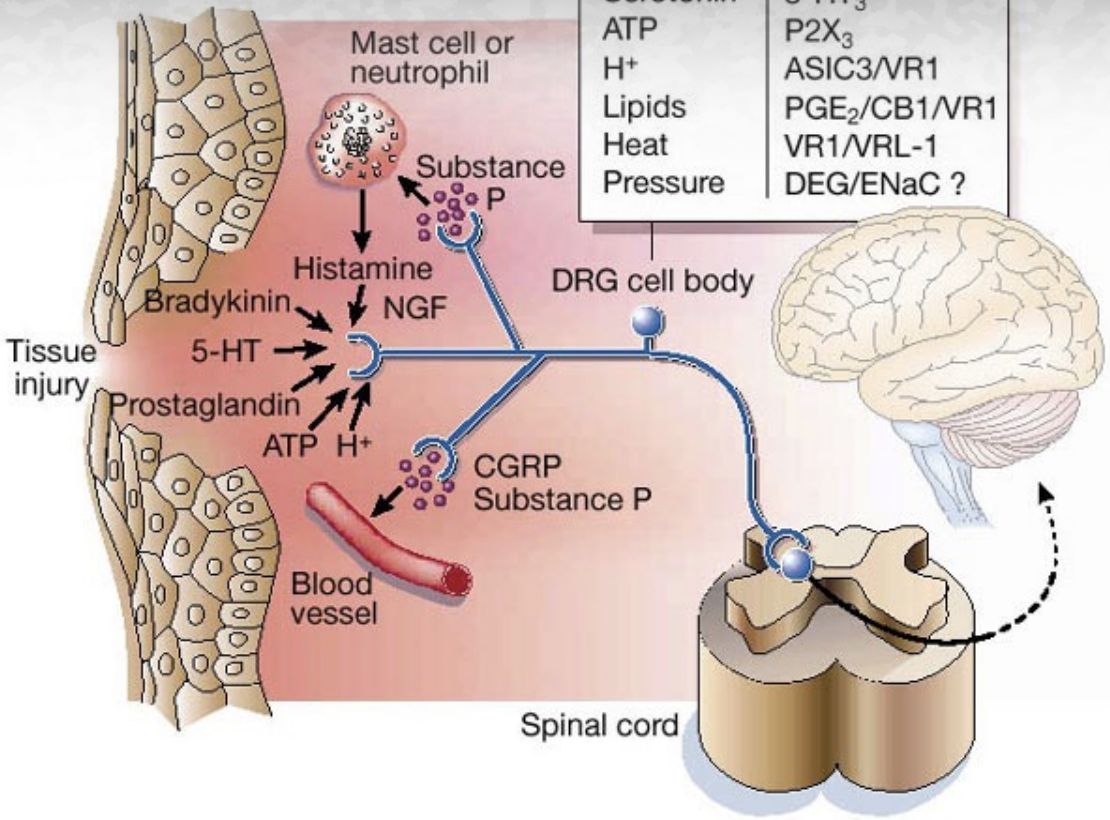
Vs.



What are "silent" or "sleeping" nociceptors?

REVIEW: Questions

Stimulus	Representative receptor
NGF	TrkA
Bradykinin	BK_2
Serotonin	$5\text{-}HT_3$
ATP	$P2X_3$
H^+	ASIC3/VR1
Lipids	PGE_2/CB1/VR1
Heat	VR1/VRL-1
Pressure	DEG/ENaC ?



What is the purpose of pain?

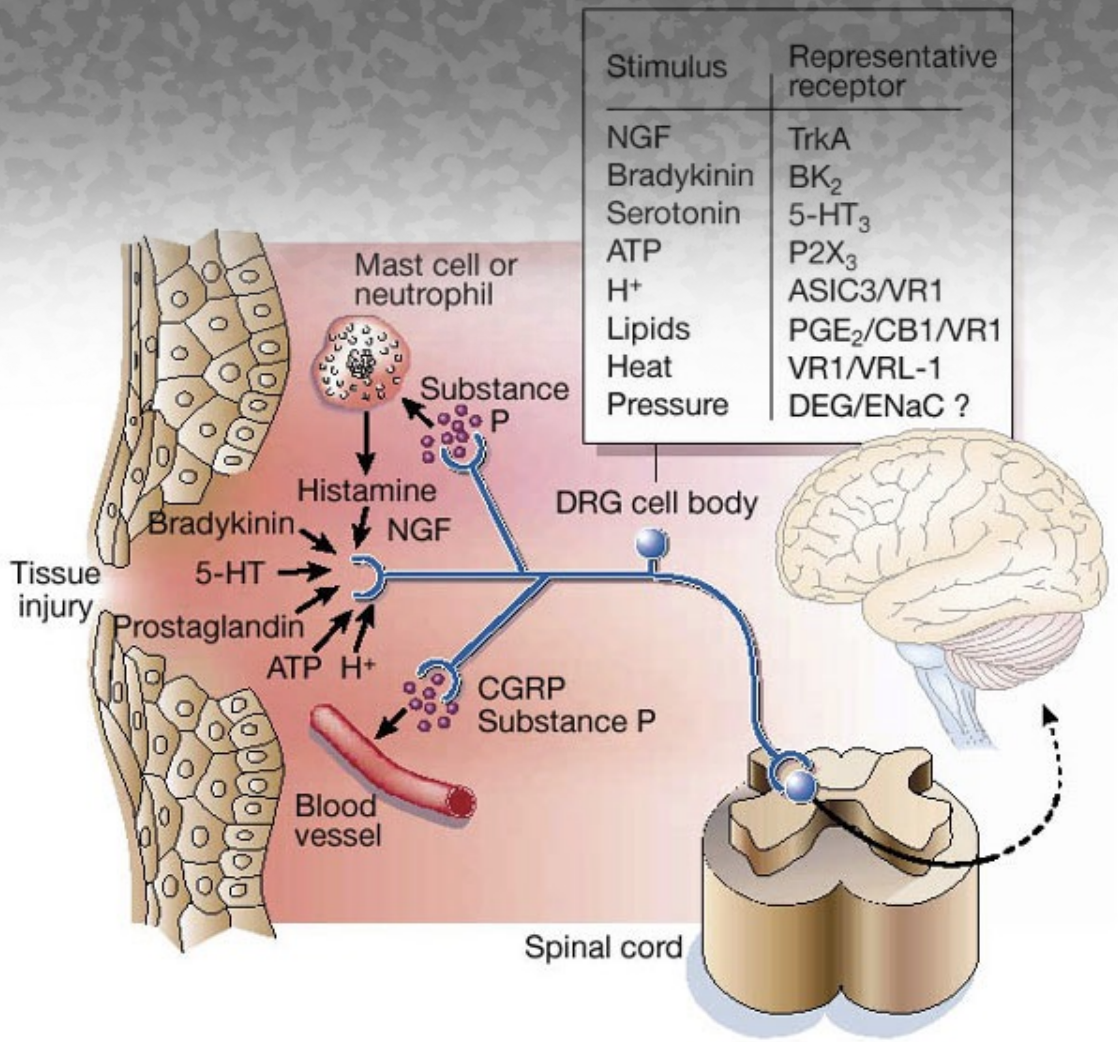
What are a few things that can sensitize or excite a nociceptor?

Where does the signal go?

Explain the relationship with ATP.

What is neurogenic inflammation?

What does Benadryl block?



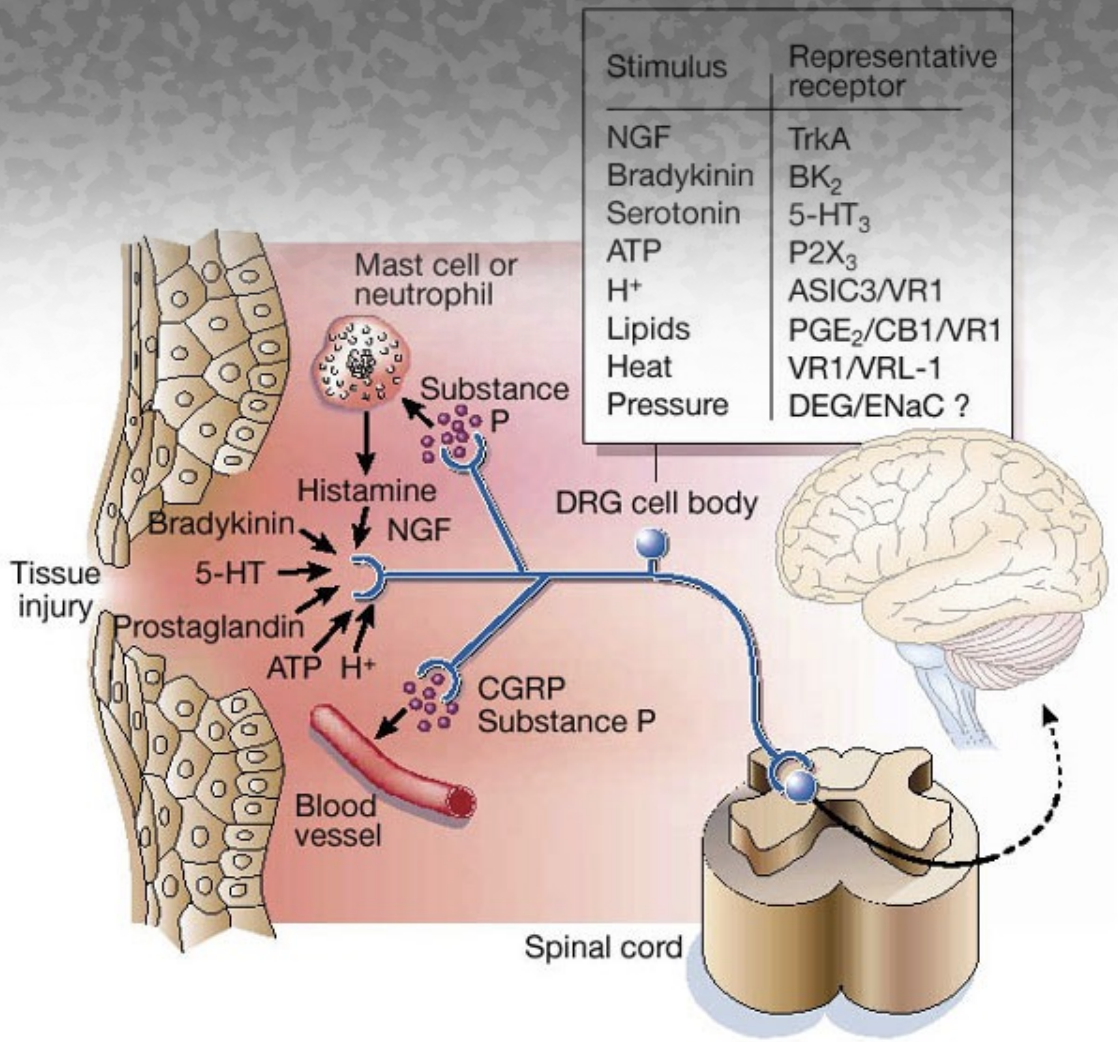
During tissue damage, a lot of stuff gets released.

Stuff: protein kinases, potassium, substance p, ATP, hydrogen ions, plasma globulins (converted to bradykinin), arachidonic acid (converted to prostaglandin), histamine, nerve growth factor, calcitonin gene-related peptide, serotonin, acetylcholine, etc.

Why does this stuff matter?

What does ”sensitizing” a nerve mean (“peripheral sensitization”)?

REVIEW: Questions



Under excessive inflammatory conditions or repeated injury, chemical receptors can be made more sensitive and can increase in number. What consequence would this have?

Talk about ATP and its role in nociception.

"Neurogenic inflammation"…?

What's the role of "Substance P"?

How does a nerve depolarize?

Where does "central sensitization" occur?

REVIEW: Questions

PHYSIOLOGICAL REVIEWS
Vol. 80, No. 2, April 2000
Printed in U.S.A.

Synaptic Control of Motoneuronal Excitability

JENS C. REKLING,* GREGORY D. FUNK,* DOUGLAS A. BAYLISS, XIAO-WEI DONG, AND JACK L. FELDMAN

How do inflammation, substance P, prolonged nociceptor activation (etc.) result in motor control changes?

What kinds of changes? And how do we measure/discover those changes?

What is the physical-therapy-related consequence of altered motor activity?

J Appl Physiol 94: 1410–1420, 2003.
First published December 13, 2002; 10.1152/japplphysiol.01183.2001.

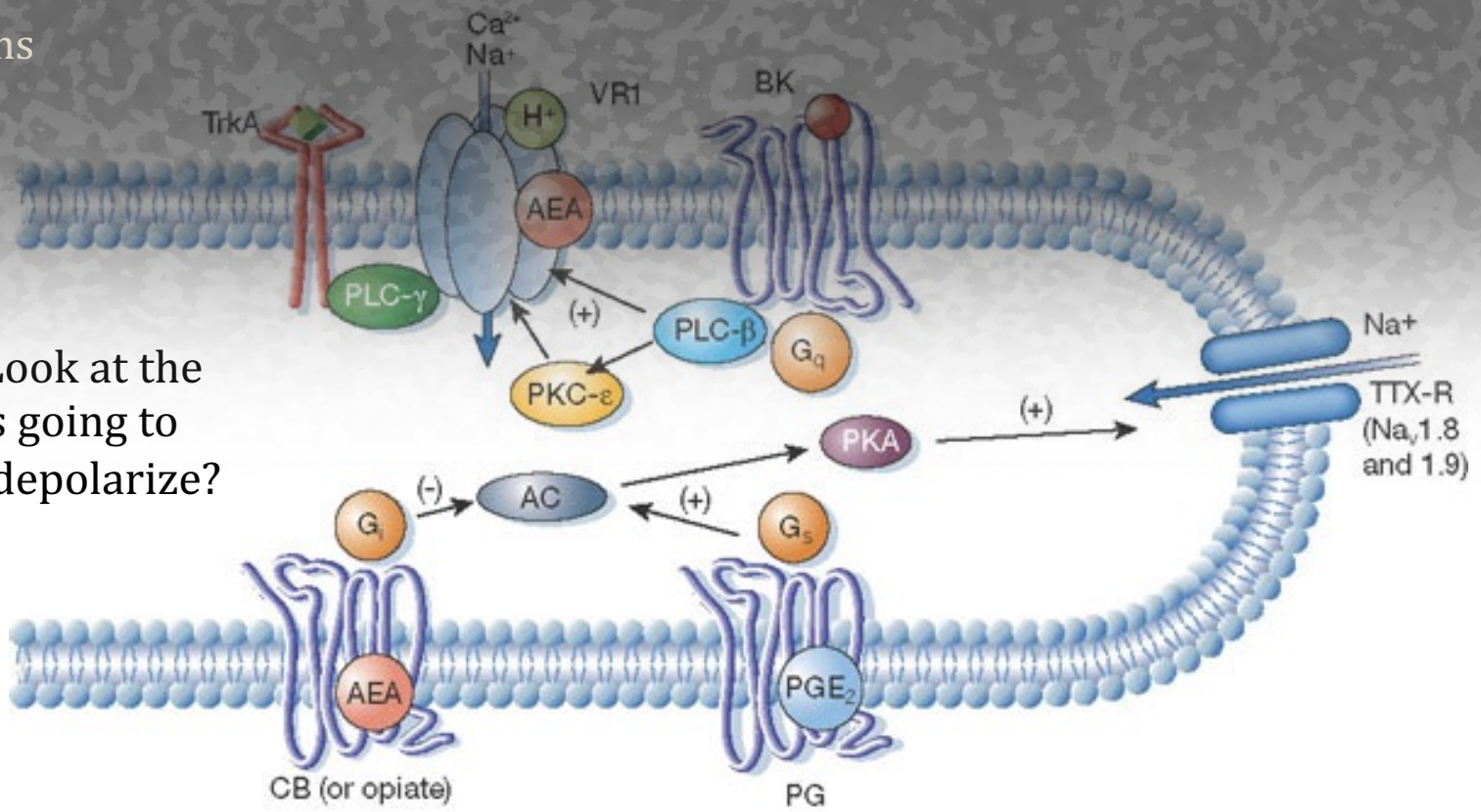
Activation imbalances in lumbar spine muscles in the presence of chronic low back pain

Lars I. E. Oddsson[1] and Carlo J. De Luca[1,2]
[1]NeuroMuscular Research Center and [2]Department of Biomedical Engineering, Boston University, Boston, Massachusetts 02215

Pain 86 (2000) 151–162

Experimental muscle pain produces central modulation of proprioceptive signals arising from jaw muscle spindles

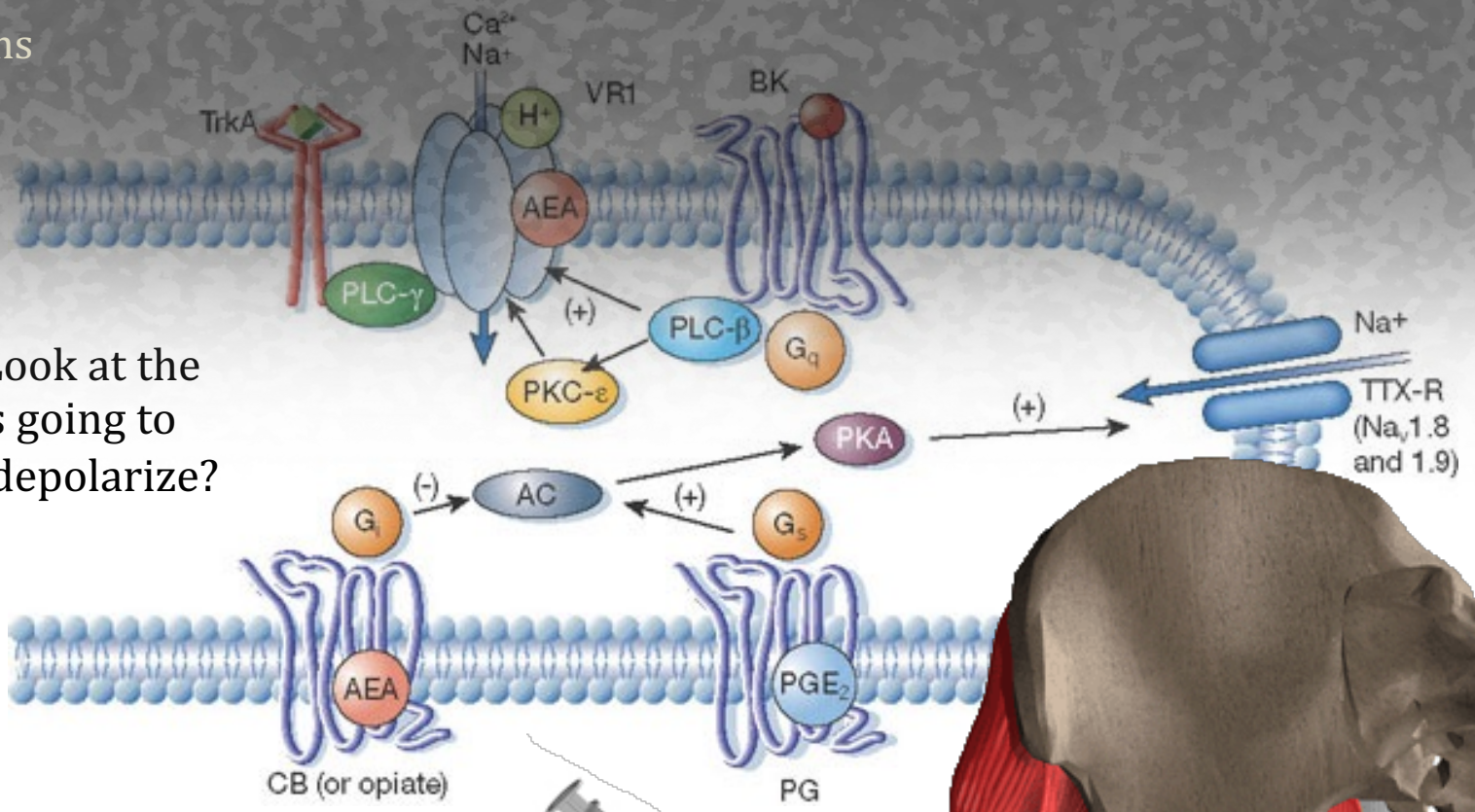
Norman F. Capra, Jin Y. Ro*



This is a nerve. Look at the far right. What is going to make this nerve depolarize?

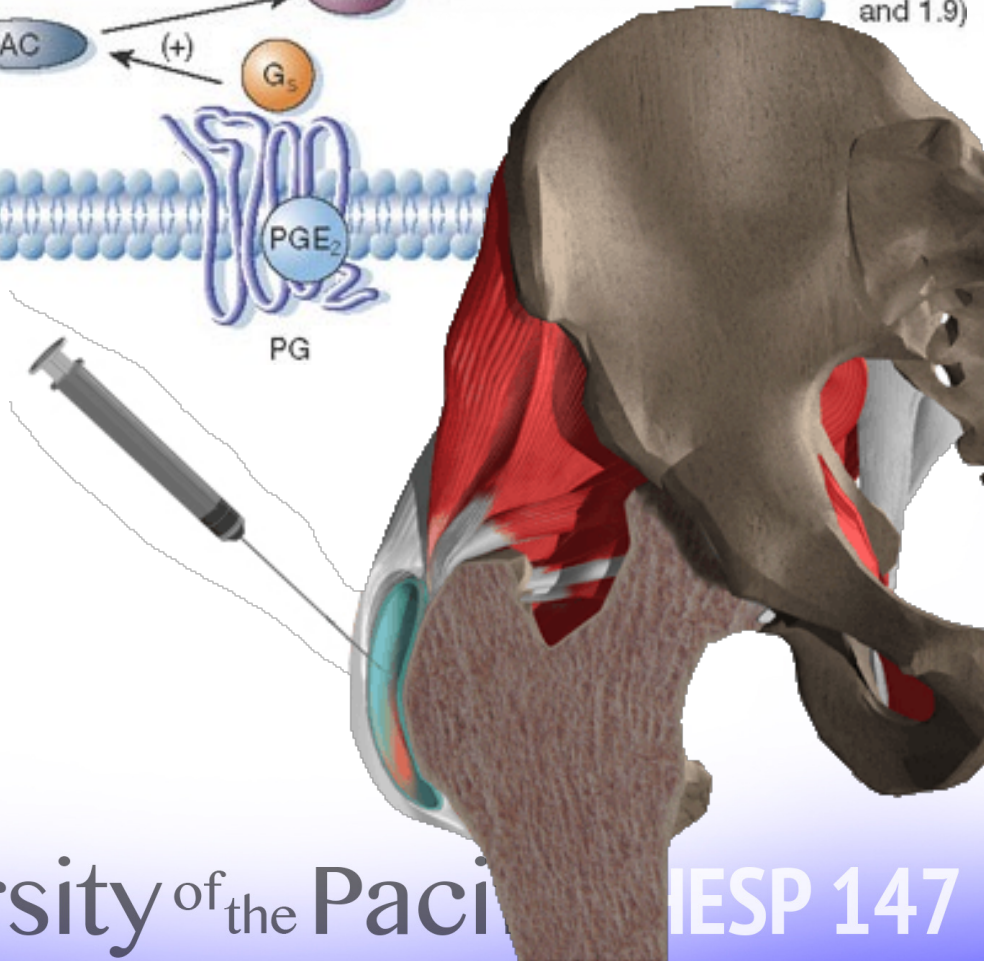
How do Aleve, Advil, and Aspirin reduce pain?

REVIEW: Questions



This is a nerve. Look at the far right. What is going to make this nerve depolarize?

How does a cortisone injection relieve pain?



What else (other than mechanical stress and heat) do **Aδ fibers** and **C fibers** respond to?

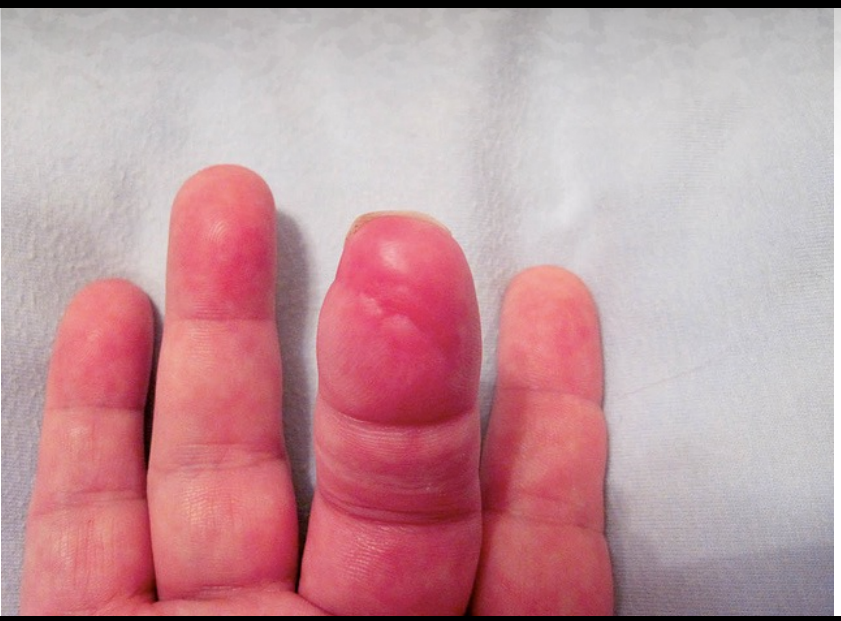
What are "silent" or "sleeping" nociceptors?

What's the difference between peripheral and central plasticity of the neuronal system?

What's the difference between allodynia and hyperalgesia?

REVIEW: Questions

?



Know the Clinical Signs:

1. **Redness:** What's causing this?
2. **Pain:** What's causing this?
3. **Heat:** What's causing this?
4. **Swelling:** What's causing this?

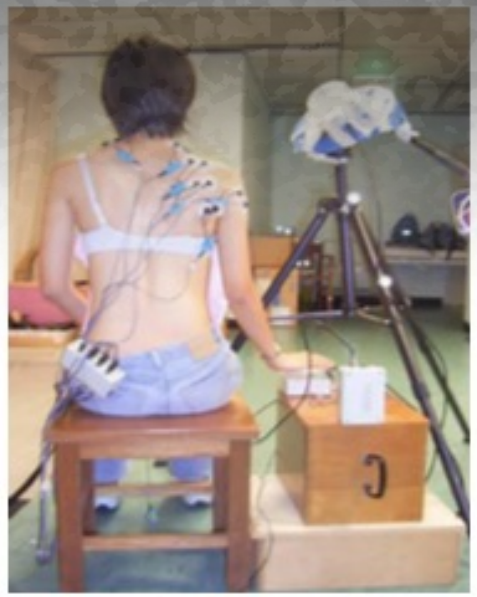
Remember: May also find a loss of function.

REVIEW: Questions

Why are we looking at pictures of 5 different amounts of hamstring damage?

Is inflammation bad?
What's a good environment and what's a bad one?
What do you need in order to heal?

REVIEW: Questions



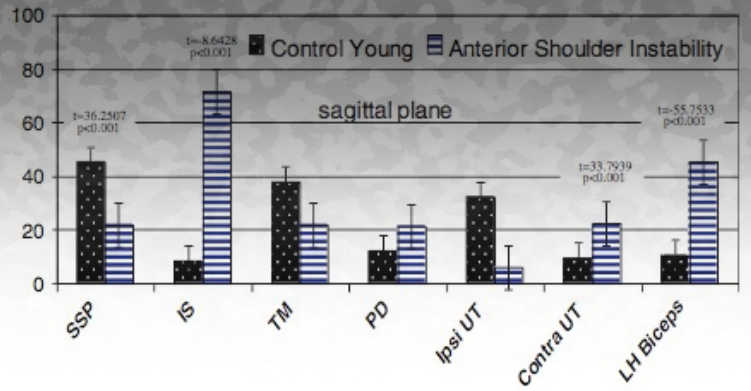
Fine Wire electrodes
(sampling rate 1800Hz)

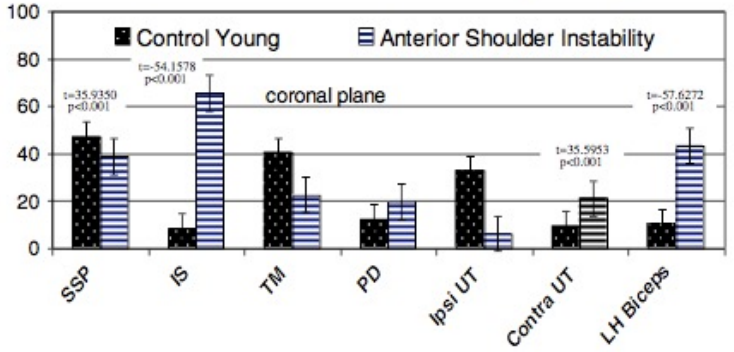
- Infraspinatus
- Supraspinatus
- Teres Major

Surface electrodes
(sampling rate 1800Hz)

- Posterior Deltoid
- Bilateral Upper Trapezius
- Biceps brachii

Figure 1 Experimental set-up.





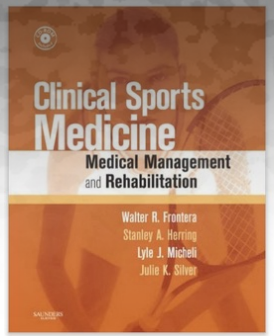
BMC
Sports Science, Medicine & Rehabilitation

Control strategies to re-establish glenohumeral stability after shoulder injury

Bala S Rajaratnam[1*], James CH Goh[2] and Prem V Kumar[2]

What happens after the injury, when the athlete returns to play?
(Talk about internal forces – *kinetics*)

REVIEW: Questions



CHAPTER 26

Shoulder Injuries

Jason Nielson and Peter Gerbino

10820 • The Journal of Neuroscience, September 2, 2009 • 29(35):10820–10826

Behavioral/Systems/Cognitive

Motor Unit Recruitment Strategies Are Altered during Deep-Tissue Pain

Kylie Tucker,[1] Jane Butler,[3] Thomas Graven-Nielsen,[4] Stephan Riek,[2] and Paul Hodges[1]

The underlying biomechanics of a serve, a strike, a throw, a swing, a pass, a kick are changed in the presence of (or following the presence of) pain. Is this detected in return-to-play tests?

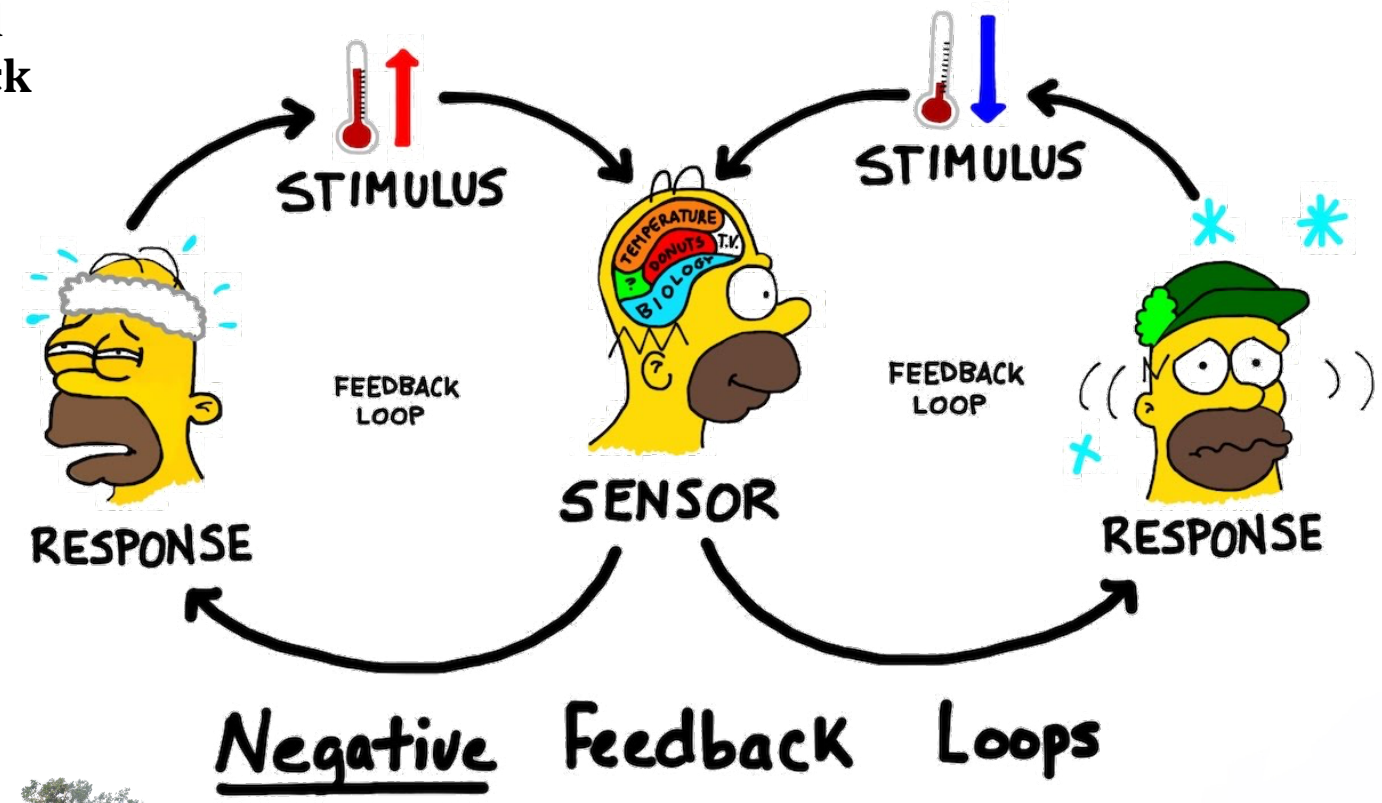
What is being evaluated in a normal testing batter? What's missing?

PART THREE

Stuff to remain somewhat familiar with from Block One. The exam won't demand much of you from the following slides; just a couple of questions.

What to Remember from Block 1

Homeostasis and **negative feedback loops:**



What to Remember from Block 1

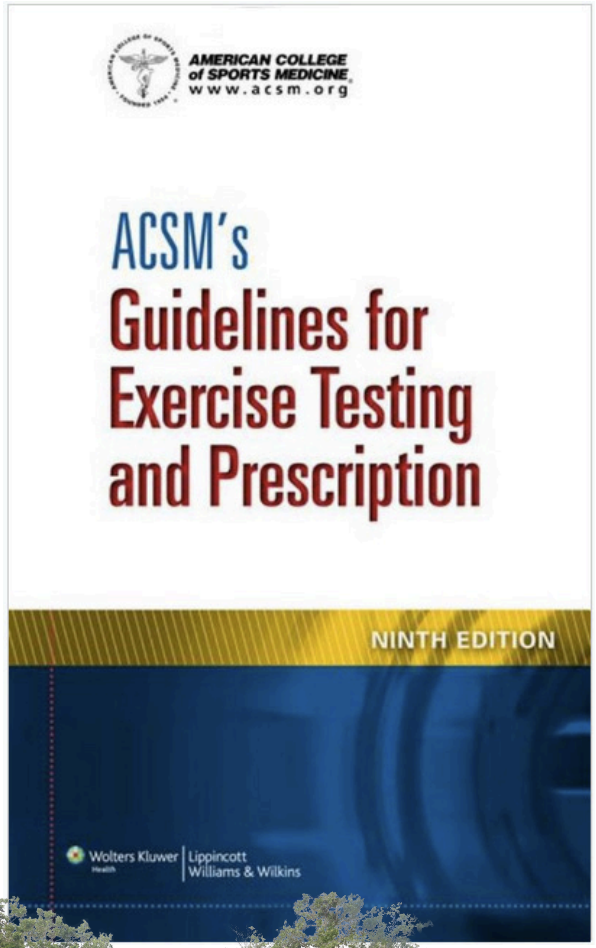


TABLE 2.2. Atherosclerotic Cardiovascular Disease (CVD) Risk Factors and Defining Criteria (26,31)

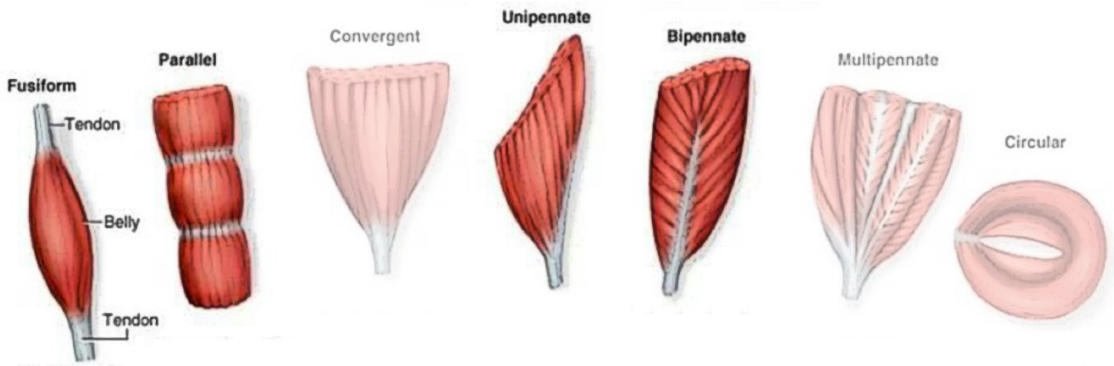
Risk Factors	Defining Criteria
Age	Men ≥45 yr; women ≥55 yr (12)
Family history	Myocardial infarction, coronary revascularization, or sudden death before 55 yr in father or other male first-degree relative or before 65 yr in mother or other female first-degree relative
Cigarette smoking	Current cigarette smoker or those who quit within the previous 6 mo or exposure to environmental tobacco smoke
Sedentary lifestyle	Not participating in at least 30 min of moderate intensity, physical activity (40%–<60% $\dot{V}O_2R$) on at least 3 d of the week for at least 3 mo (22,30)
Obesity	Body mass index ≥30 kg · m^{-2} *or* waist girth >102 cm (40 in) for men and >88 cm (35 in) for women (10)
Hypertension	Systolic blood pressure ≥140 mm Hg and/or diastolic ≥90 mm Hg, confirmed by measurements on at least two separate occasions, *or* on antihypertensive medication (9)
Dyslipidemia	Low-density lipoprotein (LDL) cholesterol ≥130 mg · dL^{-1} (3.37 mmol · L^{-1}) *or* high-density lipoprotein[b] (HDL) cholesterol <40 mg · dL^{-1} (1.04 mmol · L^{-1}) *or* on lipid-lowering medication. If total serum cholesterol is all that is available, use ≥200 mg · dL^{-1} (5.18 mmol · L^{-1}) (21)
Prediabetes[a]	Impaired fasting glucose (IFG) = fasting plasma glucose ≥100 mg · dL^{-1} (5.55 mmol · L^{-1}) and ≤125 mg · dL^{-1} (6.94 mmol · L^{-1}) *or* impaired glucose tolerance (IGT) = 2 h values in oral glucose tolerance test (OGTT) ≥140 mg · dL^{-1} (7.77 mmol · L^{-1}) and ≤199 mg · dL^{-1} (11.04 mmol · L^{-1}) confirmed by measurements on at least two separate occasions (5)
Negative Risk Factors	**Defining Criteria**
High-density lipoprotein (HDL) cholesterol	≥60 mg · dL^{-1} (1.55 mmol · L^{-1})

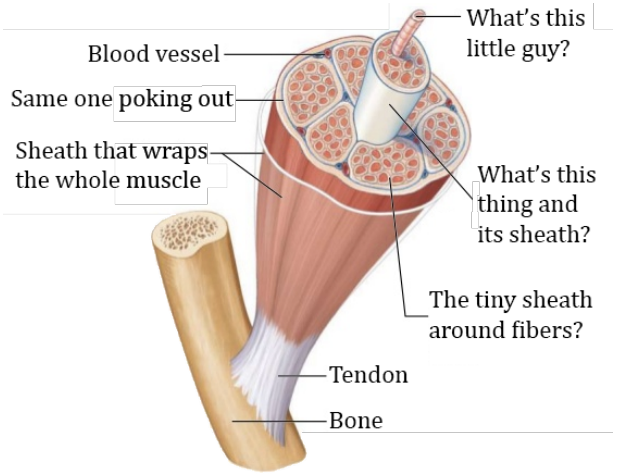
[a]If the presence or absence of a CVD risk factor is not disclosed or is not available, that CVD risk factor should be counted as a risk factor except for prediabetes. If the prediabetes criteria are missing or unknown, prediabetes should be counted as a risk factor for those ≥45 yr, especially for those with a body mass index (BMI) ≥25 kg · m^{-2}, and those <45 yr with a BMI ≥25 kg · m^{-2} and additional CVD risk factors for prediabetes. The number of positive risk factors is then summed.

[b]High HDL is considered a negative risk factor. For individuals having high HDL ≥60 mg · dL^{-1} (1.55 mmol · L^{-1}), for these individuals one positive risk factor is subtracted from the sum of positive risk factors.

$\dot{V}O_2R$, oxygen uptake reserve.

What to Remember from Block 1





European Journal of Applied Physiology and Occupational Physiology

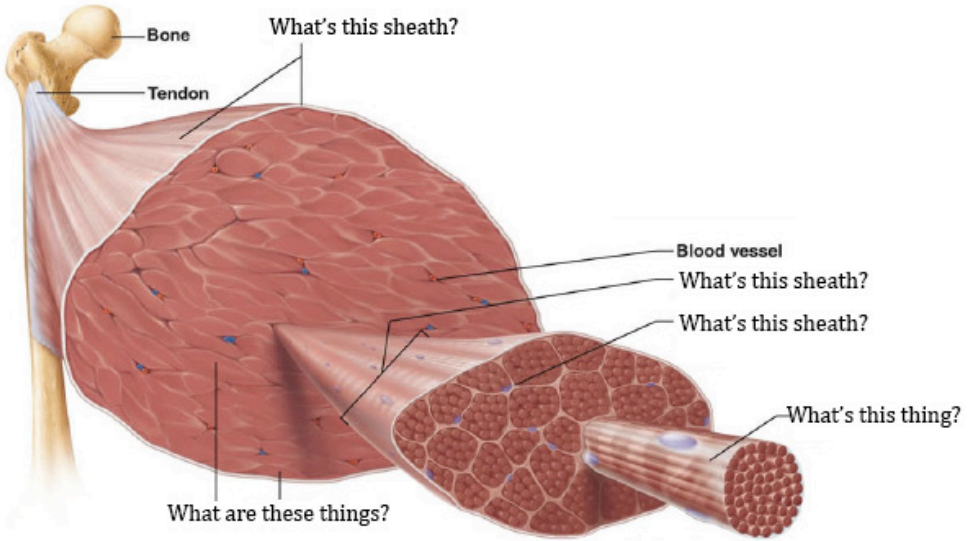
1986;55(2):137-41.

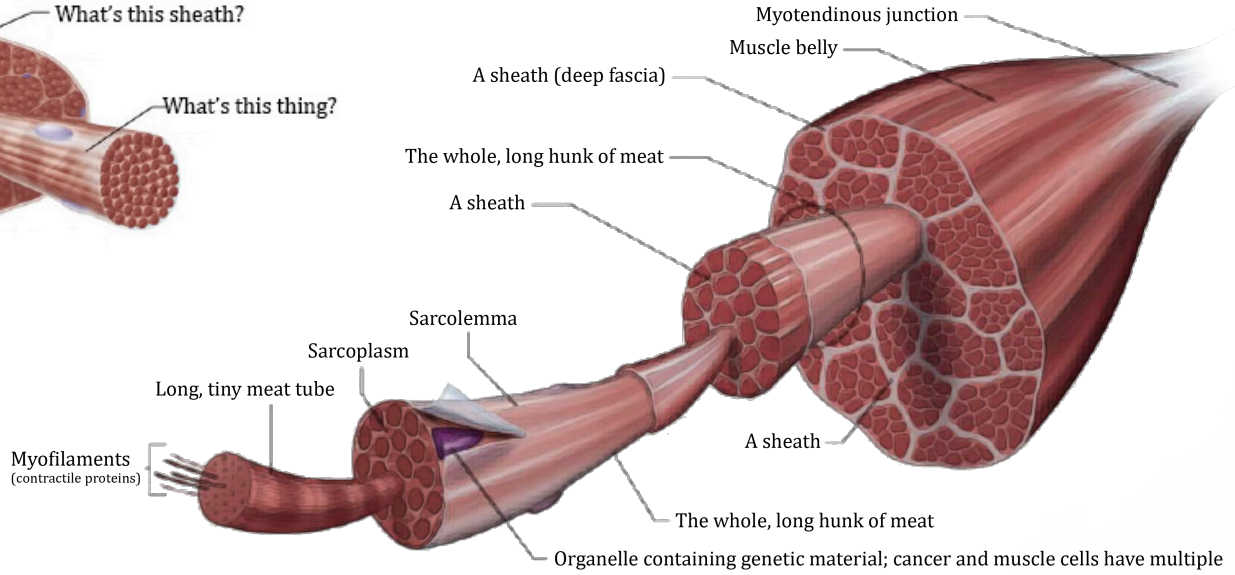
Gonyea WJ, Sale DG, Gonyea FB, Mikesky A.

Abstract

The effect of weight-lifting, which induced muscular enlargement, on fiber number was tested in the flexor carpi radialis muscle by operantly conditioning 6 cats to flex their right wrist against increasing resistance for an average of 101 weeks. The left was used as a control. At the end of training, the cats were performing "one-arm" lifts with an average of 57% of their body weight. There was an 11% greater muscle weight (P less than 0.01) and 9% (P less than 0.02) more fibers in the exercised muscles from the right limb than in the left. This study using a different method, supports our earlier observations that prolonged weight-lifting exercise significantly increases the total number of muscle fibers.

What to Remember from Block 1





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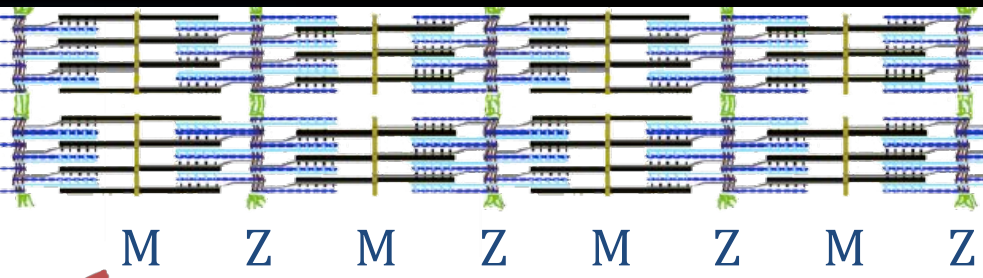
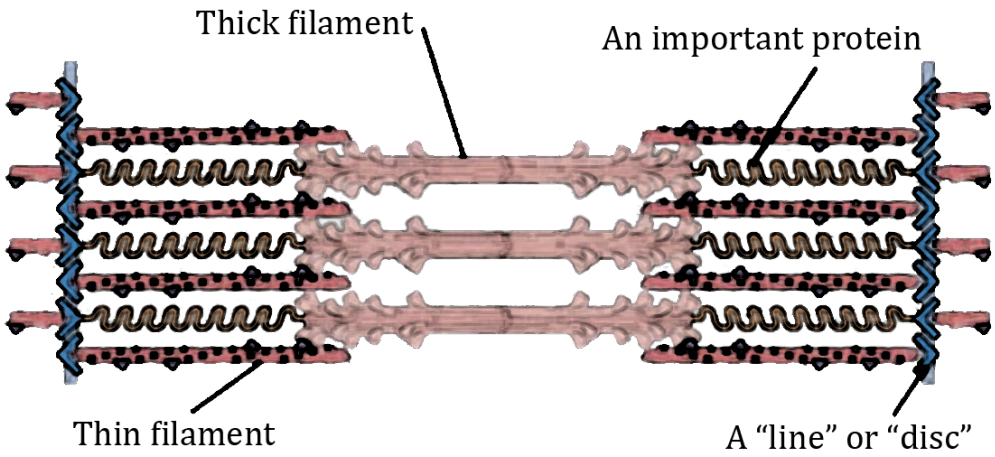
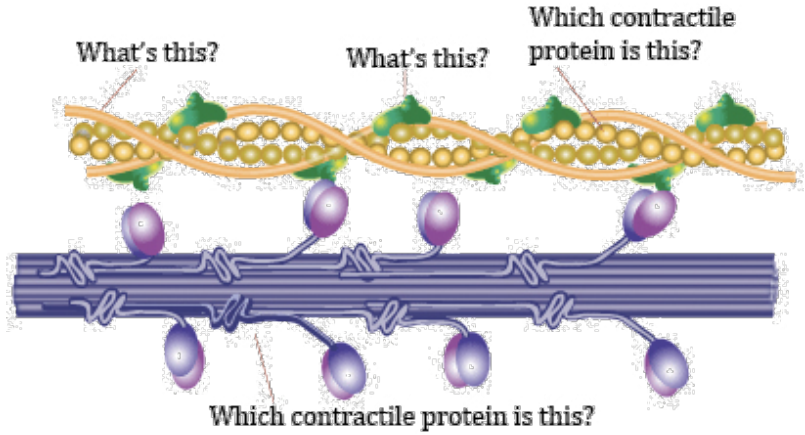


What are these letters?

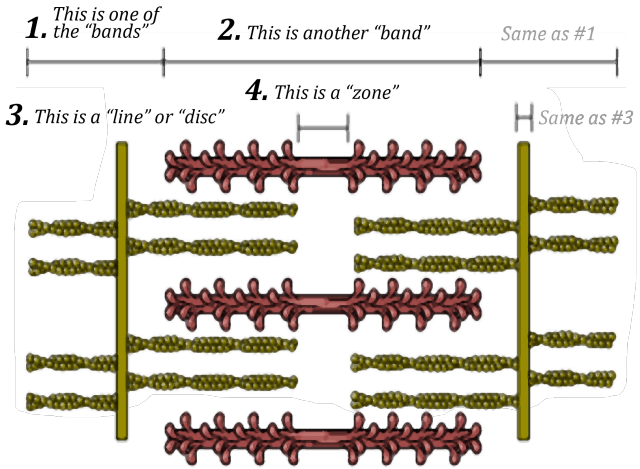
Know sarcomerogenesis
Know sarcomerolysis

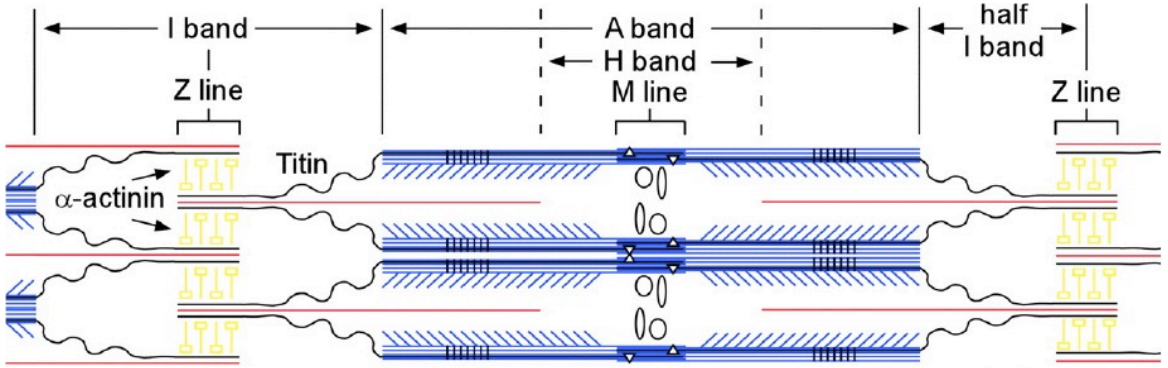
What to Remember from Block 1



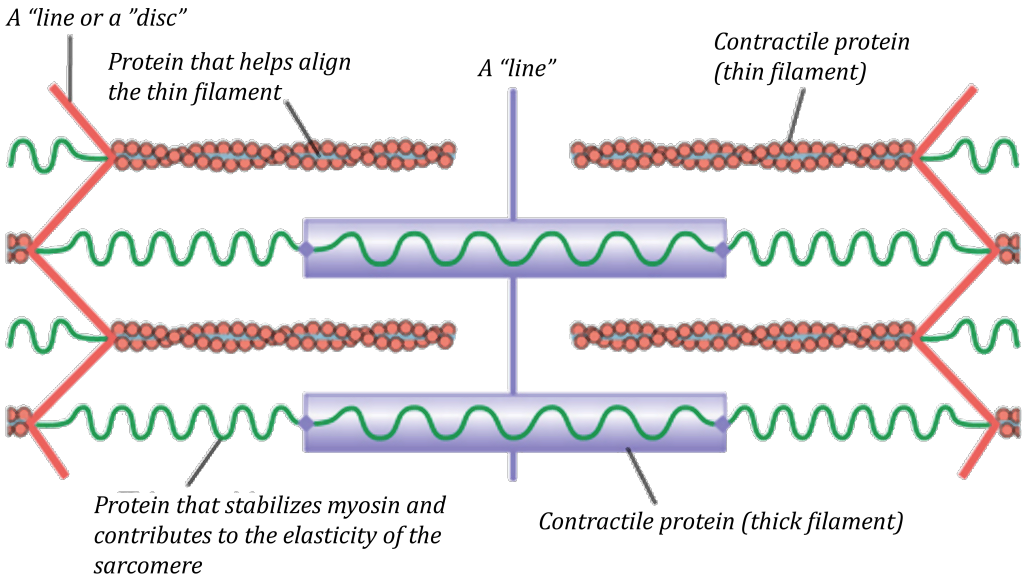


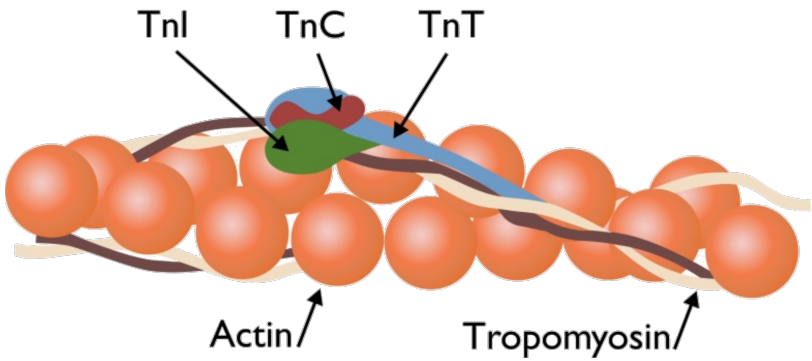
What to Remember from Block 1





What to Remember from Block 1





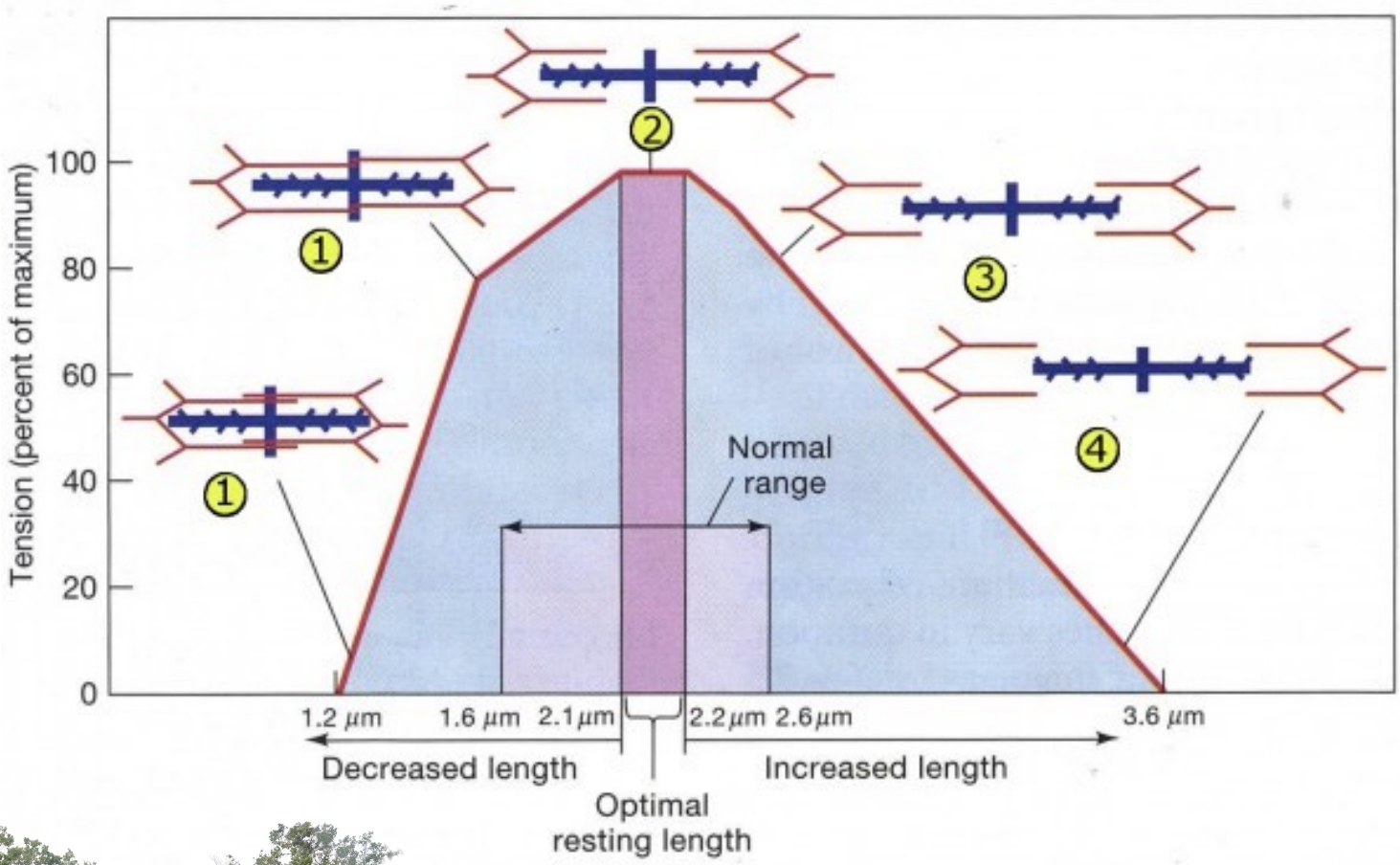
What to Remember from Block 1

Weak bond = ?

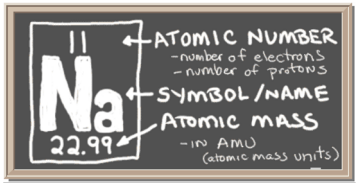
Strong bond = ?

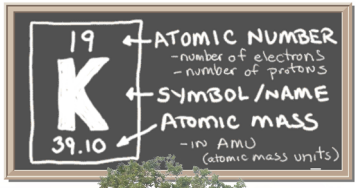
ATP hydrolysis = ?

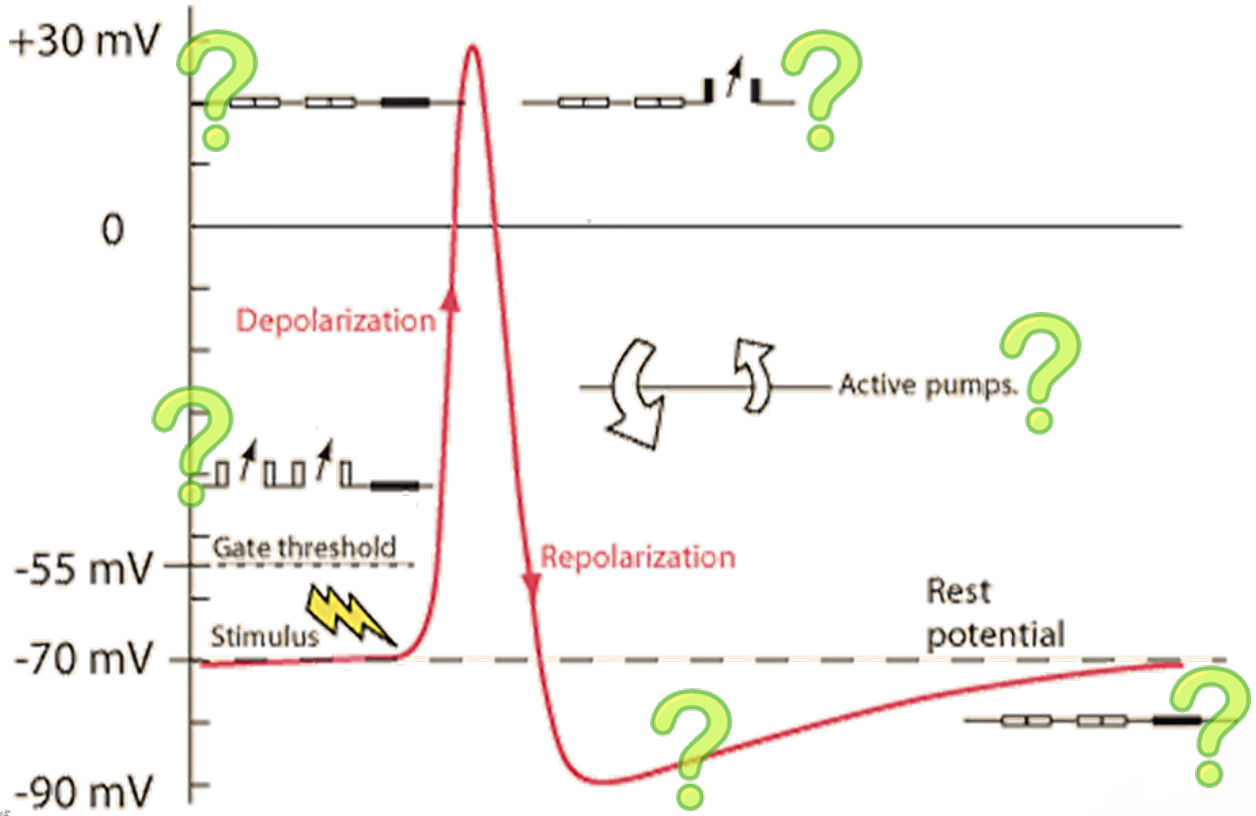
What to Remember from Block 1



What to Remember from Block 1

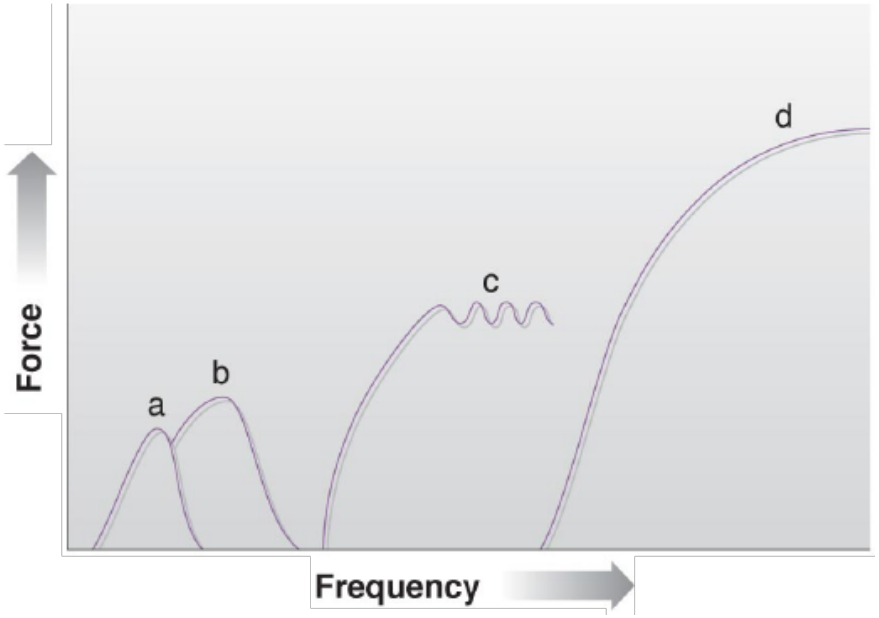


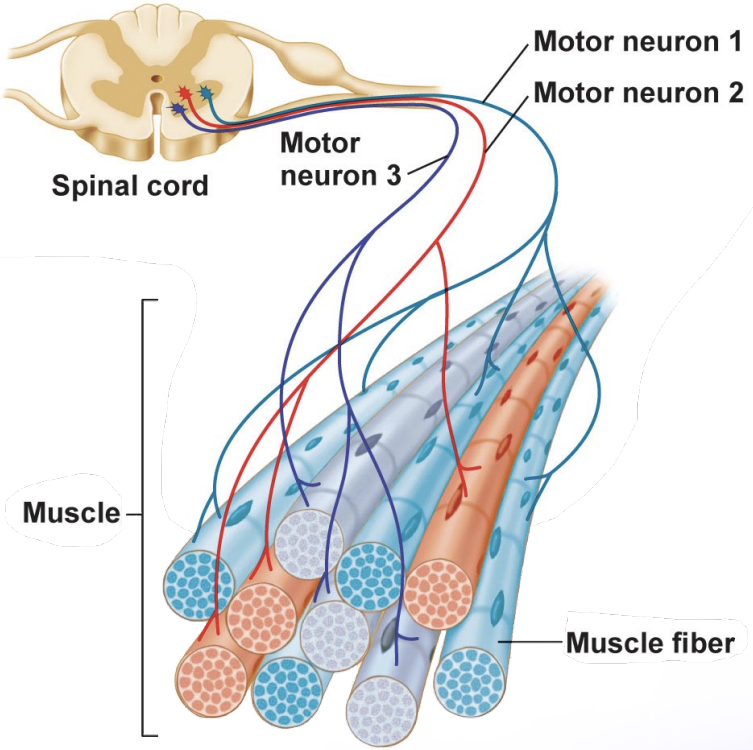




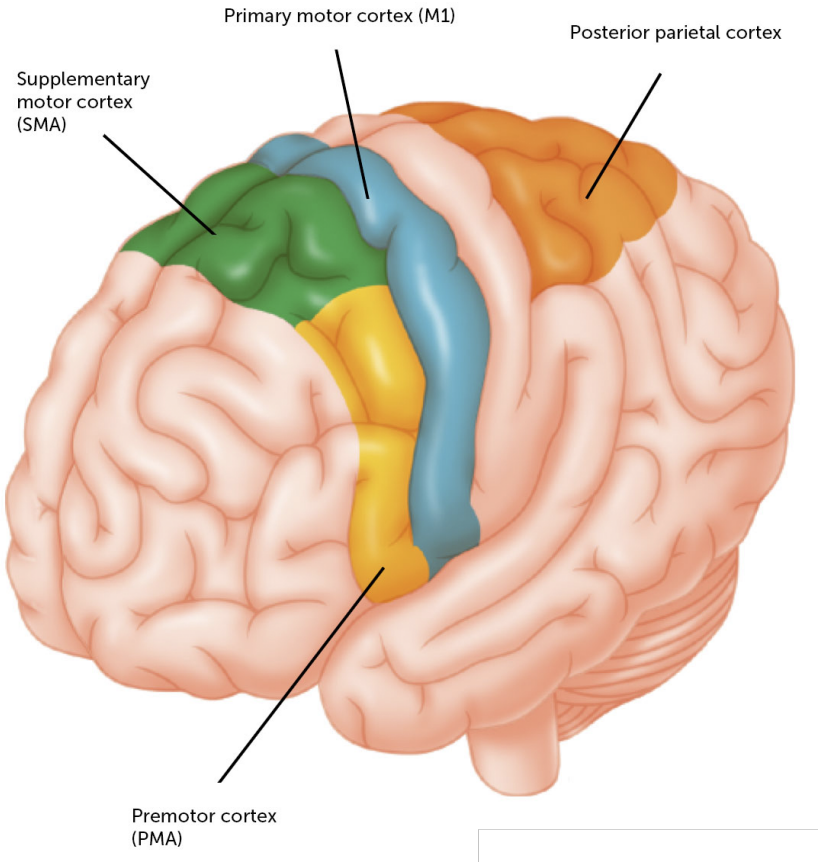
What to Remember from Block 1

All-or-none principle and rate coding…



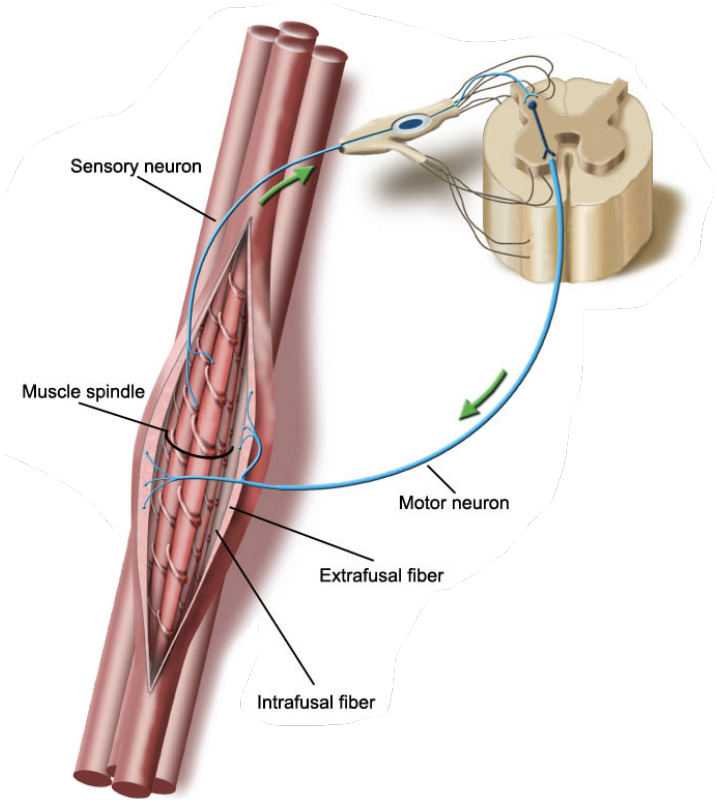


What to Remember from Block 1



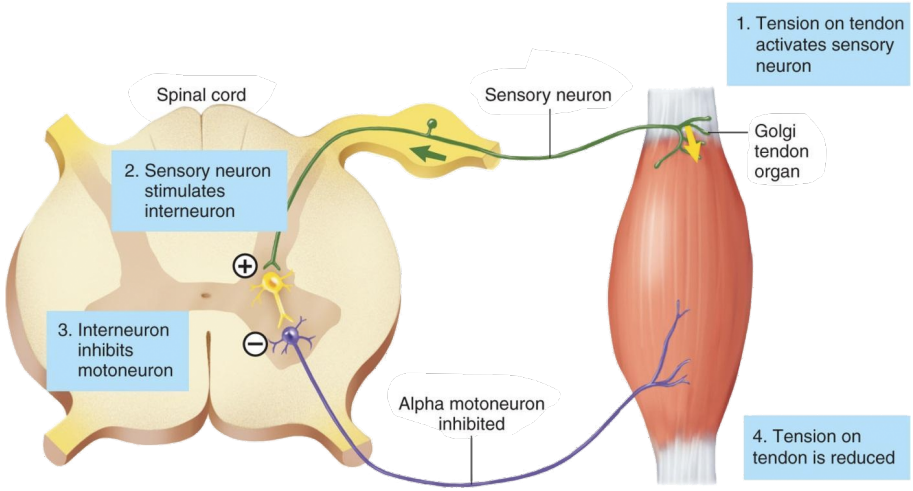
Primary motor cortex.
Alpha motor nerves.
Voluntary contraction.
Extrafusal fibers.

What to Remember from Block 1



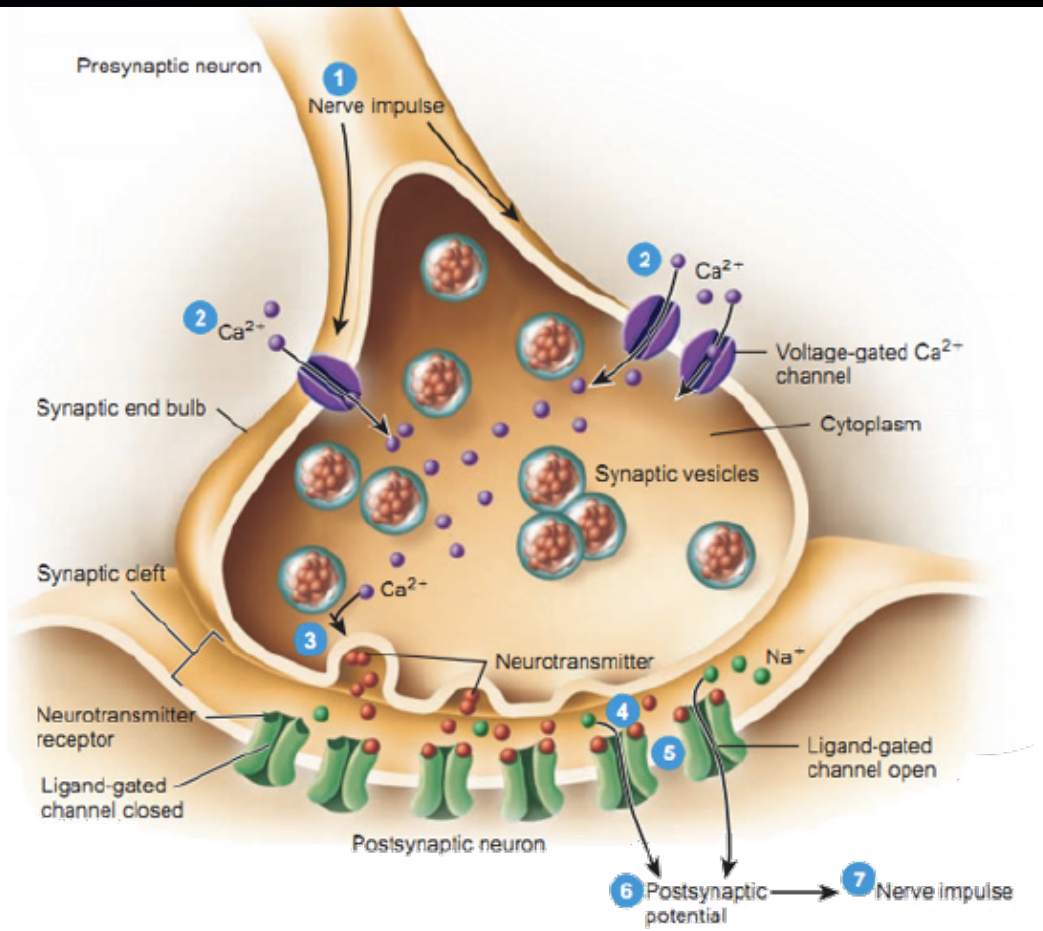
Muscle spindles.
Sense stretch.
Gamma motor nerves.
Involuntary contraction.
Intrafusal fibers.

What to Remember from Block 1



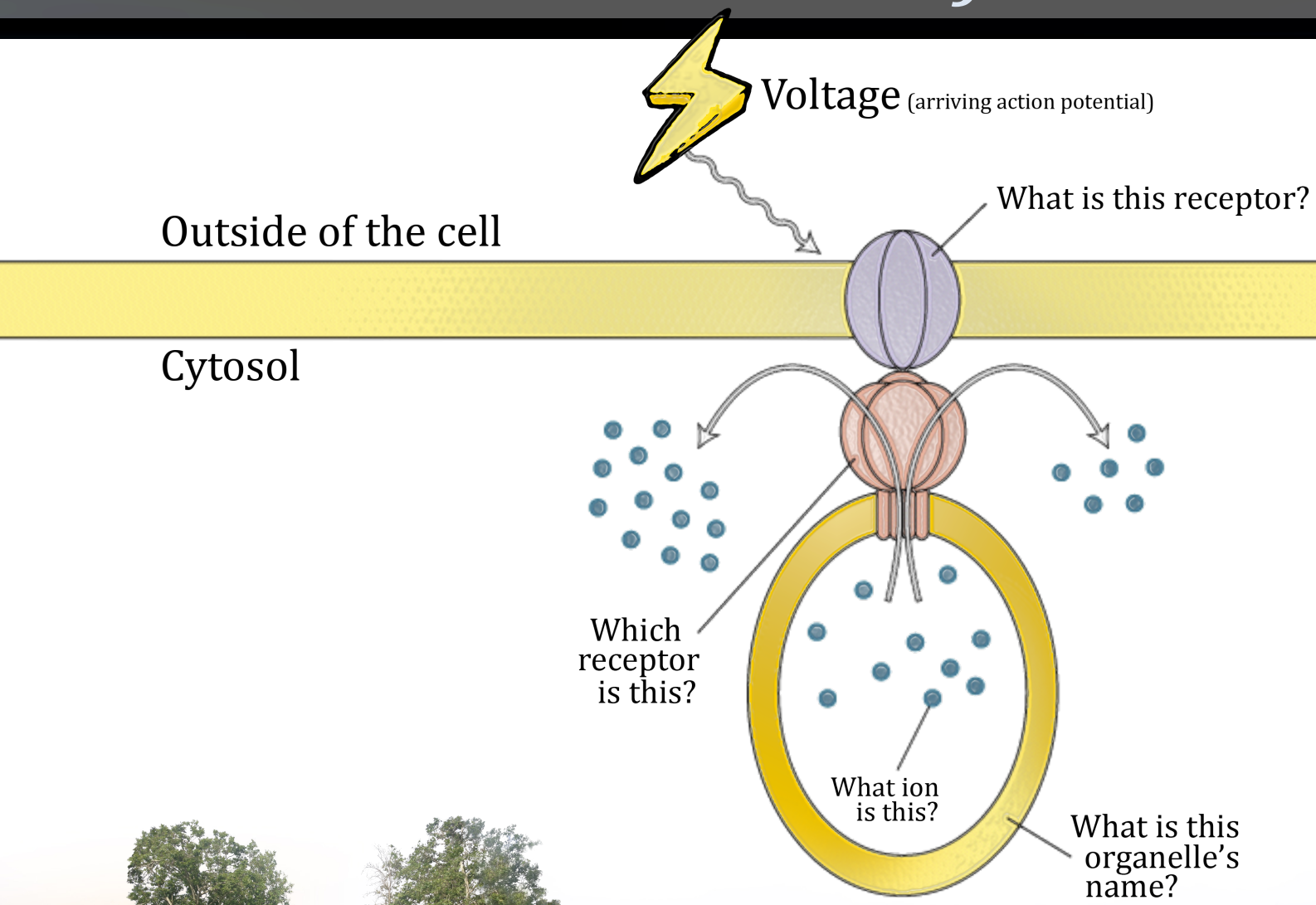
Golgi tendon organ.
Senses load/tension.
Hyperpolarizes alpha motor nerves with glycine.

What to Remember from Block 1



Remember how acetylcholine works and what inhibits it.

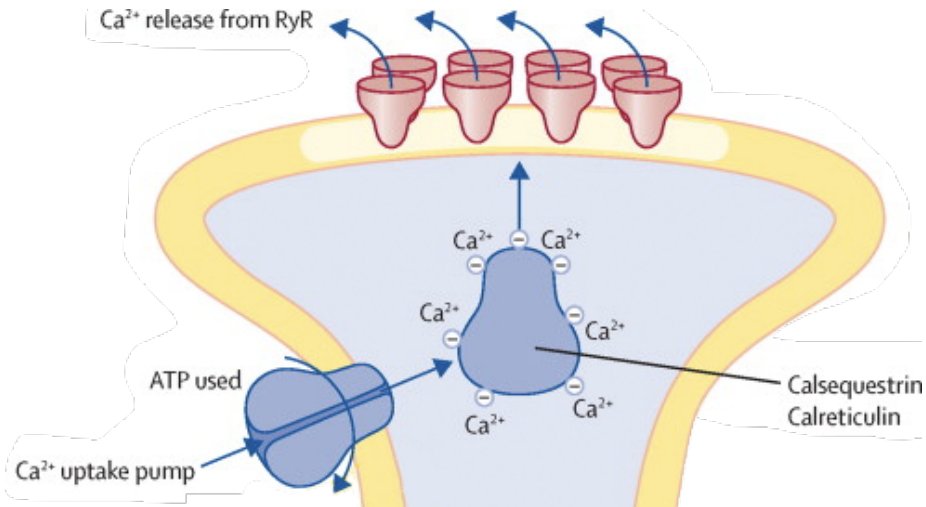
What to Remember from Block 1

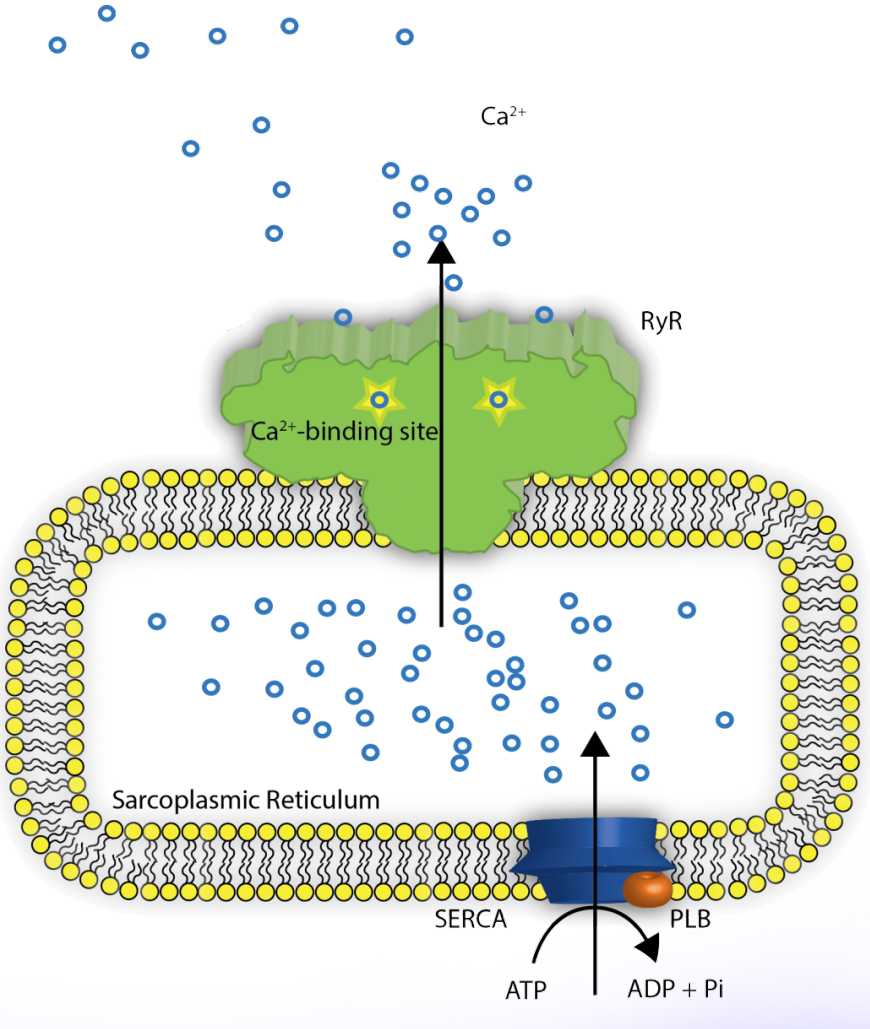


What to Remember from Block 1

DICR vs. **CICR?**

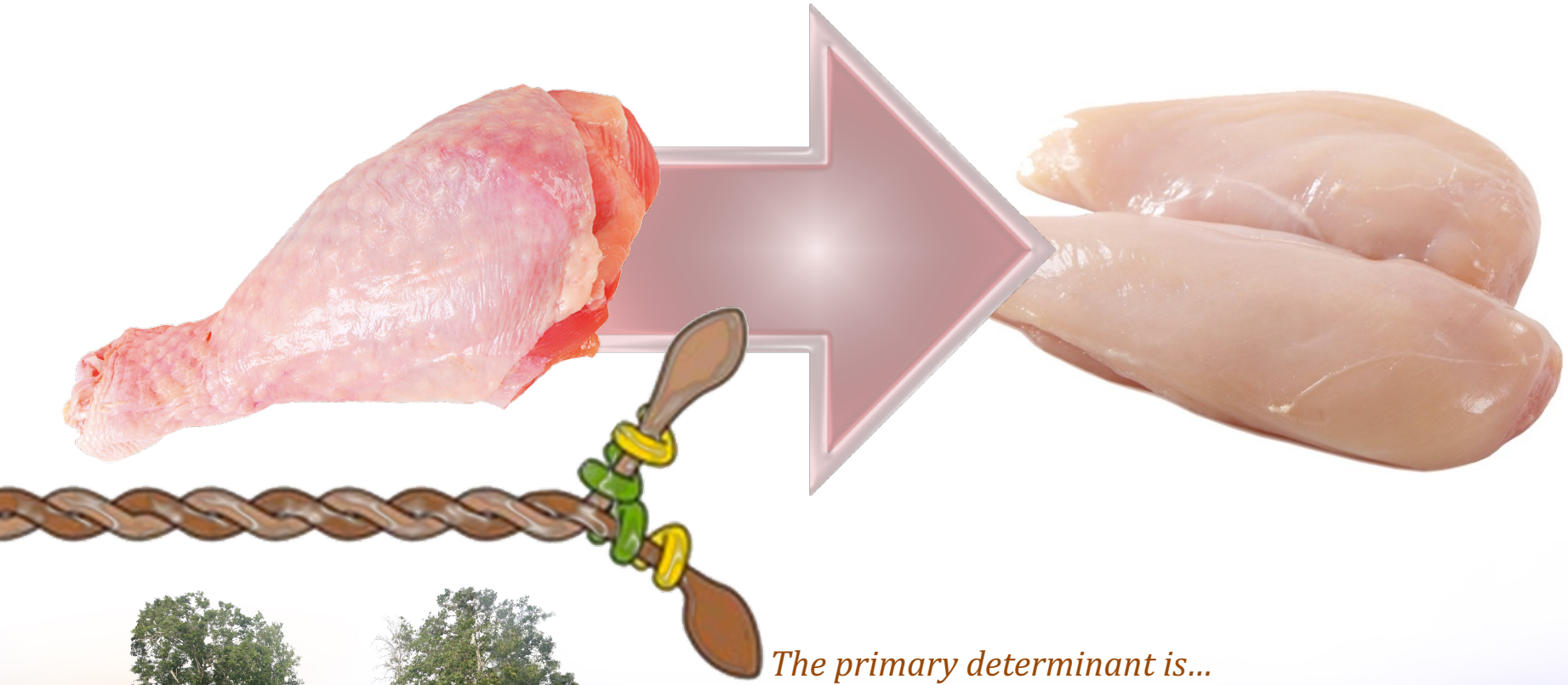
And how is Calcium put away?





What to Remember from Block 1

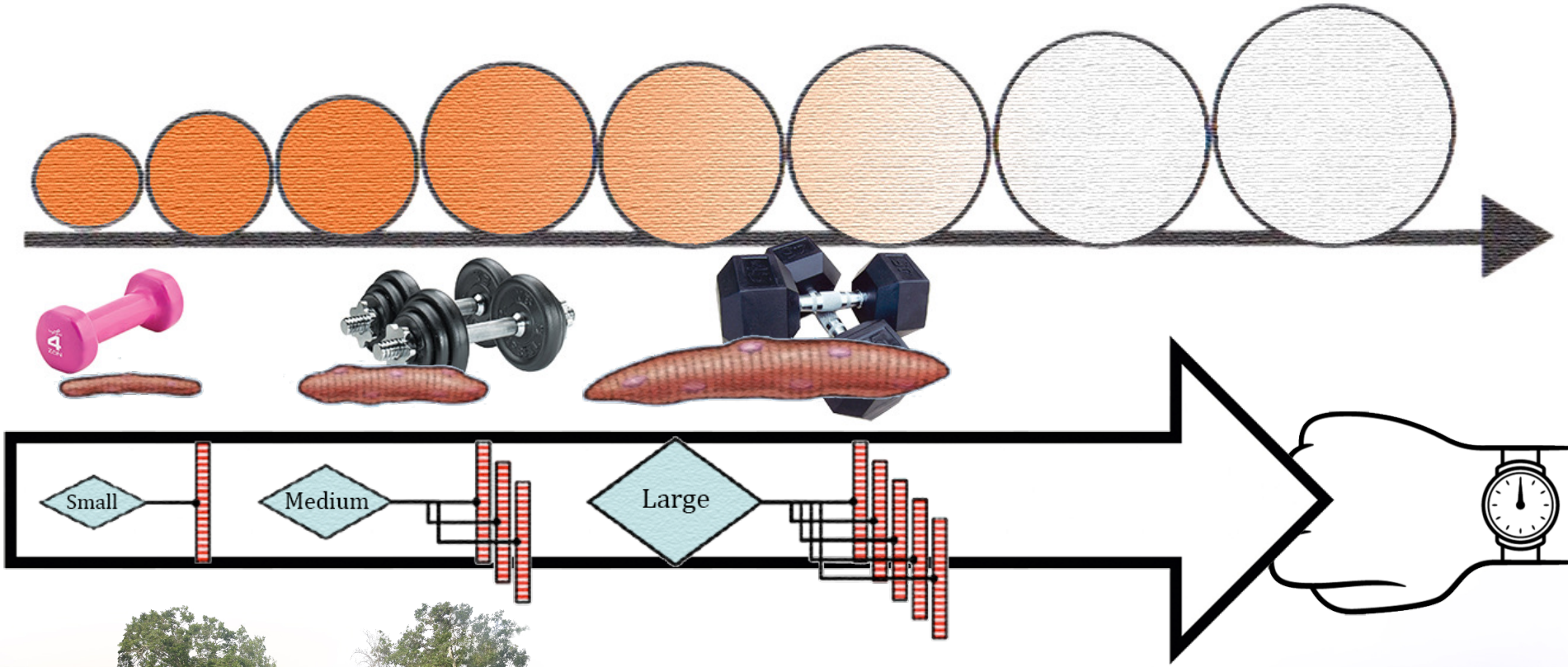
I → Ic → IIc → IIac → IIa → IIax → IIx



The primary determinant is…

What to Remember from Block 1

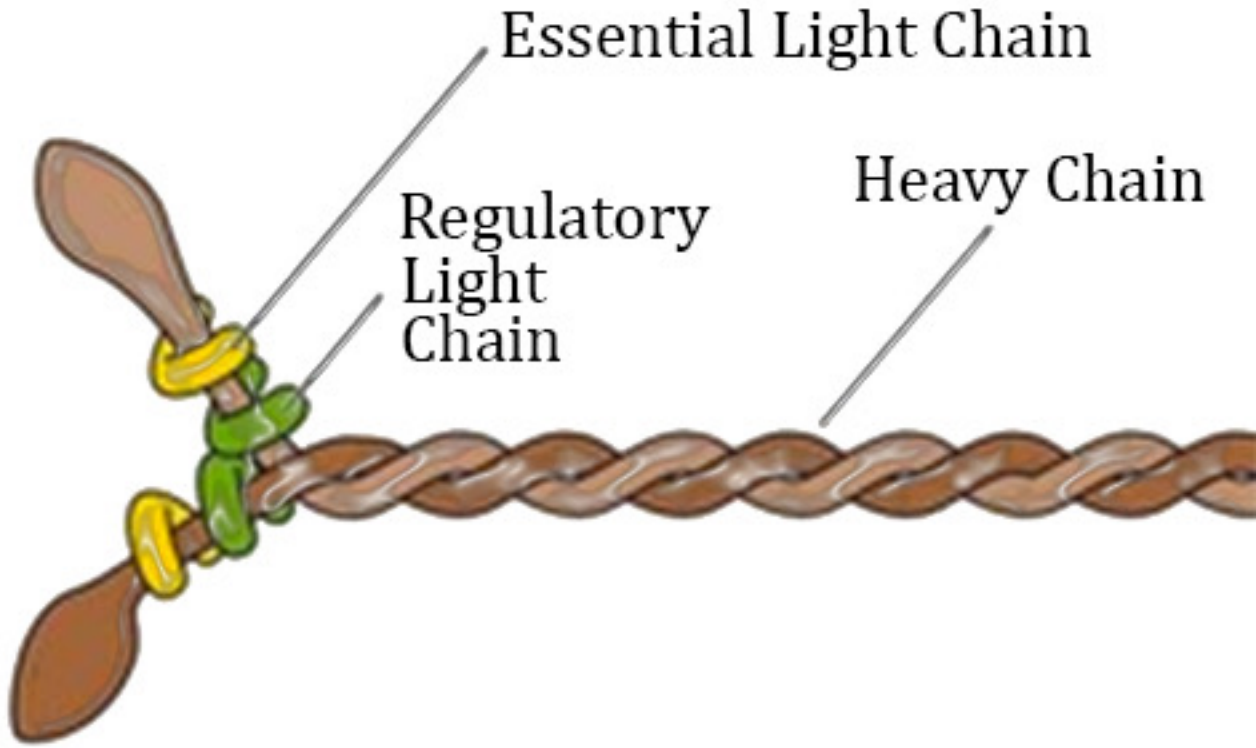
I → Ic → IIc → IIac → IIa → IIax → IIx



What to Remember from Block 1

<u>Myosin heavy chain:</u>
Head and tail of myosin.

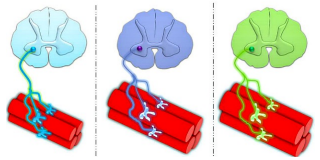
<u>Myosin light chain:</u>
Two per head, they bind heavy chains in neck to the tail.



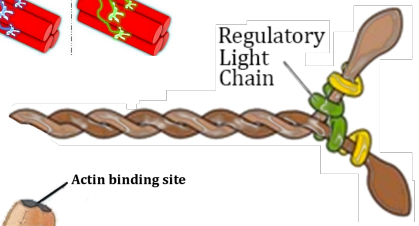
What to Remember from Block 1

Other stuff affects the velocity of muscle shortening as well:

- Alpha motor nerve innervation

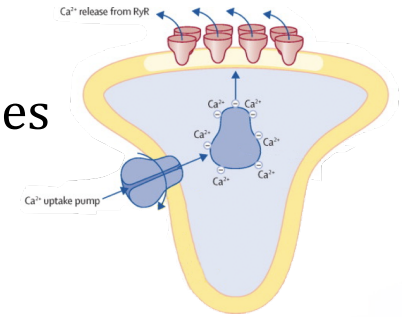


- Myosin regulatory light chain isoform



- Myosin ATPase activity

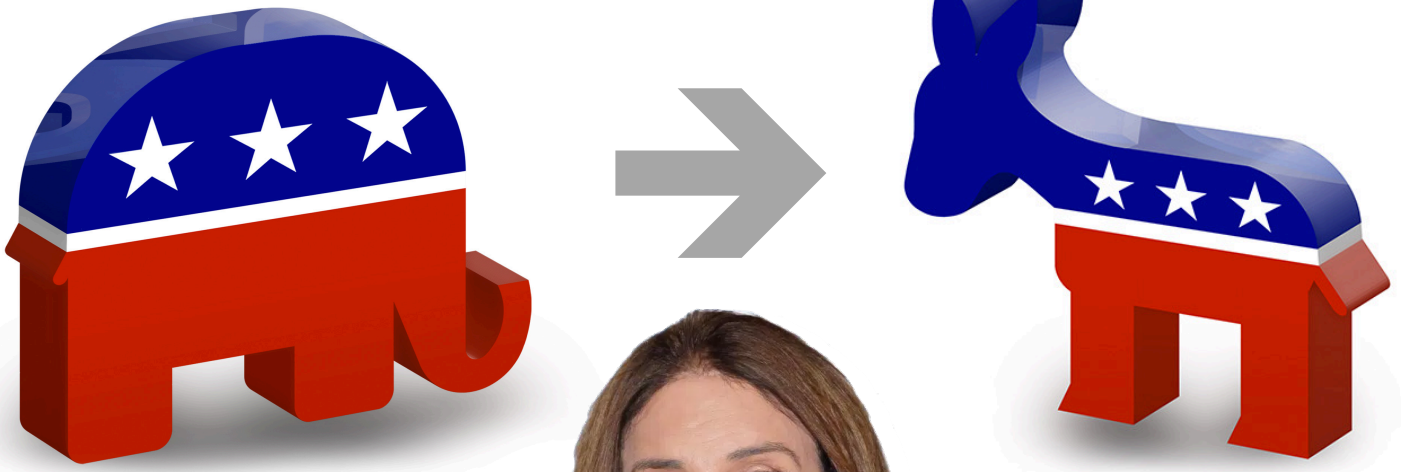
- Sarcoplasmic reticulum concentration and enzymes for calcium release and reuptake



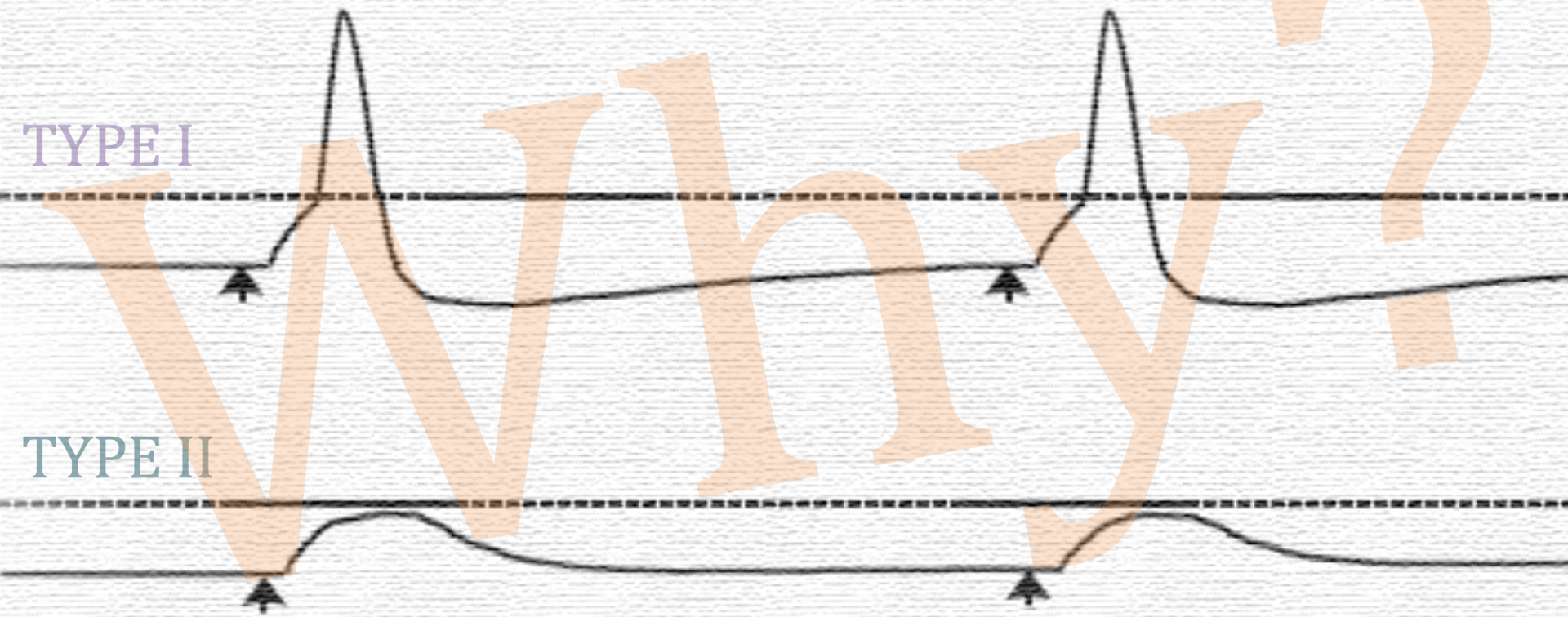
What to Remember from Block 1

Fiber type switching:

I → Ic → IIc → IIac → IIa ← IIax ← IIx



What to Remember from Block 1



Why?

What to Remember from Block 1

The International Journal of Sports Physical Therapy

CLINICAL COMMENTARY

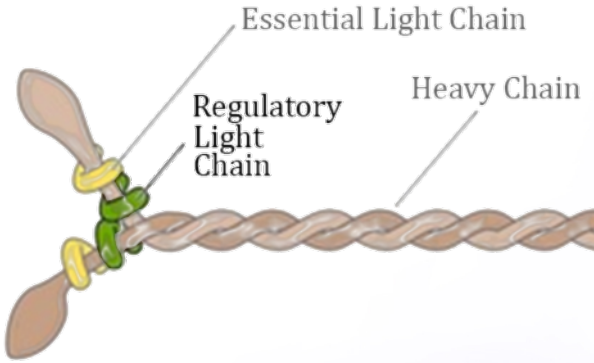
POSTACTIVATION POTENTIATION: AN INTRODUCTION

Daniel Lorenz, DPT, PT, ATC/L, CSCS
Kansas City, Kansas, USA

Volume 6, Number 3 | September 2011

The most important muscle characteristic affecting the magnitude of PAP is fiber type, with the greatest potential for enhanced PAP in muscles with the highest proportion of Type II fibers.[15,19,20] Further, PAP is greater in muscles with the shortest twitch contraction time.[17,19,21,22] Based on muscle fiber type, athletes who perform in maximal intensity activities that depend on Type II muscle fibers (i.e. sprinting, weightlifting, throwing, jumping) would also show the greatest PAP in muscles involved in their sports performance.[23]

Ca^{2+} Ca^{2+} Ca^{2+} Ca^{2+} Ca^{2+} Ca^{2+} Ca^{2+} Ca^{2+}



What to Remember from Block 1

J Appl Physiol 94: 1410–1420, 2003.
First published December 13, 2002; 10.1152/japplphysiol.01183.2001.

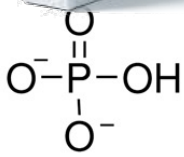
Activation imbalances in lumbar spine muscles in the presence of chronic low back pain

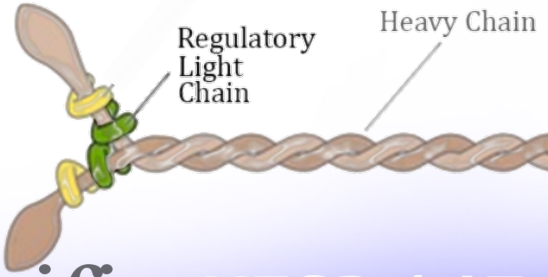
Lars I. E. Oddsson[1] and Carlo J. De Luca[1,2]
[1]*NeuroMuscular Research Center and* [2]*Departmer*
Engineering, Boston University, Boston, Massc

What stuff does the opposite of Post-Activation Potentiation? And how?

Essential Light Chain

Ca^{2+} Ca^{2+} Ca^{2+} Ca^{2+} Ca^{2+} Ca^{2+} Ca^{2+}





What to Remember from Block 1

Effects of Dynamic and Static Stretching on Vertical Jump Performance and Electromyographic Activity

Paul A. Hough,[1] Emma Z. Ross,[2] and Glyn Howatson[1]

[1]School of Human Sciences, St. Mary's University College, Twickenham, United Kingdom; and [2]Centre for Sports Medicine and Human Performance, Brunel University, Uxbridge, United Kingdom

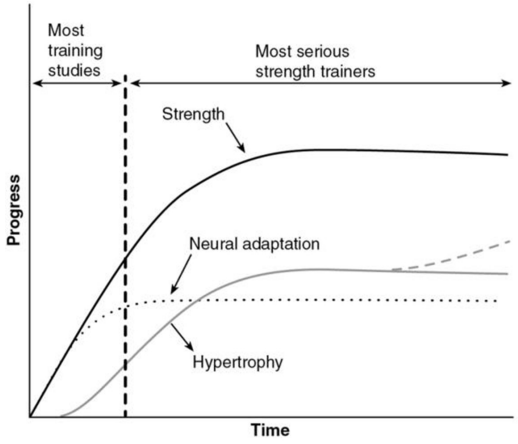
Abstract

Hough, PA, Ross, EZ, and Howatson, G. Effects of dynamic and static stretching on vertical jump performance and electromyographic activity. *J Strength Cond Res* 23(2): 507–512, 2009–The results of previous research have demonstrated that static stretching (SS) can reduce muscular performance and that dynamic stretching (DS) can enhance muscular performance. The purpose of this study was to assess the effects of SS and DS on vertical jump (VJ) performance and electromyographic (EMG) activity of the m. vastus medialis. Eleven healthy men (age 21 ± 2 years) took part in 3 conditions (no stretching [NS], SS, and DS), on separate occasions in a randomized, crossover design. During each condition, measurements of VJ height and EMG activity during the VJ were recorded. A repeated-measures analysis of variance and post hoc analysis indicated that VJ height was significantly less (4.19 ± 4.47%) after SS than NS ($p < 0.05$) and significantly greater (9.44 ± 4.25%) in DS than SS ($p < 0.05$). There was significantly greater EMG amplitude in the DS compared with the SS ($p < 0.05$). The results demonstrated that SS has a negative influence on VJ performance, whereas DS has a positive impact. Increased VJ performance after DS may be attributed to post-activation potentiation, whereas the reduction in VJ performance after SS may be attributable to neurological impairment and a possible alteration in the viscoelastic properties of the muscular tendon unit (MTU). This investigation provides some physiological basis for the inclusion of DS and exclusion of SS in preparation for activities requiring jumping performance.

What stuff does the opposite of Post-Activation Potentiation? And how?

What to Remember from Block 1

What accounts for the initial improvements experienced in strength training?

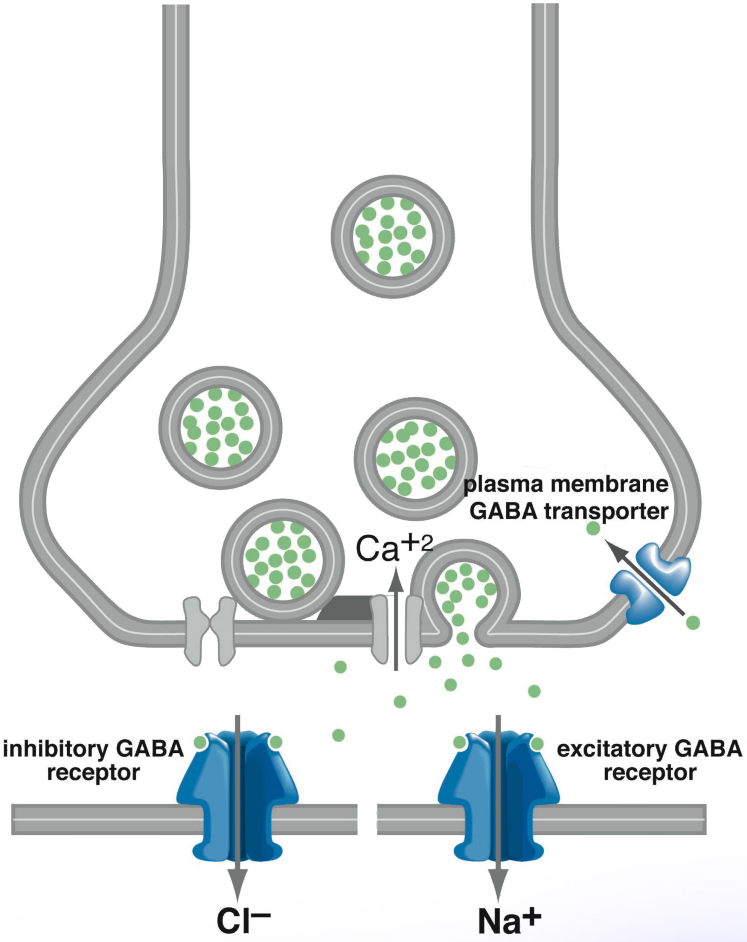


Lots of stuff:
Neurotransmitters (e.g., GABA), agonist-antagonist recruitment activity, withdrawal of inhibition by GTO, rate coding, changes to motor cortex and descending neural tracts, better motor end plate connections, etc.

What to Remember from Block 1

What accounts for the initial improvements experienced in strength training?

GABA: primary inhibitory neurotransmitter in the CNS (in the mature brain; plays excitatory roles in development). Made from glutamate (primary excitatory neurotransmitter in CNS). GABA permits chloride (negative) to enter the nerve or potassium (positive) to exit, which hyperpolarizes that nerve.



What to Remember from Block 1

Aging skeletal muscle:

Journal of Applied Physiology 2010 Jun; 108(6): 1659–1667.

Recruitment and derecruitment characteristics of motor units in a hand muscle of young and old adults

Mark Jesunathadas,[⊠] Adam R. Marmon, James M. Gibb, and Roger M. Enoka

DUE TO A SIGNIFICANT DECLINE in the number of spinal motor neurons that begins around the sixth decade of life (5, 20, 38), muscle fibers that were once innervated by these motor neurons either disintegrate and disappear or are reinnervated by surviving motor neurons (5). The result of these changes is fewer but larger motor units in the muscles of old adults compared with young adults (5, 10, 11). It has been suggested that this remodeling contributes to the decline in force control observed in old adults (1, 17, 24, 39).

What to Remember from Block 1

Aging skeletal muscle:

MUSCLE & NERVE 11:423–432 1988

METHODS FOR ESTIMATING NUMBERS OF MOTOR UNITS IN BICEPS-BRACHIALIS MUSCLES AND LOSSES OF MOTOR UNITS WITH AGING

WILLIAM F. BROWN, MD, FRCP(C), MICHAEL J. STRONG, MD, and ROBERT SNOW, BSc, MSc

Estimates in subjects over 60 years of age were about half those of subjects in the third decade. This overall decline in numbers of motor units works out to about 10–20 motor units per year, or about 1% of the total per year.

What to Remember from Block 1

Habituation vs. Sensitization:

WILLIAM GOLDMAN

"It's too far around," the Count said.

"Not for my whites."

"We'll follow as best we can," the Count said. He stared again at the Fire Swamp. "He must be very desperate, or very frightened, or very stupid, or very brave."

"Very all four I should think," the Prince replied. . . .

WESTLEY LED THE way. Buttercup stayed just behind, and they made, from the outset, very good time. The main thing, she realized, was to forget your childhood dreams, for the Fire Swamp *was* bad, but it wasn't *that* bad. The odor of the escaping gases, which at first seemed almost totally punishing, soon diminished through familiarity. The sudden bursts of flame were easily avoided because, just before they struck, there was a deep kind of popping sound clearly coming from the vicinity where the flames would then appear.

Westley carried his sword in his right hand, his long knife in his left, waiting for the first R.O.U.S., but none appeared. He had cut a very long piece of strong vine and coiled it over one shoulder and was busy working on it as they moved. "What we'll do nce I've got this properly done is," he told her, moving steadily on beneath the giant ees, "we'll attach [illegible] to each other, so that way, no matter what the darkness, 'll be close [illegible] that's more precaution than necessary, because, to tell [illegible] the truth [illegible] ted; this place is bad, all right, but it's not that bad

What to Remember from Block 1

Accommodation:

What to Remember from Block 1

Adaptation:

What to Remember from Block 1

Acclimation vs. Acclimatization:

What to Remember from Block 1

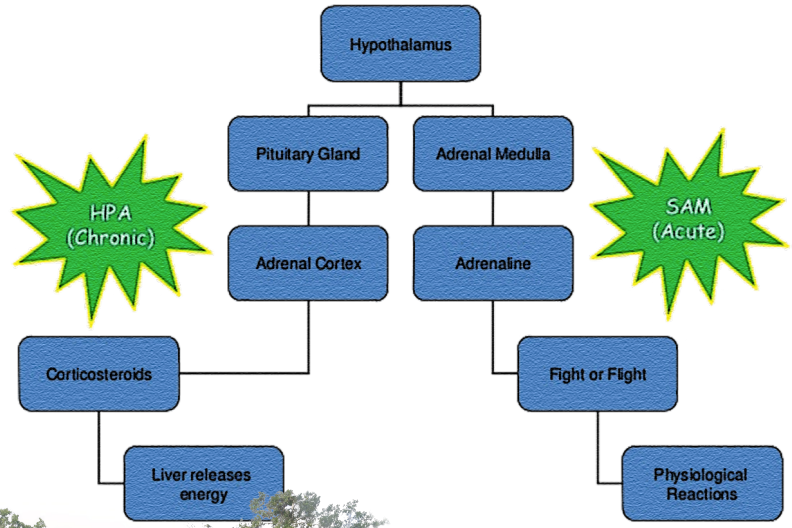
Adaptation is an attempt to improve fitness:

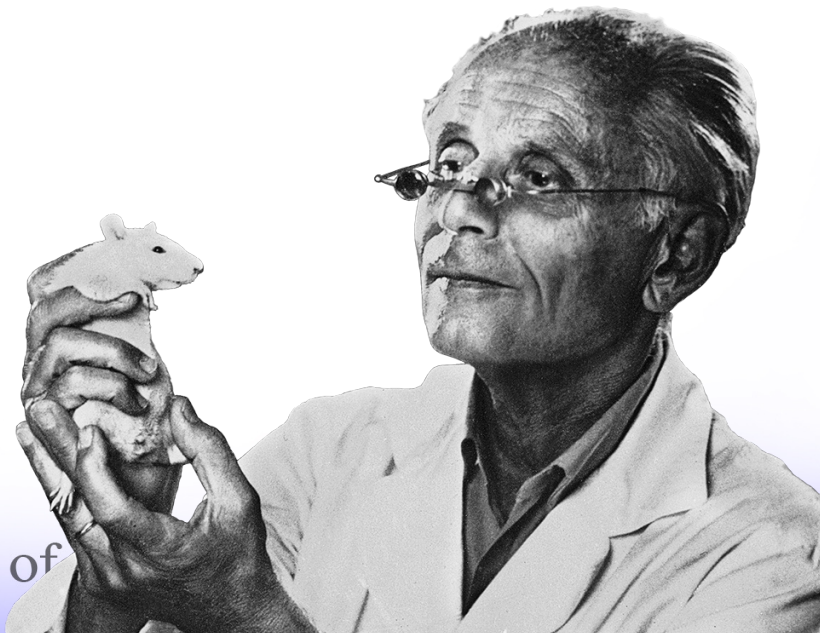
Self preservation, nature's firſt great law,
All the creation, except man, does awe:
'Twas in him fix'd, 'till lying prieſts defac'd
His heav'n-born mind, and nature's tablets raz'd.
Tell me, ye forging crew, what law reveal'd
By God, to Kings, the *jus divinum* ſeal'd?
If to do good, ye *jus divinum* call,
It is the grand prerogative of all:
If to do ill unpuniſh'd be their right,
Such pow'r's not granted the great King of night. December 1675.

What to Remember from Block 1

Hans Selye:

> "We consider the first stage to be the expression of a general alarm of the organism when suddenly confronted with a critical situation, and therefore term it "**general alarm reaction**."

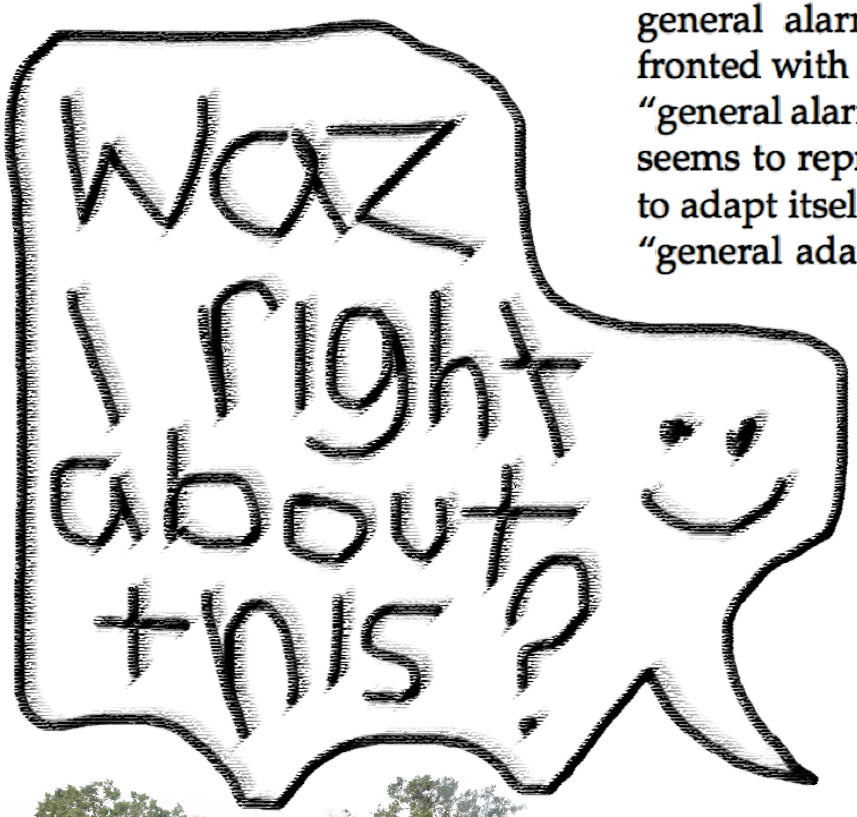




University of

What to Remember from Block 1

Hans Selye:



We consider the first stage to be the expression of a general alarm of the organism when suddenly confronted with a critical situation, and therefore term it the "general alarm reaction." Since the syndrome as a whole seems to represent a generalized effort of the organism to adapt itself to new conditions, it might be termed the "general adaptation syndrome."

General Adaptation Syndrome:
Generalized responses to stresses

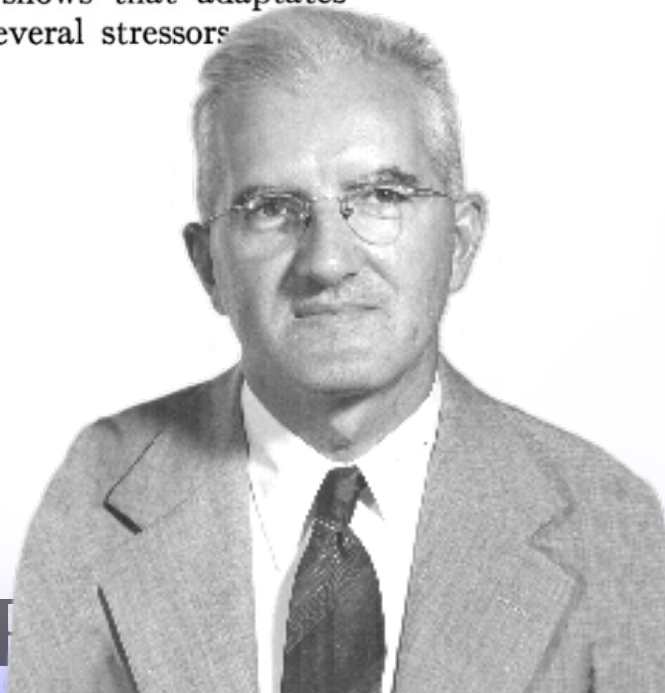
What to Remember from Block 1

Edward Adolph:

Adaptations to altitude, or to cold air, or to other circumstance (stressor) have each been studied separately. The aim of this investigation is to compare adaptations to several diverse stressors in order to see whether any two of them arouse the same modifications. It has been widely supposed that adaptations to various stressors have much in common. I ask, how much? The conclusion will be reached that combinations of modifications differ markedly from one stressor to another.

Adaptates overlap; for instance, adaptation to high altitude is not wholly separate from adaptation to cold air. Nevertheless, the combination of manifestations found is specific to the stressor. Although this possibility has been heretofore recognized (8), it has been neglected in the belief that the general syndrome predominates. The tally of specific instances now shows that adaptates are not the same for several stressors

Adolph EF. (1956). General and specific characteristics of physiological adaptations. *American Journal of Physiology*, 184(1): 18-28.



What to Remember from Block 1

Remember the broccoli

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Nuclear shipyard worker study (1980–1988): a large cohort exposed to low-dose-rate gamma radiation

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Abstract: This paper is a summary of the 1991 Final Report of the Nuclear Shipyard Worker Study (NSWS), a very comprehensive study of occupational radiation exposure in the US. The NSWS compared three cohorts: a high-dose cohort of 27,872 nuclear workers, a low dose cohort of 10,348 workers, and a control cohort of 32,510 unexposed shipyard workers. The cohorts were matched by ages and job categories. Although the NSWS was designed to search for adverse effects of occupational low dose-rate gamma radiation, few risks were found. The high-dose workers demonstrated significantly lower circulatory, respiratory, and all-cause mortality than did unexposed workers.

What to Remember from Block 1

Specificity of adaptation is specific to the profile of the stressor:

Nutrition Facts

Serving Size 1/2 muffins (57g)
Servings Per Container 8

Amount Per Serving

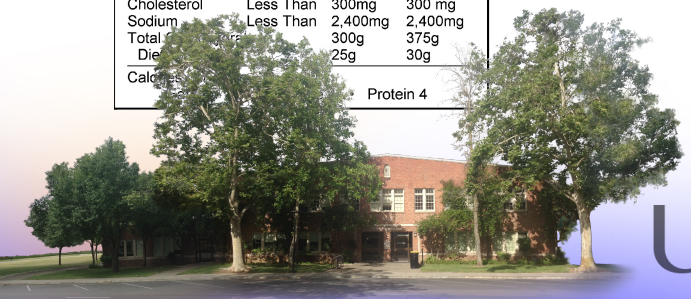
Calories 220	Calories from Fat 110
	% Daily Value*
Total Fat 12g	**19%**
Saturated Fat 2g	**9%**
Trans Fat 0g	
Cholesterol 35mg	**12%**
Sodium 170mg	**7%**
Total Carbohydrate 24g	**8%**
Dietary Fiber 1g	**4%**
Sugars 13g	
Protein 3g	

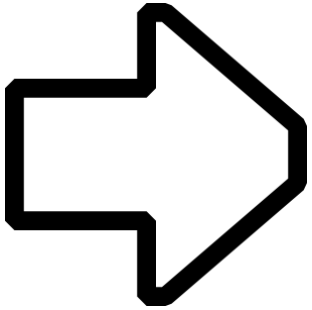
Vitamin A 2%	• Vitamin C 15%
Calcium 2%	• Iron 2%
Thiamin 6%	• Riboflavin 4%
Niacin 2%	• Folate 0%

*Percent Daily Values are based on a 2,000 calorie diet. Your daily values may be higher or lower depending on your calorie needs:

	Calories	2,000	2,500
Total Fat	Less Than	65g	80g
Saturated Fat	Less Than	20g	25g
Cholesterol	Less Than	300mg	300 mg
Sodium	Less Than	2,400mg	2,400mg
Total [illegible]		300g	375g
Die[illegible]		25g	30g

Cal[illegible] • Protein 4





Nutrition Facts

Serving Size 1 Workout (57 min)
Servings Per Container 8

Amount Per Serving

Calories 220	Calories from Fat 110
	% Daily Value*
Type of Load	**19%**
Dynamic (Con/Ecc)	**9%**
Isometric	
Magnitude of Load	**12%**
Rate of Application	**7%**
Direction of Load	**8%**
Compression or Stretching	**4%**
Torsion or Shear	
Duration of Load	

Frequency	• Variability
Location of Load	• Coordination
Range of Motion	• Number of Tissues
Temperature	• Time of Day

*Percent Daily Values are based on a 2,000 calorie diet. Your daily values may be higher or lower depending on your calorie needs:

	Calories	2,000	2,500
Total Fat	Less Than	65g	80g
Saturated Fat	Less Than	20g	25g
Cholesterol	Less Than	300mg	300 mg
Sodium	Less Than	2,400mg	2,400mg
Total Carbohydrate		300g	375g
Dietary Fiber		25g	30g

Calories per gram:
Fat 9 • Carbohydrate 4 • Protein 4

What to Remember from Block 1

Specificity of mechanotransduction:

Perhaps diet contributes. Perhaps swimming in shallow tanks contributes, as orcas don't experience the high static fluid pressure or simulated microgravity found at depth. **Most importantly, the tissues experience repeated exposure to a unique mechanical stress: tons of swimming in a counter-clockwise circle.**

What to Remember from Block 1

Specificity of Adaptation & Specificity of Degeneration

Your physical form becomes the embodiment of what it endures.

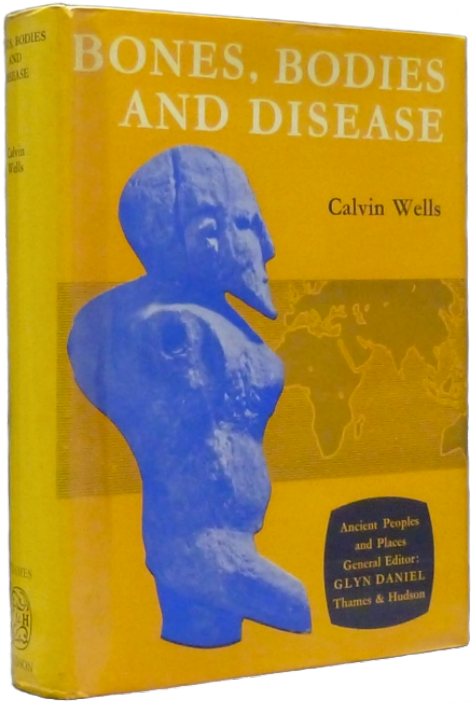
Keep moving: body attempts to retain the ability of movement.

Stop moving: future moving privileges are (slowly) revoked.

***Weird* moving:** the cause of most musculoskeletal pathology.

What to Remember from Block 1

Bodily problems are seldom the result of "physiology gone wild"



"The pattern of disease or injury that affects any group of people is never a matter of chance. It is invariably the expression of stresses and strains to which they were exposed, a response to everything in their environment and behavior."

In other words:
Your physiology isn't often naughty,
issuing illness through misbehavior.

What to Remember from Block 1

Proceedings of the Nutrition Society (2004), **63**, 331–335
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Mechanotransduction and the regulation of protein synthesis in skeletal muscle

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Specificity in mechanotransduction

In addition to being able to sense mechanical stimuli, it also appears that muscle cells can differentiate between different types of mechanical forces. For example, in skeletal muscle chronic longitudinal stretch produces growth in length but not cross-section (sarcomere deposition in series to the long axes), while chronic functional overload produces cross-sectional growth with no changes in length (sarcomere deposition is parallel to the long axes).

GOOD LUCK!

Study hard.
Get lots of sleep.
Perform well.