BIOM 5301 Biomechanics of Skeletal Systems, Motion and Tissue

Winter 2013

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Contents

Chapter 1

Course Outline[1]

Calendar Description Biomechanics of tissues central to human movement: bones, joints, muscles, nerves, ligaments, and tendons. Integration of tissues into limbs Analysis of limb movement and control. Injury, treatment, healing, prosthetics.

Lectures: Times and Location

- Monday, 11:35 pm - 2:25 pm, PA 112

Topics

Emphasis will be placed on approaching the following subject areas from the point of view of the engineer.

Structure:	Cells
	Bone
	Joints
	Ligaments
Actuation:	Tendon
	Muscle
Sensory:	Nerves
	Sense organs
Control:	Spinal Cord and Reflexes
	Brain

[1] This is the final course outline for this course and replaces any Preliminary Course Outlines.

Evaluation

Class Presentations	20%
Paper Review Report	20%
Term Project	30%
Examination	30%
Total	100%

Class Presentations Each student will give approximately five short presentations (**3 to 5 minutes**) during the term. Topics include a brief review of one (or maybe two) related journal papers or an analysis of a specific injury or condition.

Paper Review Each student will submit a written summary (**about 5 pages in length**) of the results and importance of two or three related and recent research papers within the topic areas of the course. **Due: Monday, March 4, 2013.**

Term Project The term project will involve selecting an injury that occurs to the arm or leg and writing a report on the mechanics of the injury, the biomechanics of the injured tissues, and the treatment for the injury. The report is intended to be approximately 15 pages in length. **Due: Monday, April 8, 2013**.

Academic Accomodation

You may need special arrangements to meet your academic obligations during the term. For an accommodation request the processes are as follows:

Pregnancy obligation: write to me with any requests for academic accommodation during the first two weeks of class, or as soon as possible after the need for accommodation is known to exist. For more details visit the Equity Services website .

Religious obligation: write to me with any requests for academic accommodation during the first two weeks of class, or as soon as possible after the need for accommodation is known to exist. For more details visit the Equity Services website .

Academic Accommodations for Students with Disabilities The Paul Menton Centre for Students with Disabilities (PMC) provides services to students with Learning Disabilities (LD), psychiatric/mental health disabilities, Attention Deficit Hyperactivity Disorder (ADHD), Autism Spectrum Disorders (ASD), chronic medical conditions, and impairments in mobility, hearing, and vision. If you have a disability requiring academic accommodations in this course, please contact PMC at 613-520-6608 or pmc@carleton.ca for a formal evaluation. If you are already registered with the PMC, contact your PMC coordinator to send me your Letter of Accommodation at the beginning of the term, and no later than two weeks before the first in-class scheduled test or exam requiring accommodation (if applicable). After requesting accommodation from PMC, meet with me to ensure accommodation arrangements are made. Please consult the PMC website for the deadline to request accommodations for the formally-scheduled exam (if applicable).

Required Textbooks

Access to the following books will be required for this course. They are all currently available as e-books through the Carleton library. The relevant chapters can be downloaded as pdf files which you can print if you wish. Only selected chapters from these references will be used - there is no need or usefulness in printing all of them.

- C. Ethier and C Simmons, *Introductory Biomechanics: From Cells to Organisms*, Cambridge University Press, 2007. [http://proxy.library.carleton.ca/login?url=http://www.knovel.com/web/portal/browse/display?_EXT_KNOVEL_DISPLAY_bookid=2341]

Suggested Textbooks

- M. Nordin, and V.H.Frankel, *Basic Biomechanics of the Musculoskeletal System*, 3rd Edition, Lippincott Williams & Wilkins, 2000.

Other Suggested Texts and References

- M. Kutz, *Standard Handbook of Biomedical Engineering and Design* McGraw-Hill, 2003.
- M. Latash, *Neurophysiological Basis of Movement, 2nd Edition*, Human Kinetics, 2008.
- Pamela K. Levangie and Cynthia C. Norkin., *Joint Structure and Function: A Comprehensive Analysis*, 4th Edition, FA Davis, 2005.
- Andris Freivalds, *Biomechanics of the Upper Limbs: Mechanics, Modelling and Musculoskeletal Injuries*, CRC Press, 2004
- W. Kapit, R. I. Macey, and E. Meisami, *The Physiology Coloring Book, 2nd edition.* Benjamin-Cummings, 2000
- W. Kapit, and L. M. Elson, *The Anatomy Coloring Book, 3rd edition.* Benjamin-Cummings, 2002
- R. L. Lieber, *Skeletal Muscle Structure and Function*, Williams and Wilkins, 1992
- E. N. Marieb *Human Anatomy and Physiology, 3rd edition.*, Benjamin-Cummings, 1995
- T. A. McMahon, *Muscles, Reflexes and Locomotion*, Princeton University Press, 1984
- A. M. Nahum and J. W. Melvin, eds, *Accidental Injury: Biomechanics and Prevention* Springer-Verlag, 1993
- B. M. Nigg and W. Herzog, *Biomechanics of the Musculo-sleletal System, 2nd edition*, John Wiley, 1999
- D. A. Winter, *Biomechanics, 3rd Edition*, Wiley, 2005
- Yokochi et al, *Photographic Anatomy of the Human Body, Third Edition*, Igaku-Shoin, 1989

Chapter 2

Lecture Notes

2.1 A Mechanical Review of Cells and Cell Structures.

Basic Reference Material

Sections 2.1 to 2.4 and Figure 2.58 from *Introductory Biomechanics: From Cells to Organisms*[1]

External Links

Cytoskeleton

Extracellular Matrix

Histology Review

Cell Junctions

2.2 Bone.

Basic Reference Material

Sections 9.1 to 9.6 from *Introductory Biomechanics: From Cells to Organisms*[1]

Additional Information contained in the Lecture Slides

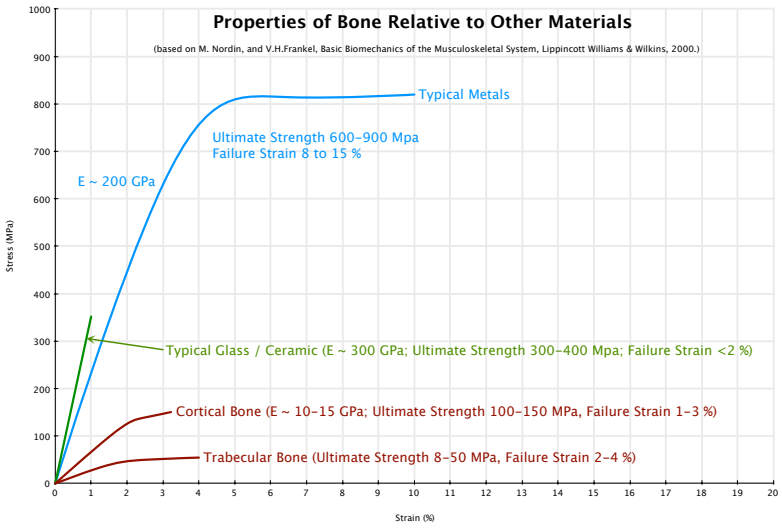
1. **Bones** See Figure 9.1 from *Introductory Biomechanics: From Cells to Organisms*[1]
2. **Bone - Distribution** See Table 9.1 from *Introductory Biomechanics: From Cells to Organisms*[1]
3. **Bone Functions** Bone is an *organ* that:
   - Protection of vital organs
   - Movement and Support
   - Mineral homeostasis (mineral storage)
   - Hematopoiesis (blood cell formation)
4. Bone is:
   - hard (only harder materials in the body are dentin and enamel in the teeth)
   - highly vascular, many nerves
   - metabolically active
5. Bone tissue is:
   - connective tissue (collagen, proteoglycans, ...)
   - high concentration of inorganic material (calcium and phosphate crystals - hypoxyapatite)
   - cells (osteocytes (mature), osteoblasts (bone-forming), osteoclasts(resorb bone))
6. **Bone - Composition** See Table 9.2 from *Introductory Biomechanics: From Cells to Organisms*[1]
7. **Four Different Bone Classifications in the Body**
   - Long bones (hollow shaft, e.g. humerus)
   - Short bones (e.g. carpal bones, patella)
   - Flat Bones (e.g. shoulder blade)
   - Irregular Bones (e.g. vertebrae, pelvis)
8. Image: FlatBone: Scapula
9. Image: Irrregular Bone: Vertebra

10. Image: Short Bone: Wrist (Carpal) Bones
11. Image: Long Bone.
12. **Two types of bone:**
    - Cortical or compact bone
    - Cancellous or spongy or trabecular bone

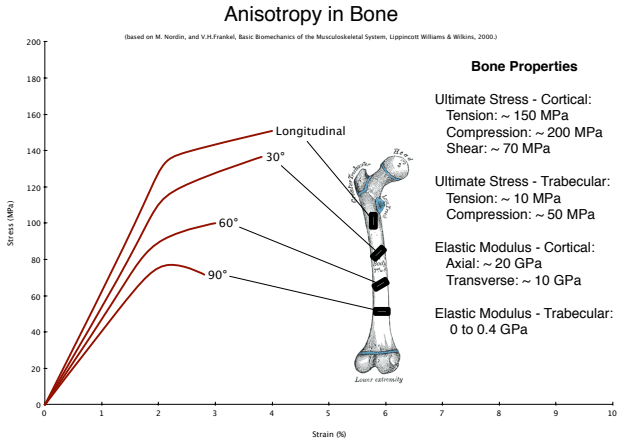
    These have similar chemistry, structure and molecular content but different mechanical and metabolic functions.
13. Image: Long Bone.
14. Image: Structure of a Long bone
15. Image: Bone types in a long bone: Compact and Spongy Bone Bone from: cancellous bone. Encyclopdia Britannica Online[7]
16. **Bone: Motivation for Relationship between Geometry and Internal Stress** See Figure 1.5 from *Introductory Biomechanics: From Cells to Organisms*[1]
17. **Bone** See Figure 9.4 from *Introductory Biomechanics: From Cells to Organisms*[1]
18. **Bone** See Figure 9.2 from *Introductory Biomechanics: From Cells to Organisms*[1]
19. **Bone Organization** See Figure 1 from THE MATERIAL BONE: Structure-Mechanical Function Relations[6]
20. **Bone Organization** See Figure 4 from Biological Composites[5]
21. **Bone** See Figures 9.3 and 9.5 from *Introductory Biomechanics: From Cells to Organisms*[1]
22. **Bone Microstructure:**[3, 4]
    - Woven Bone
    - Lamellar Bone (mature, regularly oriented collagen matrix)
      - Primary (lamellar, plexiform, primary osteons)
      - Secondary
23. **Woven Bone:**[3, 4]
    - randomly oriented collagen fibers (structurally disorganized)
    - immature (develops during growth and healing)
    - mineralizes quickly, high cell density - spreads quickly
    - can be synthesized in regions where no bone exists
    - reduced mechanical properties
24. **Lamellar Bone:Primary Lamellar Bone**
    - Forms on existing surfaces (bone or cartilage)

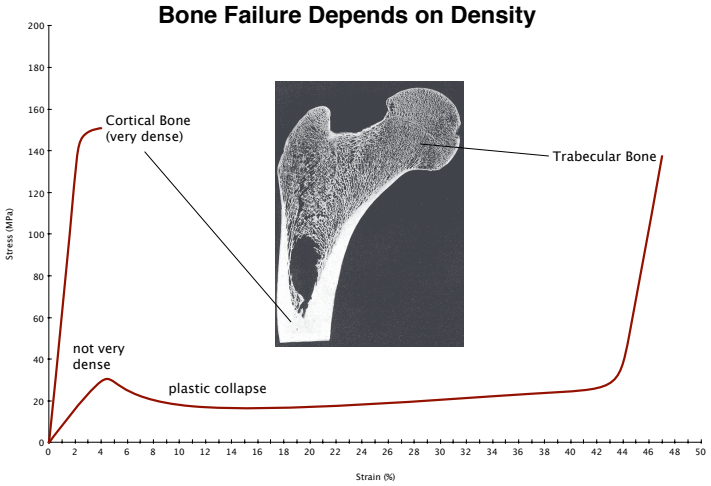
- Circumferential lamellae
- trabeculae in epiphysis of long bones are largely Primary bone
- Primary Osteons: grow around vascular channels (no cement lines)

25. **Lamellar Bone:Secondary Lamellar Bone**[3, 4]
    - Secondary bone is remodelled from primary bone (resorption and deposition)
    - Larger vascular channel (Haversian Canal) than in primary osteons
    - More lamellae than primary osteons
26. **Bone - Composition** See Table 9.6 from *Introductory Biomechanics: From Cells to Organisms*[1]
27. **Geometry - Scale**[3]
    - Osteons range in diameter from 200 to 300 μm
    - Haversian System is 300 microns in diameter and 3 to 5 mm long
    - Cement lines are 1 - 5 μm thick
28. Image: See Figure 6.1 in De Santis et al, *Mechanical Properties of Human Mineralized Connective Tissues* in MODELING OF BIOLOGICAL MATERIALS, 2007
29. Bone is fairly well modelled as a rigid, but anisotropic material.
30. Image: See Figure 1 in Dunlop and Fratzl, Biological Composites, Annual Review of Materials Research, Vol. 40: 1-24
31. **Bone Properties** See Table 9.3 from *Introductory Biomechanics: From Cells to Organisms*[1]
32. **Bone Properties**



33. **Bone Properties - Vary with Location** See Table 9.4 from *Introductory Biomechanics: From Cells to Organisms*[1]





34. **Bone: Properties dependance on density** See Figures 9.6, 9.7 and 9.8 from *Introductory Biomechanics: From Cells to Organisms*[1]

35. Failure strain depends on Bone Type [2]

    Cancellous Bone: Failure when strain exceeds up to 50 %.

Cortical Bone: Failure when strain exceeds 1.5 to 2.0 %.

36. **Data on Bone Properties** Data is available from a large number of sources grouped by bone type , for example, see Accidental Injury: Biomechanics and Prevention[4]

37. **Bone Failure - Cracks** See Figures 9.9, 9.10, 9.11 and 9.12 from *Introductory Biomechanics: From Cells to Organisms*[1]

38. **Bone Failure - Osteons** See Figures 9.13 and 9.14 from *Introductory Biomechanics: From Cells to Organisms*[1]

39. **Bone - Fracture Mechanics** See Figure 9.15 and Table 9.5from *Introductory Biomechanics: From Cells to Organisms*[1]

40. Image: Long Bone Fracture

41. **Failure Modes - Compression** See Figures 2-13 and 2-20 from Nordin and Frankel[2]

42. **Failure Modes - Tension** See Figures 2-12 and 2-18 from Nordin and Frankel[2]

43. **Failure Mode - Bending** Bone is stronger in compression - bending failure initiates on the side under tensile stress. Bending failure of a femur. See Figure 2-28 from Nordin and Frankel[2]

44. **Failure Mode - Torsion** The initial failure is due to a shear load followed by a failure along the plane of maximum tensile stress. Image: Torsional failure of a femur.

45. **Failure Mode - Fatigue** Resistance to total bone failure from local compressive fatigue failure is less than that from local tensile compressive fatigue failure. See Figure 2-38 from Nordin and Frankel[2] See Figure 9.16, 9.17 and 9.18 from *Introductory Biomechanics: From Cells to Organisms*[1]

46. **Bone - Factors that Influence Properties - Effects of age:**[2]
With age:
    - Bone mass decreases (may loose up to 50 % of trabecular bone and 25 % of cortical bone).
    - Ultimate stress remains unchanged.
    - Failure strain may be as low as 50 % of that of a young bone.
    - Reduced energy absorption capacity and greater brittleness.

47. **Growth and Remodelling** Wolff's Law: "the remodeling of bone is influenced and modulated by mechanical stress." Examples: space flight; fracture fixation plates. [2]

48. **Growth and Remodelling** Wolff's Law: "the remodeling of bone is influenced and modulated by mechanical stress." Examples: space flight; fracture fixation plates. [2]

49. **Growth and Remodelling** See Figure 6-10 from Marieb[8]

50. Image: **Bone - Remodelling** See Figure 9.20 from *Introductory Biomechanics: From Cells to Organisms*[1]

51. Image: **Bone Growth** Image: Bone Growth
52. Image: **Bone Growth** Image: Bone Growth
53. Image: **Healing and Repair** Bone Healing - Callus
54. Image: Bone Healing

Other External Links

1. Youtube Video: Introduction to Bone Biology

2.3 Tendons and Ligaments.

Basic Reference Material

Sections 9.7 to 9.10 from *Introductory Biomechanics: From Cells to Organisms*[1]

Additional Information contained in the Lecture Slides

1. See Figure 6-10 from Marieb[8]
2. **Connective Tissue - Cell Types** Main Cell Types:

   fibro*blast* immature, undifferentiated cells that secrete the ground substance and fibers (indicated with the suffix *blast*, others include osteoblast and chondroblast)

   fibro*cyte* mature cells that maintain the health of a matrix; they revert to the immature form with an injury

   Other Cells in the Matrix:

   - White blood cells
   - Fat cells
   - Mast cells
   - Macrophages
   - Plasma cells

3. **Ground Substance** The ground substance is amorphous and fills the space between the cells and fibers.

   It contains:

   Cell Adhesion Proteins *fibronectin*, *anchorin* and *laminin* serve as "glue"

   Protoglycans protein core with *glycosaminoglycans* attached; they intertwine and trap water varying in consistency from fluid to stiff (less than 1 % of dry weight in ligament)

Interstitial Fluids medium through which substances defuse from capillaries to the cells, diffusion is hindered by the fibers.

4. **Fibers** There are three primary fiber types in connective tissue:

   Collagen fibers spontaneously assemble from collagen molecules secreted by cells — tough and strong (stronger than steel !)

   Elastic fibers formed from elastin a coiled molecule; there presence gives a matrix flexibility

   Reticular Fibers fine fibers similar to collagen fibers in chemistry that form a supportive matrix

5. **Collagen**

   - Collagen makes up approximately one third of the protein in the body.
   - Type 1 Collagen is by far the most common
   - Each molecule consists of three polypeptide chains of about 100 amino acids
   - A collegen molecule is about 280 nm long and has a diameter of 1.5 nm

6. **Collagen microstructure** See Figures 4-1 and 4-2 from Nordin and Frankel[2]

7. **Collagen Fibers**[2]

   - cross-linking becomes more stable and irreducible over time
   - fibers are 1 - 20 μm in diameter; many cm long
   - may last through an entire life-time, replaced more frequently in the young and where there is an injury

8. **Elastic Fibers**

   - composed of elastin.
   - probably assists collagen fibers in elastic recovery and, perhaps, in protecting collagen.
   - virtually absent in tendon and ligaments in the extremities
   - in some flexible ligaments the ratio of elastic to collagen fibers may be 2:1 (e.g. in the *ligaments flava* that support the spine)

9. **Tendon and Ligament Anaytomy** See Figures 4-17 and 4-10 from *Photographic Anatomy of the Human Body, Third Edition*[9]

10. **Tendons**

    - attach muscle to bone
    - transmits high tensile loads with minimal energy loss
    - act to change mechanical advantage

11. **Ligaments**

    - attach articulating bones (bone-to-bone connection)

- guide joint motion
- maintain joint congruency and stability
- may serve in a sensory role

12. **Composition** Tendons and ligaments contain:

Component	Ligament	Tendon
Cellular Material: Fibroblast	20%	20%
Extracellular Matrix:	80%	80%
Water	60-80%	60-80%
Solids:	20-40%	20-40%
Collagen:	70-80 %	slightly higher
Type 1	90%	95-99 %
Type 3	10 %	1-5 %
Ground Substance	20-30 %	slightly lower

13. **Tendon Structure** See Figure 9.24 from *Introductory Biomechanics: From Cells to Organisms*[1]

14. **Collagen fibers are arranged differently in tendons and ligaments:**

Tendons the fibers are primarily parallel and are oriented to support primarily uni-axial loading

Ligament fibers may be oriented and interlaced to support loads in more than one direction (depending on the function of the ligament)

15. Image: **Comparison of Tendon and Ligament** See Figures 4-4 from Nordin and Frankel[2]

16. Image: **Crimping** See Figures 2.5.2 from *Biomechanics of the Musculo-sleletal System, 2nd edition* [3]

17. **Outer Structure - Ligaments**

- Ligaments are surrounded and protected by loose areolar connective tissue (the *epiligament*).

18. **Outer Structure - Tendons**

- Tendons are surrounded by a similar layer called the *paratenon.*
- This layer forms a sheath that enhances gliding of the tendon.
- This sheath may only be present where a tendon bends at a joint.
- Where friction may be particularly high an additional *synovial* layer called the *epitenon* lies under the paratenon
- fiber bundles within the tendon are bound together by the *endotenon*

19. **Bone Insertion**

- Bone insertions for ligaments and tendons are similar.
- The tissue gradually changes from collagen to fibro-cartelage to calcified fibro-cartelage and then to cortical bone.
- Stress concentrations are minimized by the gradual change in properties
- When joined at an acute angle some fibers blend into the periosteum.

20. Image: **Bone Insertion** See Figure 4-7 from Nordin and Frankel[2]

21. Image: **Muscle Tendon Connection** See Figures 2.6.5 from *Biomechanics of the Musculo-sleletal System, 2nd edition* [3]

22. **Muscle Insertion of Tendons**

    - increases surface area (typically 10 to 20 fold, max. 50 fold)
    - transfers load primarily in shear
    - *Microtendon*: collagen fibers in the endotenon and the edonmysium become aligned and blend together (one per muscle fiber)

23. **Vascularization**

    - Tendons and ligaments have very poor blood supply.
    - Tendons with a paratenon sheath are termed vascular tendons. Blood supply enters from many points along the length of the tendon.
    - Tendons with a synovial sheath are termed avascular tendons and receive some of their nutrition by diffusion.
    - In ligaments a network of fine blood vessels is contained in the epiligament and they penetrate into the ligament in a few places travelling along the fascicles.

24. Image: **Vascularization** See Figure 4-6 from Nordin and Frankel[2]

25. **Neural - Ligaments** Nerves in ligaments:

    - no clear function
    - contribute to reflexes
    - active for both passive and active movements
    - most active near extremes of joint motion

26. **Neural - Tendons** Nerves in tendons – the *Golgi-tendon organs*:

    - lie close to the muscle in series with contractile proteins in the muscle
    - approximately one for every 10 muscle fibers
    - each nerve cell extends around and between many collagen fibers
    - as load is applied the fibers compress initiating and neural signal

27. Image: **Collagen** See Figure 1-4 from *Dynamics of Human Biological Tissues*[10]

28. Image: **Micrographs - Relaxed and Loaded Collagen** See Figure 4-9 from Nordin and Frankel[2]

29. Image: **Effect of Crimping on Mechanical Properties** See Figure 9.28 from *Introductory Biomechanics: From Cells to Organisms*[1]

30. Image: **Stress-Strain Relationships** See Figures 4-8 and 4-10 from Nordin and Frankel[2]

31. Image: **Stress-Strain Relationships** See Figures 9.29 and 9-30 from *Introductory Biomechanics: From Cells to Organisms*[1]

32. **Toe Region:**[3, 4]
    - strain up to 2 or 3 %
    - straightening of collagen fibers
    - may also reflect shearing of interfibrillar gel and interfibrillar sliding

33. **Linear Region:**[3, 4]
    - strain from 2 or 3 % up to 4 or 5 %
    - some report linear behaviour up to 20 % for whole tendons and ligaments
    - mean of tendon moduli across all species is 1,000 MPa
    - micro-damage in tendons and ligaments can occur in loads less than one-half the maximum

34. **Variation in Failure Properties -Tendon**[4]
    - modulus varies from 300 to roughly 600 MPa
    - no change with age

35. **Variation in Failure Properties - Ligament**[4]
    - modulus *decreases* with age
    - ACL modulus is 111 MPa in the young and 65 MPa in older specimens

36. **Variation in Failure Properties - Flexible Ligaments (*ligamentum flava*, *ligamentum nuchae*)**[4]
    - maximum strains of up to 70 %
    - modulus decreases from 100 MPa to 2 MPa with age

37. **Injury and Failure** See Case Study4-1 from Nordin and Frankel[2] Failure influenced by fracture near bone insertions. Bone avulsion is the dominant failure mode in cases of advanced age or immobilization.

38. Image: **Force-Displacement Data - Ligament** See Figure 9.27 from *Introductory Biomechanics: From Cells to Organisms*[1]

39. **Ligament and Tendon - Failure**[4]

Example: ACLigament
- Failure: stress of around 40 MPa and a load of around 2000 N

Example: Patellar Tendon
- failure stress of range from approximately 30 to 50 MPa
- failure strains range from 20% to 30 %

Example: Ligamentum Flava
- 10 MPa at 70% strain (young adult)
- 2 MPa at 30% strain (older adult)

40. **Ligament and Tendon- Testing Challenges**
    - Clamping
    - Non-uniformity
    - Calculation of stress and strain from load displacement data
    - Cross-sectional area determination.
41. **Connective Tissue Properties - Summary** See Tables 9.7 and 9.8 from *Introductory Biomechanics: From Cells to Organisms*[1]
42. **Physiological Loading**[2]
    - Strain limited to 2 % to 5%
    - Peak strains only last for short durations
    - Stresses may be limited to less than 25 % of the ultimate stress
43. **Energy Losses**
    - Energy losses are very small (6% to 11%).
    - Indicated by hysteresis in load-deformation curves (area between loading and unloading curves represents the amount of energy lost.
44. Image: **Hysteresis and Energy Losses** See Figure 10.3 from *Accidental Injury: Biomechanics and Prevention*[4]
45. **Strain Rate** [4] Tensile strength and ultimate strain are increased 20% to 30% with a 1000 fold increase in strain rate. With higher strain rates failure is more often in the ligament substance than by avulsion of the bone.
46. **Cyclic Loading**[4]
    - Tendons and Ligaments undergo permanent deformation under cyclic loading.
    - Stiffness increases with repeated loading
    - Microfailures accumulate over time.
47. **Stress Relaxation**[4]
    - Tendons and ligaments exhibit stress relaxation.
    - The rate of stress relaxation is initially large and then decreases.
    - The degree of relaxation decreases with repeated loading.

48. **Creep**
    - Tendons and ligaments exhibit creep.
    - With repeated loading strain increase becomes less pronounced.
    - Clinical significance - application of constant low loads can elongate ligaments over time.
49. **Bone-ligament-bone complex**
    - At low load rates (slower than for injuries) the weakest point is the bone interface.
    - At high load rates, typical on injuries, the ligament is the weakest link.
    - No change in stiffness.
    - Possible explanation - bone shows a greater increase in strength than ligament with increases in load rate.
50. **Categories and Common Types of Injuries**
    - Category 1: Little pain no clinical indications
    - Category 2: Continuous, severe pain, reduction in strength and stiffness of up to 50 % - partial rupture
    - Category 3: Pain during injury, reduced pain following - virtually complete rupture of all collagen fibers
51. **Common Tendon Injuries**[3]
    - lacerations (tears)
    - ruptures
    - tendinitis
52. **Basic healing processes**
    - Inflammation with cellular infiltration
    - Proliferation of new ground substance and collagen
    - Remodeling of new tissue
53. **Lacerations and ruptures**[3] Problems and concerns:
    - Maintaining blood supply
    - Adhesions between tendon and tendon sheath (passive motion)
54. **Tendinitis**[3]
    - Micro-structural damage occurs during repetitive tasks (damage to collagen fibers)
    - Normal healing process is triggered (inflammation, organization, regeneration)
    - If the time between bouts of repetitive tasks is short, healing does not progress beyond the inflammation stage.
55. **Remodelling**[3]

- Large loads cause fibrocytes to change to fibroblasts without inflammation.
- One author reports that before new tendon is formed there is initial transient weakness (empty spaces have been observed).
- These loads must be large but not large enough to cause damage and inflammation.

56. **Factors that Affect Biomechanical Properties - Movement and Age**

    - Aging - strength and stiffness increase during maturation ($\tilde{2}0$ years) and then slowly decrease with age.
    - Immobilization - dramatically decreases strength (no change in cross-sectional area but higher metabolism and more immature collagen), only 90 % recovery after one year.
    - Exercise - increases strength

57. **Factors that Affect Biomechanical Properties - Disease / Biochemical**

    - Diabetes - stiffness and failure mode changes
    - Steroids - decreased strength stiffness and energy absorption with repeated use.
    - Nonstreoridal anti-inflammatroy drugs - some evidence of increased strength
    - Hemodialysis - reduced strength (36 % failures in individuals receiving dialysis.

Other External Links

None

2.4 Joints and Cartilage.

Basic Reference Material

Sections 9.7 to 9.10 from *Introductory Biomechanics: From Cells to Organisms*[1]

Additional Information contained in the Lecture Slides

1. **The primary types of joints:**
   - Synovial Joints – large range of motion (between articulating bones)
   - Cartiloginous Joints – little movement (epiphyseal bone plates during growth)
   - Fibrocartiloginous Joints – little movement (intervertegral discs)
   - Fibrous Joints (skull, interosseous ligaments)
2. Image: http://www.britannica.com/EBchecked/media/138384/Synovial-jointGeneralized Synovial Joint
3. **Examples of Joints** See Figures 8-4 and 8-11 from Marieb[8]. Look for examples of shoulder and elbow joints online.
4. **Hyaline Articular Cartilage**
   - Cartilage is an effective bearing surface.
   - Cartilage is a virtually isolated tissue:
     - devoid of blood vessels
     - no nerves
     - no lymphatic channels
5. **Hyaline Articular Cartilage - Composition:**
   - Condracytes are less than 10 % (by volume) of cartilage.
   - Collagen - 15 to 22 % (by dry weight)
   - Proteoglycans - 4 to 7 % (by dry weight)
   - Water, salts and small amounts of other proteins - 60 - 85 %
6. **Cartilage** See Figures 3-1, 3-2, 3-3, 3-4 and 3-5 from Nordin and Frankel[2]
7. Image: **Cartilage Structure** See Figure 9.26 from *Introductory Biomechanics: From Cells to Organisms*[1]
8. **Proteoglycans** Proteoglycans are a major component in ground substance. Proteoglycans are:
   - Large protein-polysaccharide
   - Consist of glycosaminoglycans (GAGs) attached to a protein core:

  - "bottle-brush appearance"
  - *agrecans* are composed of two GAGs: chondroitin sulfate (CS) and keratan sulfate (KS)

9. **Cartilage** See Figures 3-6, 3-7, and 3-8 from Nordin and Frankel[2]

10. **Proteoglycan Function**

    - Sulfate and carboxyl charge groups dissociate from CS and KS chains.
    - The chains are now covered in fixed negative ions - they repulse each other.
    - Ignoring other loads the PG aggregates straighten (drawing in fluid).
    - Other ions in solution are attracted to the fixed negative charges and serve as partial shields. (pH and salt concentration influence this effect).
    - PG aggregates exist in a matrix linked to the collagen fibers.
    - Tension in the collagen fiber matrix limits the size of the PG aggergates to 20 % of the "free solution" domain.
    - Inhomogeneously distributed in cartilage (higher in the middle zone) - may contribute to the visco-elastic behaviour.

11. **Cartilage as a Material: Cartilage resists compressive loads by:**

    - pre-stressed matrix of collagen and PGs
    - resistance to fluid flow / osmotic pressures

12. **The function of cartilage has "not yet been fully determined". [2]**

13. **Cartilage is made of :**

    "*a porous-permeable, fiber-reinforced composite matrix possessing all the essential mechanical characteristics of a solid that is swollen with water and ions and that is able to resist the high stresses and strains of joint articulation*"[2]

    Cartilage:

    - is a *mulitphasic* material — porous-permeable solid and fluid (may include an ion phase).
    - carries high loads — up to 10 times body weight.
    - has complex patterns of contact area during loading.
    - has internal stresses as high as 20 MPa.
    - viscoelastic behaviour is important

14. **Two main causes of viscoelastic behaviour:**

    - Internal friction – dominates in shear behaviour.
    - Fluid flow resistance – dominates in compression (and tension), permeability of the solid matrix is important.

15. **Porosity – ratio of fluid volume to total volume**

$$\beta = \frac{V^f}{V^T}$$

16. **Permeability – resistance to fluid flow**

$$k = \frac{\beta^2}{K}$$

K is the frictional drag coefficient

17. **Permeability – resistance to fluid flow** [2]

- Typical values range from 1.1×10^{-15} to 7.6×10^{-15} $\frac{m^4}{N \cdot s}$.
- Average pore diameter is 6 nm (molecular scale).
- Depends on compressive load – matrix compaction.

$$k = \frac{Qh}{A(P_1 - P_2)}$$

18. **Cartilage Testing** See Figure 3-11 from Nordin and Frankel[2]

19. Image: **Cartilage Testing** See Figures 9.31, 9.32, 9.33, 9.34 and 9.35 from *Introductory Biomechanics: From Cells to Organisms*[1]

20. **Tensile Behaviour**[2]

- Tensile tests do not generally account for viscoelastic effects.
- Highly anisotropic - properties vary depending on fiber direction and depth due to variations in collagen content.
- Tensile load supported by the network of collagen fibers — under loading fibers align with the load direction.
- At physiological strain levels ($< 15\%$), Young's modulus ranges from 5 to 10 MPa

21. Image: **Tensile Behaviour** See Figure 2.4.5 from *Biomechanics of the Musculo-sleletal System, 2nd edition* [3]

22. **Compressive Behaviour - Testing** Compressive properties:

- depend on orientation and depth (stiffness is greatest in deep cartilage)
- compressive stiffness increases with proteoglycan content (with more negative charges in the matrix a higher level of applied load is required in order to overcome the repulsive forces)
- viscoelatic effects dominate - creep and stress relaxation

23. Image: **Cartilage Testing** See Figures 3-9 and 3-10 from Nordin and Frankel[2]

24. **Creep**[2]

- in typical cartilage (2 - 4 mm thick) equilibrium is reached after 4 to 16 hours
- under relatively high loads ($> 1MPa$) 50 % of the fluid is exuded
- fluid content is fully recovered when the load is removed
- can be used to measure permeability (values from 1×10^{-15} to 2×10^{-15} $\frac{m^4}{N \cdot s}$ are typical)
- at equilibrium a measure of the compressive modulus of the matrix is possible (approximately 0.5 MPa)

25. **Stress Relaxation - II**

    - Stress relaxation in cartilage is rapid (a time constant of 2 to 5 seconds) and depends on the magnitude of the deformation.
    - Equilibrium is reached in as little as 15 minutes for small deformations.

26. Image: **Stress Relaxation** See Figure 12.8.3 from Y.C. Fung: Mechanical Properties of Living Tissues, 2nd edition

27. **Cyclic Loading**

    - Steady-state achieved in approximately 30 cycles (for most soft tissues)
    - Hysteresis and energy losses (but fluid flow aids in nutrient and waste transport)

28. **Shear Behaviour - for pure infinitesimal shear:**

    - no pressure gradients
    - no volume changes
    - no fluid flow

29. **Synovial Fluid Properties**[3]

    - Highly non-Newtonian.
    - at low shear rates (0.1 s) the viscosity is "a fews 10 s of Pascals"
    - at higher rates (1000 s) it is "only a 1000th of this value"

30. **Synovial Joint Lubrication - Synovial Joints are high performance and unique:**

    - The coefficient of friction in an intact synovial joint is around 0.02 !!
    - Deformation of the cartilage increases the contact area
    - Lubrication appears to be independent of the viscosity of the synovial fluid !!
    - Surface undulations are 2 to 6 μm in height.

31. **Forms of lubrication in synovial joints:**

    - Fluid-film lubrication (hydrodynamic and squeeze film)
    - Boundary Lubrication

32. **Fluid Film Lubrication - Hydrodynamic, Squeeze Film, Boundary, Mixed, Boosted** See Figures 3-19, 3-20, 3-21 and 3-23 from Nordin and Frankel[2]

33. **Initial Compressive Load Bearing**[2]
    - On initial loading hydrostatic pressure transfers the load to the bone.
    - This hydrostatic effect persists over time (1000 s) shielding the cartilage from high local stresses
34. **Compressive Load Bearing - Contact Area**[3]
    - Compressive forces in the superficial layer reduce permeability.
    - Interstitial fluid flows laterally away from the contact zone, flowing up and out of the cartilage at the edges of the contact area. This fluid is available for lubrication (fluid-film or boundary).
    - In normal activities, there is little time for significant fluid flow.
    - This flow causes lateral tension in the matrix due to drag forces – this subsides over time as the flow diminishes.
35. **Compressive Load Bearing - Contact Area Edge Effects** See Figures 2.4.16 from Biomechanics of the Musculo-sleletal System, 2nd edition[3]
36. **Wear and Failure of Cartilage**[3]
    - Acute failure — from active loads or from impact loads
    - Chronic failure (wear) — interfacial or fatigue
37. **Cartilage Wear** See Figure 3-25 from Nordin and Frankel[2]
38. **Wear**[2]
    - Interfacial Wear (Not likely significant for cartilage).
      - Adhesion - surface fragments adhere to each other and are torn away.
      - Abrasion - one surface scrapes the other
    - Fatigue Wear
      - Accumulated microscopic damage of the PG-collagen matrix (reduced load carrying capacity for the matrix)
      - PG "wash-out" - fluid flow removes PGs from the matrix - reduced compressive strength
    - Impact Loading

    Once the collagen matrix has been disrupted, further damage becomes possible due to:
    - further degradation of the matrix
    - PG wash-out
    - changes in load bearing mechanism
39. **Degeneration and Remodelling of Cartilage - Causes of degeneration and failure:**[3]

- Disuse – lack of flow of the fluids into and out of the cartilage – reduced influx of nutrients and efflux of waste

Cartilage Healing and Remodelling:

- Chondrocytes are responsible for the synthesis and degradation of the matrix.
- They are very ineffective - "cartilage has a very limited ability to remodel"

Other External Links

1. Video: human skeleton: joint movement. Video. Encyclopdia Britannica Online. Web. 17 Oct. 2011.

2.5 Active Tissues - Cell Membranes and Nerves.

Lecture Slides

1. The Cell Membrane and Membrane Potentials
2. Transport across the Cell Membrane: Diffusion
3. Transport across the Cell Membrane: Bulk Flow
4. Transport across the Cell Membrane: Osmosis
5. Transport across the Cell Membrane: Ionic Flow
6. Passive Transport: Solubility through Membrane, Protein Channel, Protein Channel
7. Active Transport: Basic transport, Co-transport, Counter-transport
8. The Sodium Pump and its Uses
9. Membrane Potential
10. Membrane Potential - The Cell
11. Nerve Fibers
12. Nerve - Dendritic Tree
13. Nerve - Synapses
14. The Action Potential
15. Stimulus causing depolarization
16. Nerve Cell Membrane - The Resting Potential
17. Nerve Cell Membrane - Depolarization
18. Nerve Cell Membrane - Sustained Depolarization
19. Depolarization — All-or-Nothing Response
20. Depolarization — Refractory Period
21. Transmission of the Nerve Impulse; Myelin
22. The Axon and Transmission of the Impulse
23. The Myelinated Axon and Transmission of the Impulse
24. Nerve - Myelin Sheaths
25. http://www.denniskunkel.com/
26. http://www.blackwellpublishing.com/matthews/animate.html

27. In a nerve:
    - Each *neuron* may be wrapped in a *myelin sheath*
    - Each *neuron* may be further wrapped in *epineurium* (A)
    - A group of *neurons* are grouped together in a *fascile* (B)
    - Each *fascile* may be wrapped in *Perineurium* (B)
    - A group of *facicles* is grouped together in a *nerve trunk* (A) and wrapped in *endoneurium* ***(C)***.
28. Image: The Nerve Trunk
29. Connective Tissue: Epineurium:
    - loose connective tissue
    - maintains blood supply
    - cushions during movement
    - most abundant near bones and joints
    - may be absent (eg in spinal nerves)
30. Connective Tissue: Perineurium:
    - very strong
    - can support an internal pressure of 1000 mmHg
    - biochemical barrier
31. Connective Tissue: Endoneurium:
    - Primarily fibroblasts and collagen
    - Normally has an elevated internal pressure of about 1.5 mmHg
32. Vascularization and Some Dimensions: Vascularization:
    - Nerves are well-vascularized (transmission of signals requires nutrient supplies).
33. Some Dimensions for individual neurons:
    - May have a diameter from less than 1 μm to 20 μm
    - May be up to a meter or more in length
    - Nodes of Ranvier are typical 1 to 2 mm apart
    - A single action potential (at 20 $\frac{mm}{ms}$ and 1.5 ms in duration) typically affects about 30 mm of the axon (17 nodes).
34. Image: Biomechanics of Nerves: A nerve fiber
35. Image: Biomechanics of Nerves: A Peripheral Nerve:
36. Injuries: The mechanical causes of injuries to nerves are

- stretching
- compression

37. Stretching - peripheral nerves
    - Nerves are very strong in tension (100 - 200 N)
    - Failure strain of 25 to 40 %
    - Elastic limit of about 20 % strain.
    - However damage to the nerve tissue becomes significant long before failure occurs.
38. Stretching - Spinal Nerves
    - Spinal nerve roots lack epineurium and perineurium
    - ultimate loads typically less than 30 N
    - ultimate strains are under 20 %
39. Image: Stress strain diagram for a typical nerve
40. Classifications of nerve injury [4]

    Neurapraxia large motor fibers affected
    - anatomic continuity is preserved
    - recovery in days to weeks

    Axonotmesis nerve is still intact but there is complete interruption of all nerve fibers (
    - complete sensory and motor loss
    - recovery by axonal regeneration starting at the cell body
    - growth at 1mm per day

    Neurotmesis complete disruption or severing of the nerve
    - surgery required; recovery prognosis is not as good...
41. Nerve damage as a function of strain - Damage is primarily due to a loss of circulation [2]

    at 6% action potential magnitude reduced by 70 % after 1 hour

    at 8 % vascular flow is greatly reduced

    at 12 % conduction was completely blocked after 1 hour (minimal recovery)

    at 15 % all intraneuronal microcirculation ceases
42. Image: Mechanics of Nerve Circulation Injuries
43. Image: Investigation of Compression Injuries
44. Nerve damage as a function of compressive pressure - Damage is primarily due to a loss of circulation. Damage increases with increased loading rates.

    at 2 mmHg healthy nerve

    at 30 mmHg reduced blood flow;

- long term viability is at risk;
- these pressure are typical of carpal tunnel syndrome
- axonal transport systems are disrupted

45. Nerve damage as a function of compressive pressure - II

    by 80 mmHg blood flow ceases (but is rapidly restored even after 2 hours)
    - edema may occur (an accumulation of fluid)
    - scar tissue may form

    over 200 mmHg permanent damage (even after short periods)
    - nerve fiber damage
    - loss of nerve function

46. Mechanics of Compressive Nerve Damage
    - damage in areas where there is a pressure gradient
    - displaced Nodes of Ranvier
    - large nerve fibers affected first
    - manner of application is important:
      - a circumferential load promotes axial displacement
      - a lateral load has deduced axial displacement but changes in cross-section
      - changes in cross-section create membrane stretching which may reduce permeability or trigger action-potentials
    - spinal nerves may be compressed between vertebrae or discs
    - nerve roots move as the extremities move (2 to 5 mm for a leg raise)

47. Action potentials:
    - are generated in sense organs
    - come from the brain
    - are spontaneously generated in some tissues
    - are transmitted to one neuron from a neighbouring neuron through a synapse

48. Image: Sense Organs

49. Image: Mechanoreceptors

50. Image: Other receptors

51. Touch - Pacinian Corpuscle

52. Touch - Action Potential Generation

53. Touch - Stimulus Strength

54. Generator Potential

55. Touch - Receptor Adaptation
56. The Synapse - Excitation and Inhibition
57. Images: ATP and the Membrane Potential
58. Image: Potassium and the Membrane Potential
59. Image: Relationship between the action potential and Na and K diffusion
60. Derivation of the Nernst Equation The work required to move δn moles of X up a concentration gradient is:

$$\delta W_c = \delta n \, RT \ln \frac{[X]_1}{[X]_2}$$

where:

- R is the ideal gas constant
- T is the absolute temperature
- $[X]_1$ and $[X]_2$ are the molar concentrations of X in compartments 1 and 2

The work required to move δn moles of X against an electrical gradient is:

$$\delta W_e = \delta n \, zFE$$

where:

- z is the charge on the ion
- F is Faraday's constant (96,500 coulombs per mole)
- E is the potential difference between compartments 1 and 2

At equilibirum $\delta W_e = \delta W_c$, so,

$$\delta n \, zFE = \delta n \, RT \ln \frac{[X]_1}{[X]_2}$$

61. The Nernst Equation

$$E = \frac{RT}{zF} \ln \frac{[X]_1}{[X]_2}$$

Evaluating the constants at 18 ° C:

$$E = \frac{25}{z} \ln \frac{[X]_1}{[X]_2} = \frac{58}{z} \log \frac{[X]_1}{[X]_2}$$

62. Image: Intra- and inter-cellular membrane concentrations
63. How many ion's need to cross the membrane ?
The charge, Q in Coulombs, on 1 cm^2 of the membrane is given by:

$$Q = C \, V$$

where,

- C is the capacitance (typically $1\frac{\mu F}{cm^2}$)
- V is the potential across the membrane(assume this to be $70mV$)

This corresponds to: $\frac{CV}{zF}$ moles per square cm or for our values, $6.8 \times 10^{-13}\ \frac{moles}{cm^2}$.
This represents a difference in ion concentration across the membrane of anywhere from 2×10^{-6} to 3×10^{-3} % depending on the axon size.

64. Cable Theory for Action Potentials

65. Image: The axon can be modelled as a long continuous cable which is described by a partial differential equation:
$$\tau_m \frac{\partial V}{\partial t} + R_m I_{ion} = \lambda_m^2 \frac{\partial^2 V}{\partial x^2}$$

66. Constants and Response. Here, the time constant is $\tau_m = R_m C_m$ and the spatial constant is given by $\lambda_m^2 = \frac{R_m}{R_i + R_o}$.
$$V_x = V_o e^{-x/\lambda_m}$$

67. Image: Typical parameter values

68. This model can be extended to include a source (the depolarization)

69. This model can be FURTHER extended to apply to myelinated nerves.

70. Transport along the Axon - Molecular Machines

71. Some nerves are very long (about 1m)
    - Diffusion along the length of the axon would take about 150 years!!
    - Molecular motors move material in both directions.
    - Fast transport — anterograde 250-400 mm/day and retrograde 100 to 200 mm/day (1m in as little as 2.5 days)
    - Intermediate speeds of 50 mm/day
    - Slow transport at 2 to 4 mm/day.

72. Image: Axon Transport — Self-assembling Microtubules

73. Image: Axon Transport — Molecular Motors

74. Image: The Neuromuscular Synapse

75. The Neuromuscular Synapse - Docking

Other External Links

The following are useful resource (available as e-books through Carleton University Library):

Electromyography - Physiology, Engineering, and Noninvasive Applications Edited by: Merletti, Roberto; Parker, Philip 2004 John Wiley & Sons.

Enoka, R. M. 2006. *Motor Unit* in the Wiley Encyclopedia of Biomedical Engineering.

Butera, R. 2006. *Nernst Potential.* in the Wiley Encyclopedia of Biomedical Engineering.

2.6 Muscle

Basic Reference Material

Chapter 8 *Introductory Biomechanics: From Cells to Organisms*[1]

Additional Information contained in the Lecture Slides

1. Image: Basic Anatomy
2. Images: Basic Microscopic Structure
3. Image: The Sliding Filaments
4. Image: Contraction — Actin, Myosin and ATP
5. Images: Contraction — Calcium and the Actin-Myosin Bond
6. Image: Force Production and the Sliding Filaments
7. Image: Isotonic and Isometric Contraction
8. Image: The Muscle Twitch
9. Image: Superposition of Twitches
10. Image: Motor Unit Recruitment
11. Three steps in the development of a muscle fiber
    - Myogenesis
    - Synaptogenesis
    - Synapse Elimination
12. Myogenesis
    - Individual cells becomes mononucleated myoblasts
    - Myoblasts begin to collect and fuse to form myotubes (single, multinucleated cells)
    - Contractile proteins fill in around the developing muscle fiber
13. Image: Myogenesis
14. ImageL Synaptogenesis
15. Synaptogenesis
    - Initially acetylcholine (ACh) receptors are distributed over the entire cell
    - As the growing nerve touches the fiber the ACh receptors migrate to the nerve (why ?)
    - Multiple nerves form synapses with a single fiber

16. Interesting Fact #1 As a nerve axon grows from the cell body at spinal chord it seeks out a specific muscles. If the muscle is moved during this growth the nerve changes direction and finds the muscle.

17. Interesting Fact #2 Muscles come in two types (fast and slow) – the type of muscle is determined by the nerve connected to it. If you surgically change the nerve attached to a muscle the muscle type will change.

18. Image: The Neuromuscular Synapse

19. Image: The Muscle Action Potential

20. Image: The Muscle - T-tubules

21. Image: Calcium and Cross-Bridge Formation

22. Image: The Cross-Bridge Cycle

23. The Motor Unit

    - smallest functional unit
    - consists of a motor nerve and the muscle fibers it innervates
    - a single nerve may control from 3 to 2000 muscle fibers

24. Motor Unit Recruitment - The Size Principle

    - "the size of a newly recruited motor unit increases with the tension at which it is recruited"
    - action potential magnitude depends on the diameter of the axon and the size of the motor unit

    from: D. A. Winter, *Biomechanics, 3rd Edition*, Wiley, 2005 and R. L. Lieber, *Skeletal Muscle Structure and Function*, Williams and Wilkins, 1992

25. Images: Motor Units

26. Image: Figure 8.1 from Introductory Biomechanics: From Cells to Organisms [1]

27. Types of Muscle

    - Smooth (in the walls of hollow organs)
    - Cardiac (the heart)
    - Skeletal

28. Images of Smooth Muscle and Cardiac Muscle

29. Image: Comparison of Muscle Types

	Skeletal	Cardiac	Smooth
Cell Type	cylindrical; multinucleated; striated	branched umi- or bi-nucleated, striated	sungle; fusiform, not striated
Myofibrils	regular thickness	irregular thickness	none
T-tubules	yes	yes (larger)	no
Sarcoplasmic Reticulum	yes	some	none
Regulation	voluntary	innvoluntary	innvoluntary
Pacemakers	no	yes	yes (in single-unit muscle)
Gap junctions	no	yes	yes in single unit muscle
Neuromuscular junctions	individual fibers	–	only in multiunit muscle
Ca^{++} Regulation	troponin	troponin	calmodulin (on myosin)
Speed	slow – fast	slow	very slow
Energy Supply	aerobic / anaerobic	aerobic	mostly anaerobic

30. Images: Muscle Anatomy
31. Image: Figure 8.2 from Introductory Biomechanics: From Cells to Organisms [1]
32. Images: Muscle Microstructure
33. Image: Figure 8.3 from Introductory Biomechanics: From Cells to Organisms [1]
34. Sarcomere size
    - A single sacromere is 2.5 μm long
    - A muscle 10 cm long and 0.1 cm^2 in diameter would contain 40,000 sarcomeres in a filament and 10^7 filaments
35. Image: Microstructure - Proteins
36. Image: Myosin
37. Image: Myosin - geometry
38. Images: Actin
39. Image: Figure 8.4 from Introductory Biomechanics: From Cells to Organisms [1]
40. Images: Basic Microscopic Structure

41. Image: The Sliding Filaments
42. Image: Figure 8.14 from Introductory Biomechanics: From Cells to Organisms [1]
43. Image: Contraction — Actin, Myosin and ATP
44. Images: Gross Muscle Architecture
45. Image: Fiber Length and Muscle Architecture
46. Image: Mechanical Properties and Muscle Architecture
47. Two important parameters
    - Penation Angle, θ
    - Physiological Cross Sectional Area, $PCSA = \frac{mass \times \cos\theta}{density \times length}$
48. Images: Figures 8.19, 8.20, 8.6, 8.7, 8.8, 8.9, 8.10, 8.11, 8.12, 8.13, 8.15, 8.16, 8.17 and 8.18 from Introductory Biomechanics: From Cells to Organisms [1]
49. : Muscle Models
50. Muscle Stiffness: The static and passive properties of a muscle can be most simply (and approximately) modeled by a spring. In general the maximum developed force is a function of PCSA and ranges from 20 to 80 $\frac{N}{cm^2}$ depending on the definition of PCSA used and the muscle.
51. Image: Muscle Stiffness
52. Image: Passive Muscle Stiffness
53. Stiffness Function The stiffness relationship in the preceding figure was

    $$\frac{d\sigma}{d\lambda} = \alpha(\sigma + \beta)$$

    where σ is the stress and λ is the Lagrangian strain, $\lambda = \frac{\ell}{\ell_0}$.

    Therefore,

    $$\sigma = \mu e^{\alpha\lambda} + \beta$$

    Despite its widespread applicability, no derivation from first principles exists for this relationship.
54. Reminder - Properties are explained by the Sliding Filament Hypothesis
55. Image: Active Muscle Stiffness [11]
56. Active Muscle Stiffness [11]
57. Muscle: Length - Velocity Relationship [11]
58. Muscle: Length - Velocity Relationship [11]

- This curve is based on experimental data.
- T_0 is the isometric tension (typically 25 $\frac{N}{cm^2}$)
- v_{max} is the shortening velocity against no load (ranges from 6 to 16 $\frac{l_0}{s}$ where l_0 is the fiber length.
- Notice the asymmetry – a lengthening muscle can have a maximum tension of up to 1.8 T_0.

59. A.V. Hill's Relationship [11] Hill proposed an empirical formulation for the concentric part for this curve:
$$(T + a)(v + b) = (T_0 + a)b$$
In normalized form,
$$\frac{v}{v_{max}} = \frac{(1 - [\frac{T}{T_0}])}{(1 + \frac{1}{k}[\frac{T}{T_0}])}$$
where $k = \frac{a}{T_0} = \frac{b}{v_{max}}$. For real muscle $0.15 < k < 0.25$.

    Note that in this formulation, $v_{max} = \frac{bT_0}{a}$.

60. Muscle Power [11] Hill's expression can be used to find that the power of a shortening muscle is:
$$P = Tv = v\frac{bT_0 - av}{v + b}$$
The maximum power output occurs at $v = 0.31\, v_{max}$ and $T = 0.31\, T_0$ and is roughly equal to $0.095\, T_0 v_{max}$

61. Image: Hill's Model - Errors [3]

62. Empirical Models and the Cross-Bridge Theories

63. Images: Models of Items with Visco-elastic Properties:
    - Damper - $\sigma = \eta\epsilon(s)$
    - Maxwell Fluid - $\sigma = \frac{\mu\eta s}{\mu+\eta s}\epsilon(s)$
    - Voigt Solid - $\sigma = \epsilon(s)[\mu + \eta s]$
    - Kelvin Body - $\sigma = [\frac{\mu_1\eta s}{\mu_1+\eta s} + \mu_2]\epsilon(s)$
    - Three Element - Matches Hill
    - Simplified Three Element - $T - T_0 = Bv = \frac{a+T_0}{v+b}v$
    - Four Element- $T = \frac{K_{SE}T_{CE}}{Bs+K_{PE}+K_{SE}} + \frac{K_{SE}(Bs+K_{PE})}{Bs+K_{PE}+K_{SE}}\epsilon$

64. Image: Parameter Identification - Quick Release Experiments [11]

65. Image: The Muscle Model - Quick release Experiemtn [11]

66. Model Parameters from the Data [11]

67. Model Parameters from the Data [11]
    - Initial slope gives data related to Hill's force- velocity relationship
    - Δx_1 is a function of the parallel elastic component
    - Δx_2 is a function of the series elastic component
68. Active State- Based on quick release experiments, we can get information on the so called "active state"
    - The active state assumes that there is an ideal force generator in the contractile element.
    - It was modified to include the capacity to shorten but incorrectly predicts that the speed of lightly loaded shortening should be proportional to the T_0
    - It does not predict the fact that with increased muscle length the muscle twitch decay rate slows.
69. Image: Measuring the Active State [12]
70. The 1957 Cross-Bridge Theory [3]
    - Prior to 1957 contraction was thought to be a result of folding or coiling of myofilaments
    - In 1954 it had been shown that no change in filament length occurred so that the filaments must slide over one another
    - The myosin heads were thought to oscillate about equilibrium due to thermal agitation
    - Myosin would spontaneously bond to the actin sites when close enough and only on one side of the equilibrium position (so that the force was only in one direction)
    - Attachment and detachment were governed by rate functions.
71. Images: The 1957 Cross-Bridge Theory [3]
72. The 1957 Cross-Bridge Theory - The theory does a good job predicting Hill's force-velocity relationship
    - The theory does not accurately predict details in the asymmetric force velocity relationship.
    - The theory does not predict heat production in muscles.
73. Image: The 1971 Cross-Bridge Theory (Reformulated) [3]
74. The 1971 Cross-Bridge Theory (Reformulated) [3] In experiments, force recovers quickly after a step change in length. In the 1957 theory, this recovery can only be due to rates of attachment which were to slow to match the data.
    The theory was reformulated in 1971 to include movement of the cross bridges to account for step changes in length. Although the 1971 theory does not explain all experimental phenomena it is the dominant theory and has yet to be replaced.
75. Muscle Metabolism

- Heat Production [12] Much progress that has been made in understanding the mechanics of force production in muscles is a result of measurements of heat production from which inferences are made regarding chemical and mechanical processes within the muscle.

  These measurements are made with very small and accurate thermopiles (groups of thermocouples) that can measure temperature with a precision of 0.000001 degrees Celcius.
- Image: Initial Heat [12]
- Resting Heat [12]
  - This is the energy released in muscle at rest.
  - It is 0.0002 cal/g/min.
- Activation Heat [12]
  - This heat is produced about 15 msec after stimulation and increases to a maximum long before tension is developed.
  - It is a function of fiber length.
  - It coincides with the active state.
- Image: Activation Heat [12]
- Heat of Shortening [11, 12]
  - A shortening muscle generates more heat than one that contracts isometrically — primarily a result of the work done by the muscle.
  - Measurements of the Heat of Shortening yield a model identical to the Hill force-velocity relationship !
  - Image. [11]
- Heat of Lengthening [12, 11]
  - Heat production is reduced in a lengthening muscle.
  - This heat reduction matches observations from the asymmetry of the length-velocity relationship.
- Thermoelastic Heat - As a material muscle exhibits a relationship between temperature and length
  - An active muscle exhibits normal thermoelasticity (expanding when heated, etc...)
  - A resting muscle exhibits "rubber" thermoelasticity (generating heat when stretched)
- Recovery or Relaxation Heat
  - After a contraction is complete muscle continues to generate heat
  - This is a result of the muscle re-establishing chemical balance.
- Image: Muscle Fatigue and Endurance [12]
- Muscle Fatigue and Endurance [12]
  - At 15% load small blood vessels start to become compressed and nutrients are prevented from reaching the msucles
  - at 70% load all blood supply is occluded and fatigue occurs quickly

  - this relationship can be approximated as

$$T_{end}(s) = \frac{1236.5}{(Strength_{\%,max} - 15)^{0.618}} - 72.5$$

- Image: ATP is the source of energy for contraction
- Image: ATP can be created anaerobically (inefficient but fast)
- Image: ATP can be created aerobically (efficient but slow)
- Image: ATP can be reversibly created from stores of creatine phosphate (PCr)

$$ADP + PCr \rightleftharpoons ATP + Cr$$

  - Equilibrium strongly favours the right hand side.
  - No drop in ATP has ever been found in intact muscle under normal circumstances.
- Image: A Schematic Model of Energy Sources [11]
- Energy for a contraction:
  - There is enough ATP present in muscle for about 8 twitches (without using PCr)
  - There is enough PCr present for about 100 twicthes.
- Image: Energy for a contraction - PCr and ATP [11]
- Light Exerecise [11]
  - Initial energy from PCr conversion
  - Anaerobic sources briefly generate ATP until aerobic sources take over
  - In steady state all energy comes from aerobic sources.
- Image: Light Exercise [11]
- Aerobic Recovery [11]
  - After contraction ends, oxidative sources continue to replenish ATP
  - The ATP is quickly converted to PCr
  - the lactic acid is re-synthesized into glucose (very slowly, time constant of 30 minutes or more)
- Image: Aerobic Recovery [11]
- Image: Heavy Exercise [11]
- Heavy Exercise [11]
  - The energy required is greater than the maximum aerobic rate of ATP production
  - Anaerobic sources generate the remaining energy
  - The lactic acid concentration begins to increase.
- Anaerobic Recovery [11]
  - Even without oxygen, PCr and ATP levels can be re-established after exercise
- Image: Energy Sources [12]
- Image: Energy Sources and Muscle Fiber Types
- Energy Examples [12]

- Energy Sources and Training [12]
- Immobilized Muscle
  - Heals more slowly.
  - The benefits from movement not from isometric contraction.
  - Type I Fibers atrophy most readily (Slow twitch, Oxidative)
  - Electrical stimulation may help.
- Exercise and stretching
  - Exercise increases muscle bulk (PSCA) and strength.
  - May change the relative area taken up by Tape I and IIa fibers relative to Type IIb Fibers.
  - The roll of stretching is not well understood - it increases flexibility but may also have an effect on neuromuscular control.

Other External Links

There are several articles (Muscle, Skeletal; Muscle Fiber Conduction Velocity; Muscle Pain; Muscle Sensory Receptors; Musculoskeletal Cell Mechanics) in the Wiley Encyclopedia of Biomedical Engineering (available as an e-book through Carleton University Library) that focus on muscle (find them in the e-book by listing reticules by title and finding muscle).

Also, you may find various chapters in the following e-book helpful:

The Biomedical Engineering Handbook, Second Edition. 2 Volume Set, Edited by Joseph D . Bronzino, CRC Press 1999

2.7 Reflexes and Motor Control

Additional Information contained in the Lecture Slides

1. Image: Two types of synapses - Inhibitory and Excitatory - from The Physiology Coloring Book, 2nd edition.
2. Axon Properties - Convergence and Divergence
3. Synapse Speed: In addition, depending on the manner of signal transmission across the cell wall, synapses can be: Fast or Slow (when secondary messengers are involved)
4. Image: Summation of synaptic potentials - Spatial Summation or Temporal Summation, from The Physiology Coloring Book
5. CNS Anatomy - Four major parts of the Central Nervous System (CNS): Spinal Cord, Brain Stem, Cerebrum (Sensorimotor cortex and basal ganglia), Cerebellum.
6. CNS Anatomy - Information on the roles of these components comes from animal studies and from studies of people with injuries or diseases.
7. Image: Major Functions of The Spinal Cord from: The Physiology Coloring Book
8. Image: Schematic of Neural Pathways from: T. A. McMahon, *Muscles, Reflexes and Locomotion*
9. If the spinal cord has been cut:
   - some reflexes still function
   - pain causes withdrawal
   - the scratch reflex still functions
   - walking can occur (with supported body weight, feet contacting a treadmill belt)
   - gate change with speed occurs
   - chickens can run and fly without their heads !!
10. The brain is divided into two parts

    Forebrain of cerebrum regulates higher functions and consists of the cerebral hemispheres, the basal ganglia and the limbic system.

    Brain Stem more primitive part of the brain that controls visceral functions and brain reflexes. It consists of the medulla, pons, cerebellum and midbrain.
11. If the neural paths are cut in the brain stem:
    - the functions from lower levels of transection still generally work
    - an animal can right itself from lying on their back or side
    - this response depends on sense organs in the ear (the semicircular canals and the otoliths)

12. The cerebellum is a part of the brain stem that
    - is a major center for motor coordination
    - smoothes fast and skilled movements
    - integrates information from the brain and from the periphery
13. Without a cerebellum (which is a focus of incoming sensory information):
    - motion sickness does not occur
    - a nearly normal range of motor behaviour can occur
    - movement is awkward and clumsy
14. The forebrain or cerebrum performs
    - perception
    - voluntary motor control
    - emotion
    - cognition
    - language
15. The Basal Ganglia are a part of the forebrain that
    - integrates voluntary and complex involuntary movements
    - lesions to the basal ganglia produce Parkinson's Disease and other movement disorders.
16. Sensorimotor Cortex: If the cerebral cortex is removed in addition to the above functions, an animal
    - can climb, display anger and reject distasteful food
    - cannot learn new skills
17. Basal Ganglia: If the basal ganglia are damaged (from injury or disease);
    - the patient sits motionless moving only occasionally
    - the patient appears not to move more than necessary
    - some movements without apparent purpose (twitching) may occur
18. Spinal Cord: Images of Bones and Discs - from The Anatomy Coloring Book
19. Spinal Cord: ruptured disk - from The Anatomy Coloring Book
20. Spinal Cord: Vertebral Canal and the Foramen - from The Anatomy Coloring Book
21. Nerves and the Spinal Cord, Images from The Anatomy Coloring Book

    A Dorsal Root (afferent; sensory nerves)

    D Dorsal Root Ganglion (cell nuclei)

    E Ventral Root (Efferent, motor nerves)

M Intervertebral Foramen

22. Organization of Nerves in the Spinal Cord - ascending and descending, white and grey matter, Images from The Anatomy Coloring Book
23. Components of the Brain - Motor Cortex, Sensory Cortex, Basal Ganglia, Cerebellum - Images from The Anatomy Coloring Book
24. Sensory Homunculus and Motor Homunculus - Images from the Web
25. Muscles Receptors - Spindle Fibers, Images from: T. A. McMahon, *Muscles, Reflexes and Locomotion*, Princeton University Press, 1984
26. Spindle Fibers - Respond to a filtered combination of stretch and rate of stretch, the response is different for a shortening msucle
27. Spindle Fibers - are active muscle fibres, controlled with a separate nerve from the main muscle, and can be modelled with the same structure as the Hill muscle model
28. The Golgi Tendon Organ - Respond to force
29. Reflexes - Synapses in the spinal cord - No involvement of the higher brain levels
30. Examples of Reflexes:
    - The Stretch Reflex - Extension of a Spindle Causes the muscle to shorten
    - Stretch Reflex Timing - 1.0 ms (spindle response) + Sensory nerve transmission (5 ms) + Synapse in the spinal cord (1 ms) + Motor nerve transmission (5 ms) + neuromuscular junction and build up of endplate potential (2 ms) + force buildup (20 ms) = 34 ms
    - "Knee Jerk" Reflex - reciprocal inhibition of the antagonist muscle
    - The Clasp-Knife Reflex - high forces sensed in the Golgi Tendon organ cause the muscle to relax (collapse)
    - Withdrawal Reflex - Pain causes the antagonist muscle to contract pulling the limb away
    - Cross-Extensor Reflex - when one foot is lifted the other foot is extended

Other External Links

None.

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