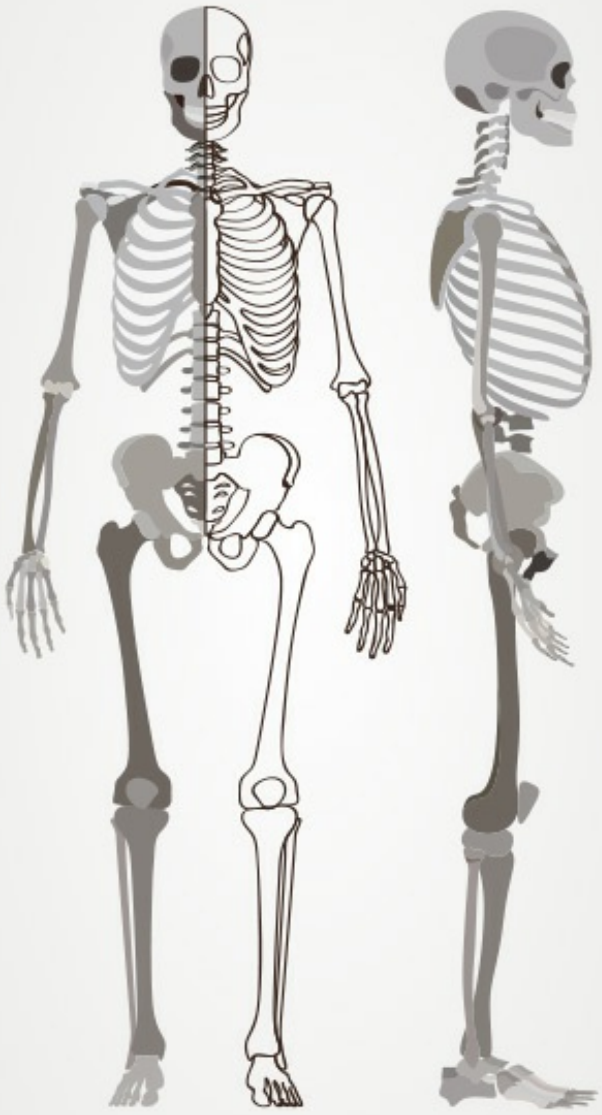
Spine and Tissue Biomechanics

Biomechanics for health sciences – A study guide part 2

Gerard Gorniak



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GERARD GORNIAK

SPINE AND TISSUE BIOMECHANICS

BIOMECHANICS FOR HEALTH SCIENCES – A STUDY GUIDE PART 2

Spine and Tissue Biomechanics
Biomechanics for health sciences – A study guide Part 2
1st edition

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To see part I download Upper and Lower Extremity Biomechanics: Biomechanics for health sciences – A study guide Part 1

ABOUT THE AUTHOR

Professor Gerard C. Gorniak PhD. PT retired in 2015 from the University of St. Augustine for Health Sciences. He has been married for 45 years, has 2 children and 3 grandchildren. He is active in his church, prayer community, neighborhood, and enjoys gardening, writing and working with his hands.

After receiving a BSPT in Physical Therapy from the State University of New York at Buffalo in 1971, he remained at the University to earn a Ph.D. in Anatomical Sciences from the College of Medicine in 1976. He then went to The University of Michigan for 5 years as a NIH post-doctoral scholar with Professor Carl Gans working in muscle transplantation, biomechanics, and functional anatomy. In 1981, Dr. Gorniak accepted an Assistant Professor position in the Department of Biological Science and Program in Medical Sciences at Florida State University and became an Assistant Director of Clinical Affairs, Program in Medical Sciences in 1986. In 1988, he joined the Physical Therapy Program at Florida A&M University as an Assistant Professor. From 1994–2015 Dr. Gorniak was at the University of St Augustine for Health Sciences, starting as an Associate professor and promoted in 1999 to full Professor. From 2000–2008, he was the Director of Physical Therapy Program at the University and the Director of the Institute of Physical Therapy from 2006–2008. From 1986–1997 he also practiced Physical Therapy part-time. Currently Dr. Gorniak is an Adjunct online Professor and Consultant at the University of St Augustine.

Dr. Gorniak began his teaching experience as an undergraduate physical therapy student, teaching physical therapy, nursing and physical education anatomy labs. In his over 40 years of teaching, he has taught mainly gross anatomy and biomechanics, but has also taught embryology, histology, neuroanatomy, pharmacology, pathology, physical therapy differential diagnosis, research, psychosocial and ethical aspects of physical therapy, and physical therapy skills and procedures. He has developed online courses in Anatomy, Biomechanics, pre-anatomy Basic Human Musculoskeletal Anatomy, and Pharmacology.

Over the years Dr. Gorniak has developed note packs for the students in Anatomy (826 pages), Biomechanics (407 pages), Pharmacology (93 pages), Differential Diagnosis (144 pages), Histology (199 pages), as well as a Lab Manual for Applied Human Anatomy (146 pages) and high definition, cadaver Video Dissector of the Human Body series (8 anatomical regions). He has published over 30 peer reviewed journal articles, most in the areas of anatomy and biomechanics, delivered many invited presentations and workshops on the extremities and pelvis, and has been a reviewer for several journals and an associate editor for the Journal of Morphology. He was an Item Writer and Reviewer for the Federation of State Boards of Physical Therapy, on the Medical Advisory Board for Medical Personnel Pool, and was active in the Florida Physical Therapy Association as a member of the Ethics Committee, Board of Directors, Practice Panel Coordinator, Research committee chair and a delegate to the National Assembly.

Dr. Gorniak is listed in American Men and Women in Science, Who's Who in the Midwest, and Who's Who in the South. He received the Richard Winzon Teaching Award four times from Programs in Medical Sciences, and outstanding instructor awards from Florida A&M University and the University of St Augustine. Dr. Gorniak received recognition from the Florida Physical Therapy Association for Ethic Committee and Research Committee Service and the Rick Shutes Committee Service Award. He has served on numerous University committees, councils, task forces, and has been involved in 4 national accreditations.

Dr. Gorniak has also been active in the community. He coached little league baseball and girls softball and grade school basketball. He was the Chairperson of BOD of St. Augustine House of Prayer and Evangelization Center, Inc.; a member of the Curisillo Secretariat, Dioceses of Pensacola-Tallahassee; made 8 missions trips to Slovakia and Czech Republic with Renewal Ministries; planned and participated in nearly 20 Dioceses of St. Augustine Catholic Charismatic Renewal Conferences, and is currently the Vice president of the Home Owner Association where he lives.

PREFACE

These study notes started in 1990 when I was teaching Physical Therapy Biomechanics and Occupational Therapy Functional Anatomy at Florida A&M University. Since that time, these have been revised 6 times. Major revisions were made in 1995 when the notes were adapted for the Biomechanics course at the Institute of Physical Therapy (now the University of St Augustine). Another major revision occurred in 2003 when study questions were added and references updated. In 2006 and 2011, the text, study questions, references and illustrations were reviewed and updated. In the current 2016, the title was modified, learning activities were added, extensive revisions made to the illustrations and text.

This study guide for Biomechanics was developed as the primary source of information for courses for Physical and Occupational Therapy students at the University of St Augustine for Health Sciences. Embedded is the guide are study questions and simple activities associated with joint movement. There are two parts to this study guide. Part 1 of the guide begins with a section on basic mechanics. Next, the mechanics of upper extremity movements are described for the shoulder complex, elbow complex, wrist complex and hand. This section is followed by the mechanics of the lower extremity, including the hip, knee, ankle complex, foot and the mechanics of gait. Partg 2 describes the mechanics of the spine, the mechanics of a good posture and the movements of the temporomandibular joint. The final sections of part 2 describe the mechanics associated with connective tissues, cartilage, bone, muscle and nerve and the effects of aging on these tissues.

I am most grateful Drs. Hilmir Augustsson, Kunal Bhanot, Steve Laslovich, Stanley Paris, Catherine Patla, Alec Stenhouse, and Jim Viti for their useful comments, help and support with this study guide and for the feedback from the many students who have studied from these materials over the years. I am also very grateful for the University of St. Augustine for Health Sciences their support and assistance.

I wish to dedicate this work to Dr Frank Kallen and Dr Carl Gans for their valuable mentorship and wisdom early in my academic career and to all those authors and researchers who have advanced our understanding in the biomechanics of the Human body.

Gerard Gorniak
Retired Professor
The University of St Augustine for Health Sciences

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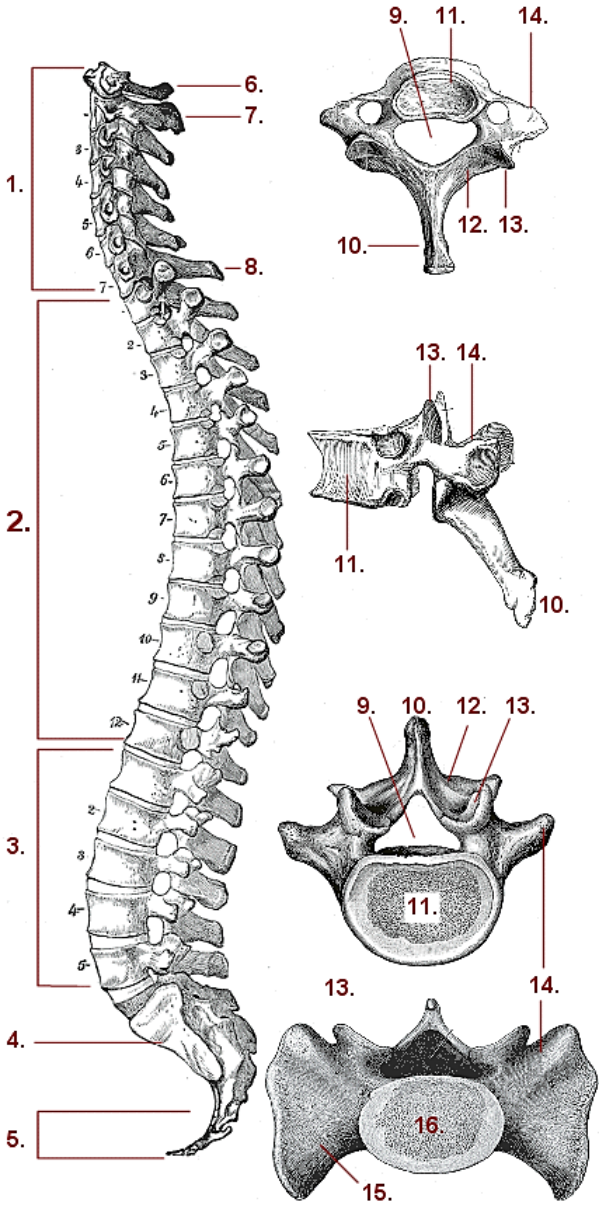
1 THE SPINE

1.1 ANATOMY OF THE SPINE

- OSTEOLOGY



Drawings of the spinal column, and typical cervical, thoracic, lumbar and vertebrae and the sacrum. 1. Cervical spine. 2. Thoracic spine. 3. Lumbar spine. 4. Sacrum. 5. Coccyx. 6. Posterior arch of Atlas. 7. Spinous process of C2. 8. Vertebra prominence (C7). 9. Vertebral foramen. 10. Spinous processes. 11. Vertebral bodies. 12. Laminae. 13. Superior articular processes. 14. Transverse processes. 15. Sacral ala. 16. Body of sacrum (Modified from Gray 1918)

- **JOINTS OF THE SPINE**

  - ATLANTO-OCCIPITAL AND ATLANTO-AXIAL JOINTS
    - The bilateral antlanto-occipital joints are formed by the condyles of the occipital bone of the skull and the atlas (C1) of the cervical spine.
    - The bilateral atlanto-axial joints are formed by the articulation between facets of the atlas (C1) and the axis (C2) of the cervical spine.

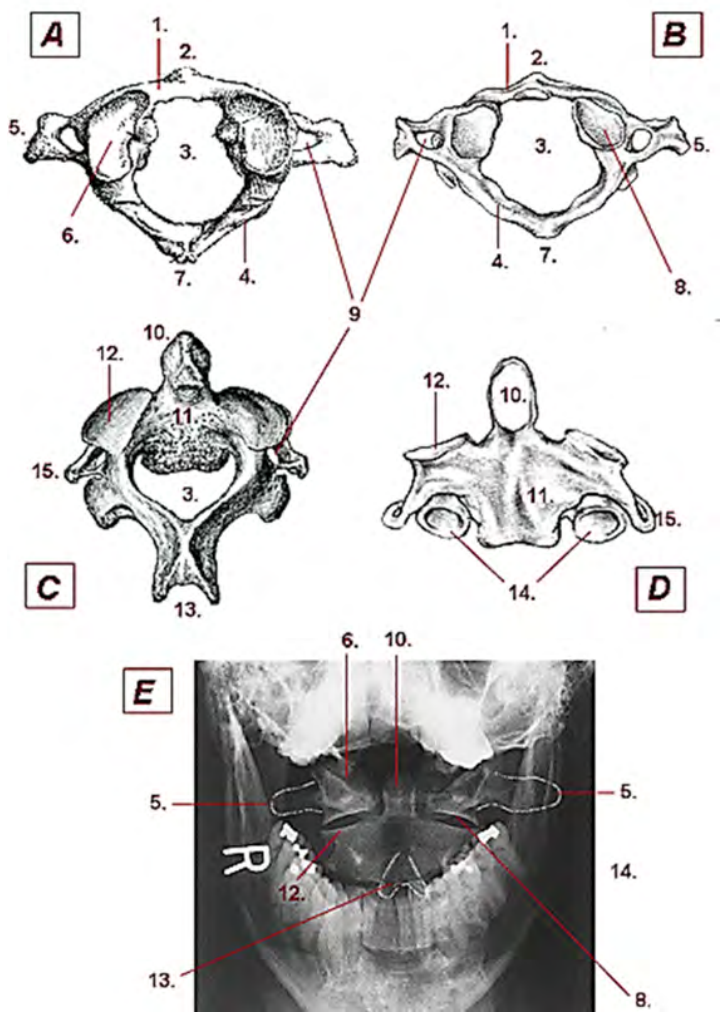


Image showing the osteology of the atlas in superior (A) and inferior (B) view; the axis in superior (C) and anterior (D) view. Radiograph (E) with the mouth open showing a frontal view of the dens, atlas, axis and atlantoaxial joint. The numbers relating to the atlas: 1. Anterior arch. 2. Anterior tubercle. 3. Vertebral foramen. 4. Posterior arch. 5. Transverse processes. 6. Superior articular facet. 7. Posterior tubercle. 8. Inferior articular facet. 9. Transverse foramen. The numbers relating to the axis: 10. Dens. 11. Body. 12. Superior articular facets. 13. Spinous process. 14. Inferior articular facets. 15. Transverse processes. (Modified from Gray 1918)

- ATLANTO-OCCIPITAL JOINTS
  - The articulation between the concave oval superior articular facets of the atlas and the rounded occipital condyles.
  - The articular plane is horizontal.

- LATERAL ATLANTO-AXIAL JOINTS
  - The articulation between flat circular inferior facets of the atlas and the flat circular superior facets of the axis.
  - The articular plane is horizontal.

- MEDIAN ATLANTO-AXIAL JOINT
  - The articulation between the dens of the axis and articular fossa for the dens on the posterior surface of the anterior arch of the atlas.
  - The articular plane is frontal.

- FACET OR ZYGOPOPHYSEAL JOINTS
  - The superior and inferior articular process of the vertebrae from C3 to L5 have an articular facet (face).
  - The facet surface of the inferior articular process of the vertebra above articulates with the facet of the superior articular process of the vertebra below.

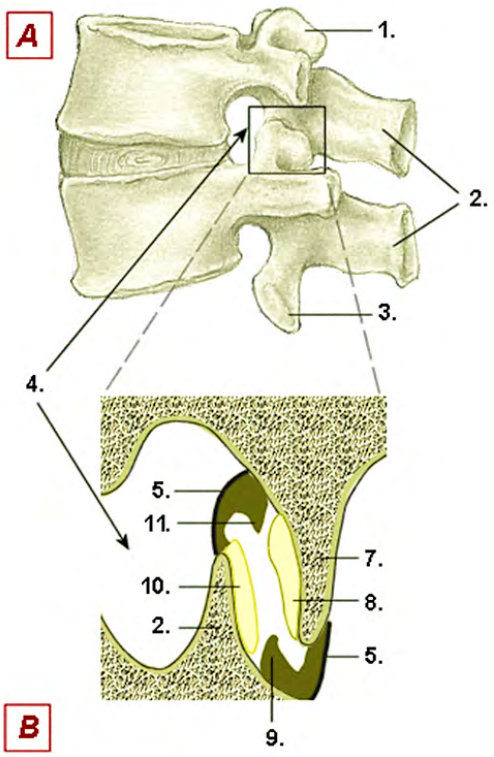


Diagram showing a sagittal section of a facet joint, with articular surfaces and menisci. 1. Superior articular process. 2. Spinous processes. 3. Inferior articular process. 4. Intervertebral foramen. 5. Joint capsule. 7. Cross section through inferior articular process. 8. Inferior articular facet. 9. Caudal meniscus. 10. Superior articular facet. 11. Cephalad meniscus.

- cervical region (C3–C7)
  + The articulating superior and inferior facets are flat and oval.
  + The inferior facets face anterioinferior and the matching superior facets face posteriosuperior.
  + Articular plane is frontal at an angle of about 45 degrees to horizontal.

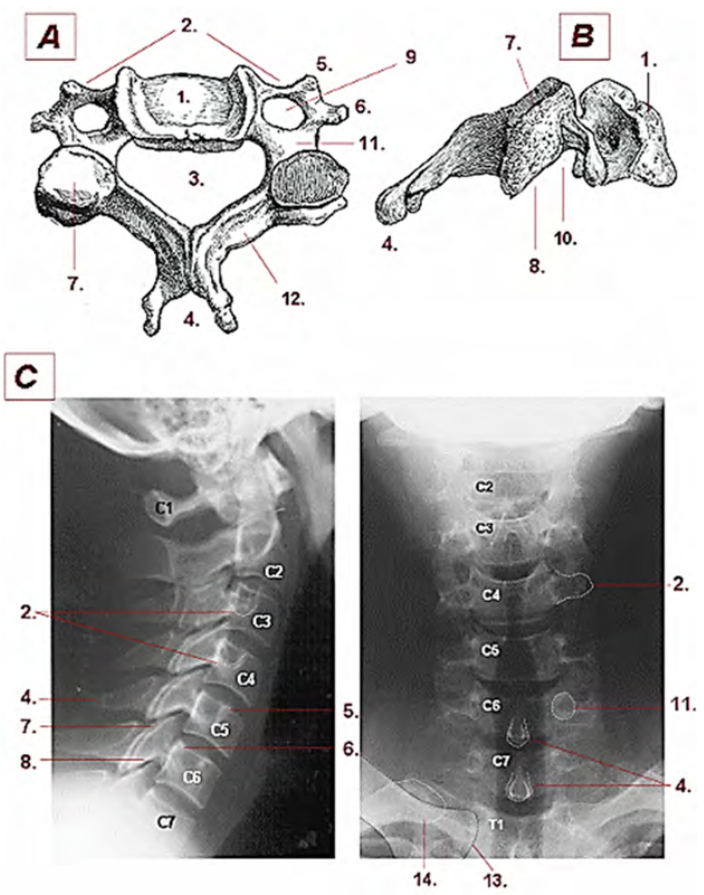
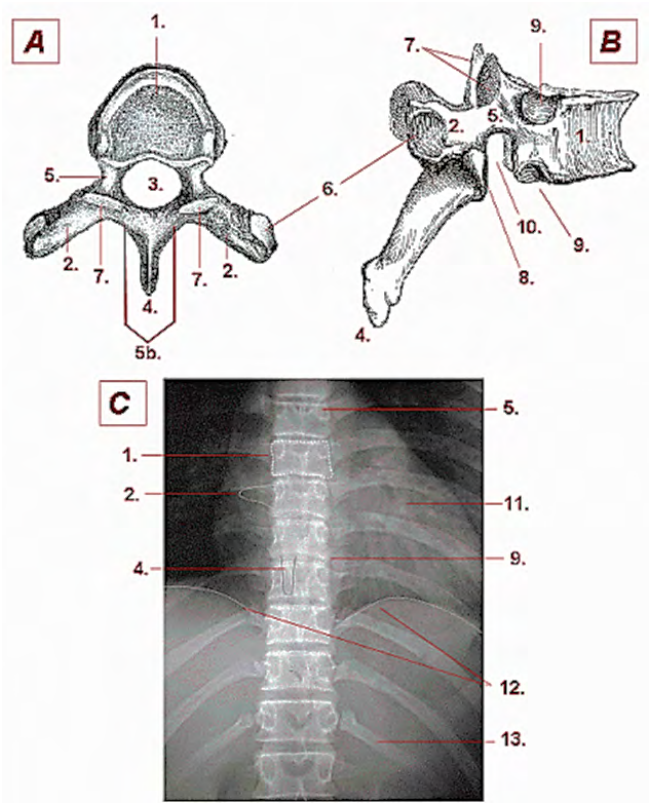


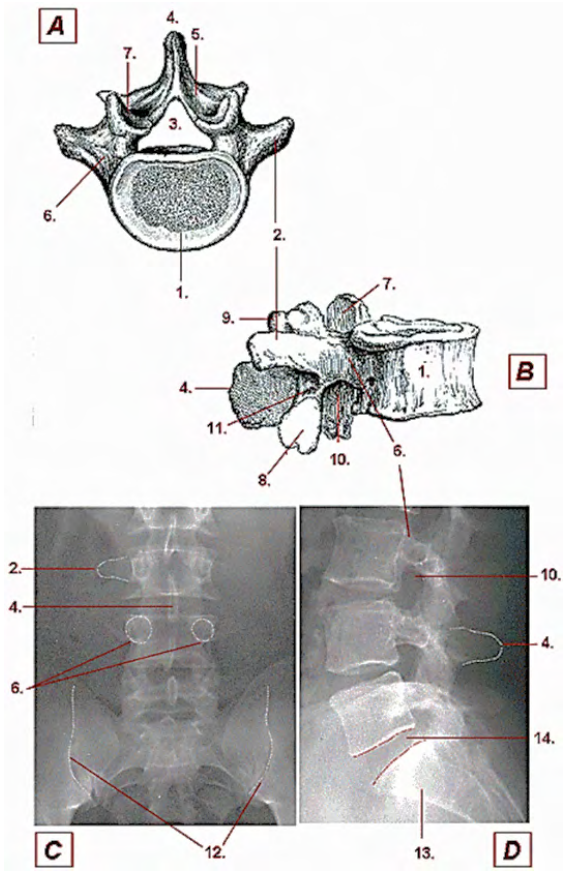
Image showing the structural parts of a cervical vertebra in superior (A) and lateral (B) views (Modified from Gray 1918). Radiographs of the cervical spine in lateral (C) and frontal (D) view. The numbers indicate: 1. Vertebral body. 2. Transverse processes. 3. Vertebral foramen. 4. Spinous process. 5. Anterior tubercle of transverse process. 6. Posterior tubercle. 7. Superior articular facets. 8. Inferior articular facets. 9. Transverse foramen. 10. Inferior vertebral notch. 11. Pedicle. 12. Lamina. 13. Clavicle. 14. First rib.

- thoracic region (T1–T12)
  + The articulating superior and inferior facets are flat and oval.
  + The inferior facets face anteriorly and the superior facets face posteriorly.
  + Articular plane is frontal at an angle of about 60 degrees to horizontal.



Drawings showing the osteology of a thoracic vertebra in superior (A) and lateral (B) view (Modified from Gray 1918) and a radiograph of the thoracic spine in frontal view (C). Notice the articulation of the ribs with the body and transverse process. The numbers in the image indicate: 1. Vertebral body. 2. Transverse processes. 3. Vertebral foramen. 4. Spinous process. 5. Pedicle. 5b. Laminae. 6. Costal tubercle facet. 7. Superior articular facet. 8. Inferior articular facet. 9. Costal facets on vertebral body. 10. Inferior intervertebral notch. 11. Heart. 12. Diaphragm. 13. Rib

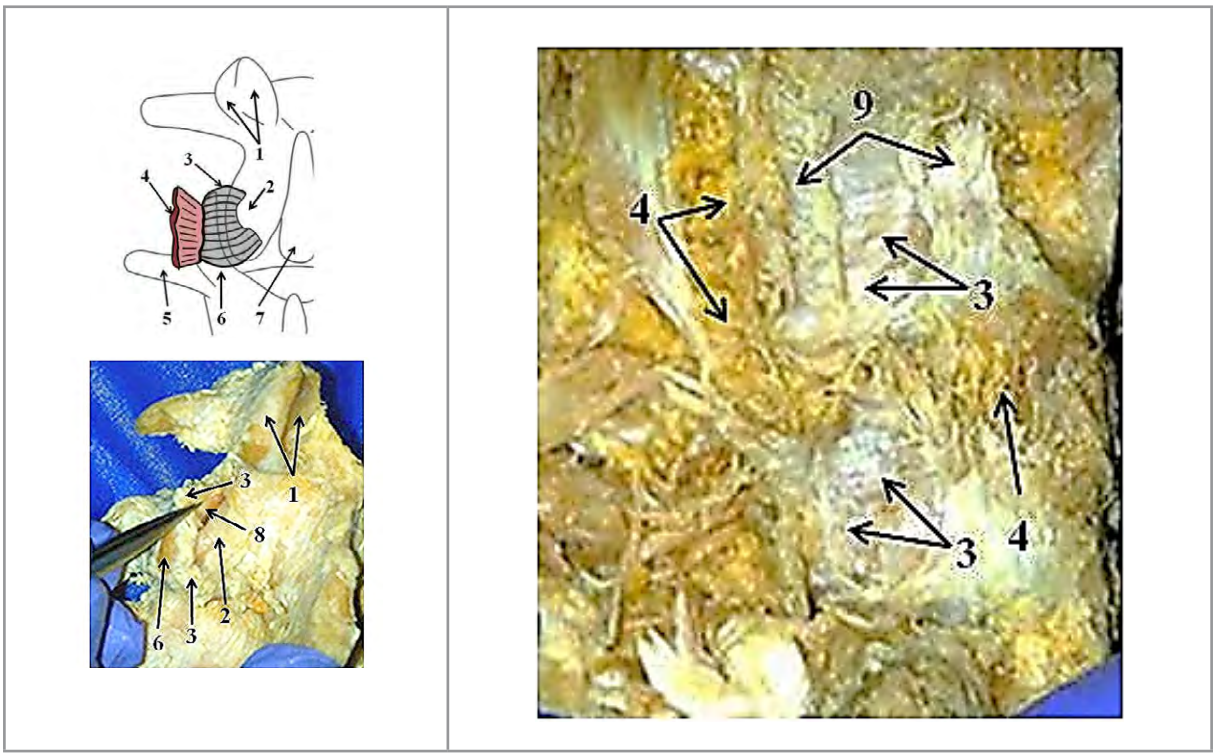
- upper lumbar region (L1–L2)
  + The superior facets are oval and slightly concave.
  + The inferior facets are oval and flat to slightly convex.
  + The inferior facets face laterally and the superior facets face medially.
  + The articular plane is sagittal at an angle of about 90 degrees to horizontal.
  + The joints have cephalad and caudal menisci that attach to joint capsule.
- lower lumbar region (L4–L5)
  + The superior facets are oval and slightly concave.
  + The inferior facets oval and flat to slightly convex.
  + The inferior facets of L4 face anteriolaterally and the superior facets of L4–L5 face posteriomedially.
  + The inferior facets of L5 face more anteriorly and less laterally than the inferior facets of L4.
  + The articular plane is diagonal to the sagittal and frontal planes at an angle of about 90 degrees to horizontal.
  + The joints have cephalad and caudal menisci that attach to joint capsule.



Drawings showing the osteology of a lumbar vertebra in superior (A) and lateral (B) view (Modified from Gray 1918). Radiographs of the lumbar spine and sacrum in frontal (C) and lateral (D) view: 1. Vertebral body. 2. Transverse processes. 3. Vertebral foramen. 4. Spinous process. 5. Lamina. 6. Pedicle. 7. Superior articular facet. 8. Inferior articular facet. 9. Mammillary process. 10. Inferior intervertebral notch. 11. Pars interarticularis. 12. Sacroiliac joints. 13. Sacrum, 14. Vertebral disc

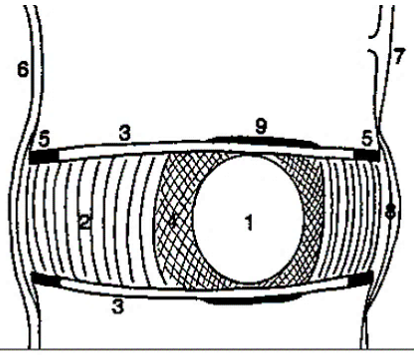
Vertebral movements are guided by the angulation of the matching articular facets between vertebrae and by ligaments. For example, the articular surfaces of the upper lumbar vertebrae lie in the sagittal plane at a 90 degree angle to the horizontal so that the inferior facet of the vertebra above lies directly medial to the superior articular facet of the vertebra below. This arrangement restricts rotation of the lumbar spine, but allows forward and lateral flexion and extension. In contrast, the articular surfaces of the cervical spine (C3–C7) lie in the frontal plane at a 45 degree angle to horizontals. This arrangement permits extensive rotation as well as flexion, extension and lateral flexion of the cervical spine. In the thoracic region, the articular surfaces lie in the frontal plane. This alignment permits rotation, flexion, extension and lateral flexion. However, the attachment of the ribs to the vertebrae limit the magnitude of these movements.

The articular cartilage of the facet joints is hyaline cartilage and it tends to be thick centrally and thin peripherally. The joint capsule is dense irregular connective tissue containing collagen and elastic fibers. The collagen fibers tense to limit the extent of facet joint movement. These fibers limit joint distraction, rotation, and the superior translation of the inferior facets on the superior facets of the vertebra below as during forward spinal flexion. The elastic fibers 1) prevent the capsule and synovial membrane from being pinched between the opposing facets, 2) allow for movement between the facets, and 3) return the capsule to its starting position. In the lumbar spine, the facet joints have a short cephalad and a short caudal menisci. These menisci can be trapped between the opposing articular surfaces, producing a painful blockage of motion at that vertebral segment.



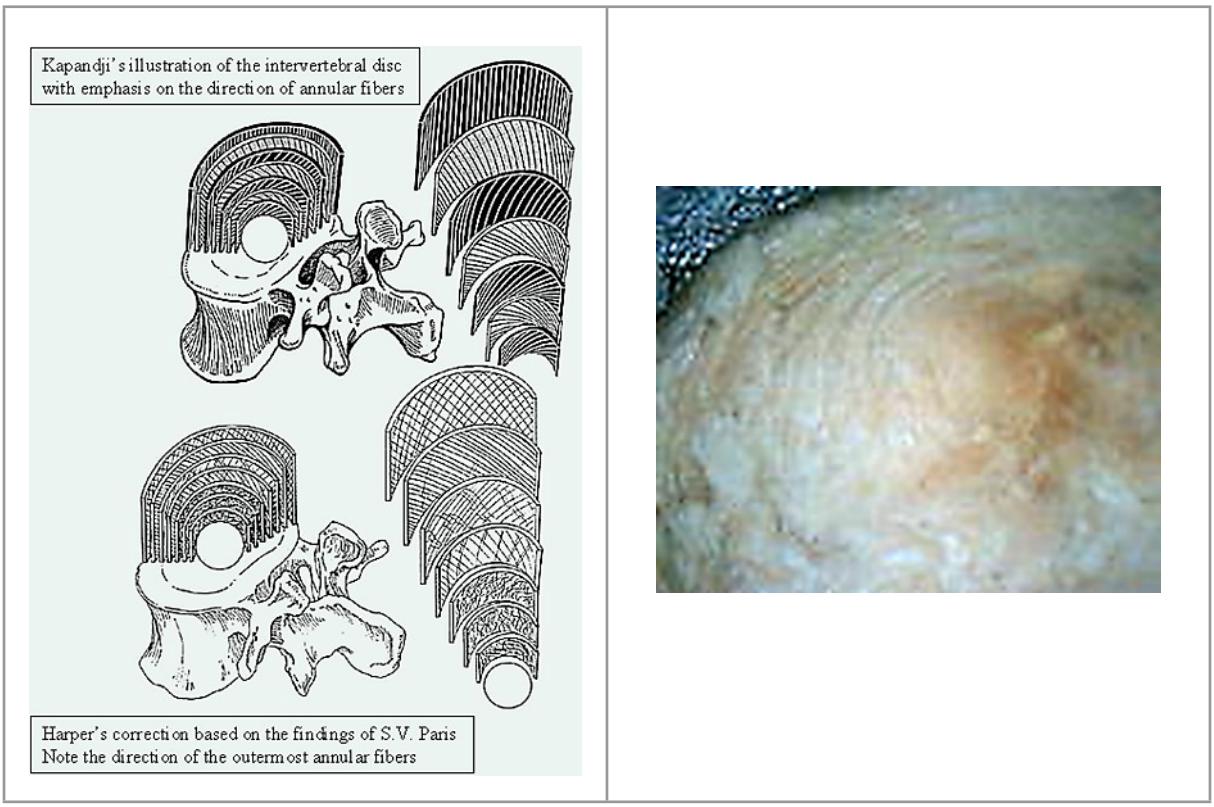
(Top Left) Drawing of a lumbar facet joint showing the capsule and attachment of the multifidus muscle. (Bottom Left) Photograph of a lumbar facet joint showing the cephalad minsicus (forceps). (Right) Photograph of 2 lumbar facet joints showing the joint capsule and multifidus muscle. 1. Superior articular process and facet, 2. Inferior articular process, 3. Joint capsule (cut in bottom left figure), 4 Multifidus muscle cut and reflected laterally, 5. Transverse process, 6. Superior articular process, 7. Spinous process, 8. Cephalad minicus held with forceps, 9. Tendonous fascia from the multifidus muscle covering the posterior joint capsule.

- **INTERVERTEBRAL DISC**
  - The intervertebral disc is a fibrocartilaginous joint between vertebral bodies.
  - The disc consists of peripheral annulus fribrosus and central nucleus pulposus.
  - Annulus fibrosus
    - The annulus has 6–10 circular rings of fibrocartilage.
    - Collagen fibers in layers surrounding nucleus pulposus are arranged loosely.
    - Collagen fibers in the outer layers are densely packed and run obliquely between the vertebral bodies.
    - Collagen fibers in the outer-most 1–2 layers have a crossing herringbone pattern which makes these layers strong in resisting tension.
    - The peripheralmost fibers attach to the smooth edge (rim) of the vertebral body with the outermost fibers attaching to the periosteum and the anterior and posterior longitudinal ligaments.
    - The other fibers attach to the hyaline cartilaginous end plate covering the rough articular surface of the vertebral body.



A diagrammatic representation of the intervertebral disc showing:
1. Nucleus
2. Annulus fibrosus
3. Cartilaginous end plates
4. Irregular connective tissue zones surrounding the nucleus
5. The epiphyseal rim
6. Anterior longitudinal ligament, continuous with the periosteum
7. Posterior longitudinal ligament seperating from the vertebral body
8. Presence of Sharpey's fibers
9. Thickened calcified layer capping the nucleus

Drawing of the intervertebral disc showing its composition and its relationships to the vertebral bodies and the anterior and posterior longitudinal ligaments (Paris and Loubert, 1990)



Drawings of the layers of the annulus fibrosus showing the arrangements of collagen fibers in each row by Kapandji (UPPER LEFT) and by Harper based on Paris (LOWER LEFT). Picture of a dissected intervertebral disc (RIGHT) showing the brownish nucleus pulposus and the whitish rings of the annulus.

- FUNCTIONS OF THE ANNULUS FIBROSUS
  - containment of the nucleus pulposus
  - stabilization of the vertebrae
  - permits multidirectional vertebral movements
  - shock absorption

- NUCLEUS PULPOSUS
  - It is a gelatinous mass consisting of collagen fibers imbedded in a mucopolysaccharide and water gel.
  - It can absorb and retain large quantities of water.
  - It is important for the exchange of nutrients between the disc and the vertebral bodies.
  - In the cervical and thoracic regions, the nucleus pulposus lies centrally within the disc.
  - In the lumbar region, the nucleus lies in the posterior one-half of the disc.

- FUNCTIONS OF THE NUCLEUS PULPOSUS
  - The absorption and retention of water.
  - Nutrition and waste removal of the annulus fibrosus.
  - Force transmission between vertebral bodies.
  - Equalizes unit stress in all directions to the annulus fibrosus.
  - Permits a "rocking" type segmental movements between vertebral bodies.

- SEGMENTAL "ROCKING" between vertebral bodies

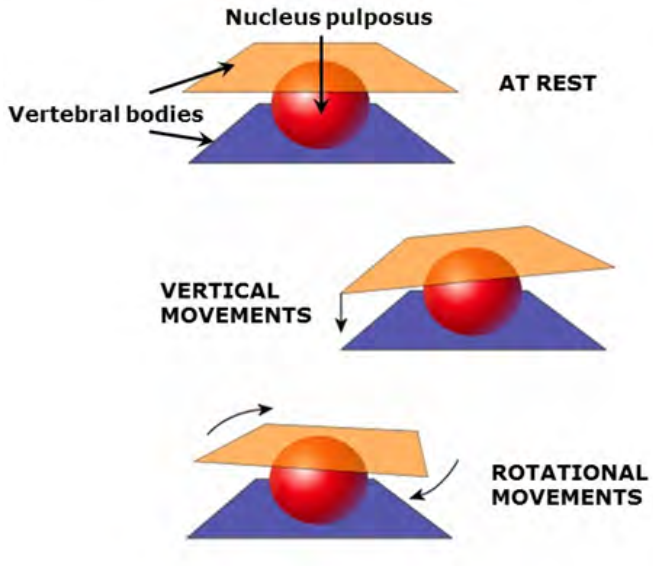


Diagram showing the "rocking" movements of the vertebral bodies permitted by the nucleus pulposus at rest (TOP), with vertical movements associated with flexion, extension and side bending or side flexion (MIDDLE) and rotational movements (BOTTOM). (Modified from Paris and Loubert. 1990)

- THE NUTRITIONAL CYCLE FOR INTERVERTEBRAL DISC
  - For nutrition of the intervertebral disc, movements between adjacent vertebrae and the ability of the annulus fibrosus and nucleus pulposus to expand are needed.
  - The nucleus pulposus expands at rest by drawing water and nutrients from blood vessels and lymphatics of the vertebral bodies.
  - When the nucleus pulposus is loaded by movement of the vertebral bodies, nutrients are forced out into the annulus fibrosus and waste products out of the nucleus into the vertebral bodies.
  - The flows of fluid inwardly during rest and outwardly during loading provide for the nutritional needs of the intervertebral disc and waste removal.

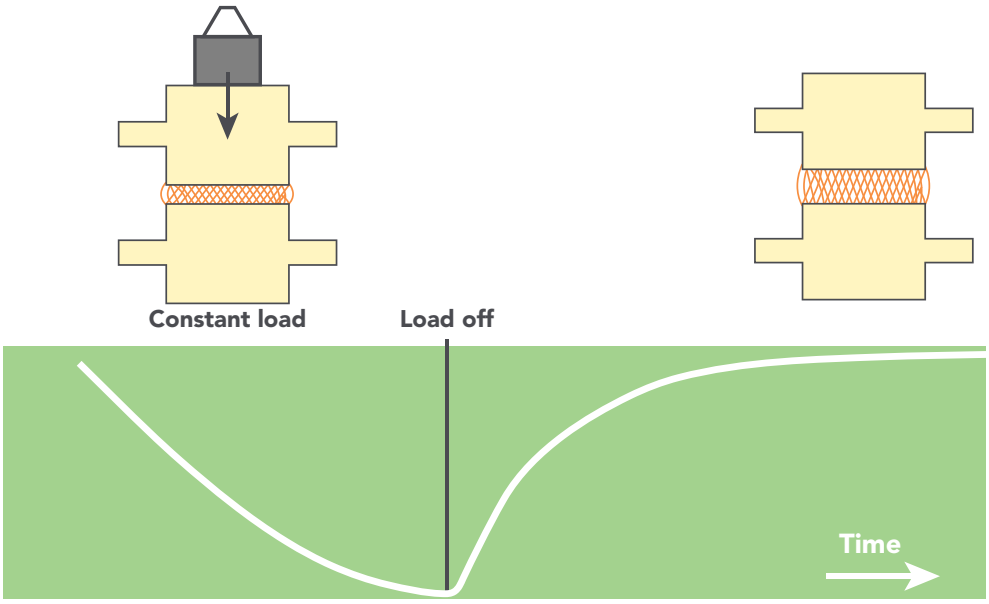


Diagram showing the change in intervertebral disc fluids when the disc is in constant load and when the disc is unloaded (modified from Paris and Loubert, 1990)

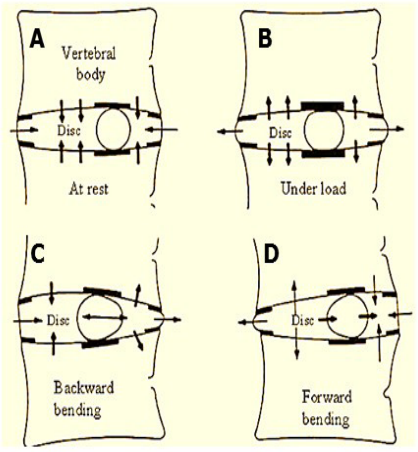
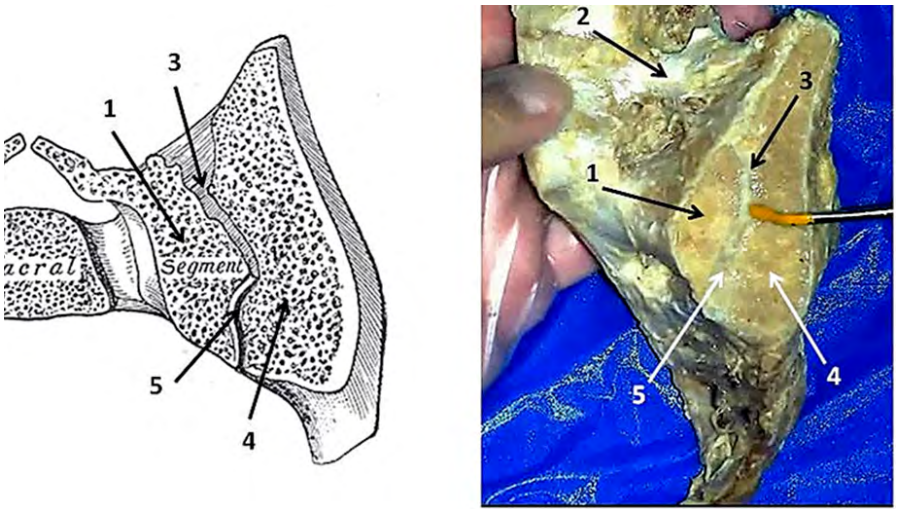


Diagram showing the directions of fluids (arrows) when (A) the disc is at rest and filling with nutrients; (B) when the disc is loaded and the nutrients and waste products are forced into the annulus and vertebral bodies; (C) when the disc is loaded in backward bending and the fluids are being absorbed anteriorly and squeezed out posteriorly;
(D) when the disc is loaded in forward bending and the fluids are being absorbed posteriorly and squeezed out anteriorly (Paris and Loubert. 1990)

- **SACROILIAC JOINT**

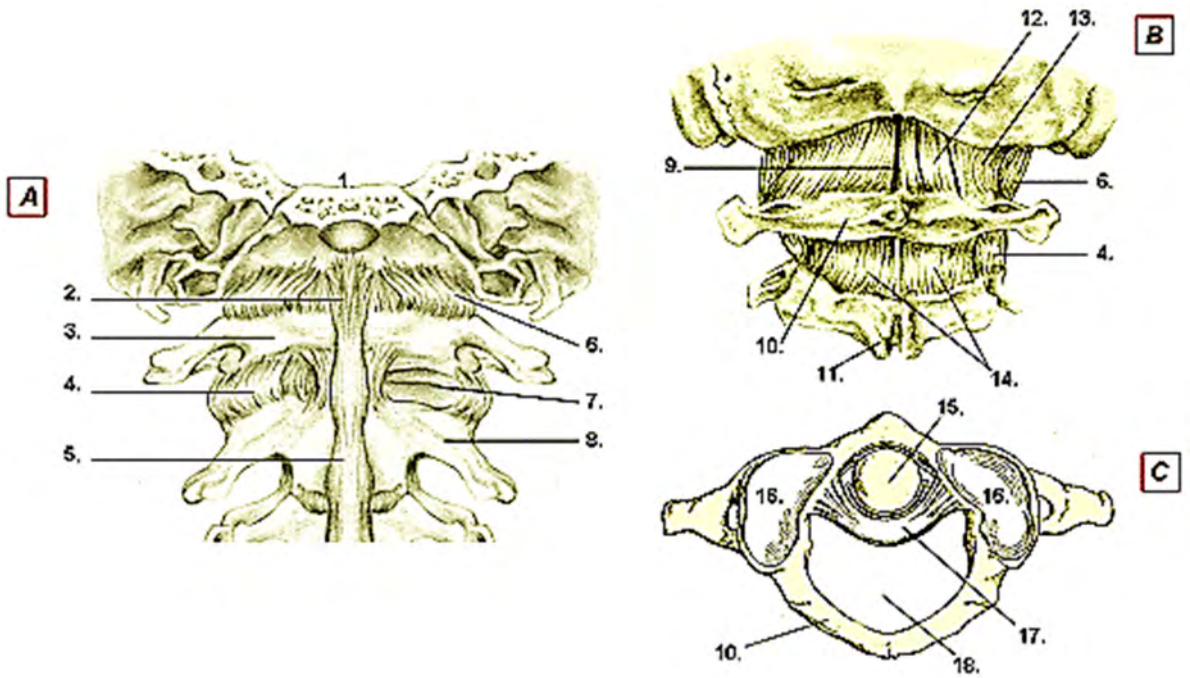
  - Consists of a synovial and fibrous articulation.
  - SYNOVIAL ARTICULATION between the auricular surface of the sacrum at the level of S1, S2, S3 and the auricular surface of the ilium lying anterior to the posterior superior iliac spine and inferior to the iliac tuberosity.
  - FIBROSUS ARTICULATION is posterior and superior to the auricular surfaces and contains the strong interosseous sacroiliac ligament that connects the iliac and sacral tuberosity.
  - Both auricular surfaces for the synovial part have an irregular pattern of ridges and grooves and both are covered with a thin layer of hyaline cartilage.



(LEFT) Drawing of an oblique transverse section of the sacroiliac joint (modified from Gray 1918). (RIGHT) Photograph of an oblique sagittal section of the sacroiliac joint. 1. Sacrum, 2. Iliolumbar ligament, 3. Fibrous interosseous part of the joint, 4. Ilium, 5. Hyaline cartilage of the synovial part of the joint.

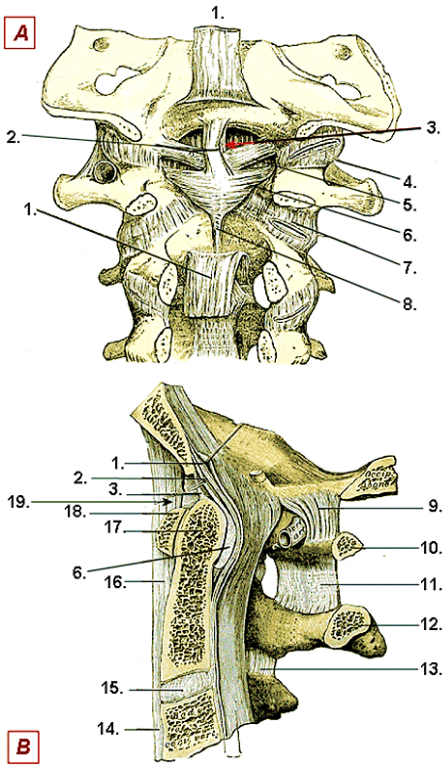
- **LIGAMENTS OF THE SPINE**

  - LIGAMENTS OF THE ATLANTO – OCCIPITAL AND ATLANTO – AXIAL JOINTS
    - **Anterior atlanto-occipital membrane** connects the anterior arch of the atlas with the base of the occipital bone and is continuous laterally with the atlanto-occipital joint capsule.
    - **Posterior atlanto-occipital membrane** connects the posterior arch of the atlas with the occipital bone and is continuous with the lateral atlanto-occipital ligament.
    - **Anterior atlanto-axial membrane** connects the body of the axis to the anterior arch of the atlas and is continuous with the capsule of the atlanto-axial joint.



Drawings of the anterior atlanto-occipital and atlanto-axial ligaments (A), the atlas in superior view (C) showing the articulation of the dens and the transverse ligament, and the posterior atlanto-occipital and atlanto-axial membrane (B). 1. Occipital bone. 2. Anterior longitudinal ligament covering anterior atlanto-occipital membrane. 3. Anterior arch of atlas. 4. Atlanto-axial joint capsule. 5. Anterior longitudinal ligament. 6. Lateral atlanto-occipital ligament. 7. Atlanto-axial joint. 8. C3 vertebra. 9. Posterior atlanto-occipital membrane. 10. Posterior arch of atlas. 11. Spinous process of axis (C2). 12 medial and 13. lateral occipito-atlantal ligament. 14. Posterior atlanto-axial membrane. 15. Dens. 16. Superior articular facet of atlas. 17. Transverse ligament of atlas. 18. Vertebral foramen (Modified from Gray 1918).

- **Posterior atlanto-axial membrane** connects the posterior body of the axis to the posterior arch of the atlas and is continuous with the capsule of the atlanto-axial joint.
- **Lateral atlanto-occipital ligament** connects the transverse process of the atlas with the occipital bone and strengthens the atlanto-occipital joint.



Images of (A) the cruciform and alar ligaments in posterior view with the tectorial membrane removed and (B) a midsagittal view of the atlanto-axial (A/A) joint showing the position of the posterior ligaments. 1. Tectorial membrane. 2. Superior longitudinal ligament of cruciform (retracted). 3. Apical ligament. 4. Atlanto-occipital (A/O) articular capsule. 5. Alar ligament. 6. Transverse ligament of atlas. 7. A/A articular capsule. 8. Inferior longitudinal ligament of cruciform. 9. Posterior A/O membrane. 10. Posterior arch of atlas (cut). 11. Posterior A/A membrane. 12. Spinous process of axis (cut). 13. Ligamentum flavum. 14. Anterior longitudinal ligament. 15. Intervertebral disc. 16. Anterior A/A membrane. 17. Dens (cut). 18. Anterior arch of atlas (cut). (Modified from Gray 1918)

- **Tectorial membrane** is the continuation of the posterior longitudinal ligament connecting the posterior arch of the axis to the occipital bone and covering the cruciform, alar and apical ligaments.
- **Cruciform ligament** consists of the **transverse ligament of the atlas** and extending vertically from it the **superior band** attaching to the occipital bone and the **inferior band** attaching to the body of the axis.
- **Transverse ligament of the atlas** cups the posterior surface of the dens and attaches to the posterior surface of the anterior arch of the atlas on each side of the median atlanto-axial joint.
- **Alar ligaments** (paired) attach to the sides and posterior surface of the dens and run laterally with a superior band attaching to the occipital bone, a middle band attaching to the lateral mass of the atlas and an inferior band attaching to the axis.
- **Apical ligament** lies anterior to the superior band of the cruciform ligament and connects the apex of the dens to the occipital bone.

- VERTEBRAL LIGAMENTS

  - **Anterior longitudinal ligament** attaches to the anterior rim of the vertebral bodies and anterior aspect of the intervertebral disc as it runs from the sacrum to the atlas where it is continuous with the anterior atlanto-occipital membrane.
  - **Posterior longitudinal ligament** attaches to the posterior rim of the vertebral bodies and posterior aspect of the intervertebral disc as it runs along the anterior surface of the spinal canal from the sacrum to the axis where it (is) becomes the tectorial membrane.
  - **Ligamenta flava** are paired segmental elastic ligaments that lie on the posterior aspect of the spinal canal from the sacrum to the axis and interconnect the lamina of adjacent vertebrae.
  - **Supraspinous ligament** attaches to the tips of the spinous processes from C7 where it is continuous with the ligamentum nuchae to about L4 where it is replaced by the erector spinae fascia.

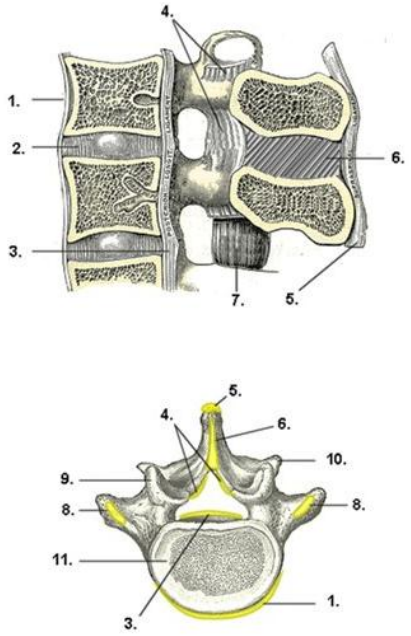


Image showing the lateral view of the lumbar spine (TOP)) with the major vertebral ligaments and the superior view of a vertebra (BOTTOM) showing the location of these ligaments relative to a vertebra. 1. Anterior longitudinal ligament. 2. Intervertebral disc. 3. Posterior longitudinal ligament. 4. Ligamentum flavum. 5. Supraspinous ligaments. 6. Interspinous ligaments. 7. Anteromedial capsule of facet joint. 8. Intertransverse ligaments. 9. Superior articular process. 10. Inferior articular process. 11. Vertebral body. (Modified from Gray 1918)

  - **Interspinous ligaments** interconnect the inferior and superior aspects of adjacent spinous processes from C7 where the ligamentum nuchae arises to L5/S1.
  - **Ligamentum nuchae** is a complex fibrous septum that runs along the posterior midline of the neck from C7 to the occiput interconnecting the tips and superior and inferior aspects of the cervical spinous processes to the occipital bone.

- **Intertransverse ligament** connects the transverse processes of adjacent vertebrae.

o VERTEBRAL LIGAMENTS DURING FLEXION AND EXTENSION

- During flexion, the anterior longitudinal ligament slackens and the supraspinous ligament, interspinous ligament, ligamentum flava and in the neck the ligamentum nuchae tighten.
- During extension, the anterior longitudinal ligament tightens and the supraspinous ligament, interspinous ligament, ligamentum flava and in the neck the ligamentum nuchae slackens.
- The posterior longitudinal ligament changes only slightly because it is close to the axis of motion for flexion and extension.

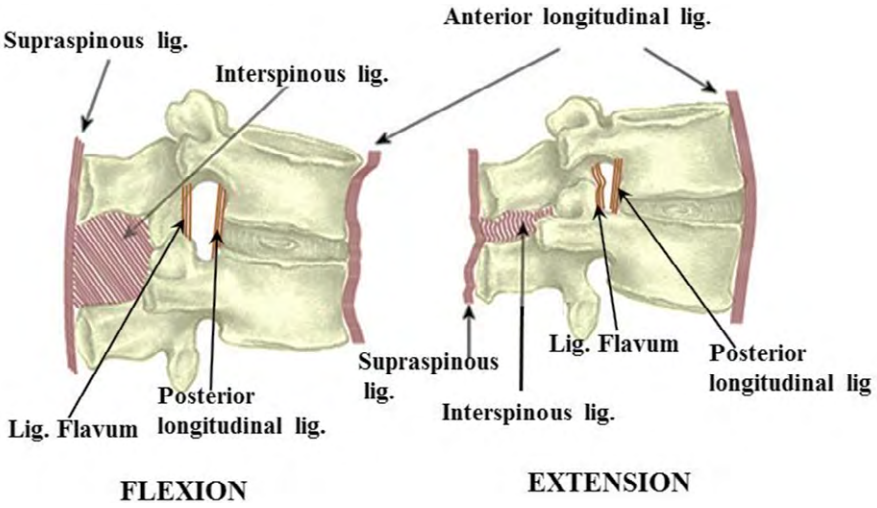
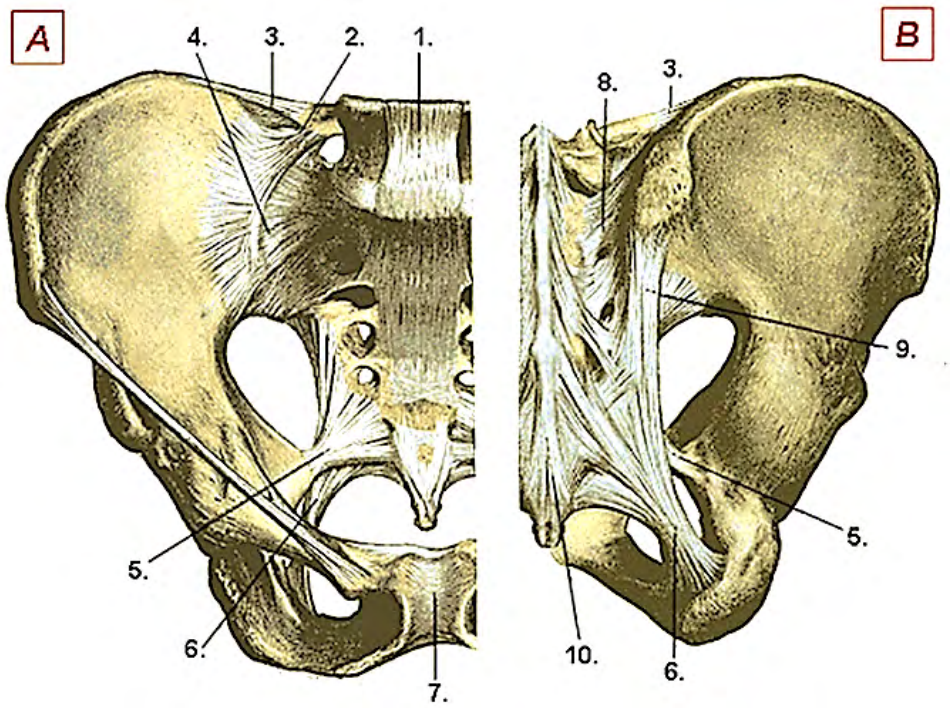


Diagram showing the responses of the vertebral ligaments (lig.) during spinal flexion and extension

- SACROILIAC LIGAMENTS

  - **Iliolumbar ligament**, which begins as a muscle but becomes ligamentous by age 40, runs from the transverse process of L5 in males and L4–L5 in females to the superior SIJ and ilium.
  - **Sacrotuberous ligament** runs obliquely from the posterior surface of the posterior inferior iliac spine, and posterolateral aspect of the lower sacrum and upper coccyx to the medial aspect of the ischial tuberosity and the ramus of the ischium.
  - **Sacrospinous ligament** runs obliquely from posterolateral surface of the lower sacrum and coccyx to the ischial spine.



Images, in anterior (A) and posterior (B) view, showing the iliolumbar and sacroiliac ligaments. 1. Anterior longitudinal ligament. 2. and 3. Iliolumbar ligament. 4. Anterior sacroiliac (SI) ligament. 56. Sacrospinous ligament. 6. Sacrotuberous ligament. 7. Pubic symphysis 8. Short dorsal SI ligaments. 9. Long dorsal SI ligament. 10. Supraspinal ligament. (Modified from Gray 1918)

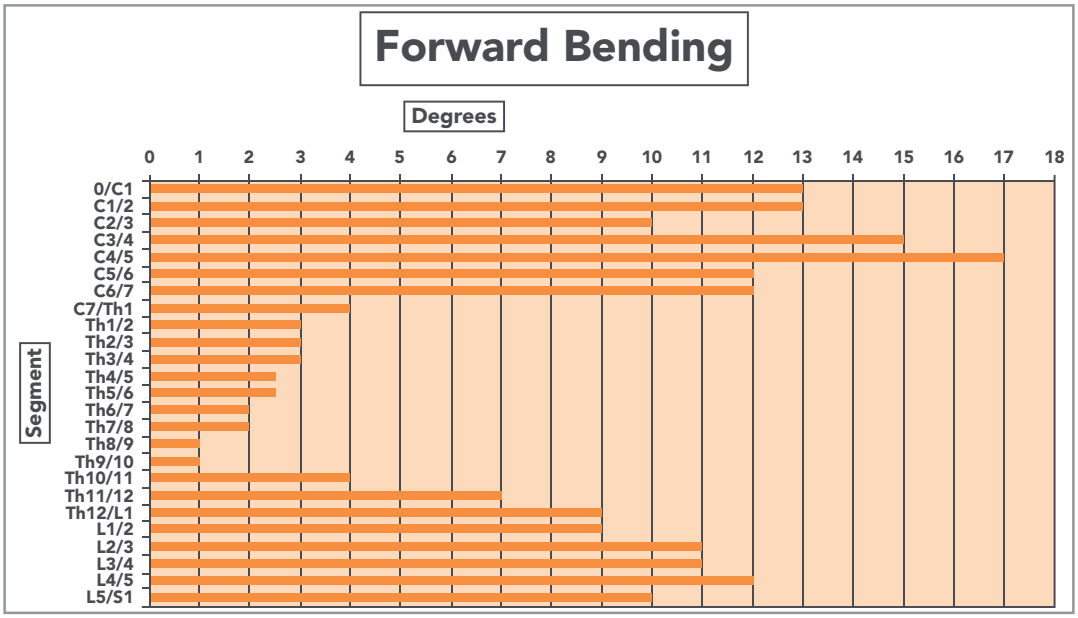
- **Interosseous sacroiliac ligament** connects the iliac and sacral tuberosities at the fibrosus part of the SIJ.
- **Short dorsal sacroiliac ligament** runs horizontally from the dorsolateral aspect of the superior part of the sacral tuberosity to the dorsal aspect of the tuberosity of the ilium.
- **Long dorsal sacroiliac ligament** runs obliquely from the dorsolateral aspect of the inferior part of the sacral tuberosity and the dorsal surface of the sacrotuberous ligament to the posterior superior iliac spine.
- **Ventral sacroiliac ligament** runs horizontally from the ventrolateral margin of the sacrum to ventral aspect of the auricular surface of the ilium.

1.2 MOVEMENTS OF THE SPINE

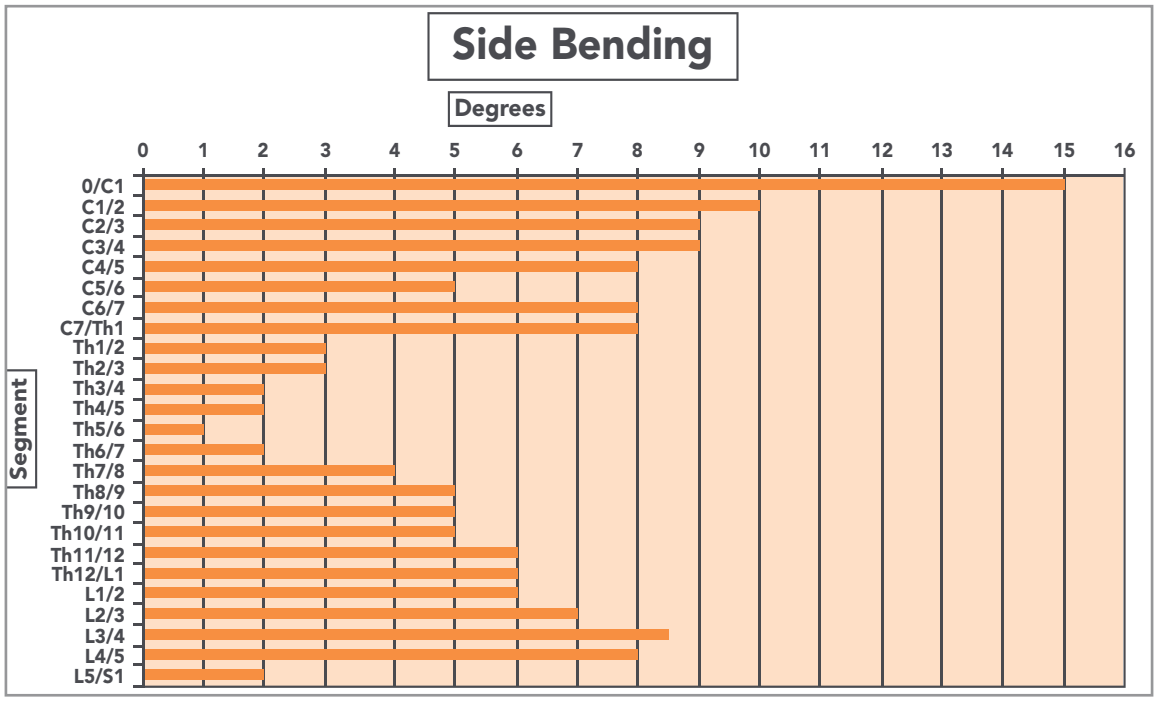
- **DEGREES OF VERTEBRAL SEGMENTAL MOVEMENT**

  - FUNCTIONAL MOTIONS OF THE SPINE
    - These are normal and usual motions of the spine.
    - These motions usually occur diagonally or obliquely to the cardinal planes.

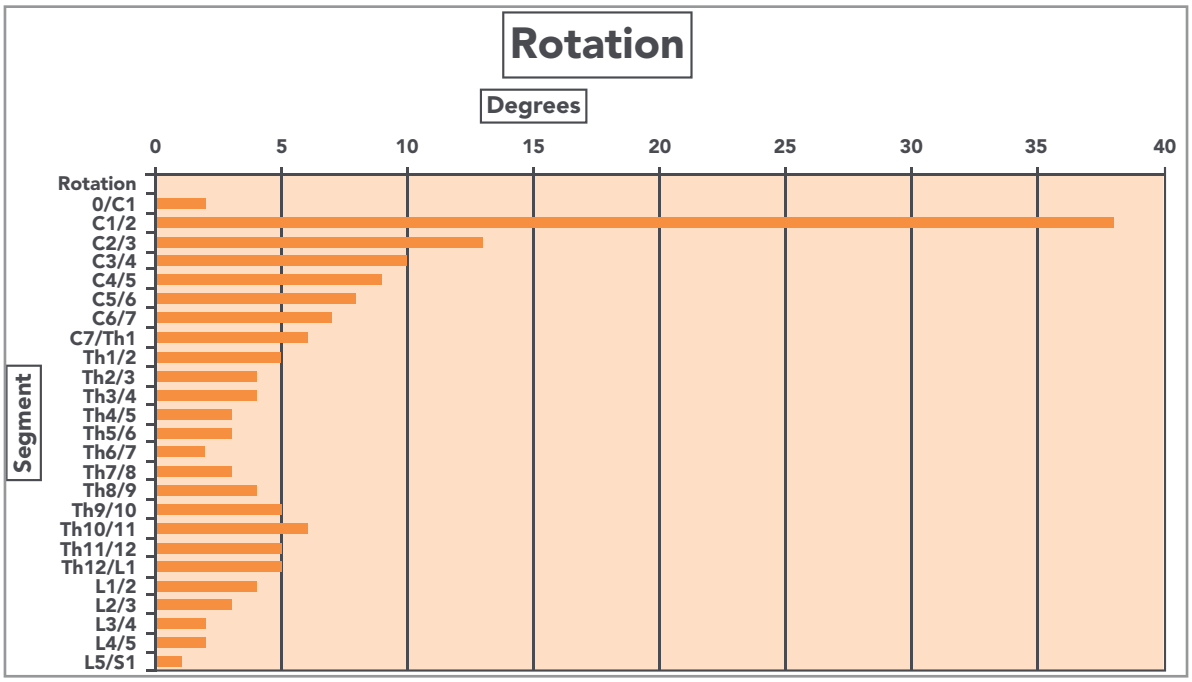
- NON-FUNCTIONAL MOTIONS FO THE SPINE
  - These motions ocurr in one of the cardinal planes.
  - These motions are usually used for evaluations.



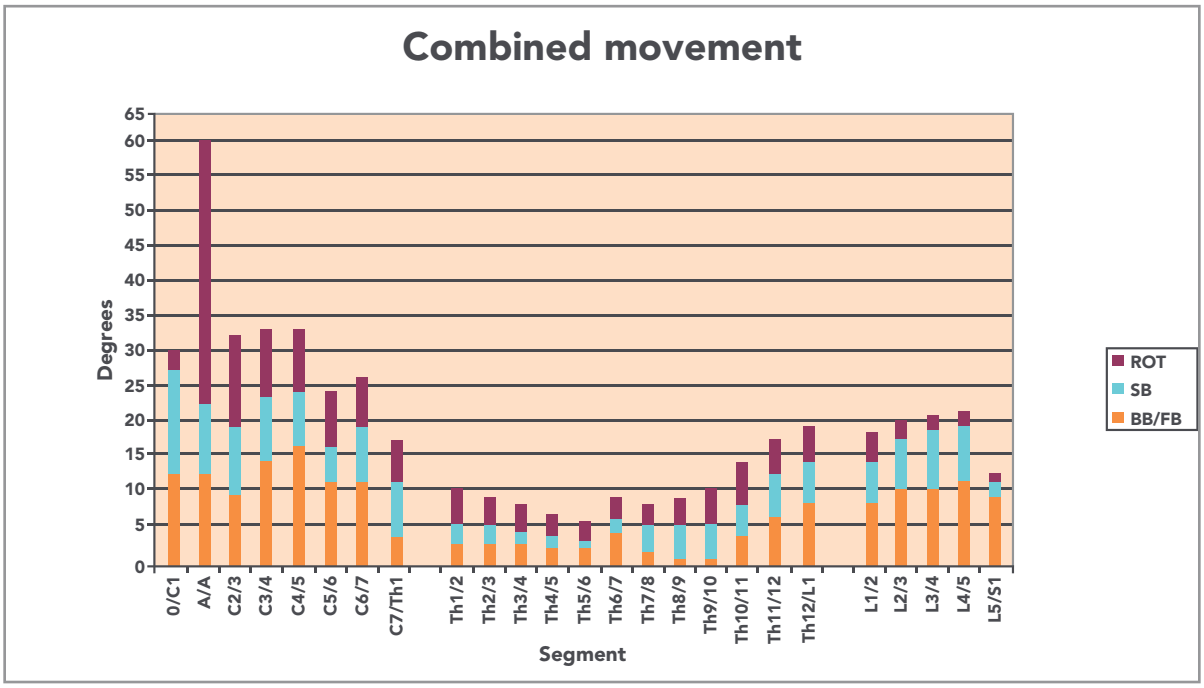
Graph showing the degrees of forward bending (Flexion) at each vertebral segment from the atlanto-occipital joints to the facets joints of L5–S1



Graph showing the degrees of side bending (Lateral flexion) at each vertebral segment from the atlanto-occipital joints to the facets joints of L5–S1



Graph showing the degrees of rotation at each vertebral segment from the atlanto-occipital joints to the facets joints of L5–S1



Graph showing the degrees of rotation (red), side bending (green) and forward bending (blue) at each vertebral segment from the atlanto-occipital joints to the facets joints of L5–S1

- **ARTHROKINEMATIC FACET JOINT MOVEMENTS**

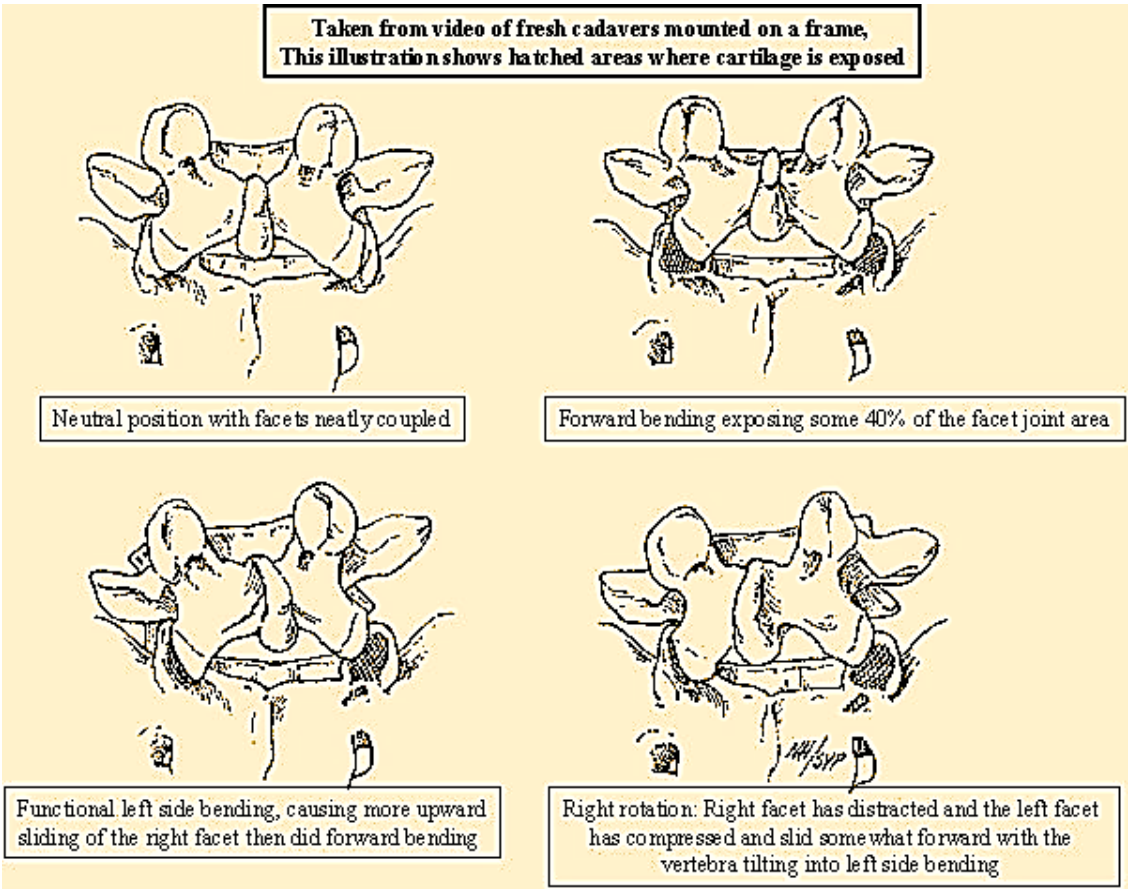
  - Depending on the spinal movements, the pattern of facet movements may be the same bilaterally or different bilaterally.
  - Below is a table, drawings and photographs showing the movements of the facets.

Table describing the bilateral movements of the condyles at the atlanto-occipital joints and the facets at the atlanto-axial, cervical, thoracic and lumbar spine during flexion, extension, side bending (side flexion) and rotation.

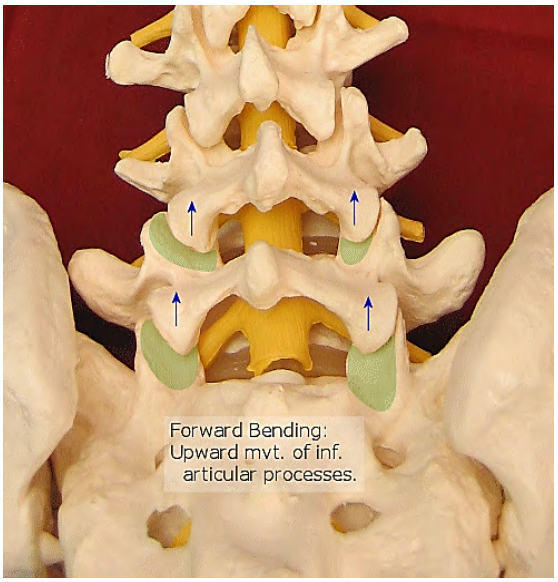
REGION	FLEXION	EXTENSION	SIDE BENDING RIGHT	ROTATION RIGHT
ATLANTO – OCCIPITAL	ROC and LOC rotate anteriorly and glide posteriorly	ROC and LOC rotate posteriorly and glide anteriorly	ROC and LOC roll right and glide left	ROC moves slightly back and LOC moves slightly forward
ATLANTO – AXIAL	RF and LF moves forward	RF and LF moves backward	Atlas sides right	RF moves back and LF moves forward
CERVICAL C2/3 –T2/3	RF and LF slide up and forward	RF and LF slide down and back	RF slides down and back and LF slides up and forward	RF slides down and back and LF slides up and forward

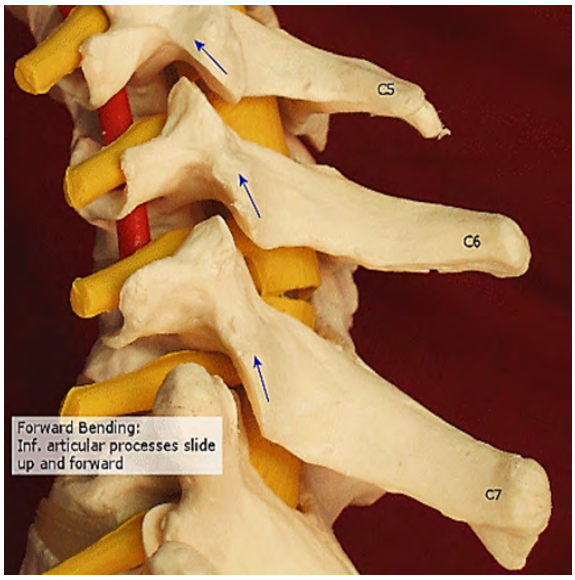
REGION	FLEXION	EXTENSION	SIDE BENDING RIGHT	ROTATION RIGHT
THORACIC T3/4 – T11/12	RF and LF slide up	RF and LF slide down	RF slides down and LF slides up	RF distracts and LF compresses and acts as fulcrum
LUMBAR	RF and LF slide up	RF and LF slide down	RF slides down and LF slides up	RF distracts and LF compresses and acts as fulcrum

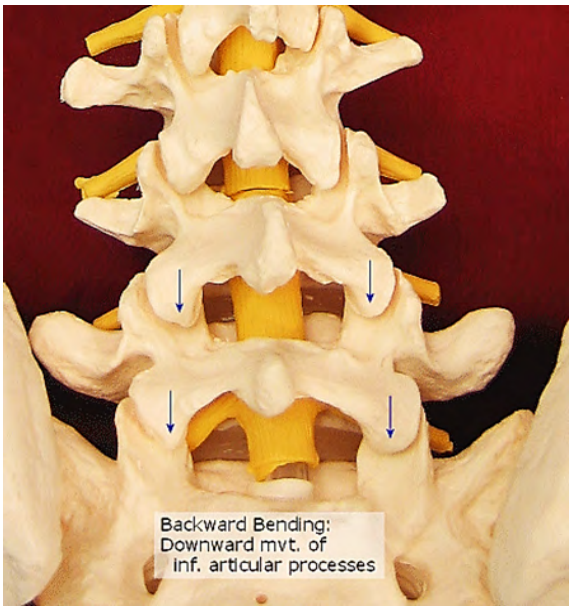
ROC = right occipital condyle; LOC = left occipital condyle; RF = right facet; LF = left facet

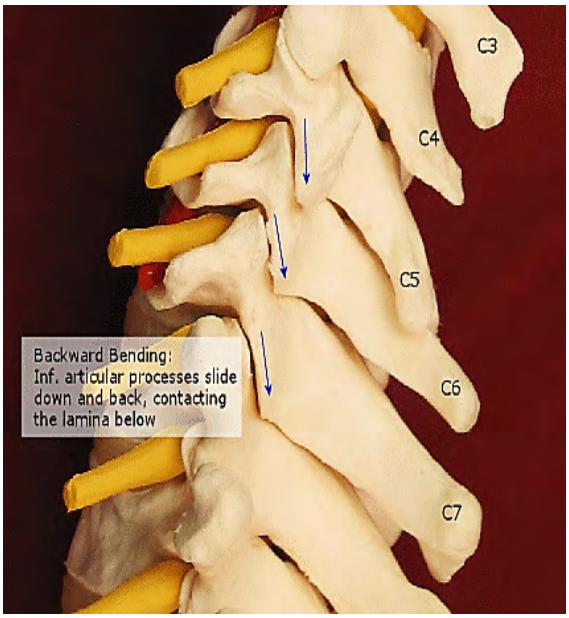


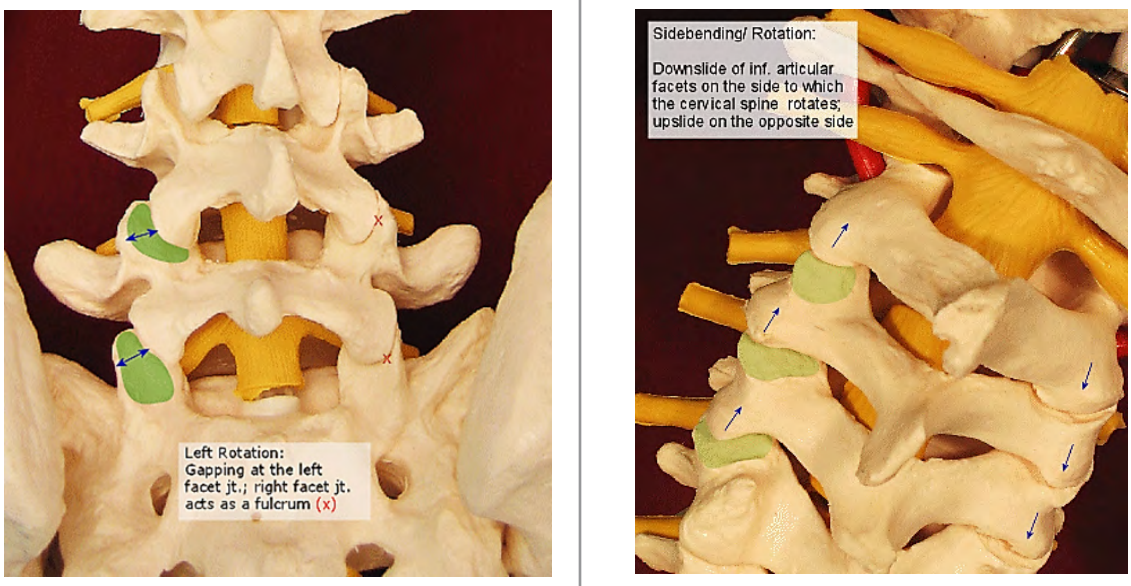
Drawings based on fresh cadaver dissections (Paris). The hatched areas show the areas where the articular cartilage on the facets is exposed (Paris and Loubert 1990).











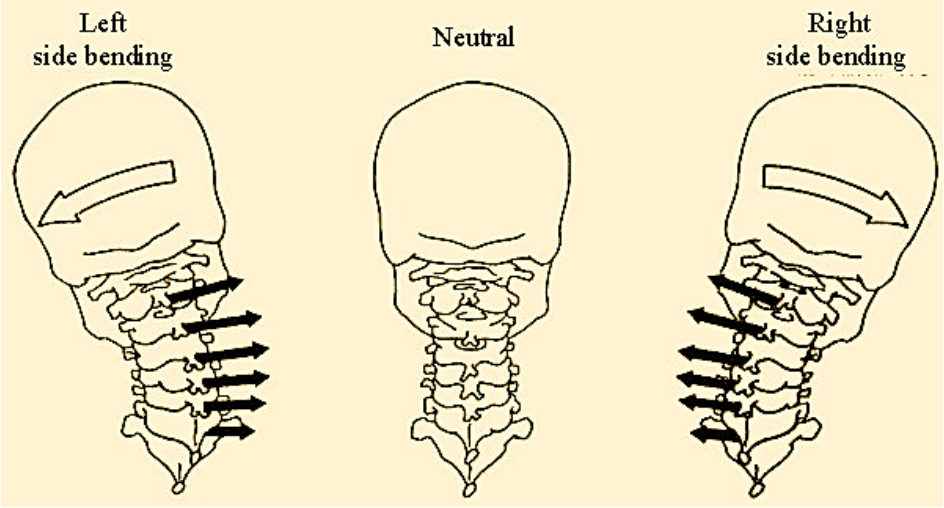
This series of photographs shows the movements of the facets (arrows) and the exposed articular facet surface (green) during a posterior view (TOP LEFT) and lateral (TOP RIGHT) of forward bending (flexion); a posterior view (MIDDLE LEFT) and lateral view (MIDDLE RIGHT) of backward bending (extension); a posterior view (BOTTOM LEFT) of rotation and a posterior view (BOTTOM RIGHT) of rotation during side bending.

- **OVERVIEW OF VERTEBRAL AND SACROILIAC JOINT MOVEMENTS**

  - VERTEBRAE FORWARD FLEXION
    - anterior tilting (rocking) of the vertebral body over the nucleus
    - compression and bulging of the anterior intervertebral disc (annulus)
    - tension on posterior intervertebral disc (annulus)
    - nucleus deforms posteriorly
    - intervertebral foramen increases in size
    - spinous processes separate
    - decreased compression on facet joints
    - tension on supraspinous, interspinous, intertransverse, ligamentum flava and posterior longitudinal ligaments
    - slack anterior longitudinal ligament

  - VERTEBRAE BACKWARD EXTENSION
    - posterior tilting (rocking) of the vertebral body over the nucleus
    - compression and bulging of the posterior intervertebral disc (annulus)
    - tension on anterior intervertebral disc (annulus)
    - nucleus deforms anteriorly
    - intervertebral foramen decreases in size
    - spinous processes converge
    - increased compression on facet joints
    - slack in the supraspinous, interspinous, ligamentum flava and posterior longitudinal ligaments
    - tension on the anterior longitudinal ligament

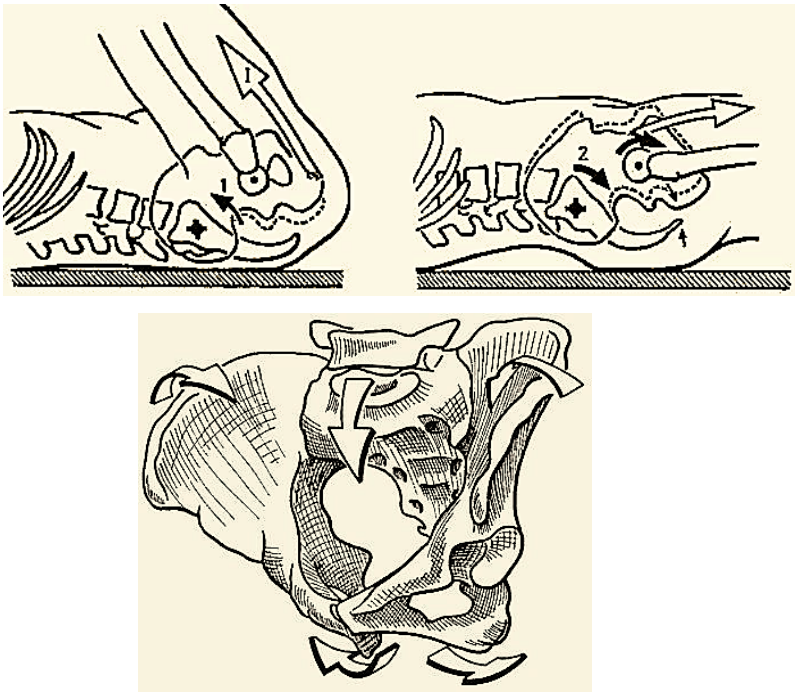
  - VERTEBRAE SIDE BENDING (LATERAL FLEXION)
    - lateral tilting (rocking) of the vertebral body over the nucleus on the side toward the movement
    - compression and bulging of the intervertebral disc (annulus) on the side toward the movement
    - tension on the intervertebral disc (annulus) on the side opposite the movement
    - nucleus deforms toward the side opposite of the movement
    - intervertebral foramen decreases in size on the side of movement
    - intervertebral foramen increases in size on the side opposite of movement
    - increased compression on facet joints on the side of movement
    - decreased compression on facet joints on the side opposite the movement
    - tension on the intertransverse ligament on the side opposite the movement
    - slack on the intertransverse ligament of the side of movement



Diagam showing the cervical side bending and rotation coupling pattern. During left side bending (LEFT), the spinous processes of the cervical spine rotate to the right. During right side bending (RIGHT), the spinous processes of the cervical spine rotate to the left. (Paris and Loubert 1990)

- VERTEBRAE ROTATION
  - increased compression of nucleus
  - shear stress on annulus fibrosus
  - increased compression on facet joint on side opposite rotation
  - decreased compression of facet joint on side of rotation
  - tension on joint capsule on side of rotation

- SACROILIAC JOINT
  - movement very limited to 1–3 degrees
  - movement is mainly in the anterioposterior direction
  - rotation and translation occur
  - TORSION is the movement of the ilium relative to the sacrum as in lying supine and bringing the knee to the chest.
  - NUTATION the is movement of the sacrum related to the ilium as during forward bending of the trunk and squatting
  - pelvic tilt.
    + anterior pelvic tilt increases the lumbar lordosis
    + posterior pelvic tilt decreases the lumbar lordosis



Diagrams showing pelvic torsion when the knees are brought toward the chest (TOP LEFT) and the knees are moved away from the chest (TOP RIGHT) and nutation (BOTTOM CENTER) when the sacrum moves forward relative to the lium. Arrows show the directions of movements. (Paris and Loubert 1990)

1.3 LOADS ON THE SPINE

- **CERVICAL SPINE**
  - Loads at the atlanto-occipital joints are lowest in full extension and highest in full flexion.
  - Loads at C7–T1 are least when the head is facing directly forward and the chin tucked in.
  - Loads at C7–T1 are slightly greater when the head is in the correct posture, greater still when the head is extended and greatest when the head is fully flexed.

- **LUMBAR SPINE**
  - Loads are least when lying supine.
  - Loads are low with relaxed standing, greater with supported sitting and still greater with unsupported sitting.
  - Compression loads on the lumbar spine are greatest near the toe off stage of walking.
  - Loads increase on the lumbar spine as the velocity of walking increases.

1.4 MUSCLE OF THE SPINE

- **SPLENIUS AND THE ERECTOR SPINAE MUSCLES OF THE SPINE**

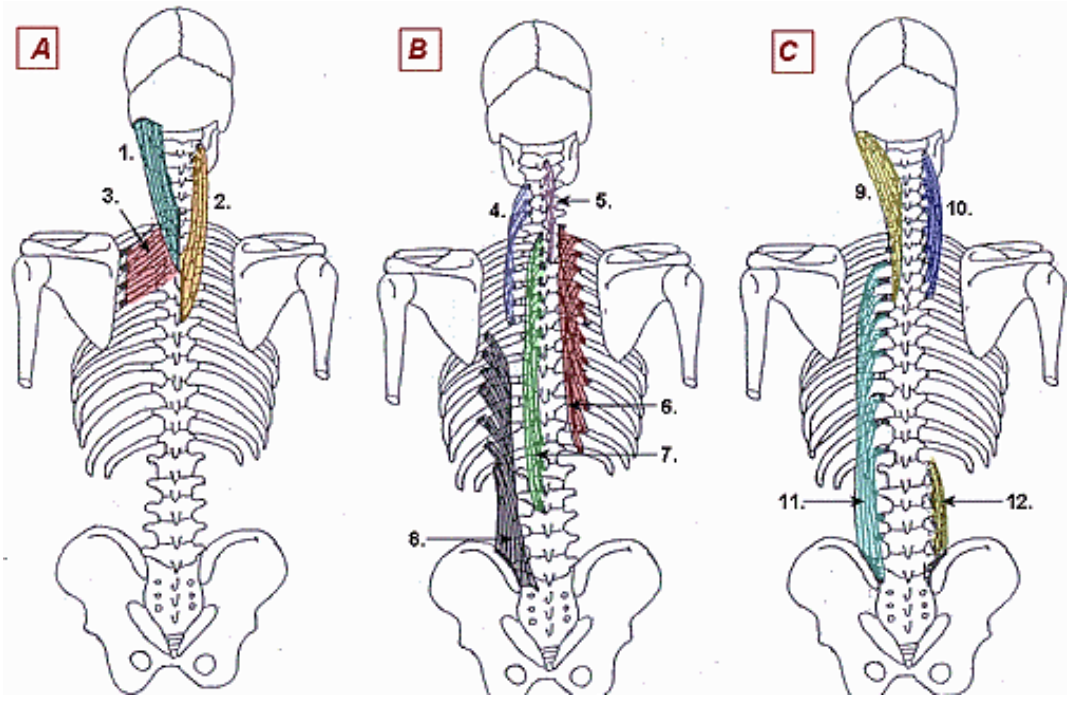


Diagram of the splenius, iliocostalis, longissimus, and spinalis muscles showing attachments. 1. Splenius capitis. 2. Splenius cervicis. 3. Serratus post. sup. 4. Ilicostalis cervicis. 5. Spinalis cervicis. 6. Iliocostalis thoracis. 7. Spinalis thoracis. 8. Iliocostalis lumborum. 9. Longissimus capitis. 10. Longissimus cervicis. 11. Longissimus thoracis. 12. Longissimus lumborum.

- **TRANSVERSOSPINALIS MUSCELS OF THE SPINE**

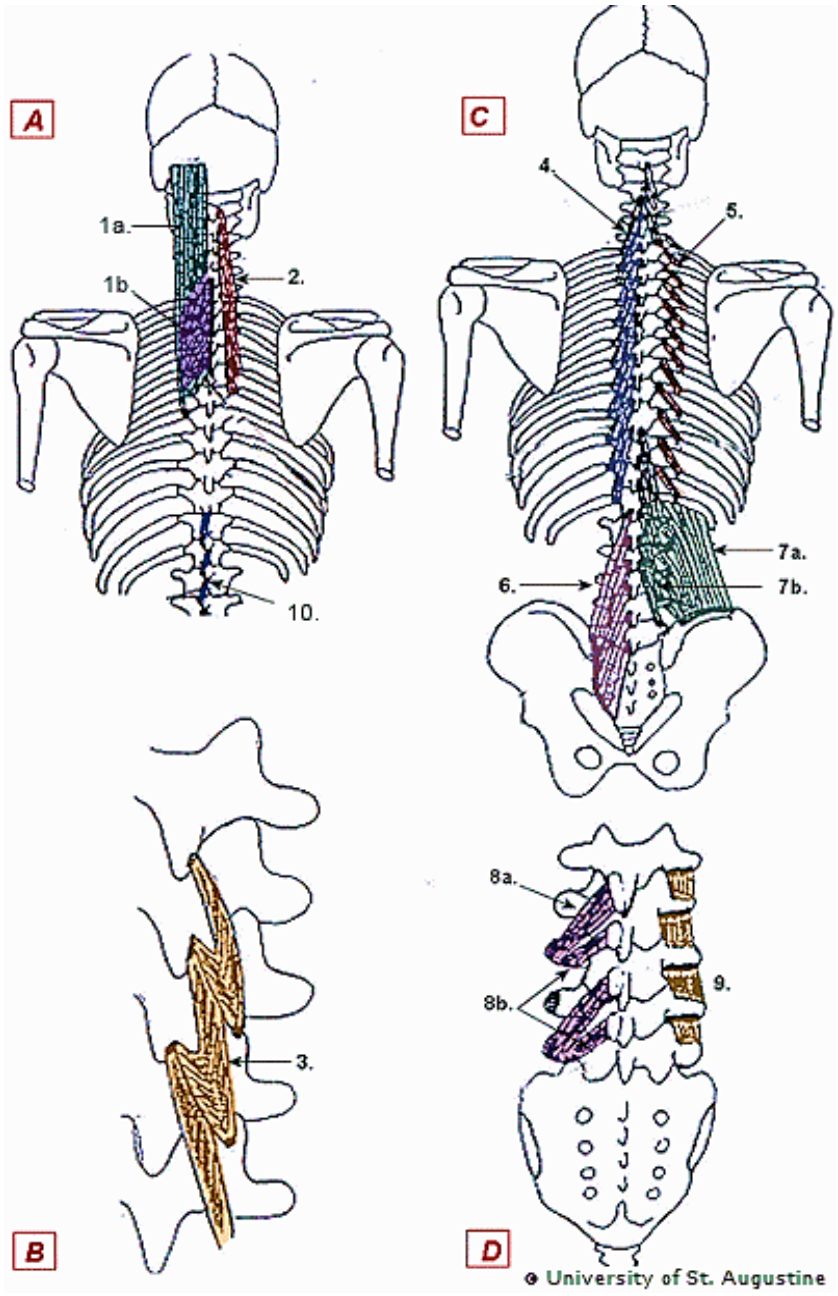


Diagram showing the semispinalis capitis (1a), thoracis (1b), and cervicis (2), multifidus lumborum (3 and 6), multifidus cervicis (4), levator costorum (5), quadratus lumborum – iliocostal (7a) and iliotransverse (7b), rotators – long (8a) and short (8b), and intertransversarius (9), interspinalis (10) Notice the bipinnate arrangement of the multifidus

- **MUSCLE ACTIONS ON THE SPINE**

  - FORWARD TRUNK FLEXION FROM STANDING
    - Abdominal muscles and iliopsoas move trunk forward.
    - Gravity pulls trunk downward.
    - Erector spinae muscles eccentrically control downward movement.
    - At 60 degrees of trunk flexion, the pelvis rotates anteriorly at hips.
    - Gluteus maximus and hamstrings eccentrically control the anterior pelvic rotation.

- TRUNK EXTENSION FROM FULL FLEXION
  - Erector spinae very active at full flexion in stabilizing spine.
  - During initial 30 degrees of extension, the pelvis rotates posteriorly at the hips.
  - Gluteus maximus and hamstrings concentrically produce this pelvic rotation.
  - Last 60 degrees of extension is produced by concentric action of the erector spinae.

- TRUNK SIDE BENDING
  - Quadratus lumborum, erector spinae and abdominal muscles on the side of movement (say right) initiate trunk side bending.
  - Gravity then pulls the trunk further laterally (increase right side bending).
  - Erector spinae, quadratus lumborum on the side opposite the movement (left side) act eccentrically to control the rate and the distance of gravity produced (right) side bending.
  - The return to an erect posture is produced by concentric activity of the erector spinae and quadratum lumborum on the side opposite (left side) the gravity produced (right) side bending.

- **HEAD AND SPINE MUSCLE ACTIONS**

Table below showing the muscles (X) that are active during extension (Ext.); Flexion (Flex.); Lateral Flexion (Lat. Fl.) or side bending and Rotation (Rot.)

HEAD AND SPINE MUSCLE ACTIONS				
HEAD				
Muscles	Ext.	Flex	Lat. Fl.	Rot.
Splenius Capitis	X		X	S
Longissimus Capitis	X		X	S
Simispinalis Capitis	X			O
Rectus Posterior Major	X			S
Rectus Posterior Minor	X			
Obliquus Capitis Superior			X	
Obliquus Capitis Inferior				S
Longus Capitis		X		
Trapezius	X		X	O
Sternocliedomastoid	X		X	O
CERVICAL SPINE				
Splenius Cervicis	X		X	S
Iliocostalis Cervicis	X		X	S
Longissimus Cervicis	X		X	S
Semispinal Cervicis	X			O
Multifidus	X			O
Spinalis	X			
Rotatores Longi & Brevi				O
Interspinalis	X			
Intertransvarius			X	
Longus Coli		X	X	O
Trapezius	X		X	O
Scalenus Ant., Med., Post.		X	X	
THORACIC SPINE				
Intercostalis Thoracis	X		X	S
Longissimus Thoracis	X		X	S
Semispinalis Thoracis	X			O
Multifidus	X		X	O
Spinalis	X			
Rotatores Longi. & Brevi				O
Interspinalis	X			
Intertransvarius			X	
LUMBAR SPINE				
Iliocostalis Lumborum	X		X	S
Multifidus	X		X	O
Rotatores Longi & Brevi				O
Interspinalis	X			
Intertransvarius			X	
Quadratus Lumborum	X		X	
Psoas Major		X	X	

S = same side; O = opposite side

ACTIVITIES: This activity is called the spine dance. You can do this standing or sitting. Place both of your hands chest high in front of you. Have you palms facing away from you and angled slightly downward at about 30 degrees. The palms of your hands represent the left and right vertebral facets. This is the start position for the 5 moves of the dance. 1) Bend your trunk and hand simultaneously forward. Both hands should go up and forward, showing the motions of the cervical, thoracic and lumbar facets during flexion of the spine. 2) From the flexed trunk position straighten your spine. Both hands should go down and back, showing the motions of the cervical, thoracic and lumbar facets during extension of the spine. 3) Return to the start position and laterally bend your trunk to the right. Your right hand should move down and your left hand move up, showing the motions of the facets during cervical, thoracic and lumbar right side bending (lateral flexion). If you bend to the left, your left hand should move down and the right hand should move up. 4) Now go back to the start and rotate your trunk to the right. Your right hand should move down and back and your left hand move up and forward, showing the motions of the cervical facets during right side rotation. If rotation is to the left, the left hand should move down and back and your hand should move up and forward. 5) Facet movements during thoracic and lumbar rotation of the spine differ from those in the cervical spine. Now return again to the start position and rotate your trunk to the right. Hold your left hand steady and move your right hand back mimicking a distraction motion of the right facet. With rotation to the left, hold the right hand steady and move the left hand back. Now if you sequence and repeat these movements from flexion to rotation you have the spine dance.

Study Questions (spine)

1. How are each of the following involved is motion of the spine:
    a. artricular facet angulation
    b. annulus fibrosus
    c. nucleus pulposus
    d. cruciform ligament
    e. alar ligament
    f. anterior longitudinal ligament
    g. ligamentum flavum
    h. supraspinous ligament
    i. rib cage
2. How do movements of the facets in the lower cervical, thoracic and lumbar regions compare during the following:
    a. forward flexion
    b. side bending
    c. rotation
3. What occurs at the spine during the following vertebral movements:
    a. forward flexion
    b. backward extension
    c. side bending
4. What are the sequence of events that occur during the following:
    a. forward trunk flexion from upright standing
    b. side bending from upright standing
5. Which muscles extend, laterally bend and rotate vertebrae to the same side?
6. Which muscles extend and rotate vertebrae to the opposite side?

2 POSTURE

2.1 POSTURAL POSITION OF THE SPINE

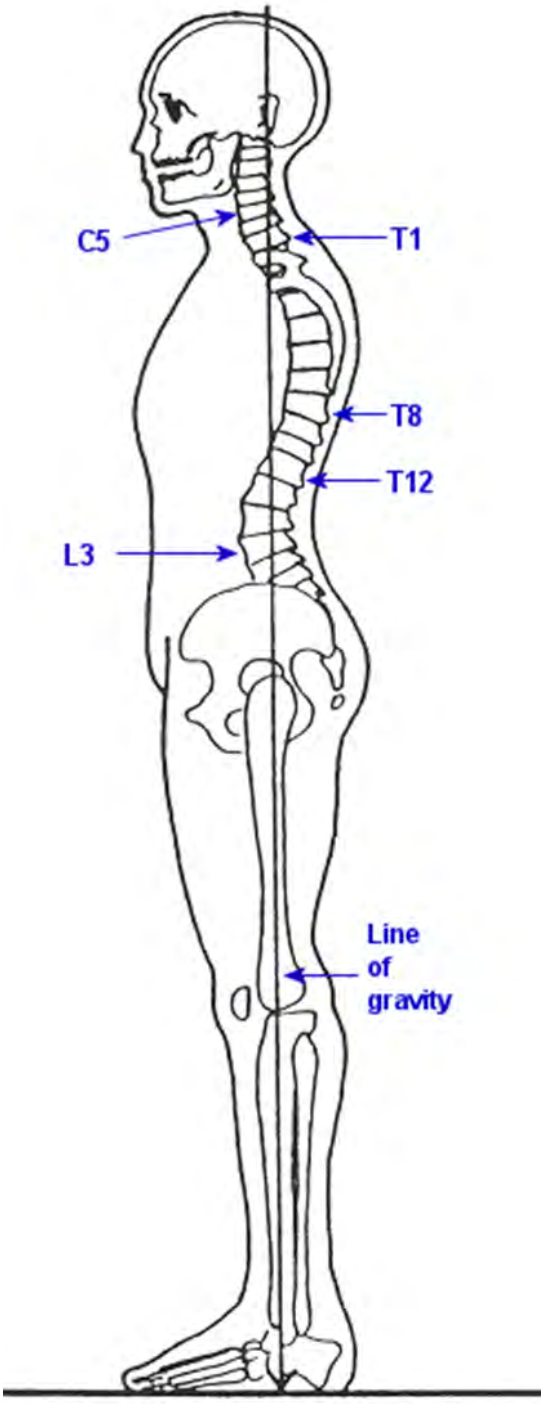


Diagram of the line of gravity (LOG) relative to the spine, hips, knee, and ankle for proper postural alignment.

- **LINE OF GRAVITY (LOG)**
  - Passes through the dens of C2, bodies of T1, T12, S2.
  - Maximal gravitational torque occurs at C5, T8 and L3 where the apex of each spinal curve lie farthest from the line of gravity.

- **FORCES PRODUCING NORMAL ALIGNMENT**
  - Relative to the joints of the spine, the LOG produces moments to move the joints in a particular direction, just as the GRF does during the stance phase of gait.
  - At these joints there are passive forces and active forces to hold the joints in proper postural alignment by counterbalancing the moments producesd by the LOG at these joints.
  - Passive forces are mainly ligamentous structures.
  - Active forces are muscular forces.
  - When there is postural malalignment, the passive and active forces are effected.

- ATLANTO-OCCIPITAL JOINTS
  - LOG is anterior to the joints.
  - moment: force to produce flexion of the joints
  - passive forces: lig. nuchae, tectorial membrane
  - active forces: posterior neck muscles

- LOWER CERVICAL SPINE (C3–C7)
  - LOG is posterior to the joints.
  - moment: force to produce extension of the joints
  - passive forces: ant. longitudinal lig., lig. nuchae
  - active forces: Anterior scalene, longus capitis and coli

- THORACIC SPINE
  - LOG is anterior is the joints.
  - moment: force to produce flexion of the joints
  - passive forces: post. longitudinal lig., lig. flavum,
  - supraspinous lig., interspinous lig.
  - active forces: extensors of the thoracic spine

- LUMBAR SPINE
  - LOG is posterior to the joints.
  - moment: force to produce extension of the joints
  - passive forces: ant. longitudinal lig., lig. flavum
  - active forces: rectus abdominis, external and internal obliques

- SACROILIAC JOINTS
  - LOG is anterior to the joints.
  - moment: force to produce flexion of the joints
  - passive forces: sacrospinous lig., sacrotuberous lig., sacroiliac lig.
  - active forces: transverse abdominis

- **EFFECTS OF POSTURAL MISALIGNMENT**
  - EXCESSIVE ANTERIOR PELVIC TILT
    - increases compression of posterior bodies
    - increases L5–S1 disc pressure
    - increases the lumbosacral angle
    - may result in potential slippage of L5 on S1
    - abdominal muscles are stretched
    - iliopsoas muscles are shortened

  - EXCESSIVE LUMBAR LORDOSIS
    - increases compression of posterior bodies
    - increases compression of facet joints
    - increases disc pressure
    - produces narrowing of intervertebral foramen
    - increases tension on anterior annulus
    - anterior longitudinal lig. is stretched
    - posterior longitudinal lig., supraspinous lig., interspinous lig., lig. flavum, and the lumbar extensor muscles are shortened

  - EXCESSIVE THORACIC KYPHOSIS
    - increases compression of anterior bodies
    - increases disc pressure
    - increases tension on facet capsule and posterior annulus
    - thoracic extensor muscles and dorsal scapular muscles are stretched
    - posterior longitudinal lig., supraspinous lig. interspinous lig., lig. flavum are stretched
    - anterior longitudinal lig. is shortened
    - ant. shoulder girdle and upper abdominal muscle shortening

- EXCESSIVE CERVICAL LORDOSIS
  - increases compression of posterior bodies
  - increases compression of facet joints
  - increases disc pressure
  - produces narrowing of intervertebral foramen
  - increases tension on anterior annulus
  - anterior longitudinal lig. and longus capitis and longus coli muscles are stretched
  - Lig nuchae, posterior longitudinal leg. and leg. flavum are shortened
  - posterior neck muscles are shortened

ACTIVITIES: These activities require 2 people. Find an inside doorway that has a hard floor. Open the door fully and hang a plumline from the top center of the door frame. 1) Have one person stand side wards in the doorway, and align the plumline along that side of that person. Now, based on the LOG diagram at the start of this section, have the other person determine if the cervical, thoracic and lumbar spines and the hip and knee joints are aligned in proper posture. Then switch places. 2) Proper postural alignment is also determined by viewing the anterior and posterior exteriors of a person. Have one person stand in the doorway with their back to the second person. Have the plumline aligned with the midline of the body. Now look for asymmetry between the right and left acromion processes, scapulae, elbows, hands, iliac crests, hips, knees and ankles. Is the spine aligned along midline or is there a lateral shift of the spine and where is that shift? Are the legs of equal length or the knees bowed in or out? Now switch places. 3) After you have assessed the alignment from a posterior view, repeat #2 from an anterior view of the body. This time look for asymmetry between the right and left acromion processes, clavicles, elbows, hands, iliac crests, hips, knees and ankles. Also look at the sternum and rib cage. When this complete, switch sides. 4) Compare what you found with each other and if there is a postural malalignment, what might be the cause and how might you correct the postural malalignment.

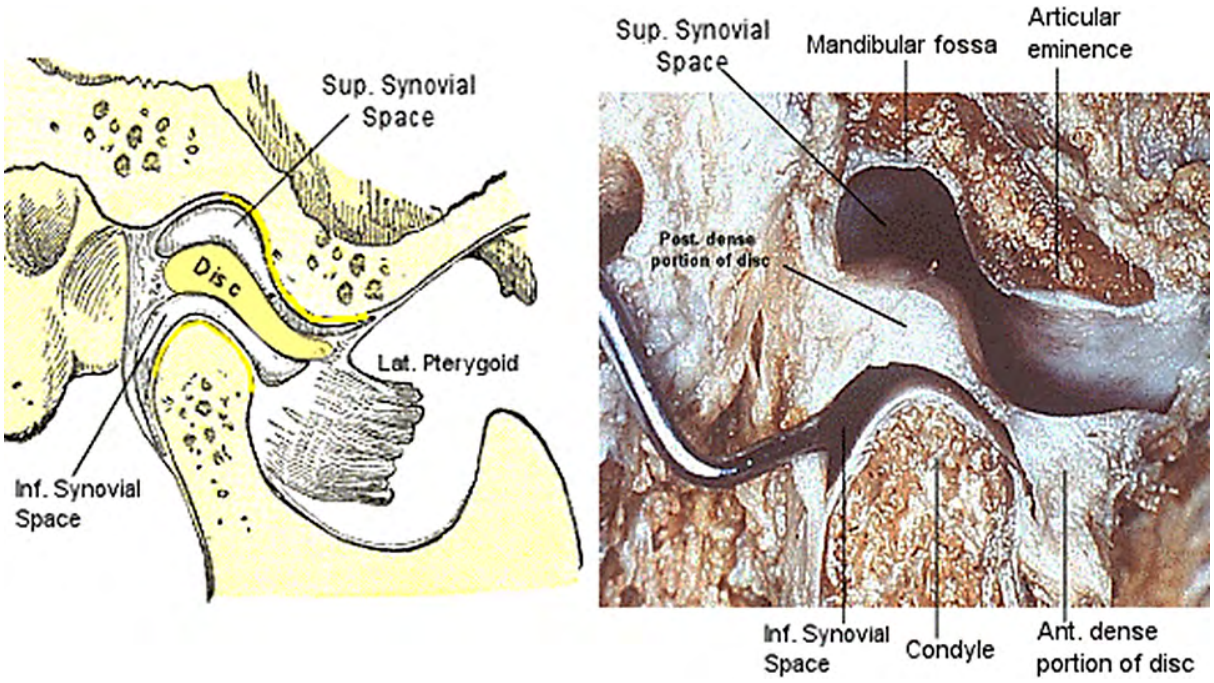
Study Questions (Posture)

1. What is the LOG moment for each of the following regions and what are the active and passive forces that counteract this moment to maintain good posture?
   a. lower cervical spine
   b. thoracic spine
   c. lumbar spine

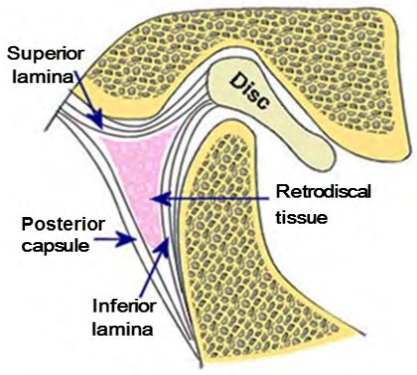
2. What structures would be stretched and shortened for each of the following postural misalignments?
   a. excessive anterior pelvic tilt
   b. excessive thoracic kyphosis
   c. excessive lumbar lordosis

3 TEMPOROMANIBULARJOINT (TMJ)

- **ANATOMY OF TMJ**
  - The TMJ is formed by the mandibular (glenoid) fossa of the temporal bone and the mandibular condyle.
  - Anterior to the mandibular fossa is the articular tubercle and posterior to it is the postglenoid tubercle.
  - The temporomandibular joint contains an fibrocartilage interarticular disc that is attached anteriorly to lateral pterygoid muscle, through the joint capsule, and posteriorly to a superior and inferior lamina of the posterior ligament.



Drawing of the TMJ (LEFT) showing the intraarticular disc with its anterior and posterior attachments and the attachment of the lateral (lat.) pterygoid muscle to the anterior joint capsule (modified from Gray 1918). Dissection of the TMJ (RIGHT) showing the posterior (post.) and anterior (ant.) parts of the disc, and the superior (sup.) and inferior (inf.) synovial spaces.



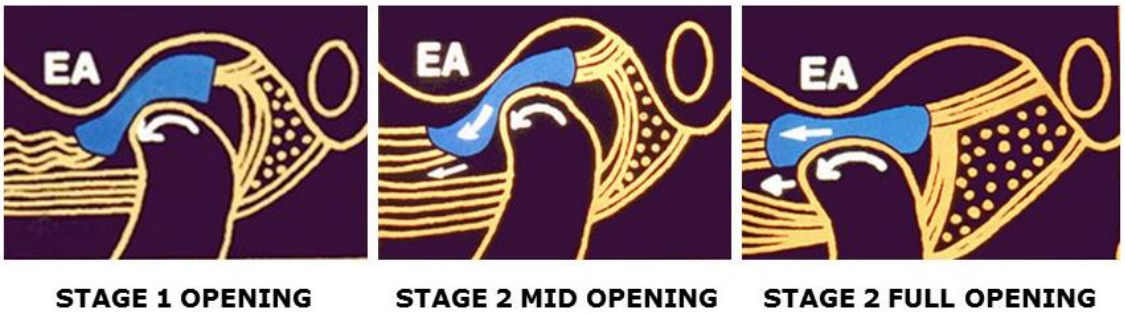
Drawing of the TMJ showing the elastic superior lamina and non-elastic inferior lamina attaching to the posterior disc. The retrodisc tissue contains nerves and blood vessels going to the TMJ. (Modified from Gray 1918)

- The superior lamina and inferior lamina attach to the posterior disc.
- Superior lamina is elastic and stretches when the disc moves anteriorly and recoils to move the disc posteriorly.
- Inferior lamina is collagen and tightens when the disc moves anteriorly to restrict the amount of anterior disc movement but it slackens when the disc moves posteriorly.

- **JAW OPENING**

  - STAGE 1 of OPENING: initial opening (11–25 mm between incisors)
    - The mandibular condyle rotates anteriorly in the fossa and on the disc.
    - The disc remains in place in the fossa.
    - The superior and inferior laminae are relaxed.

  - STAGE 2 of OPENING: terminal opening (40–50 mm between incisors)
    - The mandibular condyle rotates anteriorly and translates anteriorly over the articular eminence.
    - The articular disc translates anteriorly with the condyle as the condyle moves over the articular eminence.
    - The superior lamina stretches and inferior lamina tightens.

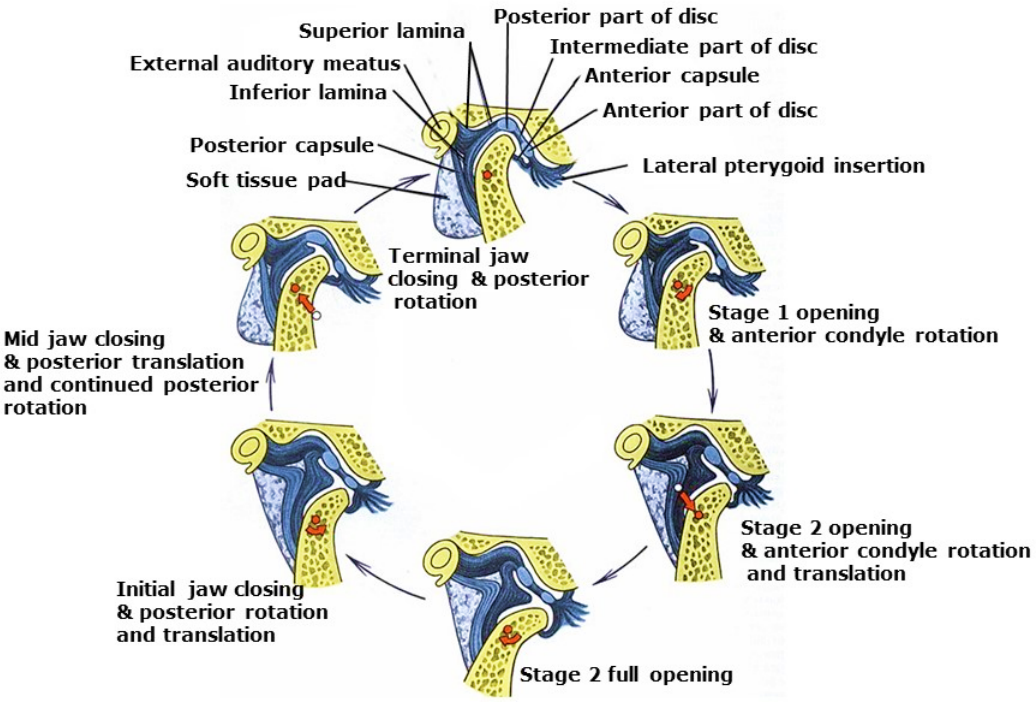


Drawing showing the movements of the mandibular condyle and disc during jaw opening. EA = articular eminence

- **JAW CLOSING**

  - NITIAL JAW CLOSING (from 40–50 mm of stage 2 opening to 11–25 mm of stage 1 opening)
    - The mandibular condyle rotates posteriorly and translates posteriorly back into the mandibular fossa.
    - The articular disc translates posteriorly with the condyle as the condyle returns to the mandibular fossa.
    - Recoil of the superior lamina pulls the disc posteriorlyinto the fossa.
    - The inferior lamina slackens as the disc moves posteriorly into the fossa.
    - Contraction of the lateral pterygoid muscle controls the rate of posterior disc movement.

- TERMINAL JAW CLOSING (from 11–25 mm of opening to full closure)
  - The mandibular condyle rotates posteriorly in the fossa and on the disc.
  - The disc is in the fossa.
  - The superior and inferior laminae are relaxed.



Drawing showing the movement of the mandibular condyle and disc during jaw opening and closing

ACTITIVITY: On the right side of your head, place your right index finger in front of your right external auditory meatus. Do the same with your left index finger on the left side of your head. Now open and close your jaw. You should be able to feel the movements of the mandibular condyles. Move your jaw slowly and see if you can feel the anterior rotation and anterior translation of the condyles during opening and the posterior rotation and translation of the condyle during closing. Now more your jaw to the right and then to the left. What are the movements of the condyles during side wards jaw motion?

Study Questions (TMJ)

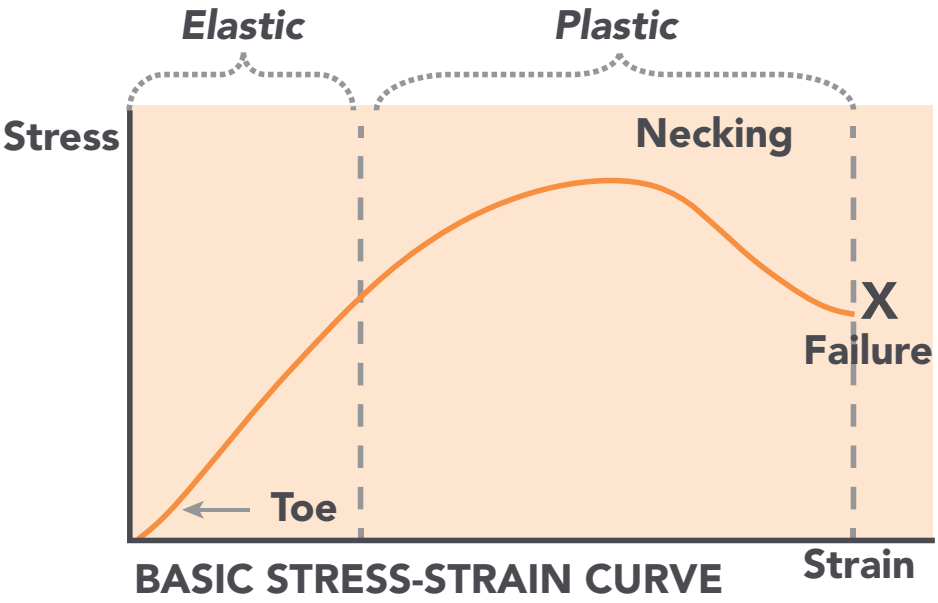
1. What occurs at the TMJ during stage 2 (terminal opening)?
2. What occurs at the TMJ during initial jaw closing?

4 TISSUE MECHANICS

INTRDUCTION

In clinical practice, many tests, procedures, treatments, dysfunctions and pathologies involve the responses of tissues to mechanical forces. Knowledge of the biomechanical responses of tissue can assist the clinician in the interpretation of assessments and in the development or modification of the treatment plans. This section begins with a basic concepts in tissue mechanics. Following this, the mechanics of connective tissue proper, cartilage, bone, muscle and nerve will be described.

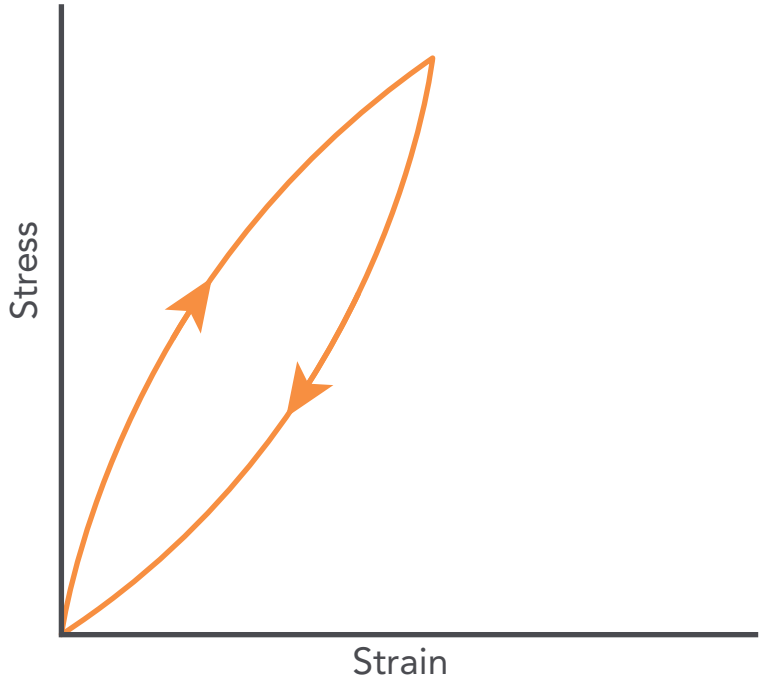
- **STRESS AND STRAIN RELATIONSHIPS**
  - STRESS – DEFORMATION or STRAIN CURVE
    - External forces of loads applied to tissues result in stresses within the tissues and these stresses produce a deformation or strain of the tissues
    - The relationship between the amount of stress/force and the amount of strain/ deformation provides a stress/strain curve that is divided commonly into 4 parts.



Graph of a basic stress-strain curve showing the toe region, elastic region, plastic region and failure point.

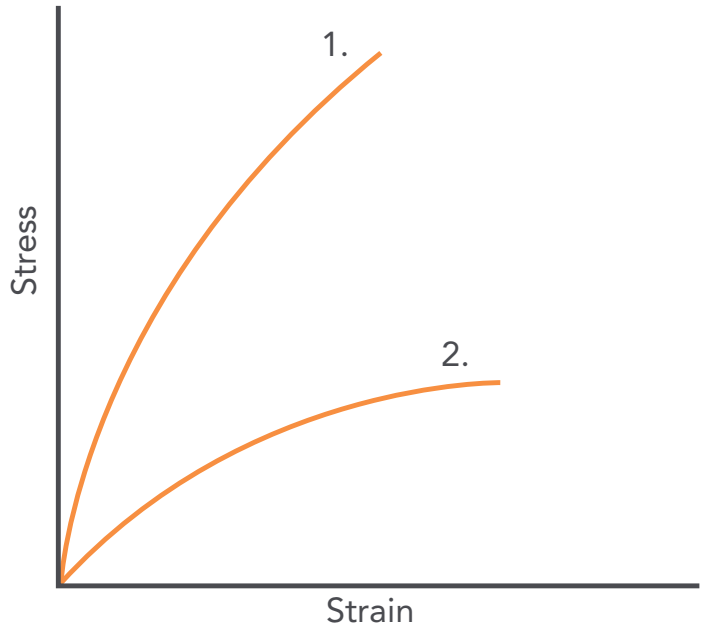
- **The toe region** is seen at the start of the curve. It is commonly the position where the material is in slack so that a small amount of stress produces proportionally more strain.
- **The elastic or linear region** follows the toe region. Here, the material is tightening so that the amount of stress is proportional to the amount of strain. It is also the region where by the release of stress would result in the material returning to its original point of not being deformed. The ability of a material to return to its non-deformed starting point defines the elastic region of the material.
- **The plastic region** follows the elastic region. In this region, the material is damaged and the degree of damage increases as the stress increases. This results in the amount of strain/deformation increasing more relative to an increase in stress. At the **necking point** of the plastic region, the degree of material damage is so extensive that the amount of deformation remains large with a decrease in stress. The material in the plastic region is permanently deformed and as such it will not return to its original position when the stress is removed.
- **Failure** is the point where the material completely breaks or ruptures.

- ELASTIC MATERIALS
  - As stress/force is applied, there is deformation.
  - When the stress/force is stopped at the top of the curve, the material goes right back to the beginning points or shape or size.
  - Now there is work lost during this process because the work required to deform the material is not totally recaptured once the forces are removed.
  - Although there is work lost as reflected by the area between the curves, this material is still considered an elastic material because it returns to its starting point.



Graph of a stress/strain curve for an elastic material.

- YOUNG'S MODULUS OF ELASTICITY
  - Youngs modulus of defined as $\mathbf{E = \frac{STRESS}{STRAIN}}$
  - Youngs modulus defines stiffness (resistance to deformation)
  - high Young's modulus = high stiffness
  - low Young's modulus = low stiffness

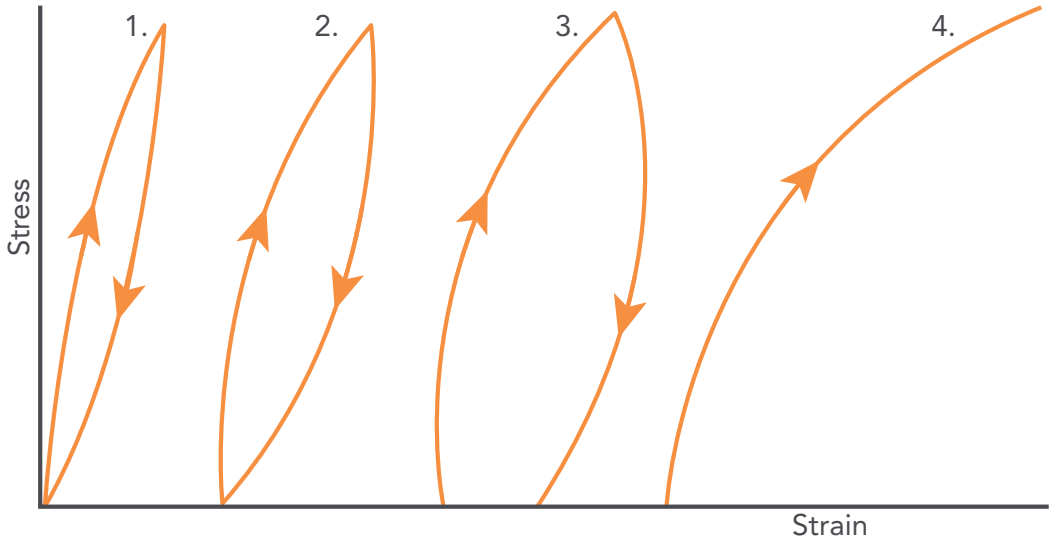


Graph showing the stress-strain curves of 2 materials

- Curve number 1 shows relatively high stress and little deformation/strain while curve number 2 shows low stress but a lot of deformation/strain.
- Young's modulus for number 1 would be higher than that for number 2. That is, number 1 would be stiffer than number 2.

- RESILIENCE
  - Resilience is defined as the mechanical work loss during deformation.
  - Its formula is $\mathbf{R = \frac{W - \Delta W}{W}}$

  - The greater the loss of work, the less the resilience, while the less the loss of work, the greater the resilience.
  - R = 1 is a perfectly resilient material.

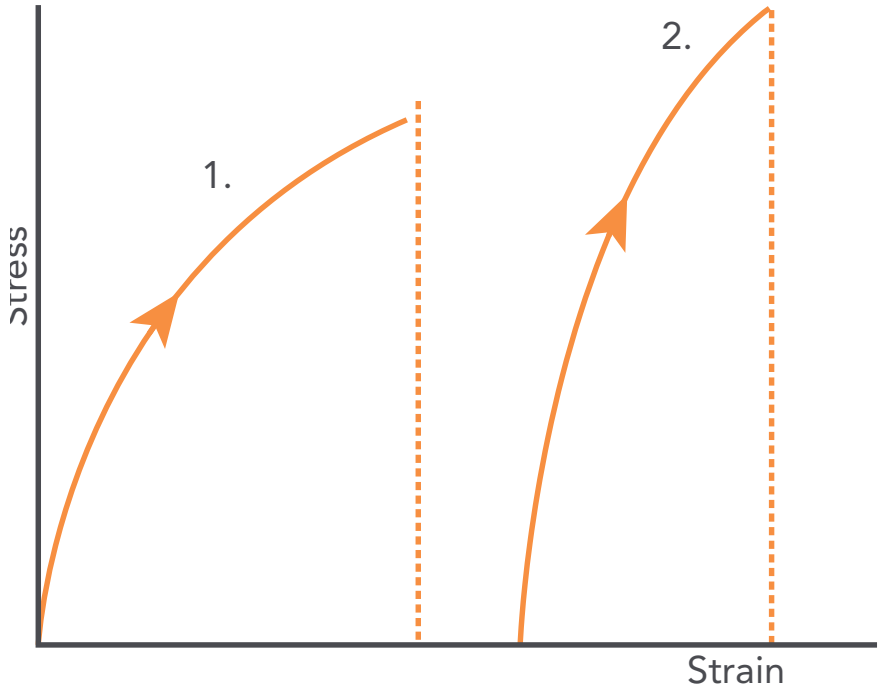


Graph showing the stress-strains curves of 4 materials. The loss of mechanical work is equal to the area under the curve for each material.

- In the graph above, the amount of work loss is represented by the area between the lines in curves 1–3 and the under the line in curve 4. The amount of work loss is least for curve #1 and greatest for curve #4.
- #1 = shows good resilience and elastic material; #2 = is less resilient than #1 but still an elastic material; #3 = shows poor resilience and is not an elastic material; #4 = is not resilient but is a good damping material (Styrofoam)

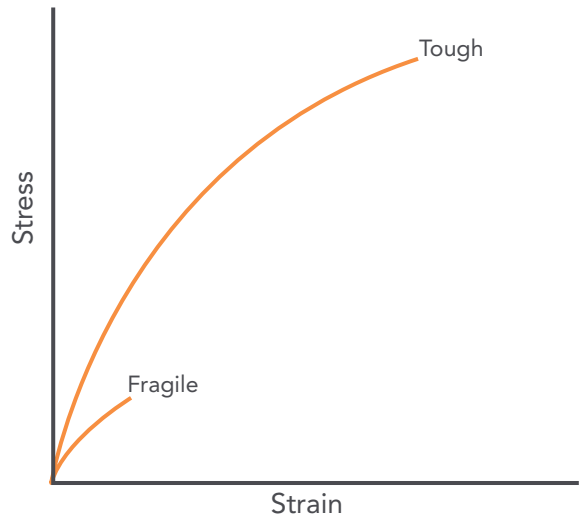
- DAMPING
  - Damping is the opposite of resilience
  - Its formula is **D = 1 – R**
  - Styrofoam is an excellent damping material.

- TOUGHNESS
  - Toughness is defined as the resistance to mechanical failure.
  - It is the amount of energy a material will absorb before it breaks as represented by the area under the curve.
  - Toughness is not necessarily equal to strength.
  - Strength is the magnitude of the force needed to break a material.



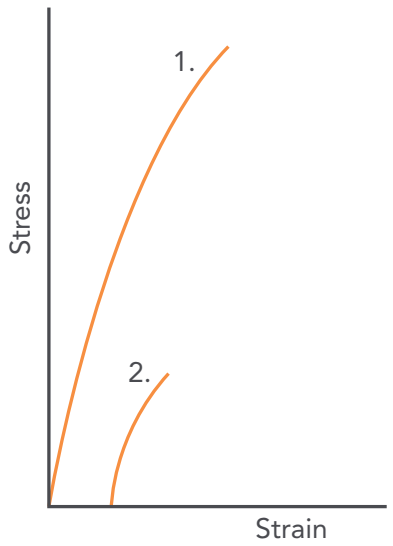
Graph showing the stress-strain curve for 2 materials. The area under the curve is greater for #1 than #2 and so #1 is tougher than #2, but #2 is stronger

- FRAGILITY
  - Fragility is the opposite of toughness.
  - A fragile material absorbs little energy before it breaks.



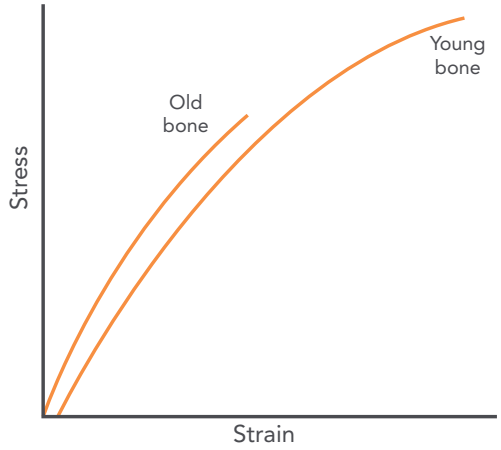
Graph showing the stress-strain curves of a tough material and a fragile material

- BRITTLENESS
  - Brittle materials strain (deform) very little before failure.
  - Brittleness is not necessarily related to strength.
  - Both materials #1 and #2 are brittle because they deform very little when stressed, but #1 is very strong (drill bit) compared to #2.



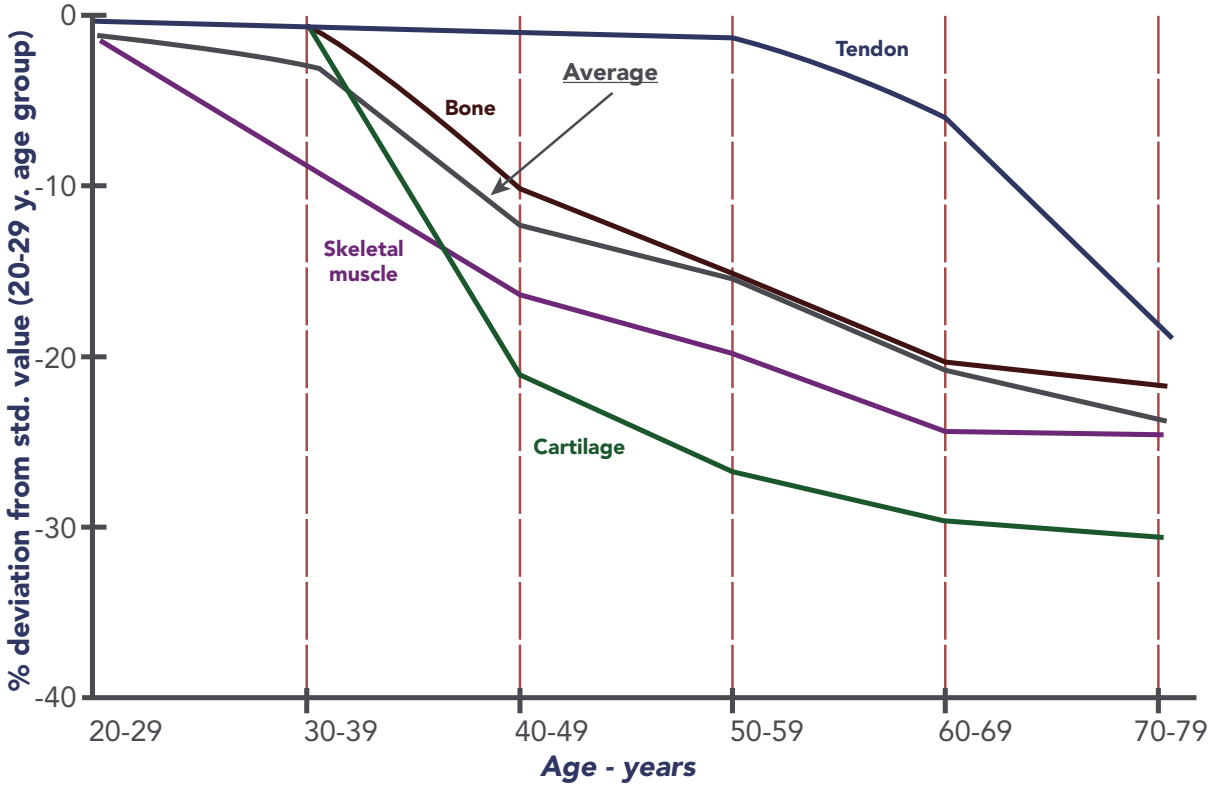
Graph showing the stress-strain curves of a strong but brittle material (#1) and a fragile and brittle material (#2).

- DUCTILITY
  - Ductility is the ability of a material to deform progressively in tension without breaking.
  - Young bone is more ductile than old bone because young bone deforms farther when tension stresses are applied than old bone.



Graph showing the stress-strain curves when tension is applied to old and young bones. Note that young bone deforms more that old bone in tension, which makes young bone more ductile that old bone.

- **TISSUE CHANGES WITH AGE**
  - Below is a graph showing the general changes in the mechanical properties of bone, tendons, skeletal muscle and cartilage with age.
  - Notice that these tissue respond differently with aging.
  - For each of these tissues, a more detailed description of the changes in stress/strain properties with age will be described.



Graph showing the stress-strain curves of tendon, bone, skeletal muscle and cartilage from age 20 to age 79 (Yamada 1970)

Study Questions (General Tissue Mechanics)

1. What is the difference between the elastic and plastic regions of a tissue stress vs. strain curve?
2. What is Young's modulus and how does it relate to tissue stiffness?
3. What is the difference between resilience and toughness?
4. What is the difference between toughness and brittleness?

4.1 CONNECTIVE TISSUE PROPER

4.1.1 ANATOMY OF CONNECTIVE TISSUE PROPER

- **CONNECTIVE TISSUE FIBERS**
  - COMPOSITION of COLLAGEN FIBERS

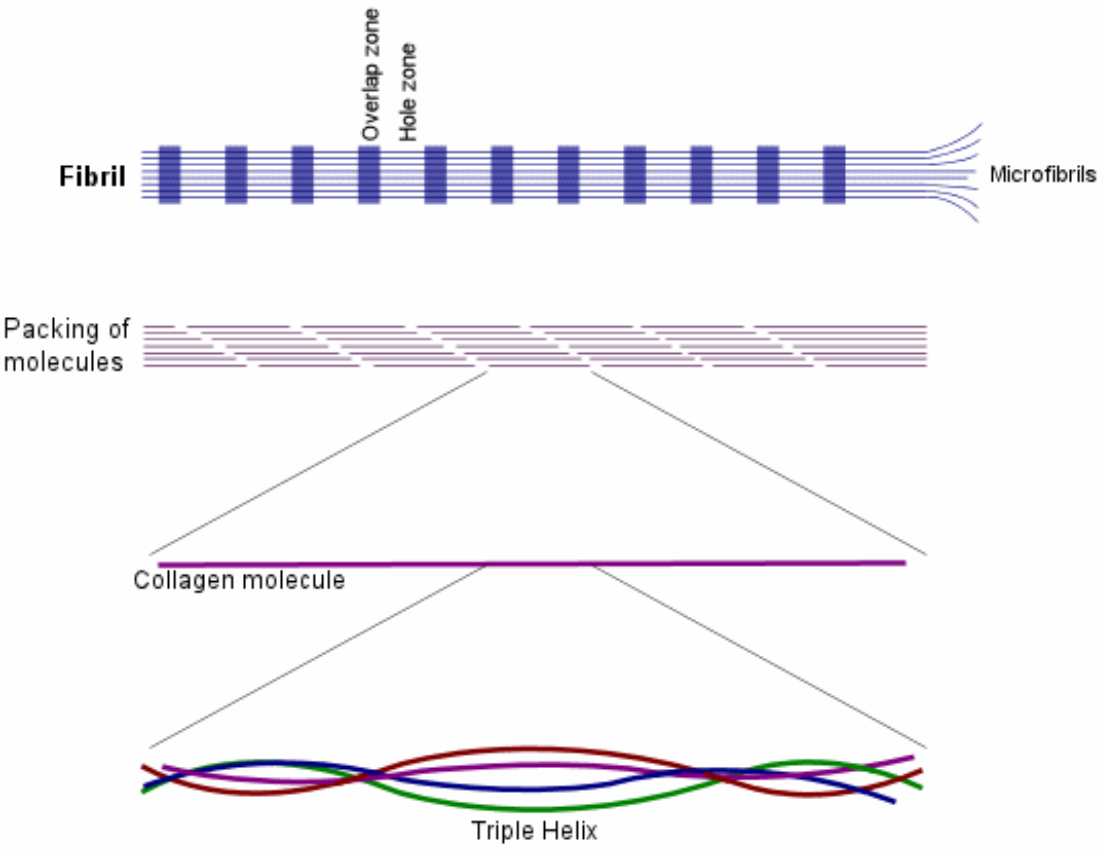


Diagram showing the composition of collagen fibers

- Collagen fibers are the predominant type of fiber found in connective tissue.
- These fibers are formed from tropocollagen molecules with each consisting of 3 polypeptide chains called alpha units that are wound about each other to form a triple helix.
- The tropocollagen molecules are packed end to end and stacked side to side to form a collagen fibril.
- The molecules are bound together by intra- and inter-chain bonds or cross-links between lysine and hydroxlysine.
- The cross-links also bind fibrils together like the rungs of a ladder.
- The cross-links contribute to the high tensile strength, stability and stiffness of collagen so that collagen fibers with many cross-links are stiffer than collagen with few cross-links.

- COMMON TYPES OF COLLAGEN
  - Some twenty- eight types of collagen have been identified but most of the collagen found in the body are Types I–V (Ross and Pawlina 2015).
  - **Type I collagen** is found in the dermis of skin, bone, tendon, ligament, fibrocartilage, and fascia. It forms 90% of the collagen in the body and functions to resist tension and stretching.
  - **Type II collagen** is found mainly in hyaline cartilage but it is also found in elastic cartilage and fibrocartilage. It functions to resist tensile strain that occurs when cartilage is compressed.
  - **Type III collagen** is found in the connective tissue of organs, such as the liver, spleen, lungs, and intestines, and in blood vessels, nerves and muscles. It functions as a structural support system in those structures and is important in wound closure.
  - **Type IV collagen** is found in the basement membrane of epithelium and functions to support the surface epithelium and as a filter.
  - **Type V collagen** is found in the basal lamina of smooth and skeletal muscle cells and Schwann and glia cells of the nervous system. It functions as a support system in these structures.

- RETICULAR FIBERS (a Type III collagen)
  - These fibers are thin and delicate.
  - The fibers form lace-like networks of fibers around smooth muscle cells, the sarcolemma of striated muscle, and the endoneurium of peripheral nerves.

- ELASTIC FIBERS
  - These fibers vary in quantity in connective tissue.
  - The contain a specific protein called elastin.
  - Elastic fibers are abundant in some ligaments, large arteries, the trachea and the dermis of the skin.

- **CONNECTIVE TISSUE CELLS**

- COLLAGEN PRODUCING CELLS
  - fibroblasts in connective tissues proper
  - chondroblasts and chondrocytes in cartilage
  - osteoblasts in bone
  - skeletal muscle cells in skeletal muscle
  - smooth muscle cells in blood vessels and some organs.

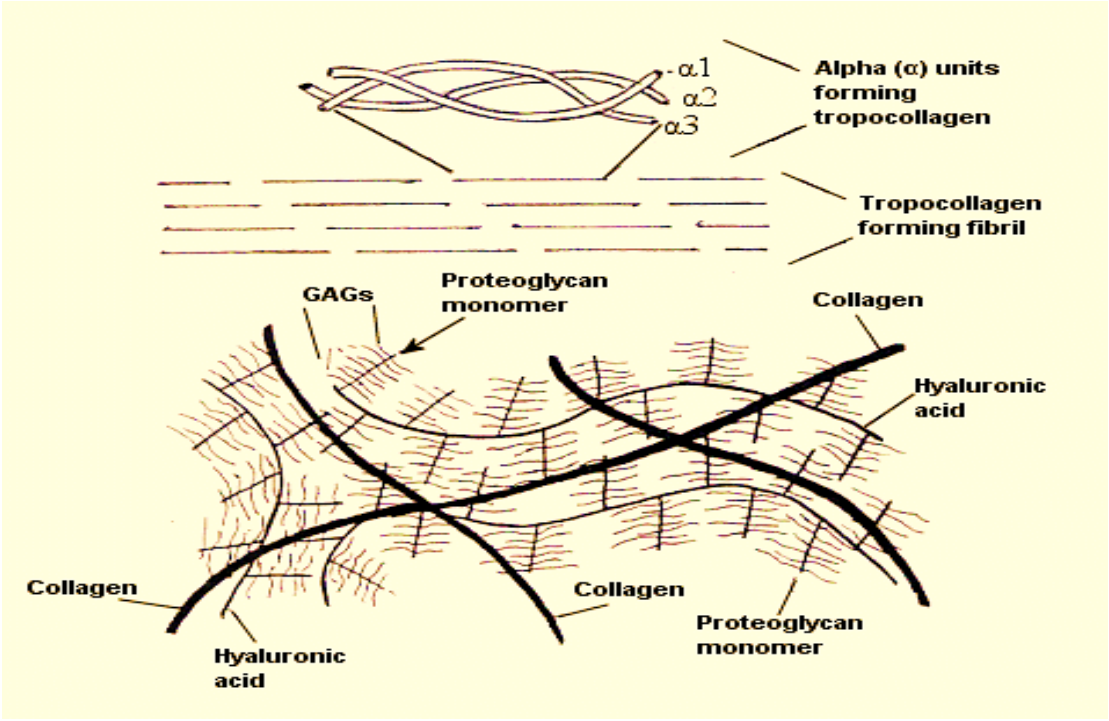
- FIBROBLASTS
  - Fibroblasts produce collagen and elastic fibers.
  - These cells produce glycoaminoglycans which form proteoglycans which are a component of the ground substance that surrounds the fibers.

- MYOFIBROBLASTS
  - These cells have properties of fibroblast and smooth muscle.
  - The cells produce collagen but contain myofilaments.
  - Myofibroblasts are abundant at sites of inflammation.
  - These cell are involved in wound closing.

- **GROUND SUBSTANCE**

- The material that surrounds the cells and fibers in connective tissue.

- CONTENTS of the ground substance
  - water, glycoaminoglycans (GAGs) and proteoglycans
  - glycoaminoglycans are polymers of disaccharide units
  - glycoaminoglycans form proteoglycans



Drawing of the ground substance surrounding collagen fibers. A main component is water. The GAGs are attaching to a protein core to form a proteoglycan monomer.

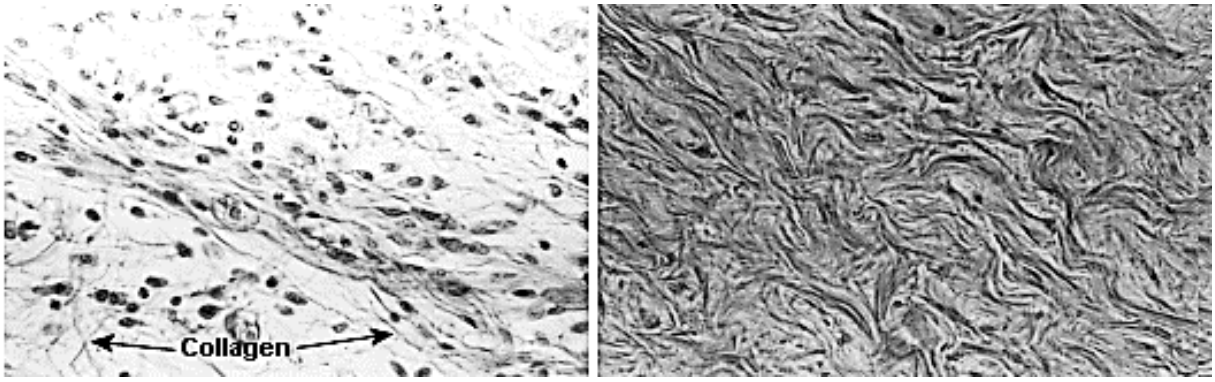
- GLYCOAMINOGLYCANS (GAGS)
  - GAGs attach at one end to a protein core and radiate outwardly from this core like the branches of an evergreen radiating from its trunk.
  - This arrangement of GAGs form a proteoglycan monomer.
  - Proteoglycan monomers attaching to a hyaluronic acid forms a proteoglycan aggregate.
  - Glycoaminoglycans produce a negative charge over the periphery of the proteoglycans monomers.
  - This negative charge repels adjacent negatively charged proteoglycan monomers as these approach each other.
  - This negative to negative charge repulsion results in increased tissue stiffness.
  - Glycoaminoglycans of proteoglycans are also hydrophilic so that these units attract and hold water which is important for cartilage nutrition and in producing the compressive stiffness of cartilage

TABLE of Glycosaminoglycan (GAG) types in human tissues

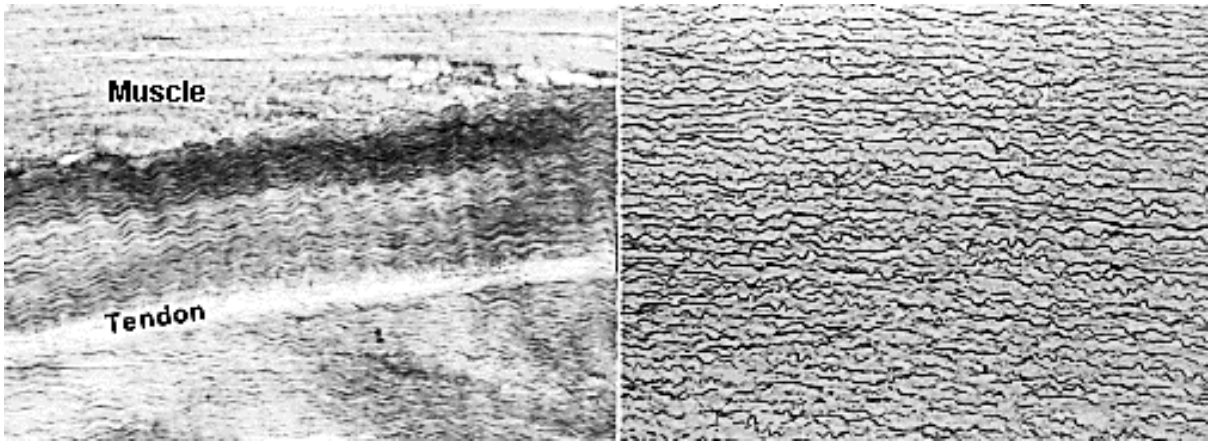
GAG type	Location
Hyaluronic acid	Basic central chain of proteoglycans; found mainly in connective tissue proper, cartilage and synovial fluid
Chondroitin-4-sulfate	Hyaline and elastic cartilage, bone, large blood vessels, nucleus polposus
Chondroitin-6-sulfate	Hyaline and elastic cartilage, tendon, intervertebral disk
Dermatan sulfate	Dermis, tendon, capsule, ligament, fascia, fibrocartilage, nerve, arteries
Keratan sulfate	Cartilage, bone, nucleus pulposus, annulus fibrous, cornea
Heparin sulfate	Basil lamina, aota, lungs, liver, smooth muscle, endoneurium

- **TYPES OF CONNECTIVE TISSUE PROPER**

LOOSE CT **DENSE IRREGULAR**



DENSE REGULAR (tendon) **ELASTIC TISSUE**



Photomicrographs of the 4 types of connective tissue (CT) proper.

- LOOSE (areolar) CONNECTIVE TISSUE

Fibers: few loosely arranged collagen fibers, with a few reticular and elastic fibers
Cells: many cells including fibroblasts, myofibroblasts, macrophages, plasma cells, mast cells, eosinophils, basophils, lymphocytes, fat cells
Locations: superficial fascia, epimysium, myofascia, papillary layer of the dermis, tunica adventia around blood vessels
Functions: permits movement of neurovascular bundles during limb and trunk movements; permits adjacent muscles to contract individually; allows blood vessels to enlarge.

o DENSE IRREGULAR CONNECTIVE TISSUE

Fibers: many densely packed collagen bundles arranged in many different directions, few elastic fibers
Cells: few fibroblasts, macrophages
Locations: dermis of the skin, periosteum, perichondrium, joint capsules, capsules around organs, aponeuroses
Functions: resist multidirectional tensile forces and shear force; stabilizes joints; protection

o DENSE REGULAR CONNECTIVE TISSUE

Fibers: many densely packed collagen bundles arranged in parallel rows running in the same direction, few elastic fibers
Cells: few fibroblasts
Locations: tendons, ligaments, fascia, aponeuroses
Functions: transmit unidirectional tensile forces, stabilize joints

- ELASTIC CONNECTIVE TISSUE

Fibers: an abundance of elastic fibers interwoven among collagen fibers
Cells: fibroblasts
Locations: ligamentum flavum, wall of large (elastic) arteries, vocal ligaments of larynx.
Functions: dampen high pressure in arteries; return structures to resting position

4.1.2 BIOMECHANICS OF CONNECTIVE TISSUE PROPER

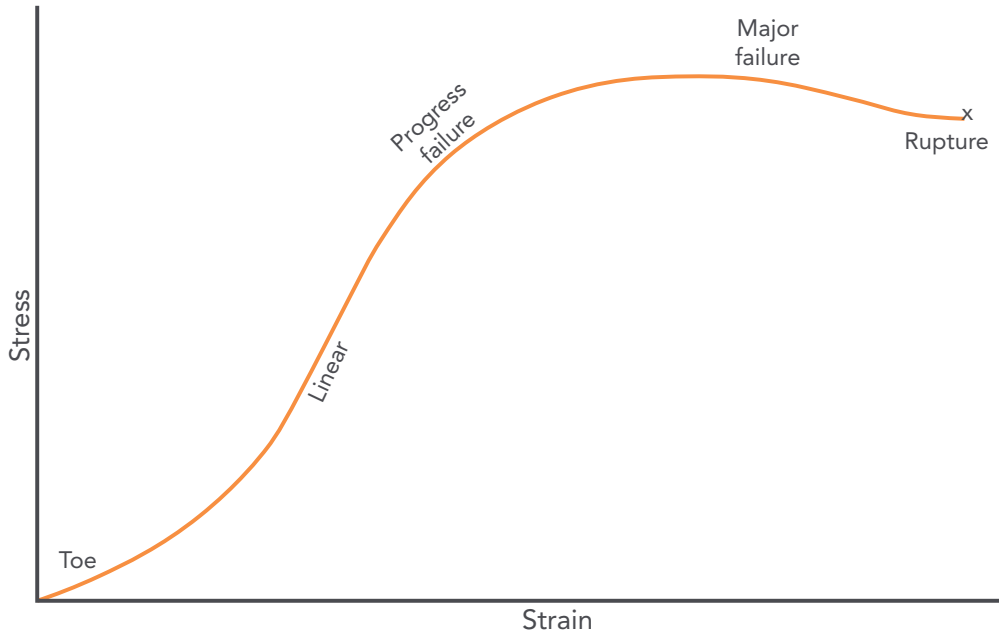
- **FIBERS**

  - COLLAGEN FIBERS
    - Show very little elongation (< 10%) in tension, but bend easily in compression (Parry and Craig, 1988; Yamada, 1970).
    - Show a greater resistance to shear stress than elastic fibers.
    - Structures, such as tendons that transmit muscular forces and ligaments that provide joint stability, are composed mainly of collagen fibers, making these structures strong and stiff in tension.

  - ELASTIC FIBERS
    - less stiff than collagen fibers
    - Easily elongate to 1.6–2.0 times its length when tension is applied (Fung,1981; Parry and Craig, 1988).

- **TENDON/LIGAMENT STRESS-STRAIN CURVE**

  - The stress-strain curves for tendons and ligaments are the basically the same so that all descriptions relating to the stress-strain curve applies to both structures.



Graph of the stress-strain curve for both tendons and ligaments

- TOE REGION: Collagen fibers are on slack so that small stress produce a lot of deformation as the fibers tighten.

- LINEAR OR ELASTIC REGION: Collagen fibers are tightening and as these tighten greater stress is need to produce the strain or deformation.

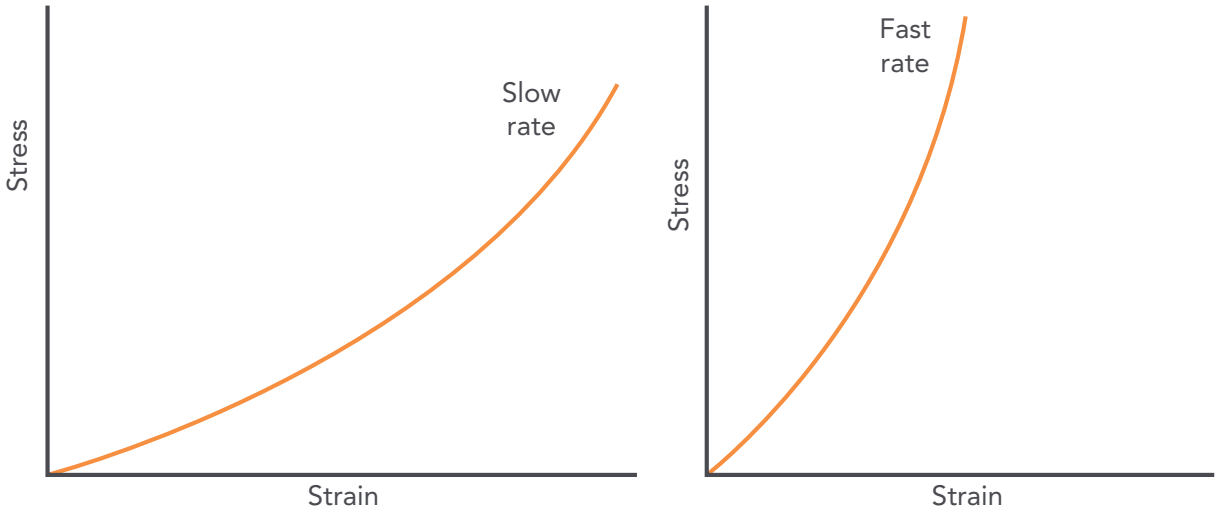
- PROGRESSIVE FAILURE: Some of the collagen fibers are broken and the structure is damaged and permanently deformed. A small increase in stress results in a proportionately large deformation.

- MAJOR FAILURE: Most of the collagen fibers are broken and the structure is weak and permanently deformed. Maintained or decrease stresses continue to produce a proportionately large deformation.

- RUPTURE: All collagen fibers are broken.

- **TENDONS/LIGAMENTS RESPONSE TO RATE OF STRESS**

SLOW RATE OF TENSION **FAST RATE OF TENSION**



Graphs of stress-strain curves showing the amount of strain (deformation) for tendon and ligaments when the stresses are applied slowly (LEFT) and rapidly (RIGHT).

- As the rates of applied tension to tendons and ligaments increase, tendons and ligaments show an increase in stiffness and a decrease in the amount of elongation.

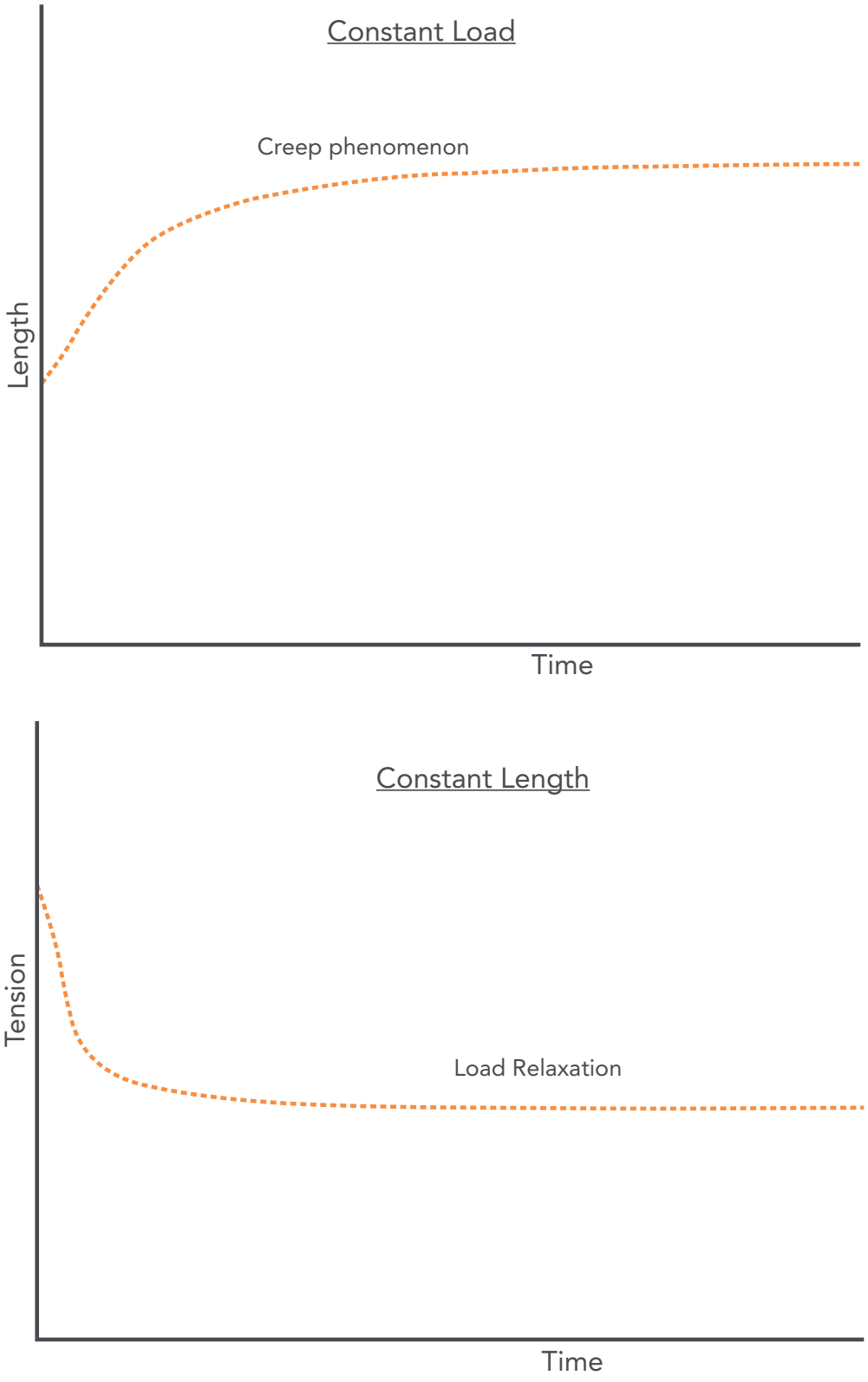
- This property suggests that elongation of ligaments and tendons is best obtained by applying low force for a long period of time.

- **OTHER TENDON/LIGAMENT RESPONSE TO STRESS**
  - As the magnitudes of tensile stress to tendons and ligaments increase with time, tendons and ligaments may respond by adding collagen and increasing the number of cross-links, which increases its strength.
  - Tension on tendons and ligaments at sub-failure levels stimulates fibroblasts to produce new collagen fibers.
  - In surgically repaired tendons and ligaments, tension accelerates healing, increases the strength of the connective tissue scar at the repair site, and directs the orientation of the collagen fibers along the directions of stress (Cummings and Tillman, 1992).
  - The addition of new collagen fibers results in an increase in the thickness and stiffness of that structure (Nordin and Frankel, 1989; Noyes, 1977; Woo et al., 1988).
  - The body strengthens tendons and ligaments to match the demands of increased muscle force and increased joint traction that occur with resistive exercise, increased work loads, and with growth.

The stiffness of collagen in tension changes with temperature (Cummings and Tillman, 1992; Tillman and Cummings, 1992). Cold makes collagen fibers brittle. Heat makes collagen fibers extensible. Collagen fibers exposed to cold and low tensile loads break prematurely. Collagen fibers exposed to heat and low tension force elongate farther than normal before breaking.

- **CREEP AND LOAD RELAXATION PHENOMENA FOR TENDONS AND LIGAMENTS**

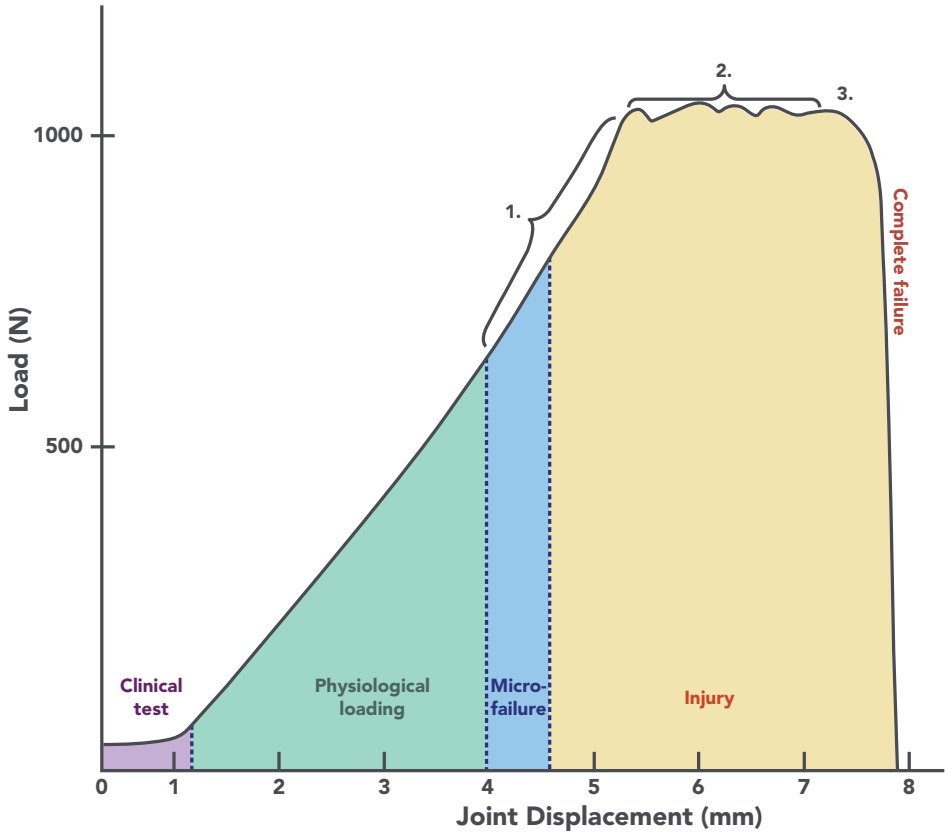
  - Tendon and ligaments are viscoelastic materials.
  - These materials show the phenomena for creep and load relaxation.
  - **Creep** is an increase in deformation that occurs over time when the load is constant.
  - **Load relaxation** is a decrease is stress over time when the magnitude of the deformation is constant.
  - These phenomena are the result of a reorganization of the collagen and proteoglycans in the materials.



Graphs showing the creep (TOP) and the load relaxation (BOTTOM) phenomena. Tendons and ligaments will increase in length when a constant tension load is applied over time as seen in the top graph. Tension stresses within tendons and ligaments will reduce over time as seen in the bottom graph. This reduction in stresses results from the redistribution of proteoglycans and water to reach an equilibrium state.

- **TENDON/LIGAMENT INJURY**

  - When high levels of tension and frequenctly repeated levels of tension are applied to tendons and ligaments, not all the collagen fibers fail at the same time.
  - Failure of only some of the collagen fibers is referred to as **microfailure.**



Graph of the stress (load) – strain (displacement) curve for the anterior cruciate ligament showing areas of testing, physiological loading and injury. (Modified from Noyes et al 1974)

  - Microfailures result in some pain and a slight weakening in the ligament/tendon but no joint instability (area 1).
  - When enough collagen is damaged and the ligament/tendon passes its yield point, there is permanent deformation and there is noticeable inflammation and joint instability (area 2).
  - When all the fibers fail, then the ligament/tendon ruptures (#3).
  - Collagen damage to a ligament is a sprain, while damage to a tendon is a strain.

The laxity of ligaments, either locally or systemically, produces an increase in joint mobility that is called HYPERMOBILITY. Hypermobility at a joint creates marked joint instability. This instability can result in a SUBLUXATION in which a bone is partially displaced from its normal joint position or a DISLOCATION in which a bone is completely displaced from its normal joint position. Hypermobility also exposes the opposing joint surfaces to abnormal forces. Abnormal forces, in turn, produce abnormal wear and degeneration on the articular surfaces, causing pain and inflammation not only of the joint but also of the tendons, ligaments and muscles associated with movement at that joint.

- **TENDON/LIGAMENT REPAIR** (replacements differ – see below)
  - DAYS 2–4: CELLULAR STAGE
    - Clot forms.
    - Infiltration of macrophages and fibroblasts
    - Weak and unstable type III collagen firbers are produced.
    - Wound connection is basically cellular and very fragile.
    - Stretching of wound tears the connection.

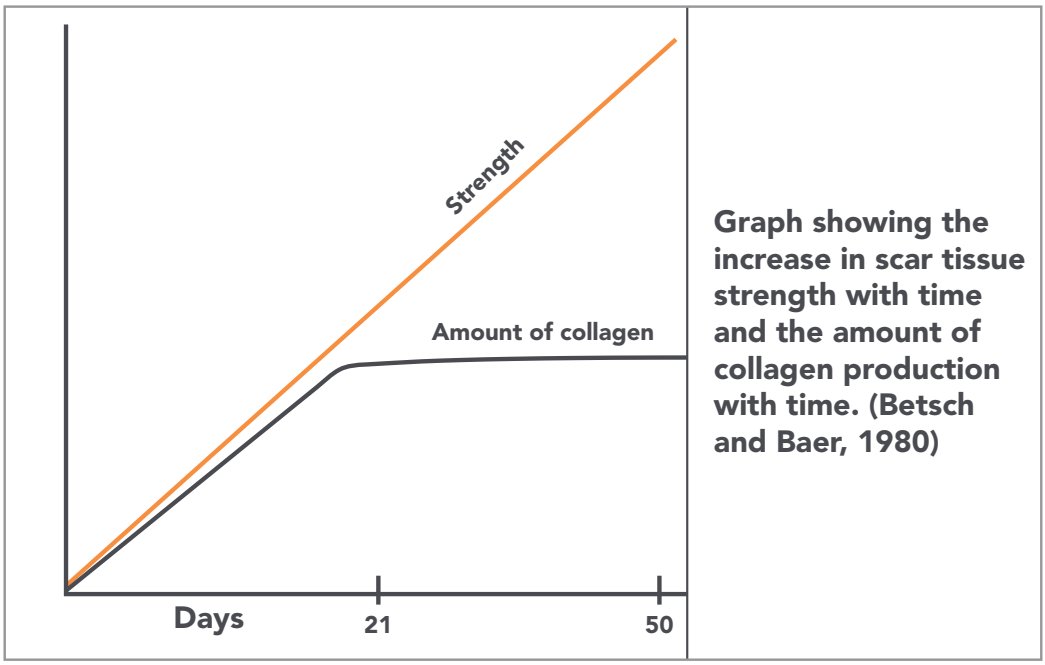
- DAYS 5–21: FIBROPLASIA
  - Wound is still very cellular.
  - Collagen production increases.
  - Collagen synthesis is high.
  - Collagen matrix is remodeling.
  - Good time to increase range of motion and joint function.
  - Tension helps heal wound.
    + Tension directs collagen fiber direction.
    + Tension increases strength of wound connection.
    + Tension increases healing rate and completeness of healing.

- DAYS 21–60: CONSOLIDATION
  - Collagen is well organized.
  - Wound connection is more fibrous than cellular.
  - Wound connection increases in the strength with the increase in collagen and increase in number of stable bonds (cross links).
  - There is a decreasing tissue response to treatment with time.

- DAYS 60–360: MATURATION
  - Large type I collagen is predominant.
  - Wound connection is mainly collagen.
  - Stable cross links
  - Poor response to treatment.

- TENSION IMPORTANT DURING FIBROPLASIA AND CONSOLIDATION
  - Tension activates fibroblast production of collagen.
  - Tension controls the direction collagen fibers are aligned.
  - Tension produces large collagen bundles.
  - Tension increases scar strength by increasing the alignment, amount, size and stability of collagen.
  - Tension increases healing rate and the completeness of healing.

- GRAPH OF COLLAGEN AND SCAR TISSUE STRENGTH



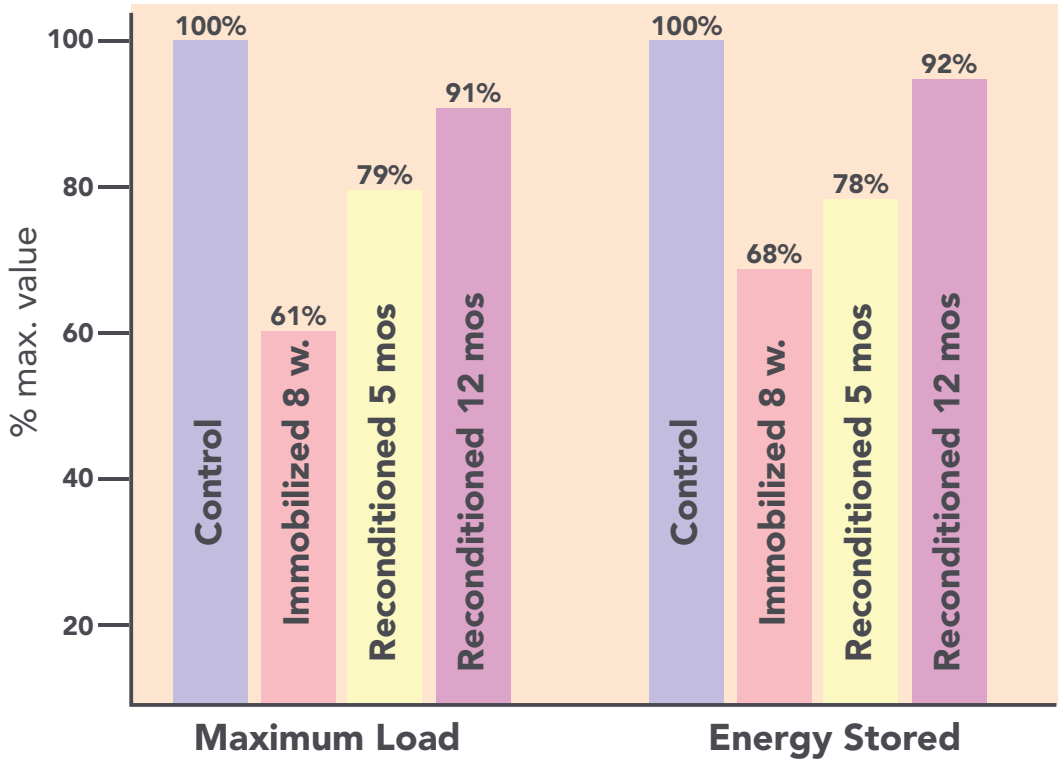
Graph showing the increase in scar tissue strength with time and the amount of collagen production with time. (Betsch and Baer, 1980)

- DAYS 2–21: Collagen increases in amount and the strength of the scar increases.
- DAYS 21–60: Collagen amount levels off and is fairly constant but strength of the scar increases. Increase in strength is due to fiber alignment, large bundles of collagen forming and increase in cross link formation.

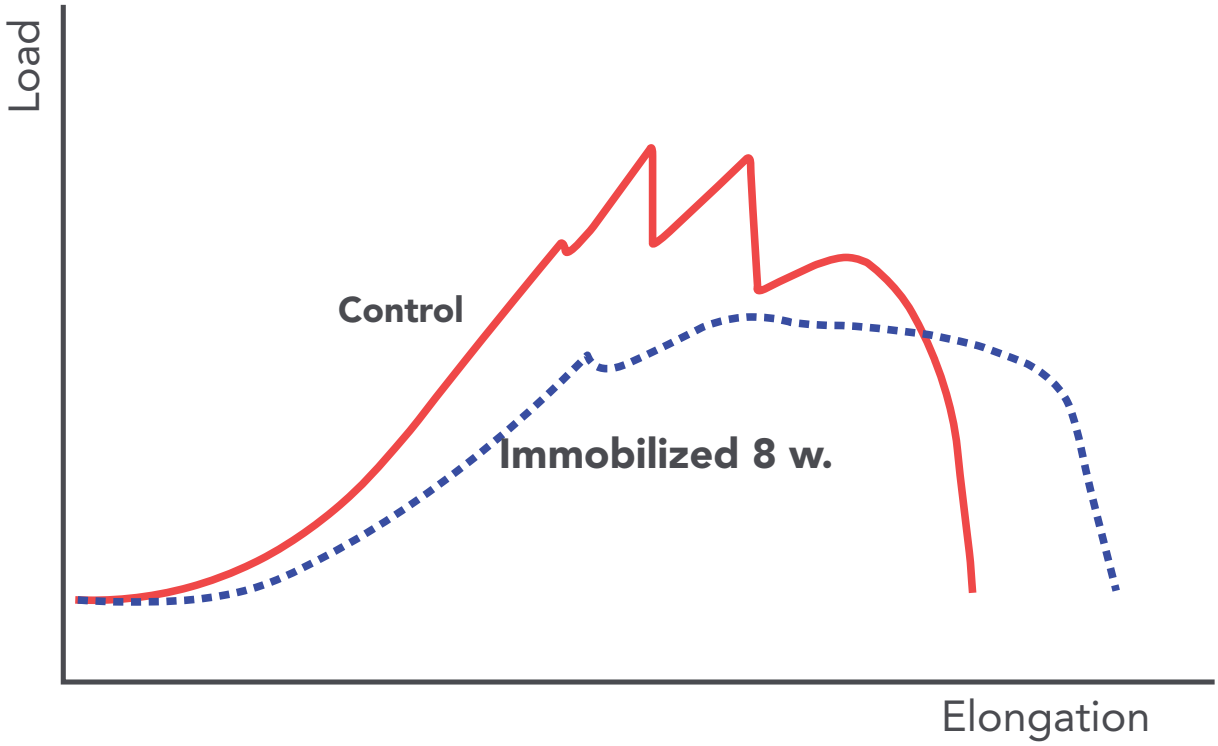
With ligament and tendon replacements, there may be several weeks before the replacement can safely be loaded in tension. This is because the replacement tissue degenerates initially. Thereafter, new collagen is produced. With the production of new collagen, tension becomes important to align collagen fibers, and strengthen and heal the replacement, just as above.

- **IMMOBILIZATION**

  - Results in the loss of tissue stiffness and strength.
  - 7 weeks of immobilization does not tend to affect the ligaments or capsule of a joint if there is no inflammation.
  - If joint inflammation is present, then adhesions and joint involvement can occur within 4 weeks of immobilization.
  - Important to prevent and quickly eliminate joint inflammation when a joint is immobilized.



Graphs showing the percentage loading strength and percentage toughness (energy stored) of a ligament after 8 weeks of immobilization, and after 5 and 12 months of reconditioning activities. (Noyes, 1977)

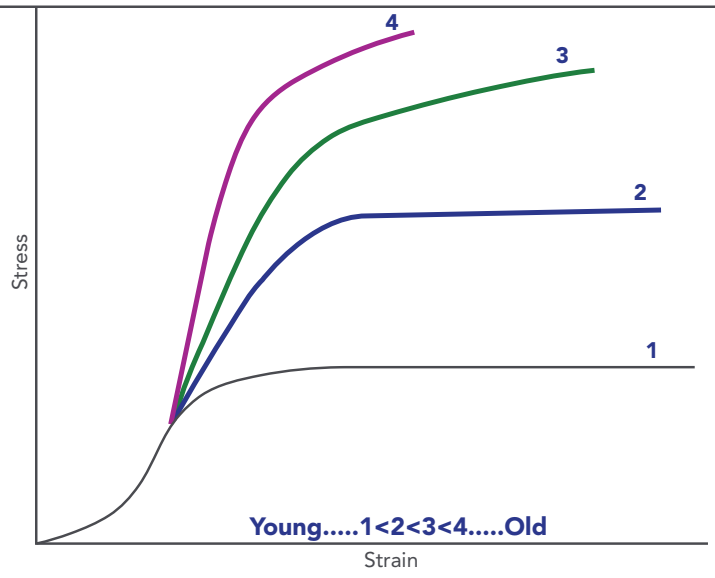


Graph showing the stress-strain curves of a ligament with no immobilization (control) compared to a ligament immobilized for 8 weeks. Note that the immobilized ligament shows greater elongation with less stress than the control ligament. The immobilized ligament is less stiff that the control ligament. (Noyes, 1977)

- **AGING**

  - Stiffness of tendons/ligaments in tension changes with age.
  - Young collagen fibers elongate more with less force than mature collagen fibers.
  - Young collagen fibers are less stiff in tension than mature fibers.
  - As collagen matures, strength and stiffness increase because of increased collagen production and cross-link formation.

- **CHANGE IN TENDONS WITH AGE**



Graph of stress - strain curves for young tendons (1) and progressing to old tendons (4). (Yamanda, 1970) Note the increase in stiffness with age.

- With advancing age the tensile strength and elasticity of ligaments and tendons decrease.
  - The amount of collagen decreases.
  - The number of large bundles of collagen fibers decreases.
  - Elastic fibers are damaged.

- The decrease in tensile strength is slight until about 70 yo (Yamada 1970).
  - Up to 50 yo there is a decrease in tensile strength of about 5%.
  - From 50 to 70 yo the decrease in tensile strength remains slight at about an additional 5%.
  - After 70 yo there is a decrease in tensile strength of about 20%.

- Elongation of tendons/ligaments to tension changes by only about 5% up to age 70 and then about another 10% thereafter (Yamada 1970).

- These changes in tensile strength and elongation appear to be greater for ligaments than for tendons with aging (Yamada 1970).

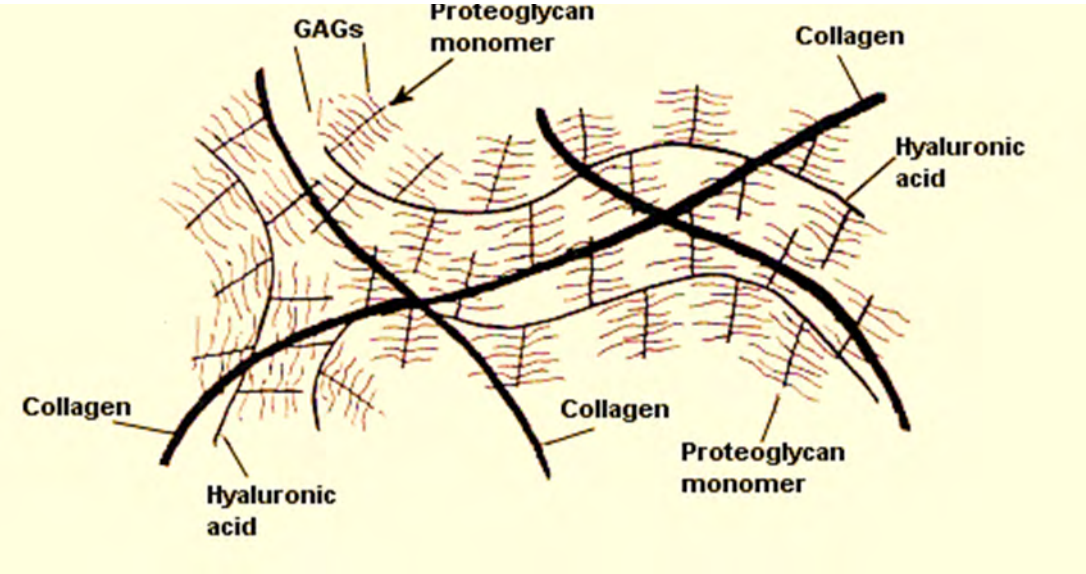
Study Questions (Connective Tissue Proper)

1. What are the functional benefits of Loose connective tissue and Dense Irregular connective tissue?
2. How do tendons/ligaments respond to slow and fast rates of tension and what are the functional implications of these responds?
3. How do tendons/ligaments respond to tension?
4. How does the tendon/ligament load vs. joint displacement (stress vs. strain) curve relate to tendon/ligament injury?
5. How does tension affect wound healing and when is the most effective time to obtain the best response and why?
6. How does the repair process for a tendon/ligament wound differ from that for a ligament replacement?
7. How are tendons/ligaments strengthened?
8. What are the affect of aging on the mechanical properties of tendons/ligaments?

4.2 CARTILAGE

4.2.1 ANATOMY

- **FIBERS AND GROUND SUBSTANCE**
  - Cartilage is typically avascular.
  - Cartilage cells called chondroblasts secrete collagen fibers and components of the ground substance.
  - Cartilage cells called chondrocytes lie in lacuna (little lake).
  - Ground substance contains water, glycoaminoglycans (GAGs) proteoglycans, hyaluronic acid, fibronectin and collagen fibers.
  - Water and proteoglycans give cartilage its hardness and its resistance to compression.
  - Collagen fibers give cartilage its resistance to tension.



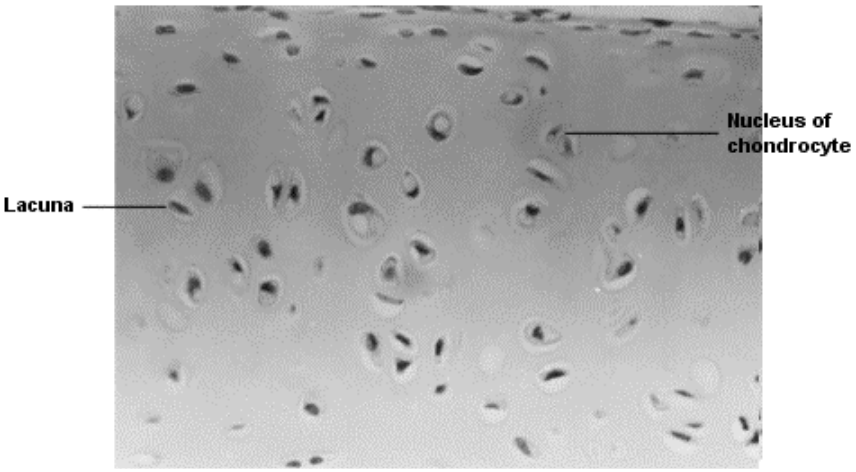
Drawing of the ground substance surrounding collagen fibers. A main component is water. The GAGs are attaching to a protein core to form a proteoglycan monomer.

Compaction of this ground substance squeezes water out from the articular cartilage as it deforms. As the cartilage loses water, the proteoglycans are compacted. Repelling negative ionic forces occur among the tightly packed proteoglycans and the rigidity of the ground substance increases. When the articular cartilage is then unloaded, the proteoglycans draw water back into the cartilage, returning it to its pre-loading stiffness. Because proteoglycans interact with water to produce stiffness, the maintenance of proteoglycans is critical for normal articular cartilage function. Loss of proteoglycans greatly reduces the loading capability of the cartilage (Tammi et al., 1989).

- **TYPES OF CARTILAGE**

  - HYALINE CARTILAGE

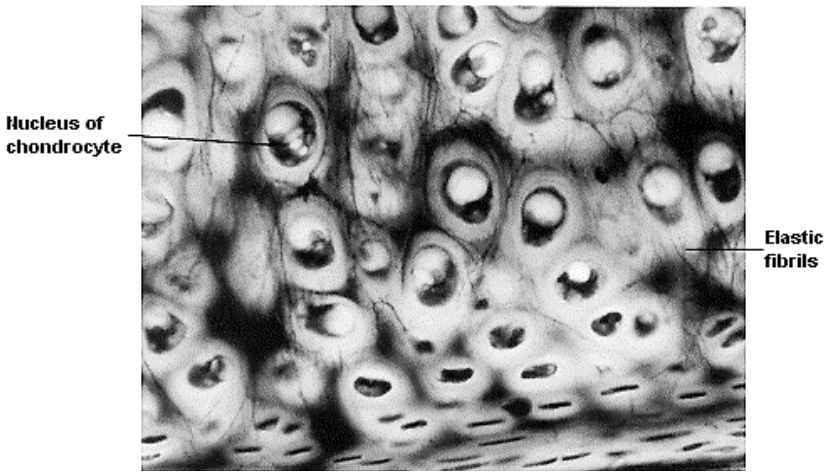
FIBERS: fine network of type II collagen fibers
CELLS: chondrocytes
LOCATIONS: costal cartilage, articular cartilage of joints, epiphyseal growth plate, trachea, skeleton of larynx, nasal septum



Photomicrograph of hyaline cartilage

- ELASTIC CARTILAGE

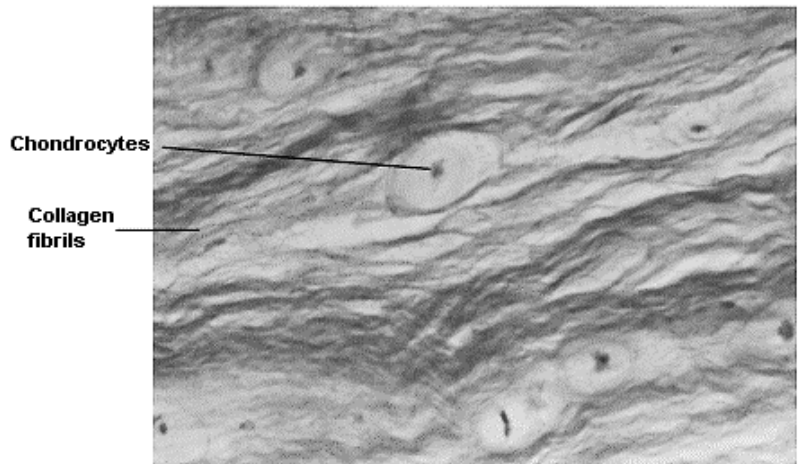
FIBERS:	abundance of densely packed elastic fibers interwoven among type II collagen fibers
CELLS:	chondrocytes
LOCATIONS:	auricle of ear, epiglottis, Eustachian tube, wall of external auditory canal



Photomicrograph of elastic cartilage. Black fibers are elastic fibers.

- FIBROCARTILAGE

FIBERS:	dense network of type I and type II collagen fibers but the amount of type II fibers varies
CELLS:	chondrocytes, fibroblasts
LOCATIONS:	menisci of knee, temporomandibular joint articular condyle, sternoclavicular joint disc, acromioclavicular joint disc, annulus of intervertebral disc, disc at pubic symphysis, tendo-osseous junction



Photomicrograph of fibrocartilage

- **ARTICULAR (hyaline) CARTILAGE STRUCTURE**

  - ZONES (LAYERS) OF ARTICULAR CARTILAGE
    - Articular cartilage has zones that differ in the arrangement of collagen fibers and in function.
    - The articular cartlage attaches to a layer of calcified cartilage.

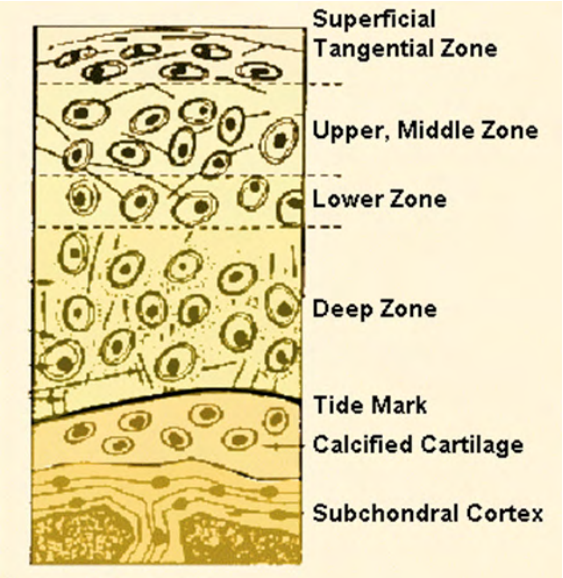


Diagram of the zones of articular cartilage (modified from Mow and Hung, 2001)

- superficial tangential zone
  + Surface layer makes up 10–20% of the articular cartilage.
  + Collagen fibers run parallel to the articular surface and are tightly interwoven.
  + This layer is very tough and durable zone.
  + Ths surface isvery smooth and slippery (surface estimated as 5 × slipperier than ice).
  + Important for the containment of proteoglycans within the articular cartilage.

- upper and lower middle zones
  + These zones make up middle 40–60% of the articular cartilage.
  + Collagen fibers are arranged randomly and loosely.
  + High concentrations of proteoglycans and water located between loosely arranged fibers.

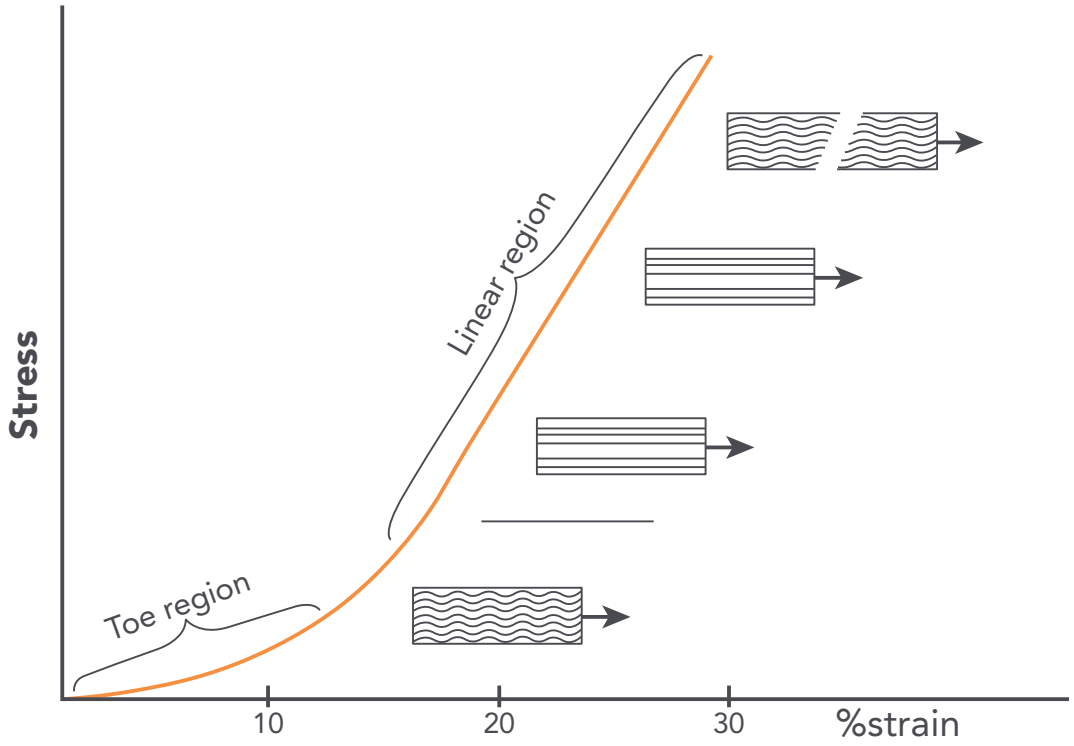
- deep zone
  + Makes up bottom 30% of the articular cartilage.
  + Collagen fibers are larger than in the superficial andmiddle zones.
  + Highest concentration of proteoglycans but lowest amount of water.
  + Fibers run vertically, nearly perpendicular to the surface of the bone.
  + Fibers penetrate into the calcifies cartilage layer which attaches the cartilage to the subchondral bone.

- NUTRITION OF ARTICULAR CARTILAGE
  - Articular cartilage is avascular.
  - It depends on the movement of water during joint loading and unloading for nutrition and the removal of waste products.
  - When articular cartilage is compressed (loaded), water with waste products leave.
  - When articular cartilage is unloaded, the proteoglycans draw water and nutrients into the cartilage.
  - When the superficial zone is damaged, water and proteoglycans are lost and nutrition to the articular cartilage is decreased which can lead to damage of the cartilage.

4.2.2 BIOMECHANICS OF CARTILAGE

- **TENSILE STRESS-STRAIN RELATIONSHIP OF HYALINE CARTILAGE**

  - The stress-strain relationship of hyaline cartilage has a toe region, linear region and a point of failure.
  - Because the type II collagen in articular cartilage is less dense and the fibers smaller than the type I collagen in tendons/ligaments, and the amount of water and proteogylcans are greater in articular cartilage than in tendons/ligaments, articular cartilage tends to failure without there being a yield point and plastic region as in tendons/ligaments.



Graph of a tension stress-strain curve for hyaline cartilage

  - TOE REGION: Collagen fibers are on slack but being loaded in tension and aligning in a direction to resist tension.
  - LINEAR REGION: Collagen fibers are stretching and becoming more and more tense as the stress increases.
  - FAILURE: Collagen fibers break and the cartilage tears.

- **CREEP AND LOAD (STRESS) RELAXATION OF ARTICULAR CARTILAGE**

  - Articular cartilage is a viscoelastic material and as such shows the phenomena of creep and load (stress) relaxation.
  - CREEP
    - Occurs after initial compression and the extrusion of water.
    - Creep is further deformation (compaction) of the articular cartilage in time with constant stress (load) being applied.
    - Results from collagen and proteoglycans reorganizing to reach an equilibrium state.

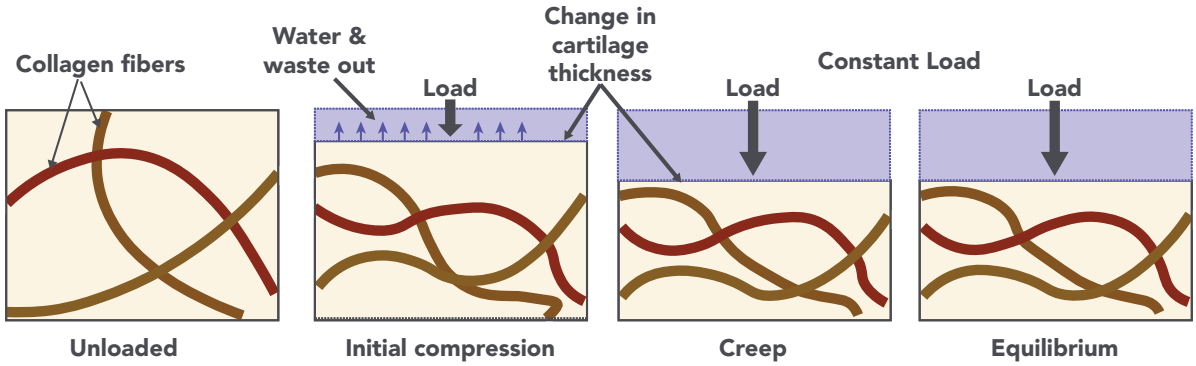


Diagram showing the mechanism for articular cartilage creep (modified from Mow and Hung 2001)

  - LOAD (STRESS) RELAXATION

    - After initial compression and the extrusion of water, stresses are high towards the superficial zone of the articular cartilage low at the subchondral bone.
    - With deformation (compaction) of the articular cartilage remaining constant with time, there is a redistribution of water and proteoglycans and a reorganization of collagen fibers.
    - The result is an even redistribution of stresses from the superficial zone of the articular cartilage to the subchondral bone producing a unit force equilibrium state.

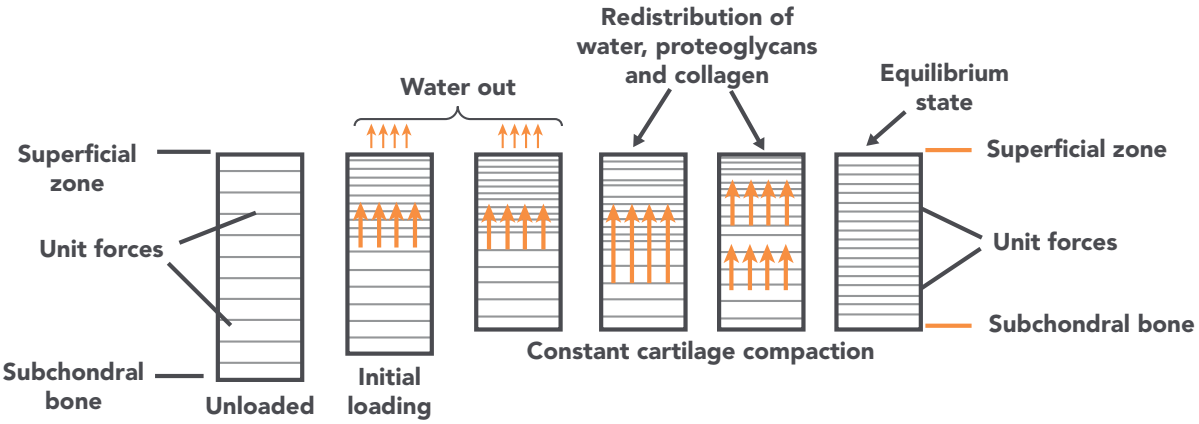


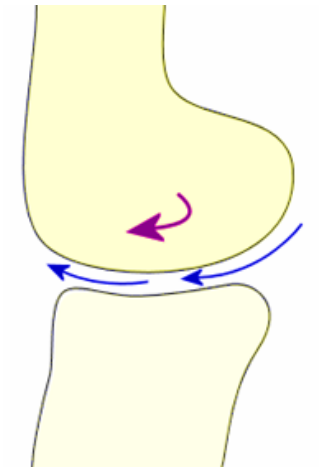
Diagram showing the mechanism for stress (load) relaxation in articular cartilage (modified from Mow and Hung 2001)

4.2.3 SYNOVIAL JOINT LUBRICATION

- Synovial joints are lubricated 1) Boundary lubrication; 2) Hydrodynamic lubrication; Squeeze film lubrication; and 4) a Combination of hydrodynamic and squeeze films lubrications.

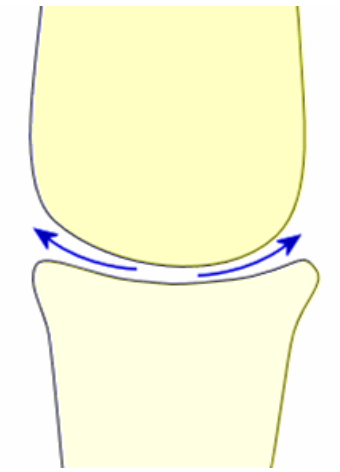
- **BOUNDARY LUBRICATION**
  - Lubricant (glycoprotein) is in the surface layer of the articular cartilage.
  - With extreme loading conditions, the lubricant is extruded to lubricate the articular surface.
  - It is the last method for protecting the articular surface when other lubricating methods fail.

- **HYDRODYNAMIC LUBRICATION**



- Used in joints where the opposing joint surfaces are not in parallel to each other.
- Synovial fluid is drawn between the surfaces as the opposing articular surfaces move on each other.

- **SQUEEZE FILM LUBRICATION**



- Used in joints where the opposing joint surfaces are in parallel with each other.
- Synovial fluid tends to pool in the concave surface of the joint and is squeezed out between the joint surfaces when the joint is compressed.

4.2.4 ARTICULAR CARTILAGE WEAR

- **INTERFACIAL WEAR**

  - Interfacial wear consists of 1) adhesive wear and 2) abrasive wear.
  - Adhesive Wear
    - Chronic inflammation and disease may result in adherence of the opposing joint surfaces.
    - Movements pull and tear at these adhesive areas causing surface wear and damage.

  - Abrasive Wear
    - Particles of articular cartilage or bone are between opposing joint surfaces.
    - Povements compresses these particles between the joint surfaces and cause abrasion of the surface.
    - Crepitus is often the result of this type of wear.

- **FATIGUE WEAR**

  - Repeated stresses on articular cartilage producing tissue failure with time.
  - Generally occurs with old age.
  - Can result from low loads of high repetition for a long time as with aging.
  - Can also result from high loads of high repetition of a short time as in some sports or occupations.

- **IMPACT WEAR**

  - Caused by rapid and very high impact loading on the joint.
  - Few repetitions involved.

- **ARTICULAR CARTILAGE DEGENERATION**

  - FIBRILLATIONS
    - Sign of early articular cartilage degeneration.
    - Results from fraying of the collagen fibers in the superficial Zone.

  - CAVITATION
    - Follows fibrillations.
    - Cavities form in the cartilage between collagen bundles.

- VERTICAL SPLITTING
  - As the cavities deepen, clefts occur in the cartilage.
  - These clefts extend as vertical splits from the superficial to the deep zone of articular cartilage and even to the subchondral bone.

- CONTINUED EROSION
  - Final stage of degeneration
  - In the area of the splits, cartilage erosion continues.

4.2.5 ARTICULAR CARTILAGE REPAIR

- **NATURAL REPAIR**: The natural repair processes for full thickness defects involves bleeding from the subchondral bone into the damaged area and the formation of a fibrous scar. (Hunziker 2001; Buckwalter et al 2006; Redman et al 2005)

- **ARTHROSCOPIC REPAIR**: Arthroscopic debridement and irrigation are commonly used for pain relief but this procedure does not cause articular cartilage repair (Redman et al 2005). Arthroscopic abrasion, Pridie drilling and microfracture procedures are used in the area of damaged cartilage to expose subchondral bone and produce bleeding, clot formation and inflammation, causing fibrocartilage scar formation (Hunziker 2001; Redman et al 2005).[1] With drilling, 4 weeks of constant mobilization has been shown to help in fibrocartilage restoration. A modification of these bleeding procedures is to produce a fibrous paste that is formed by centrifuging blood of the recipient before surgery. This paste is then spread over the damaged articular cartilage to initiate a blood clot and a fibrocartilage scar.

- **PERIOSTEAL GRAFT**: The layer of periosteum that lies adjacent to the bone contains chondrocyte precursor cells (Hunziker 2001; Redman et al 2005). After a full thickness defect is made and a 1 mm of subchondral bone removed, a periosteal graft in implanted in to the defect with the layer contain chondrogenic cells facing the side of the articular surface. The graft is secured using a fibrin glue (Carranza-Bencano et al 2000 in Redman et al). In this graft, potential chondrocytes can be derived for the periosteum to form mesenchymal cell that differentiate into chondroblasts. The outcome of this procedure varies but age and post-op continuous motion are essential factors for success.

- **OSTEOCHONDRAL TRANSFER**: "Mosaic Plasty" and "OATS" are terms used for osteochondral transfers (Hunziker 2001;Redmen et al 2005). A plug of subchondral bone is removed from a non-weight bearing region of articular cartilage. For large defects and allogenic cadaver grafts, this procedure is commonly used. For small defects, an autogenic graft from the patient is usually used. In small and medium size full thickness defects, decreased pain and increased joint function have been reported (Hangody et al 2004; Jakob et al 2002 in Redman et al). However, the viability of the osteochondral transfer is questionable (Fitzgerald J et al 2008; Evans et al 2004 in Redman et al). Because the transfer comes from articular cartilage at a non-weight bearing site, the ability of the transfer to withstand loading force is in question. Some studies show chondrocytes dying at the margins of the transfer and predict tissue degeneration of the graft (Evans et al 2004). Others studies show fibrocartilage hypertrophy or the lack of fibrocartilage growth at the joint but both are accompanied with increased joint stiffness. (Ahmad et al 2002; LaPrade and Botker 2004 in Redman et al 2005). Further, the effects of removing articular cartilage at the doner site in an autogenic transfer are unknown.

- **AUTOLOGOUS CHONDROCYTE TRANSPLANTATION**: In this procedure, healthy articular cartilage only is removed by arthroscopy from a non-weight bearing site in the surgical joint of the patient (Hunziker 2001; Redman et al 2005). The chondrocytes are enzymatically separated from the matrix and place in a culture with natural growth factors. Once the culture is ready, the damaged area is debrided arthroscopically down to the subchondral bone. A periosteal graft is taken from the tibia and suture over the debrided area. The cultured chondrocytes are then inserted beneath the periosteal graft. In patients with femoral condyle repairs, over 70% showed a "hyaline-like" cartilage repair whereas only 14% of the patella repairs were "hyaline-like" (Redman et al 2005). This difference suggests that outcomes may vary with joint. However, clinical results are very promising using the procedure (Fitzgerald J et al 2008; Hettinga, 1990; Salter, 1998). After 12–24 months, patients showed improved function and quality of life. In 3 years, functional outcomes of the knee improved in 84% of the patients (Micheli et al, 2001). In a 2–9 year follow-up study (Peterson et al 2000), 90% of patients treated for femoral condylar articular cartilage damage had good outcomes. It is not clear if these good outcomes are the result of the cultured chondrocytes or the result of a combination of the cultured cells, chondrocyte precursors from the periosteal graft and mesenchymal cells from the subchondral bone.

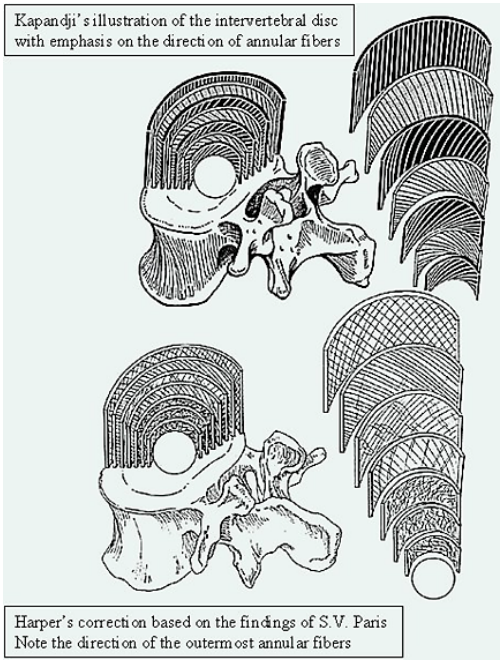
- **JOINT LUBRICANTS**: These are viscosupplements substances, such as Hyalgen, Orthovisc and Synvisc, that are injected into mainly osteoarthritic joints for lubrication and to stimulate the production of synovial fluid (Ciccone 2007 Pharm in Rehab). These injections may provide some relief of pain from 3 months to 1 year.

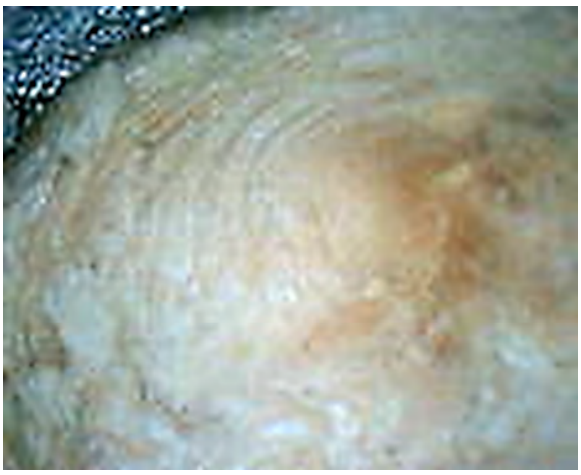
- **NUTRITIONAL SUPPLIMENTS**: Chondroitin and glucosamine supplements are over the counter dietary supplements. Studies have shown that in some patients there is a generalized reduction in joint pain or discomfort in multiple joints (more systematic than precise) accompanied by improved function. Some standard imaging studies also suggest a reduction in articular cartilage degeneration (Ciccone 2007). However, further well controlled studies on the benefits of these supplements are still needed. Currently, no adverse side effects have been reported with the use of these supplements, but patients should still consult with their physician to eliminate potential drug interaction other prescribed medications.

4.2.6 FIBROCARTILAGE

- **INTERVERTEBRAL DISC**

  - The disc contains a central gel-like nucleus pulposus surrounded by 8–10 rings of fibrocartilage making up the annulus fibrous.
  - The annulus contains nearly equal amounts of type I and type II collagen.
  - The innermost rings show a loose arrangement of collagen to allow for deformation of the nucleus.
  - The middle rings show a dense arrangement of collagen with the fibers running mainly in one direction.
  - The fiber directions of sequential middle rings are nearly perpendicular to each other.
  - Outermost rings show a dense arrangement of collagen with the fibers showing a crossing (herringbone) pattern which is indicative of a material that is strong in tension and shear.



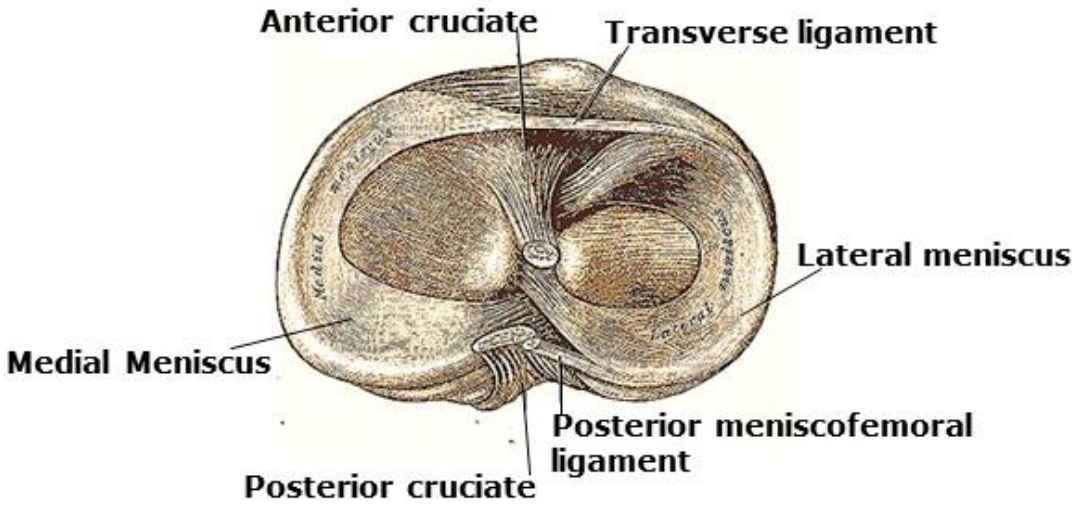


Drawings of the layers of the annulus fibrosus showing the arrangements of collagen fibers in each row by Kapandji (UPPER LEFT) and by Harper based on Paris (LOWER LEFT). Picture of a dissected intervertebral disc (RIGHT) showing the brownish nucleus pulposus and the whitish rings of the annulus.

- DISC TENSILE STRENGTH
  - nucleus pulposus = 3kg/m2
  - inner rings = 45kg/m2
  - middle rings =53kg/m2
  - outer rings = 80kg/m2

- **MENISCI**

  - Consists of fibrocartilage with mainly type I collagen and a small amount of type II collagen.
  - The superficial collagen fibers are crossed showing a herringbone pattern which indicates strength in tension and shear.
  - The deep fibers run circumferentially which would strengthen the menisci in anterioposteriorly directed tension.
  - With compression loads, the proteoglycan resist the compression but shear and anteroposterior and medial-lateral tension is resisted by the collagen fibers.



Drawing of the medial and lateral menisci of the knee (modified from Gray 1918)

- FUNCTIONS OF THE KNEE MENISCI

  - Increase surface area for force distribution which decreases unit forces on the femoral and tibial condyles.
  - Increases surface area for lubrication of the condyles.
  - Joint proprioception because of innervation of the anterior and posterior horns of the menisci.

- BLOOD SUPPLY OF THE KNEE MENISCI

  - Only the peripheral 10%–30% of the menisci have a blood supply in teens and adults.
  - Central 90%–70% of the menisci is avascular which results in a very poor healing capability in this portion of the menisci.

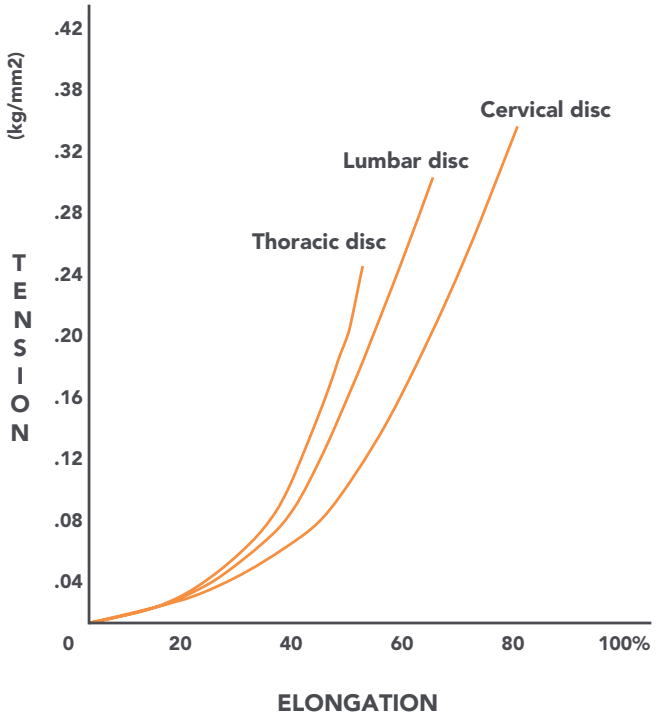
4.2.7 STRESS-STRAIN PROPERTIES AND AGING

- **HYALIN CARTILAGE**

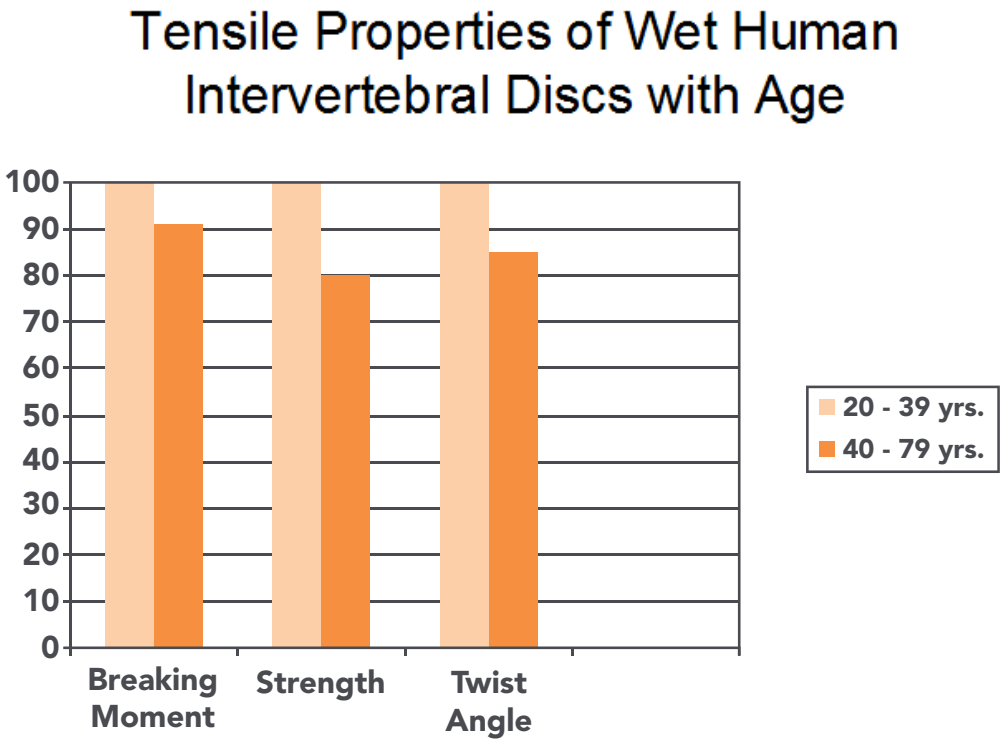
  - Tensile properties (strength and elongation) begin to show noticeable decline at about age 40 of about 20%. After age of 50, the decline reaches about 40% and the decline continues so that after 60 there is a 60% reduction in hyaline cartilage tensile properties.
  - Compression properties (strength and compaction) show a decline of about 5% at about age 40. After age 50 there is a decrease of 20% but then little to no decrease up to the age of 70. Decreases in compressive properties of hyaline cartilage much are less over time than the tensile properties.
  - Decrease in compressive properties are less over time than the tensile properties.

- **DISC FIBROCARTILAGE**

  - The disc reaches its peak of health at around 25 years of age.
  - Around the age of 30, the annulus begins to show macroscopic signs of use and abuse, usually reflecting life style choices.
  - Disc protrusions usually begin at age 30 and continue to about age 50 after which protrusions reduce. This reduction occurs, although the annulus continues to weaken with age, because the nucleus is also aging but faster than the annulus. As a result, the aging nucleus is less able to imbibe fluids and internal disc pressures are low, reducing the likelihood of a disc herniation.
  - By age 60, protrusions of the nucleus are rare.
  - TENSILE PROPERTIES OF THE DISC
    - The breaking loads are least for the small cervical disc, intermediate for the intermediate size thoracic disc and greatest for the large lumbar disc.
    - The breaking loads correspond directly to disc size.
    - When disc sizes are considered and the strengths calculated, the cervical disc is stronger than the lumbar and the thoracic less than both cervical and lumbar discs.
    - Elongation strains are greatest for the cervical disc, intermediate for the lumbar disc and lowest for the thoracic disc.
    - With age, disc elongation decreases with age. Cervical disc elongation decreases 21%, upper thoracic elongation decreases 25% and low thoracic decrease 30%. Lumbar disc elongation decrease 25%.
    - With age, cervical disc strength decreases about 12%, upper thoracic strength decrease about 17%, but lower thoracic strength decreases by only about 12%. Lumbar disc strength decreases by 20%.

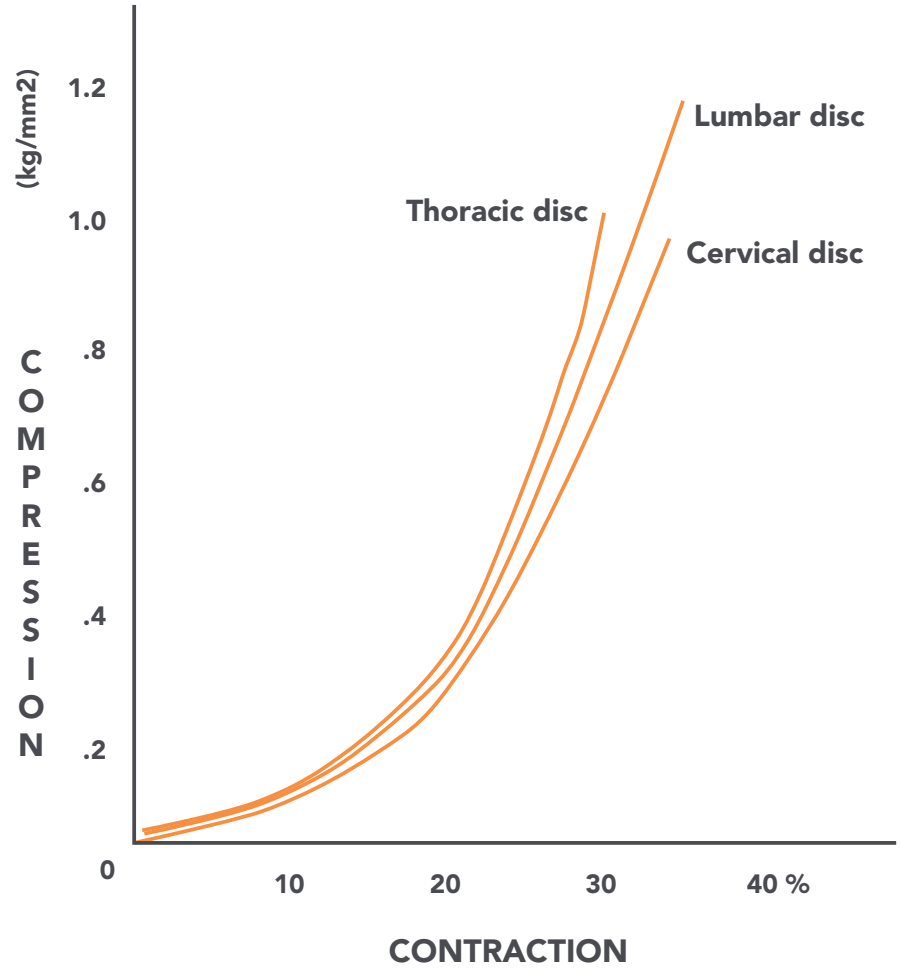


Graph showing the tension stress-strain curves for the cervical, thoracic and lumbar discs. (Yamada 1970)



Graphs showing the average changes in tensile loads, strengths and strains between an age group of 20–39 year old and a group of 40–79 years old.

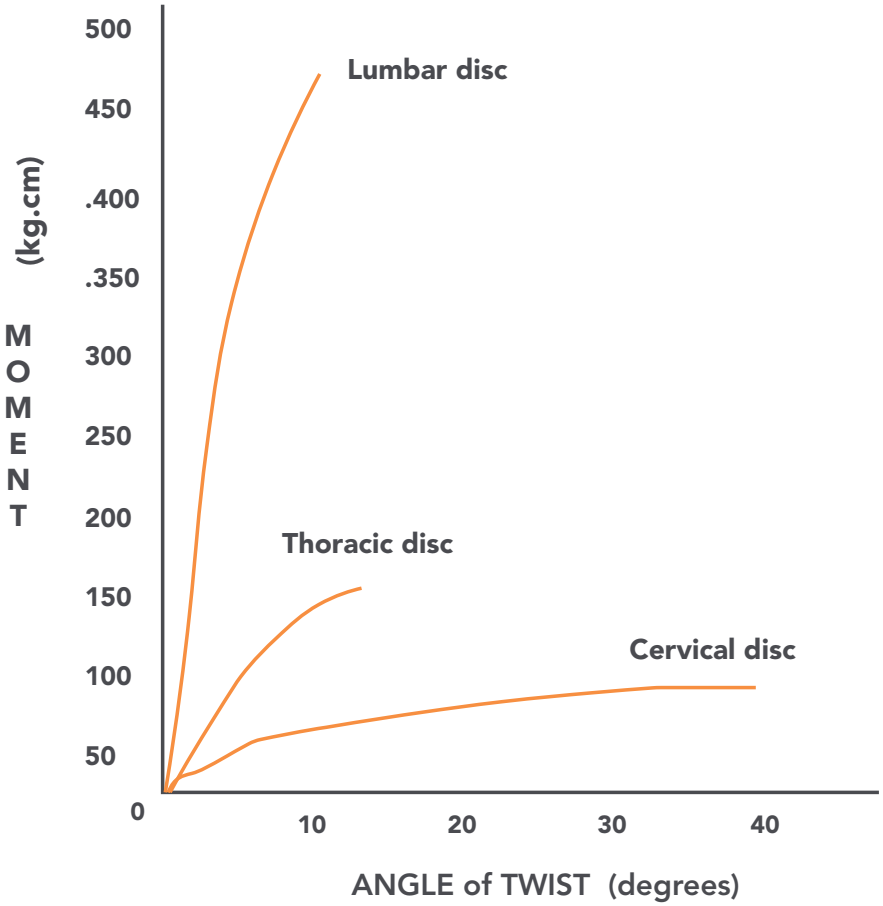
- COMPRESSION PROPERTIES OF THE DISC
  - Compression strengths are greatest for the lumbar discs, less for the thoracic discs and least for the cervical discs.
  - Compaction (contraction) is greatest for the cervical discs, less for the lumbar discs and least for the thoracic discs.
  - Compression breaking loads are 40–59% less in females than males.
  - With age, there is a 60–70% decrease in strength in both males and females.



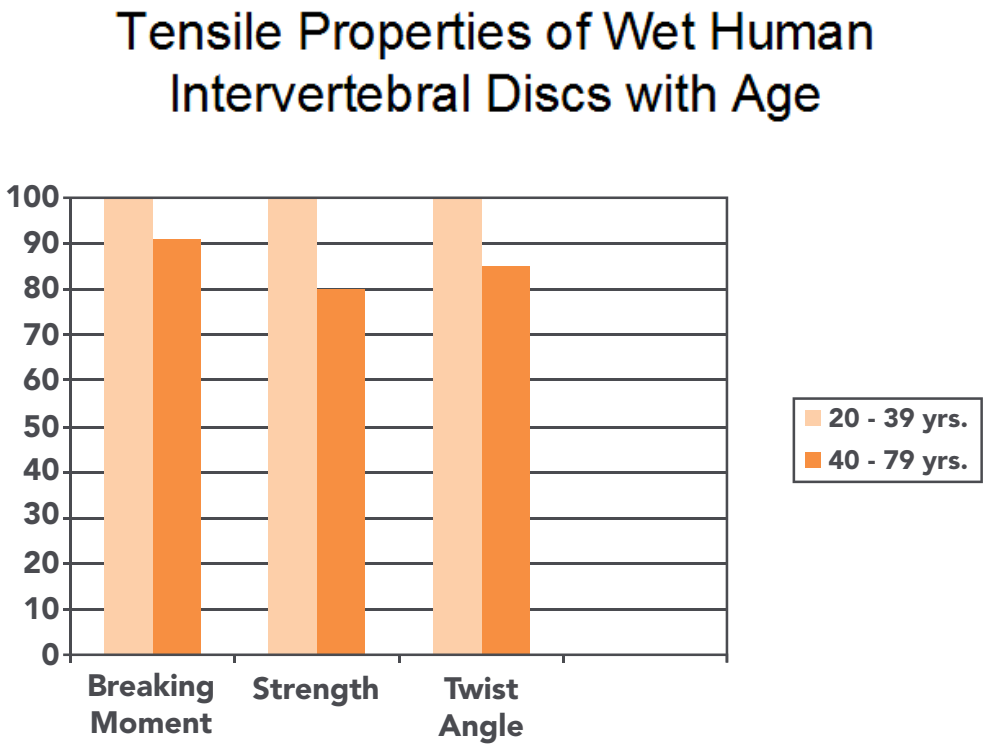
Graph showing the comprerssion stress-strain curves for cervical, thoracic and lumbar intervertebral discs. (Yamada 1970)

- TORSIONAL PROPERTIES OF THE DISC
  - The forces that harm the disc are many but torsional forces are perhaps the most injurious especially when the direction of forces suddenly changes.
  - Torsional breaking loads are least for the cervical disc, intermediate for the thoracic disc and greatest for the lumbar disc.
  - Torsional breaking loads are nearly 9 times greater for the lumbar than the cervical disc.
  - Torsional breaking loads correspond directly to disc size.
  - When disc sizes are considered and the strengths calculated, the cervical and lumbar torsional strengths are nearly the same and the thoracic strengthens are slightly less than both cervical and lumbar discs

- With age, torsional disc cervical strength decreases about 12%, upper thoracic disc strength decreases about 21%, middle thoracic strength decreases about 11% and lower thoracic disc strength decreases about 9%. Lumbar disc strength decreases about 10%.
- The angle of twist at which the disc breaks also decreases with age. The cervical angle of twist decreases about 19%. In the thoracic region, the upper thoracic angle decreases about 18%, the middle disc angle decreasing about 17% and lower disc angle decreases about 12%. The lumbar angle of twist decreases about 13%.
- The angle of twist appears to correspond with the amount of rotational movement that can occur in that region of the spine.



Graph showing the torsional stress-strain curves for cervical, thoracic and lumbar intervertebral discs. (Yamada 1970)



Graphs showing the average changes in tensile loads, strengths and strains between an age group of 20–39 year old and a group of 40–79 years old.

Study Questions (Cartilage)

1. How are tension and compression resisted in articular cartilage?
2. What are the functions of proteoglycans in articular cartilage?
3. What is the difference between creep and load (stress) relaxation and howare these properties similar?
4. What are the four types of joint lubrication and how do they differ?
5. What are the effects of aging on articular cartilage and fibrocartilage?

4.3 BONE

4.3.1 ANATOMY OF BONE

- **TYPES OF BONE**

  - Fibrosus bone shows an irregular and unorganized pattern of bone and is found during embryonic bone formation and development, in fracture healing, and in pathological bone.
  - Lamellar bone consists of rows or concentric circles of bones and is the typical bone of the skeleton.
  - SPONGY OR CANCELLOUS LAMELLAR BONE
    - Internal network of delicate processes called trabeculae.
    - Abundant at the ends of long bones in the epiphyses.
    - Trabeculae align to resist the common stresses placed on the regions of the bone where these are located.
    - The trabeculae are arranged in rows of bone cells and not osteons.

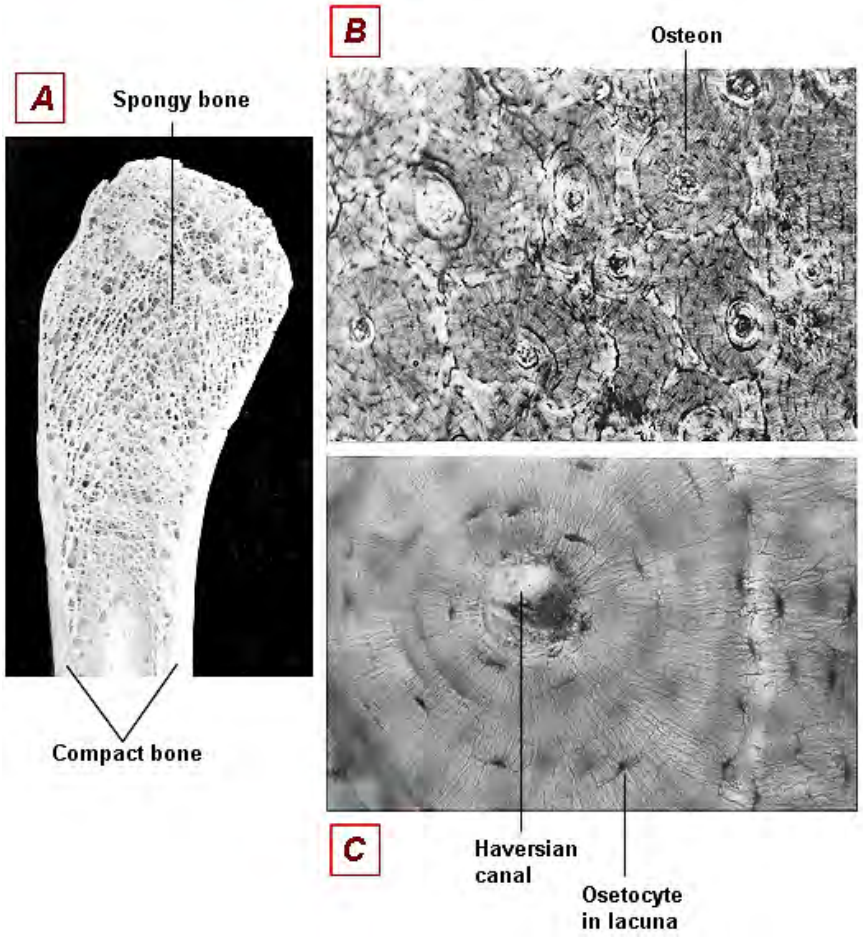
  - DENSE OR COMPACT LAMELLAR BONE
    - Solid bone tissue found on the periphery of bones.
    - Thin in the region of the epiphyses but usually thick in the diaphyses of long bones.
    - Consists of concentric circular layers of bone forming an osteon or Haversian system.

  - COVERINGS OF BONES
    - Bones are covered externally, except in the areas of articular cartilage, by a dense connective tissue layer called periosteum.
    - Periosteum contains osteogenic cells (bone forming cells) and is anchored to the bone by Sharpey's fibers.

- The internal surfaces of bones are covered by a thin cellular layer called the endosteum.
- Endosteum also contains osteogenic cells and bone absorbing cells called osteoclasts.

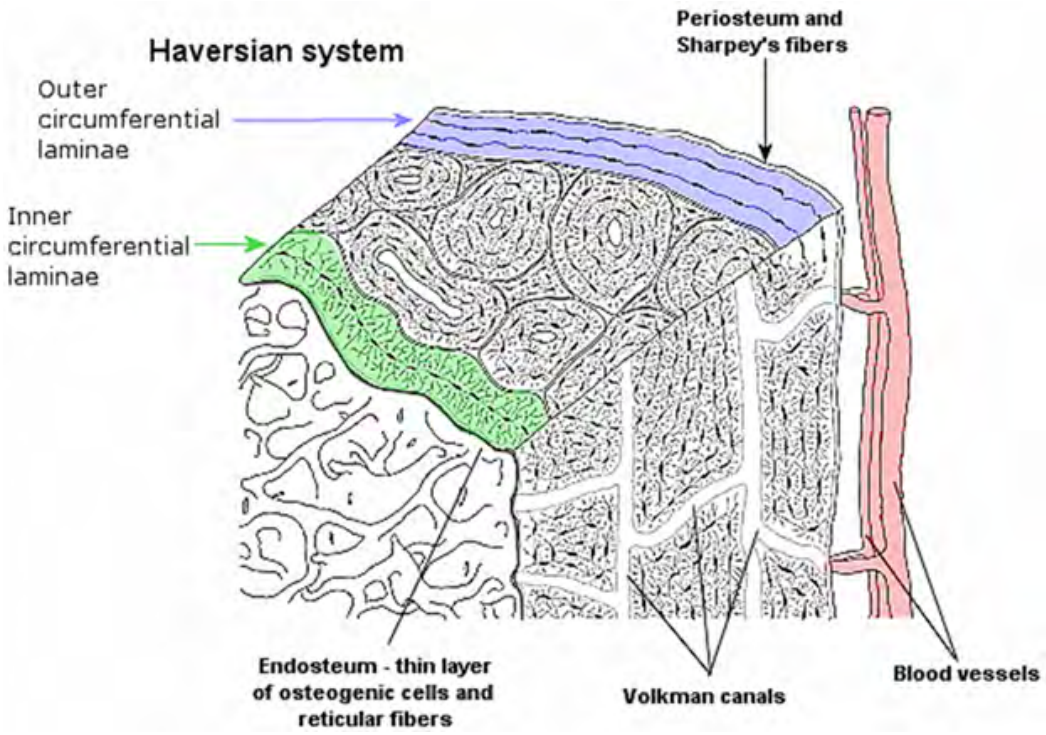
• MACROSCOPIC ANATOMY OF BONE

- Bone tissue is ridged connective tissue composed of Type I collagen fibers embedded in a mineralized ground substance.
- The collagen and ground substance = matrix.
- About 65–70% of the matrix is composed of inorganic salts mostly calcium phosphate and calcium carbonate.
- About 25–30% of the matrix is composed of organic compounds of which 95% is collagen, and the other 5% are proteoglycans and glycoproteins.

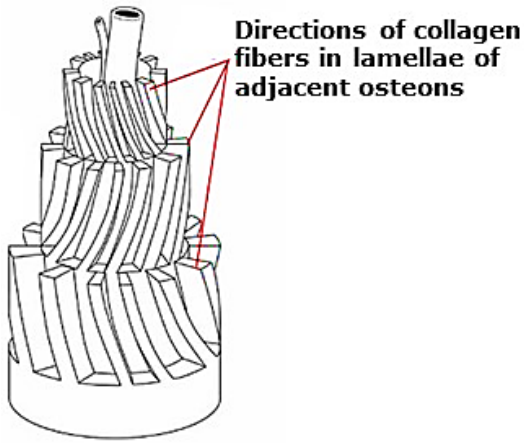


A) Photograph of a frontal section through a long bone showing the location of solid compact bone and trabecular organization of the spongy bone. B) Photomicrograph of a cross-section through compact showing the arrangement of osteons. C) High magnification photomicrograph of an osteon showing the Haversian canal, circular lamella and osteocytes in lacunae.

- The osteon is the structural unit of compact bone.
- Osteons lie between peripheral layers of the external circumferential system and the medullary layers of the internal circumferential system.
  - Periosteum attaches by Sharpey fibers to the external circumferential system.
  - Endosteum attaches to the internal circumferential system.
- Between osteons are interstitial bony tissues which are mainly remnants of old osteons after bone remodeling.
- Each osteon contains a central Haversian canal that contains blood vessels to provide nutrients to the bone cells called osteocytes and remove waste products, lymphatic channels and fine nerve fibers.
- Volkmann's canals run transversely to Haversian canals and transport blood vessels, lymphatic vessels and fine nerves to the Haversian canals from nutrient foramen that lie on the surface of the bone.
- Each Haversian canal is surrounded by rings of calcified bone called lamella and between the rings are bubble-like structures called lacuna containing mature bone cells called osteocytes.
- Adjacent lamellae of a osteon contain Type I collagen fibers that run at an angle to to each other.



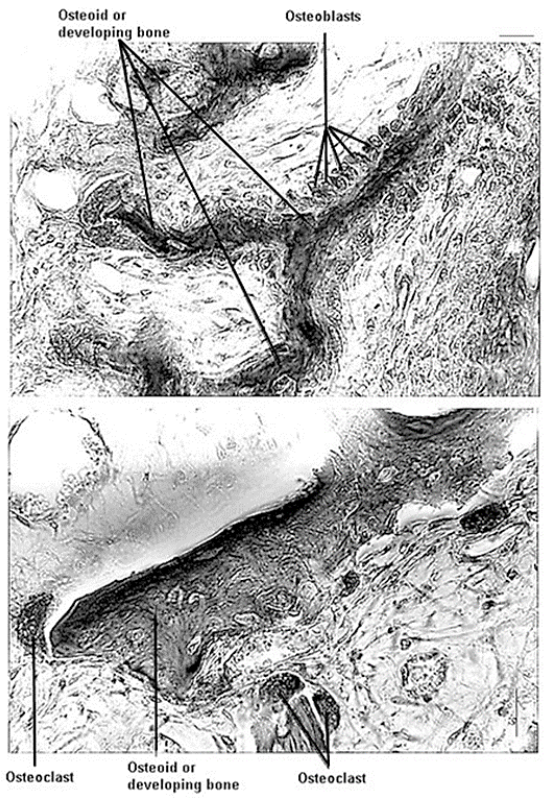
Drawing showing a section of the shaft of a long bone with its components and coverings. (modified from Paris and Loubert 1990)



Drawing showing the type I collagen directions in three adjacent lamellae of an osteon (modified from Paris and Loubert 1990)

- BONE CELLS
  - Osteocytes are mature bone cells that lie in lacunae of bone and are derived from osteoblast.
  - Osteogenic cells arise from embryonic mesenchymal cells from the periosteum and endosteum of mature bone and differentiate into osteoblasts.

- Osteoblasts are immature bone cells that secrete collagen and a non-calcified ground substance called osteoid and become osteocytes when the surrounding matrix is calcified.
- Osteoclasts are large multinucleated cells that remove calcified bone and osteoid and are important in bone growth, bone remodeling, fracture healing and maintaining blood calcium levels.

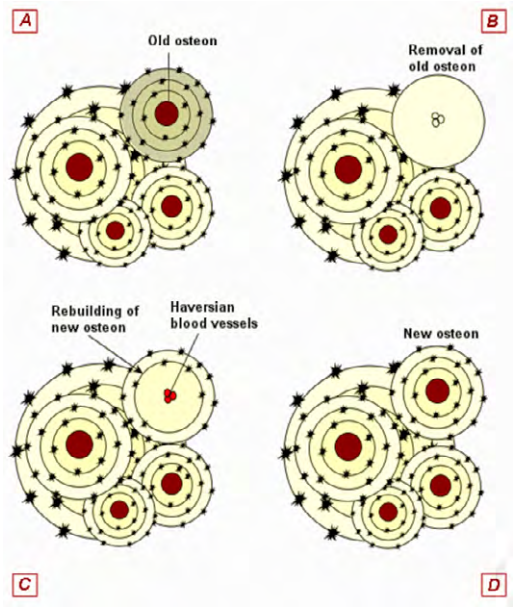


(TOP) photomicrograph of developing bone showing osteoblasts lined up and depositing uncalcified bone. (BOTTOM) photomicrograph of developing bone showing osteoclasts removing bone.

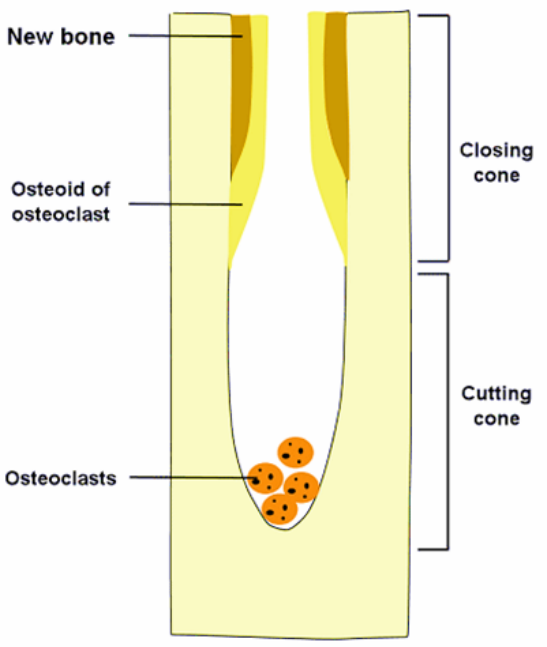
- **BONE REMODELING**

  - REMODELING DURING BONE GROWTH
    - Surface bone remodeling involves the simultaneous process of depositing new bone in one area by osteoblasts and the reabsorption of bone in a different area by osteoclasts.
    - In cylindrical regions of bone, osteoblasts in the subperiosteal area deposit bone, while osteoclasts in the subendosteal area reabsorb it.
    - In conical regions of bone, osteoblasts in the subendosteal area deposit bone, while osteoclasts in the subperiosteal area reabsorb it.

- INTERNAL BONE REMODELING
  - Internal remodeling begins with the removal of old lamellar bone by osteoclasts that bore out through the bone a longitudinal cylindrical cavity called a cutting cone.
  - This cutting cone is relatively long and reaches the diameter of the new osteon.
  - With the osteoclasts in the cutting cone are blood vessels, perivascular connective tissue and numerous cells in mitosis that appear to give rise to osteoblasts.
  - Osteoblasts deposit collagen and osteoid (uncalcified bone) along the walls of the cone and the cutting cone now becomes the closing cone.
  - The closing cone is refilled from the outside inwardly with concentric circular layers of lamellar bone until the Haversian canal remains.



Drawing showing the replacement an old osteon (A). First the osteon is removed by osteoclasts (B) and then filled from the outside inward by osteoblasts (C) to form a new osteon (D) (modified from Ross and Pawlina 2006).



Drawing showing the cutting cone removing an old osteon and the closing cone forming the new osteon (modified from Ross and Pawlina 2006).

- BONE REMODELLING THROUGHOUT LIFE
  - Old osteons and interstitial bone are removed and new osteons formed throughout the lifespan.
  - The reasons for bone remodeling are:
    + Removes bone that has been damaged by microfractures because of bone fatigue or strains.
    + Replenishes osteocytes and maintains the organic and inorganic components of the bone.
    + Adapts bone to long term changes in stresses.
    + Makes calcium stored in bone available to the body.

- **BONE FRACTURES**

  - TYPES OF FRACTURES
    - A fracture is complete when it transects the entire bone and incomplete when it penetrates through only part of the bone.
    - A fracture is simple or closed when the surrounding tissue is intact.
    - It is comminuted when there is splintering of the bony ends, and compound when the bony ends are displaced, disrupting the surrounding tissue and the skin.

  - FRACTURE SITES
    - The site of the fracture will depend on the distribution of spongy and compact bone in an area and the type of load applied.
    - Spongy bone is weaker than compact bone so those areas containing primarily spongy bone, such as the epiphyses and tuberosities of long bone, will be more prone to fractures than the shafts which contain mainly compact bone.
    - Spongy and compact bones are weaker in tension than compression so the areas receiving tensile forces are more susceptible to fracture than those receiving compression forces.

  - REGIONAL BONE COMPRESSION STRENGTH
    - The Table below shows the percentage of compression strength based on 100% for the middle of the shaft of the bone, which is mainly compact bone.
    - With the exception of the ulna, the proximal and distal ends are weaker in compression strength than the middle shaft.
    - The proximal end of the ulna contains the olecranon process which is mainly compact bone and larger than the
    - narrow middle shaft of the ulna.
    - The proximal and distal ends of the other long bones contain mainly spongy bone which is weaker than compact bone.

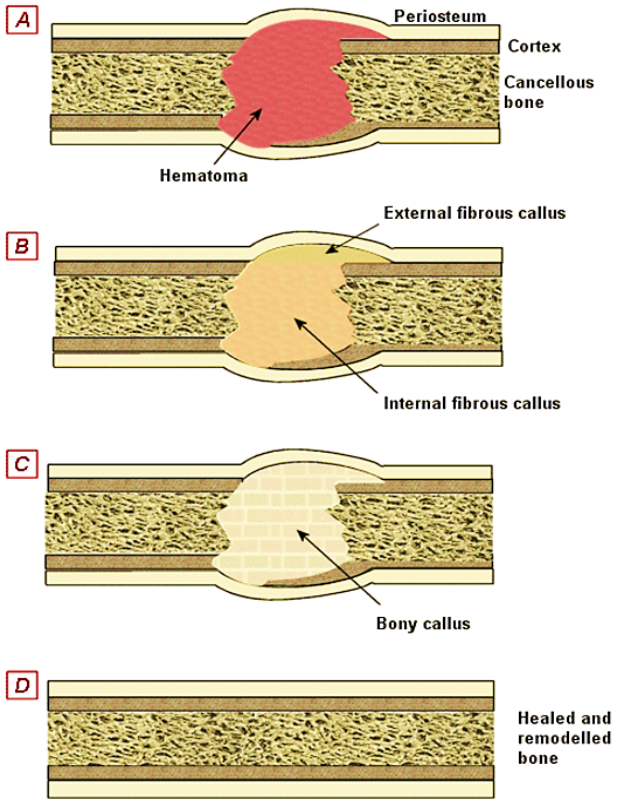
Bone	Proximal End*	Middle Shaft*	Distal End*
Femur	80	100	62
Tibia	73	100	71
Fibula	89	100	93
Humerus	86	100	88
Radius	76	100	84
Ulna	129	100	62

* % of compression strength

- COMMON CAUSES OF FRACTURES
  - Trauma (accidents, athletic and work injuries, violence).
  - Osteoporosis is a disorder characterized by a decrease in the mass of both spongy and compact bone but no abnormality in the composition of the bone.
  - Osteomalacia is a disorder that is characterized by reduced mineralization of bone during remodeling which results in "soft bone" and appears to be associated with the lack of vitamin D.
  - Paget's disease (osteitis deformans) is a metabolic disorder characterized by marked bone reabsorption followed by the formation of patches of new bone that lacks the strength of normal bone, even though it is thick.
  - Osteogenesis imperfecta (brittle bone) is an inherited disorder that results in abnormal collagen synthesis and absorption making bone brittle.

- **BONE HEALING**

1. The fracture results in a localized hematoma due to the rupture of blood vessels in the area.
2. A blood clot then develops at the fracture site.
3. Capillaries grow into the clot and form a vascular network. At the same time, connective tissue grows into the site forming a granulation tissue. Macrophages remove dead tissue and osteoclasts remove bone fragments.
4. The granulation tissue becomes a dense connective tissue, in which hyaline cartilage and fibrocartilage develop. This tissue is called a fibrocartilaginous callus. It is divided into a large external callus and a small internal callus.
5. The disruption of the periosteum and endosteum at the fracture site stimulates osteogenic cell activity.
6. Osteogenic cells then differentiate into osteoblasts, which migrate along the capillaries into the fracture site and secrete osteoid. At the same time, the fibrocartilaginous callus is being absorbed.
7. The osteoid calcifies, forming a bony callus of fibrous bone. This callus is not organized along mechanical lines of stress but the layers of bone are aligned along the direction of the capillaries.
8. With time and the return of function, the bony callus is remodeled and the fibrous bone converted to lamellar bone.



Drawing highlighting the process of bone healing.

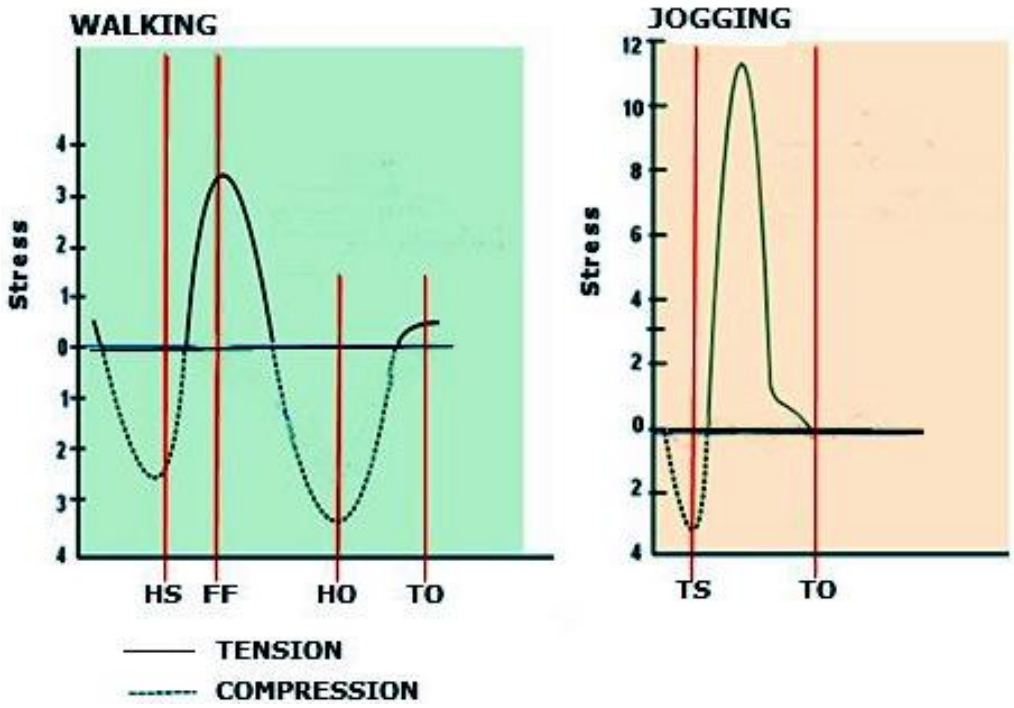
- Movements that produce rotation and traction at the fracture site should be avoided during early bone healing as these displace the opposing bony ends and can prevent union of the bones.
- Exercises that reflect functional activities will strain the fracture callus to direct bone remodeling so that the remodeled bone aligns to resist appropriate mechanical loads.

• FACTORS THAT EFFECT BONE

- EXERCISE
  - Strenuous exercise results in muscle fatigue muscle fatigue decreases the shock absorption capability of muscle and produces altered movements and abnormal loading on the bone.
  - Abnormal loading changes the force distribution and strain pattern on the bone resulting in microfailures and possibly fractures.

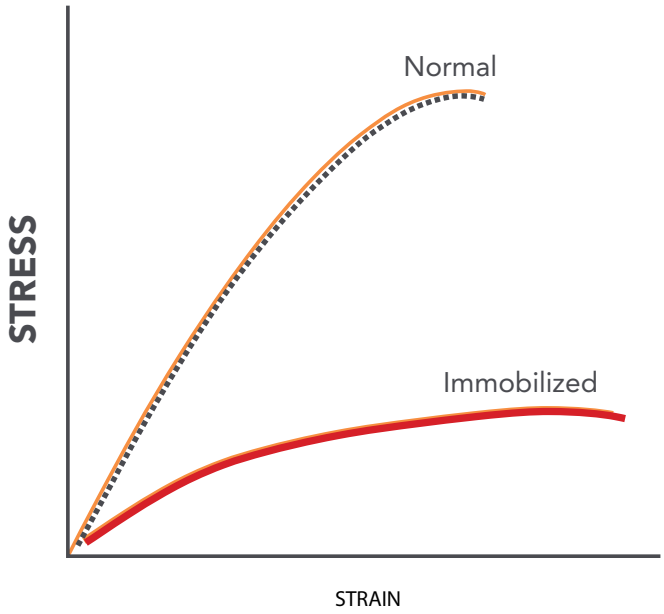
STRENUOUS EXERCISES → FATIGUES MUSCLES → DECREASES THE SHOCK ABSORPTION FUNCTION OF MUSCLE AND ALTERS MOVEMENTS → ABNORMAL LOADING FORCES → CHANGING THE FORCE AND STRAIN PATTERNS ON BONES → CAUSING MICROFAILURES → FRACTURES

- JOGGING
  - Compression forces on the tibia at toe strike (TS) during jogging are nearly 2 times greater than at heel strike (HS) during walking.
  - Tensile forces on the tibia from toe strike to toe off (TO) are almost 4 times greater than from foot flat (FF) to heal off (HO) during walking.



Graphs showing the forces on the tibia during walking and jogging. HS = heel strike. FF = Foot flat, HO = Heel off, TO = Toe off, TS = Toe strike. (modified from Norkin and Frankel 2001)

- IMMOBILIZATION
  - Results in a 1% loss in bone mass per week.
  - Bone decreases in strength, stiffness and toughness.

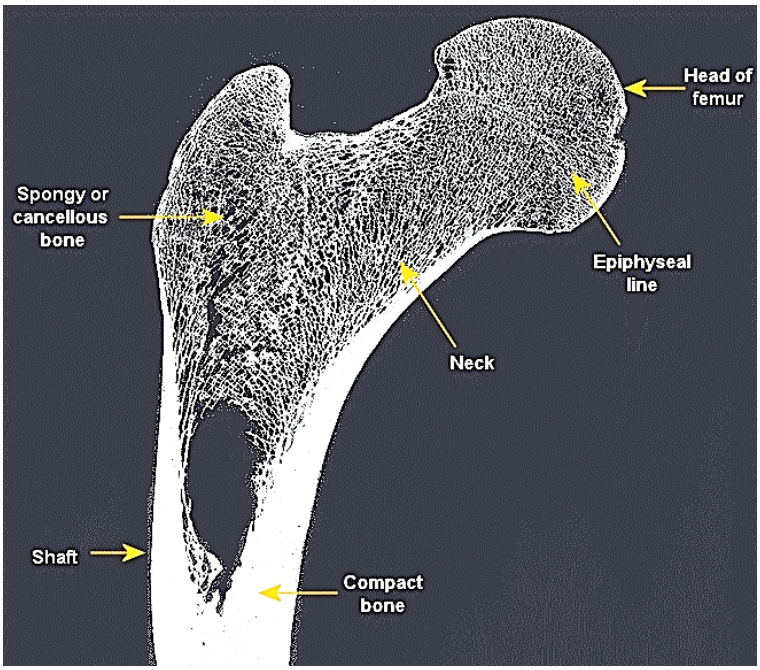


Stress-strain graph for normal bone and immobilized bone. (modified from Norkin and Frankel 2001)

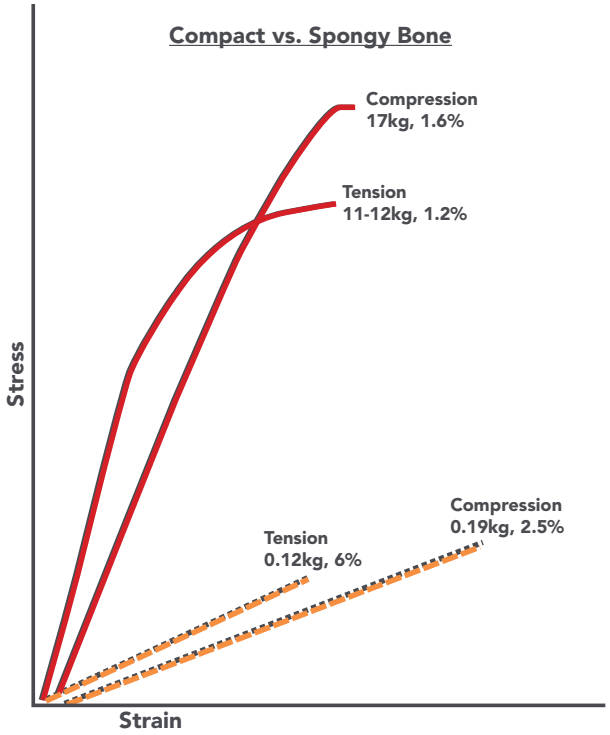
4.3.2 BONE STRESS AND STRAIN

- COMPACT AND SPONGY BONE

  - Compact bone is stiffer and stronger in breaking force than spongy bone when compression, tension, and shear stresses are applied.
  - Compact bone is most resistant to compression, less resistant to tension, and least resistant to shear.
  - Trabeculae of spongy bone and the osteons of compact bone are aligned to resist the most frequently occurring stresses.
  - Wolff's law states that bone is deposited where needed to resist stress and is absorbed where not needed.
  - Wolff's law seems to apply also to all connective tissues, not only bone.



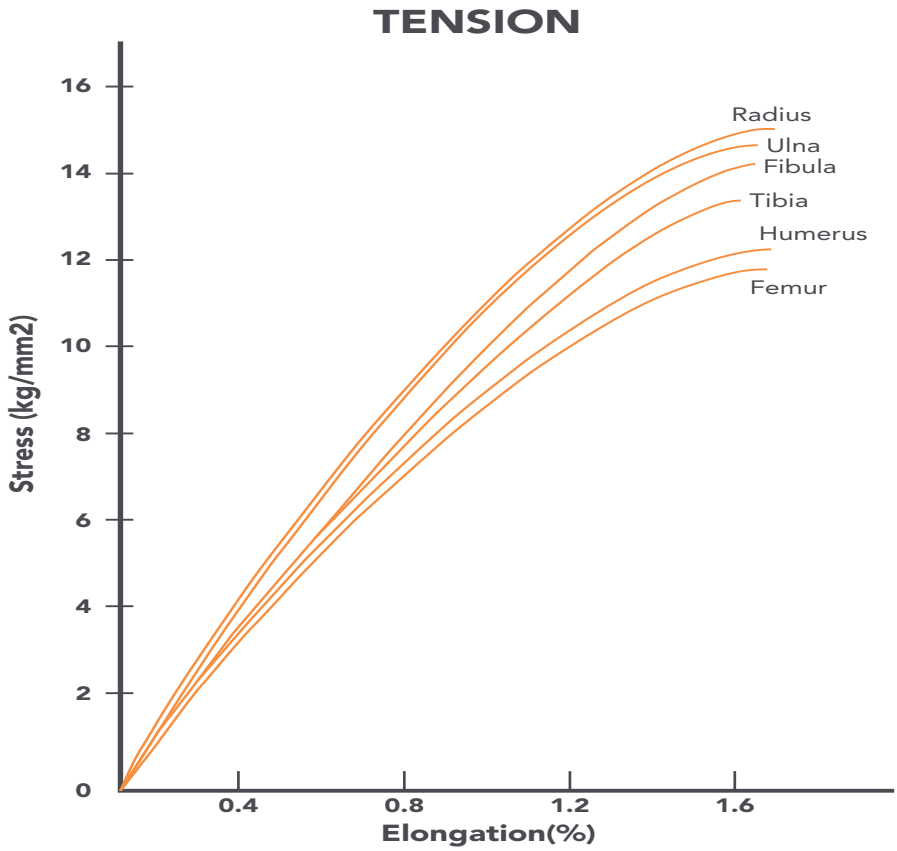
Photograph of the femur showing spongy and compact bone



Graph showing the stress-strain curves for compact bone (upper 2 lines) and spongy bone (lower 2 lines) for tension and compression. (modified from Norkin and Frankel 2001)

o LONG BONES

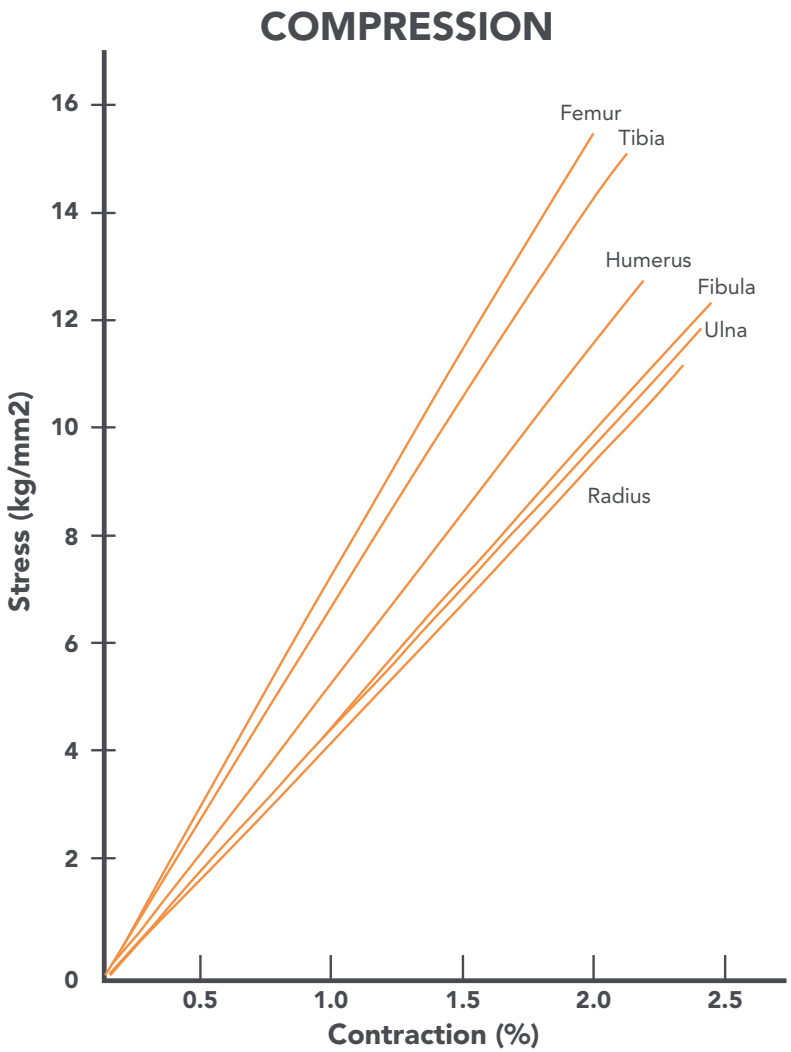
- During routine activities of daily living, long bones of the body are subjected to multiple stresses and strains by muscle actions, gravity and resistive forces.
- Activities stress the bone in tension, compression, bending and torsion and because the long bones differ in shape and size, the ability of these bones to resist these forces differ.
- Terms used to describe biomechanical properties of bone.
  + Breaking load = the amount of force needed to break the bone without consideration of the size of the bone; total force needed to break the bone.
  + Strength = the amount of force needed to break a bone relative to the size and area of the bone; unit force need to break the bone.
  + Elongation, contraction, deflection, angle of twist are all terms used to describe deformations or strains of bone.



Graph of tension stress-strain curves for eachof the long bones. (Yamada 1970)

STRENGTH: Thin long bone (radius, ulna) are stronger in tension than the thick ones (femur, humerus). The thin diameter bones have a very small medullary cavity compared to the large diameter bones. Tension applied to the thin bones is similar to applying tension to a solid rod. Tension applied to large diameter bone results in the cavitation and breaking of the shaft because of the large medullary cavity.

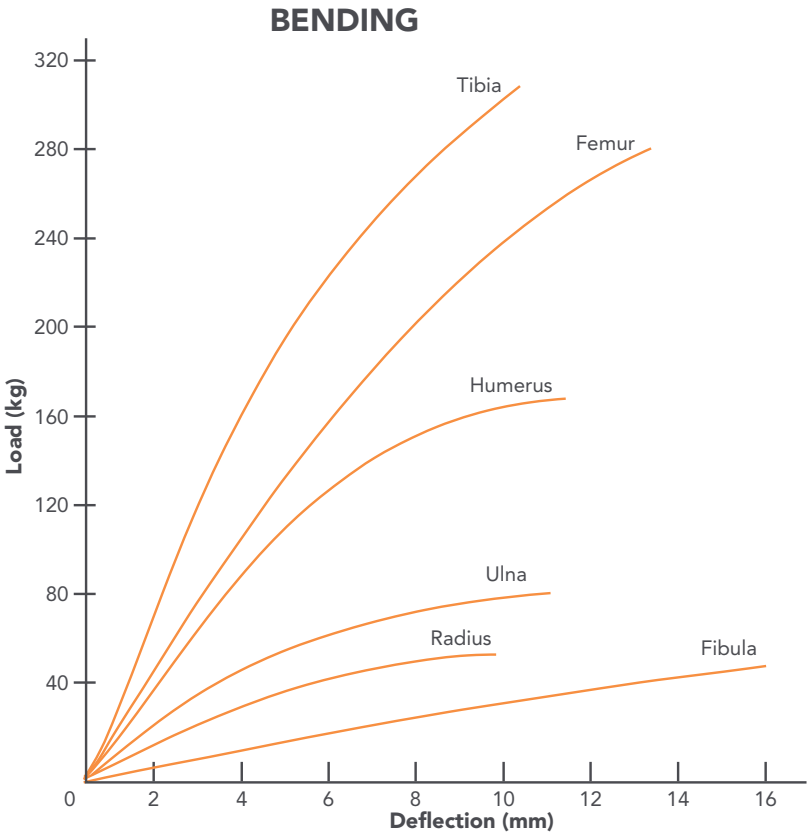
STRAIN: Thin long bones (fibula, radius) elongate more than thick bones (femur, humerus) when loaded in tension.



Graph of compression stress-strain curves for eachof the long bones. (Yamada 1970)

STRENGTH: Thick long bones (femur, humerus) are stronger in compression than the thin long bones (ulna, radius). Compression strength is directly related to the cross-sectional area of a bone. The larger the cross-sectional area (diameter), the greater is the resistance to compression.

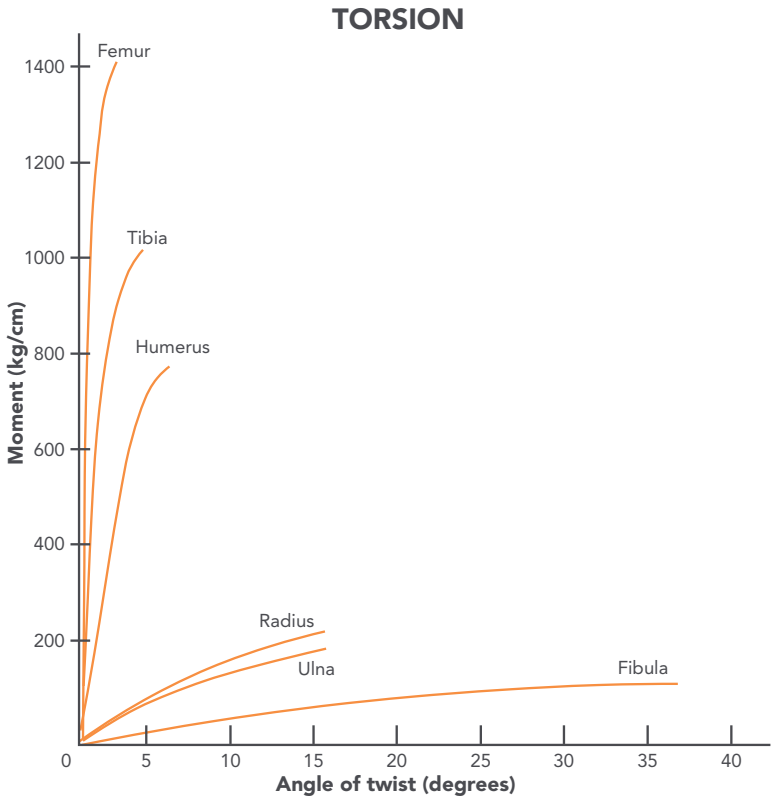
STRAIN: Thin long bones (fibula, radius) compress more than the thick long bones (tibia, femur) when loaded in compression. The degree of compression of a long bone is inversely related to the cross-sectional area (diameter) of the bone. The smaller the cross-sectional area, the greater is the amount of compression (compaction).



Graph of stress-strain curves for bending of each of the long bones. (Yamada 1970)

STRENGTH: Thick long bones (tibia, femur) are stronger in bending than thin long bones (fibula, radius). Bending strength is directly related to the cross-sectional area of a bone. The larger the cross-sectional area (diameter), the greater the resistance is to bending forces (bending strength).

STRAIN: Thin long bones (fibula, radius) bend more than thick long bones (tibia, femur). The degree of bending of a long bone is inversely related to the cross-sectional area (diameter) of the bone. The smaller the cross-sectional area, the greater is the amount of bending displacement.



Graph of stress-strain curves for torsion of each of the long bones. (Yamada 1970)

STRENGTH: Thick long bones (tibia, femur) are stronger in torsion than thin long bones (fibula, radius). Torsional strength is related directly to the cross-sectional area (diameter) of the bone. The larger the cross-sectional area, the greater is the resistance to torsion (torsional strength).

STRAIN: Thin long bones (fibula, radius) twist more than thick long bones (tibia, femur). The degree to which a bone twists (angle of twist) is inversely related to the cross-sectional area(diameter) of a bone. The smaller the cross-sectional area, the greater is the degree (angle) of twist.

With the exception of tension, the strengths of long bones are directly related to the cross-sectional diameters of the bone. Large diameter bones are stronger in compression, bending, and torsional forces than small diameter bones. However, deformations are inversely related to the cross-section diameters of the bone. Small diameter bones deform more than large diameter bones.

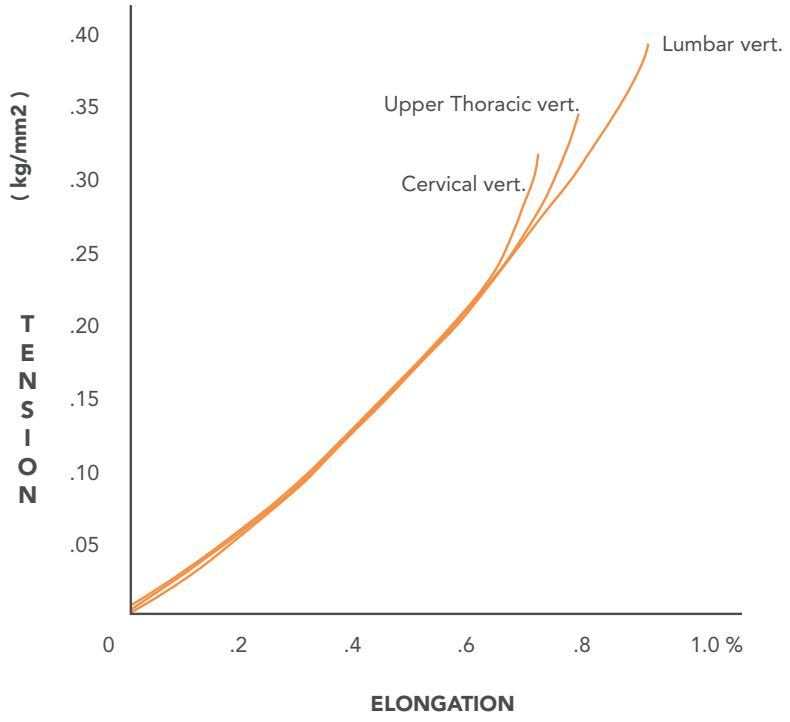
4.3.3 AGING

- Bone reaches its maximum strength in males and females between 20–29 years old.
- Strength then begins to decrease slowly in both genders between 30-39 years of age.
- Before the age of 30, osteoblasts are more active than osteoclasts which results in increased thickness of the cortical bone.
- After the age of 40, osteoblast activity decreases in males and females but osteoclast activity is unchanged, the result of which is a slow decrease in cortical bone thickness.
- Below 50 years of age, the mechanical properties of bone are similar in males and females, but males tend to have more bone than females.
- After 50, the strength and the amount of trabecular bone decreases more rapidly in females than in males.

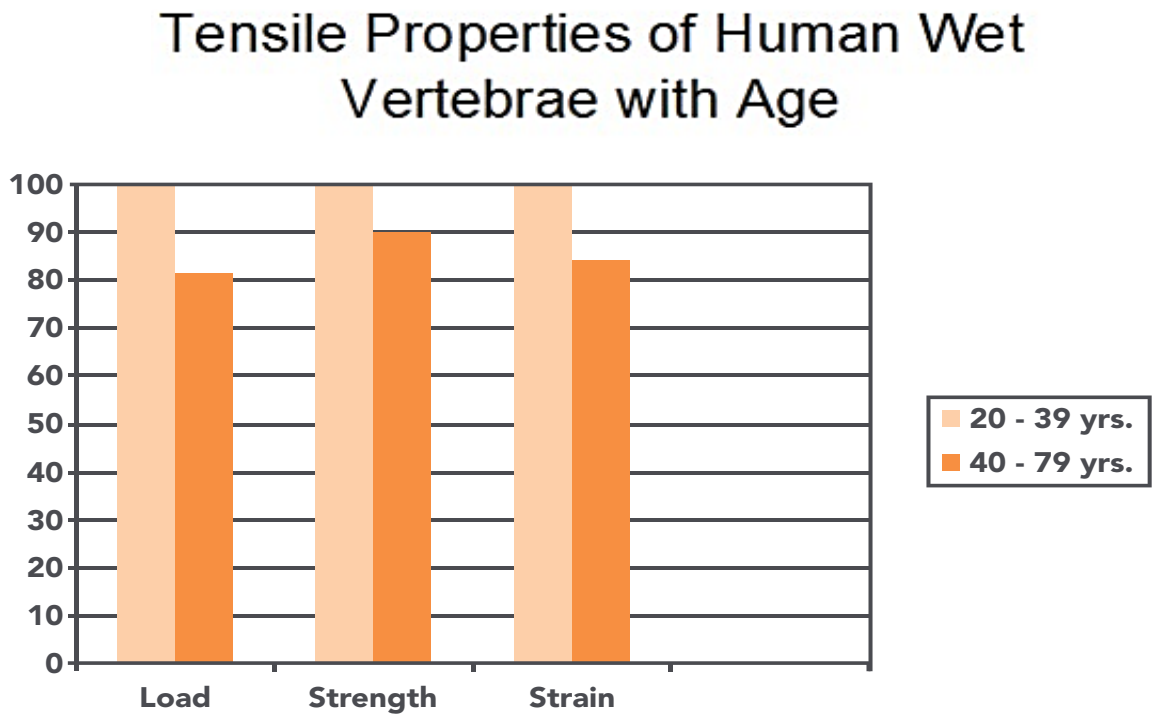
- **AGING LONG BONES** (Yamada 1970)
  - After the age of 50, tensile strength is still about 75% of maximum (a loss of about 25%).
  - Compression strength is about 85% of maximum (about 15% loss).
  - Bending strength is about 75% of maximum (about 25% loss).
  - Torsional strength is about 85% maximum (about 15% loss).

- **BIOMECHANICAL PROPERTIES AND AGING IN VERTEBRAE**
  (Yamada 1970)

  - TENSION
    - Tensile breaking loads in lumbar vertebral bodies are about 4 times greater than cervical vertebral bodies.
    - Tensile breaking loads in lower thoracic vertebral bodies are more than 2 times that of the upper thoracic vertebrae and both are less than the lumbar vertebrae and greater than the cervical vertebrae.
    - Tension breaking loads in vertebral bodies are a function of the sizes of the vertebrae.
    - When vertebral body sizes are considered, tensile strengths are greatest for the lumbar vertebral body, the least for the cervical and intermediate for the thoracic vertebral bodies.
    - Elongation strains are also similar among the vertebrae.
    - With age, the tensile properties in lumbar, thoracic and cervical vertebral bodies are less than 20%.
      + Tensile breaking loads decrease about 20%.
      + Tensile strengths decrease about 10%.
      + Tensile strains decrease about 15%.



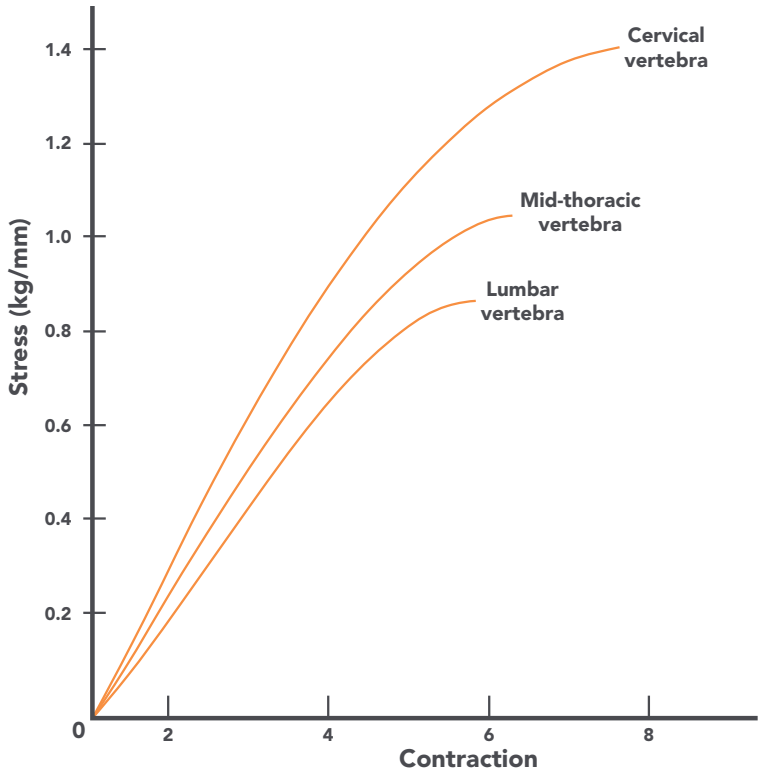
Graph of the tension stress-strain curves for the lumbar, thoracic and cervical vertebral bodies. (Yamada 1970)



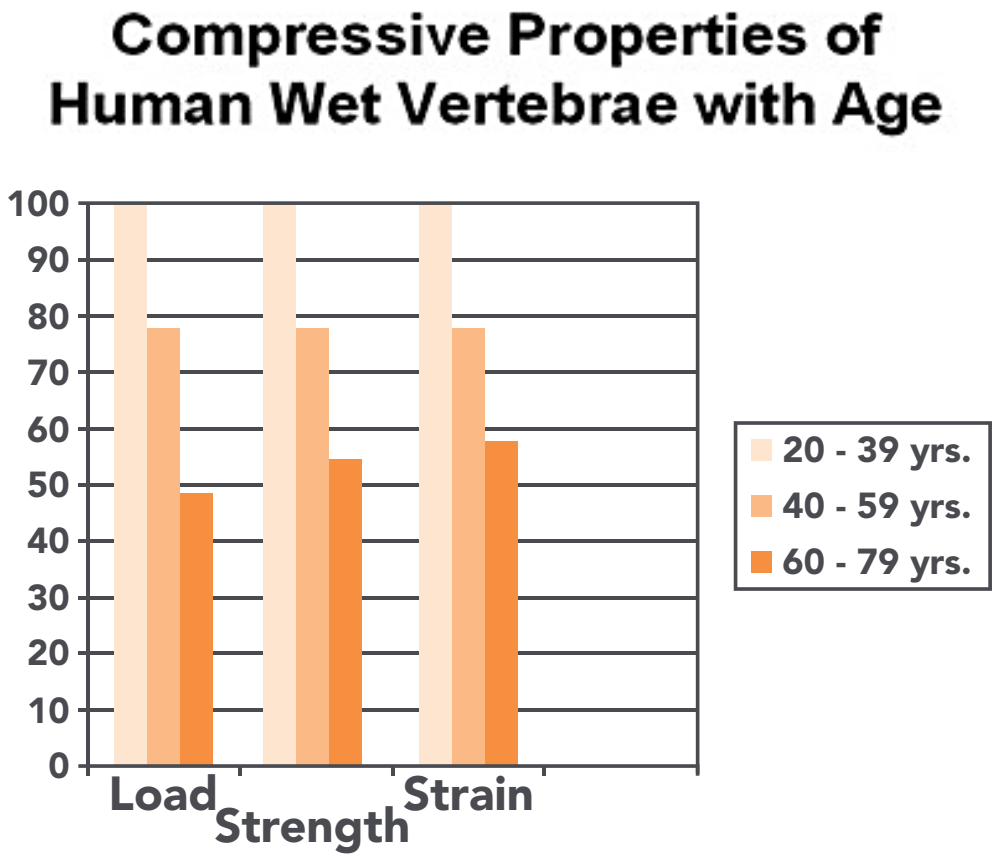
Graph showing the tensile properties of the vertebral bodies between 20–39 year old and 40–79 years old.

- COMPRESSION (Yamada 1970)

  - Compression breaking loads of lumbar vertebral bodies are about 2 times greater than the cervical vertebral bodies.
  - Breaking loads of the lower thoracic vertebral bodies are about 1.5 times that of the upper and middle thoracic bodies.
  - All the breaking loads of the thoracic vertebral bodies are less than the lumbar bodies and greater than the cervical vertebral bodies.
  - Compression breaking loads of the vertebral bodies are a function of the sizes of the vertebrae.
  - When vertebral body sizes are considered, compression strengths of the cervical vertebrae are greatest, followed by the thoracic; the lumbar vertebral bodies are least.
  - Cervical vertebral bodies are about 2 times greater in strength than the lumbar bodies but the thoracic vertebral bodies are only about 20–25% greater in strength than the lumbar bodies.
  - Compression strains are greatest in the cervical vertebral bodies and about 30% less in the lower thoracic and lumbar bodies.
  - With age, the compressive properties decrease between 40–50%.
    + Breaking loads decrease about 50%.
    + Strengths decrease about 45%.
    + Strains decrease about 40%.



Graph of the compression stress-strain curves for the lumbar, thoracic and cervical vertebral bodies. (Yamada 1970)

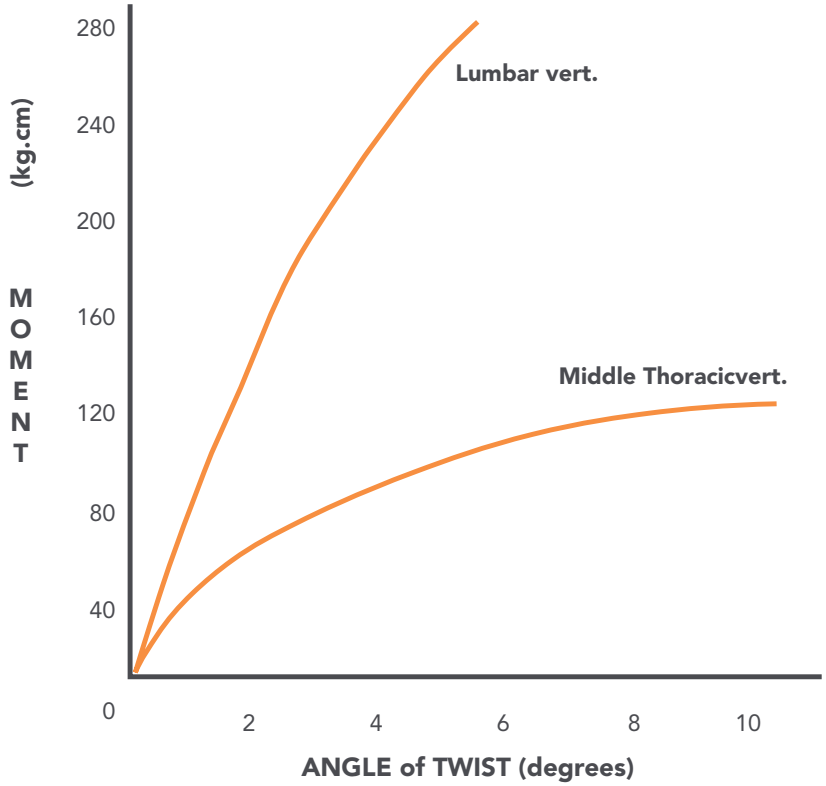


Graph showing the compression properties of the vertebral bodies between 20–39 years old, 40–59 years old, and 60–79 years old.

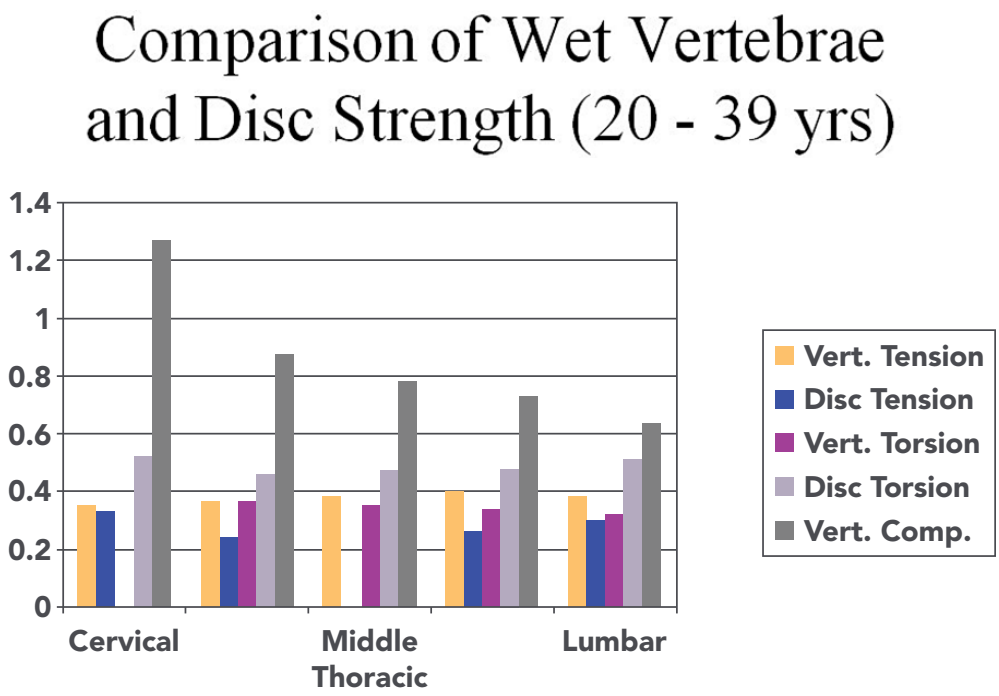
- TORSION (Yamada 1970)

  - Torsional breaking loads of lumbar vertebral bodies are about 4 times greater than upper thoracic bodies but only about 35% greater than the lower thoracic vertebral bodies.
  - Torsional breaking loads of the lower thoracic vertebral bodies are about 60% greater than the upper thoracic; the breaking loads of the middle thoracic bodies are about 45% greater than the upper thoracic vertebral bodies.
  - All the torsional breaking loads of the thoracic vertebral bodies are less than the lumbar vertebral bodies.
  - Torsional breaking loads are a function of the sizes of the vertebral bodies.
  - When vertebral body sizes are considered, torsional strengths of the upper thoracic region are greatest, but strengths are very similar between the thoracic and lumbar vertebral bodies.

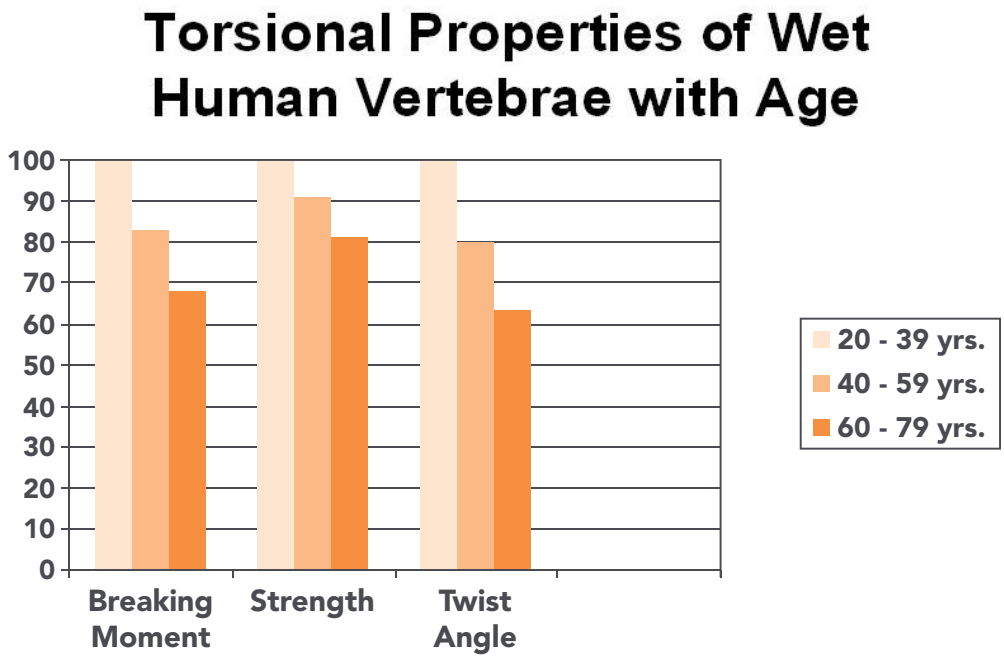
- The angles of twist (deformation) are greatest in the upper thoracic vertebral bodies (and probably also in the cervical bodies) and less in the lumbar vertebral bodies.
- The angles of twist of the upper and middle thoracic vertebral bodies are 2 times greater than the angles of twist of the lumbar bodies; the angles of twist of the lower thoracic vertebral bodies are about 60% of the angles of twist of the lumbar vertebral bodies.
- With age, torsional breaking loads and angles of twist decrease about 40%; torsional strengths decrease by about 20%.



Graph of the torsional stress (t) – strain (angle of twist) curves for the lumbar, thoracic and cervical vertebral bodies. (Yamada 1970)



Graph comparing the strength of the cervical, middle thoracic and lumbar vertebral (vert.) discs and vertebral bodies in tension and torsion and the vertebral bodies in compression.



Graph showing the torsional properties of the vertebral bodies between 20–39 years old, 40–59 years old, and 60–79 years old.

Study Questions (Bone)

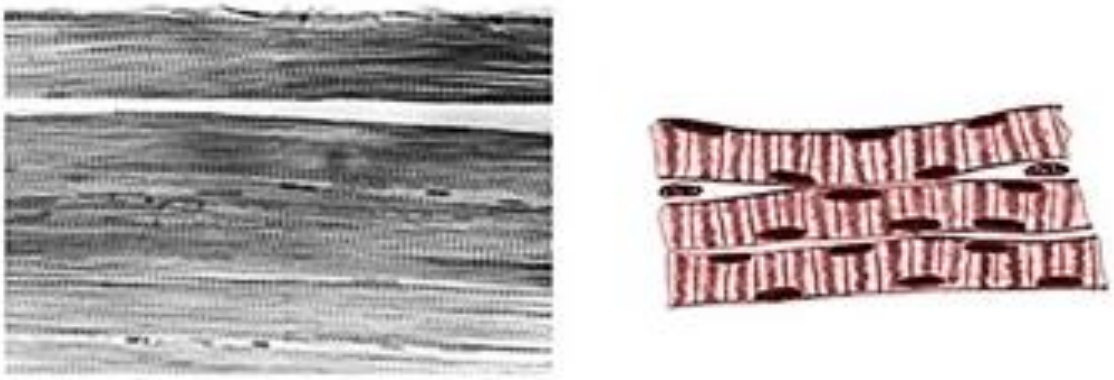
1. What resists tensile and compressive forces in bone?
2. Why is it necessary to remodel bone even in old age?
3. What are the differences between spongy and compact bone and do theserelate to the strength of long bones (ie radius, femur, etc.)?
4. How does the cross sectional area of a long bone relate to the mechanicall properties of that bone?
5. How does aging affect the following:
    a. general properties of bone
    b. long bones
    c. vertebral bodies

4.4 MUSCLE

4.4.1 ANATOMY OF SKELETAL MUSCLE

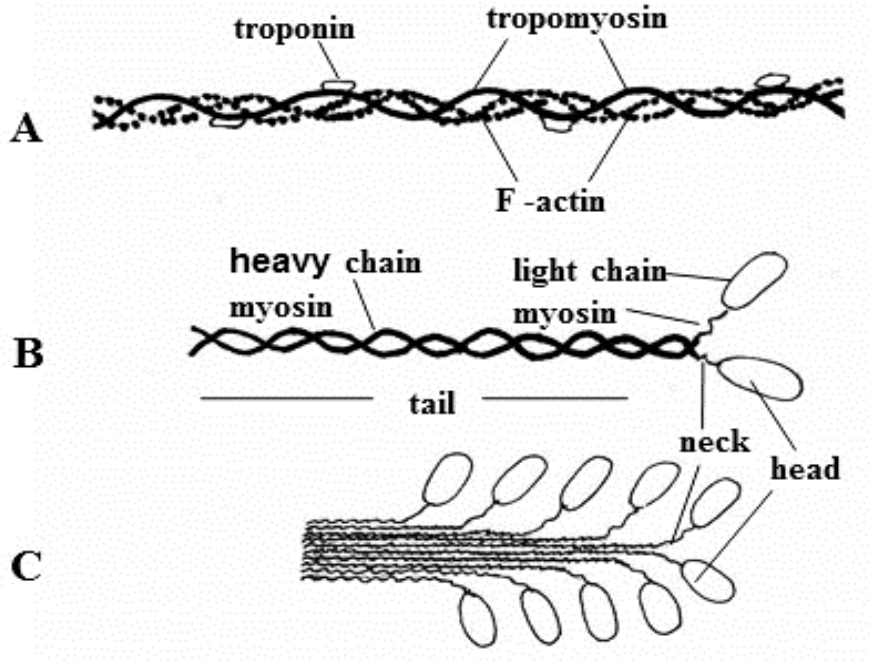
- SKELETAL MUSCLE FIBERS (CELLS)

  - NUCLEI: multiple nuclei lie along the periphery of the muscle fiber (cell)
  - STRIATIONS: yes, dark A bands and light I bands
  - LOCATIONS: upper and lower extremities, trunk, back, pelvis, head, neck
  - FEATURES:
    - Actin and myosin are arranged to form organized A and I bands or striations.
    - Fibers do not usually split but may divide to form another fiber.
    - The ends of the fibers taper and connect to connective tissue.
    - Fibers run the length of the muscle belly so some fibers are very long (sartorius) and others short (lateral rectus of eye).
    - Skeletal muscles are controlled by the somatic (voluntary) motor system.



Photomicrograph (LEFT) of skeletal muscle and a drawing (RIGHT) of 3 muscle fibers or muscle cell showing the striations and position of the nuclei.

- COMPOSITION OF SKELETAL MUSCLES
  - Skeletal muscles are composed of muscle fibers or muscles cells and connective tissue.
  - The muscle fibers are usually arranged in groups called fascicules.
  - The fascicles in a muscle of divided by connective tissue called perimysium.
  - Muscle fascicles can be arranged into functional areas in a muscle called compartments.
  - The muscle fibers (cells) of muscles fascicles fiber contain myofibrils and each myofibril contains thin **actin** and thick **myosin** myofilaments.



Drawing of A) actin filament; B) myosin molecule; C) myosin filament. (Modified from Ross and Pawlina 2006; Guyton 1991)

ACTIN filaments are composed of spherical actin molecules that line up to form a double helix. Associated with actin filaments are two proteins: **tropomyosin** and **troponin**. Both proteins lie on the surface of the actin filament. Tropomyosin is a double helical structure that stiffens the actin filaments. Troponin is a globular protein, consisting of three polypeptide subunits: Troponin T, Troponin I, and Troponin C. Troponin T attaches to tropomyosin, and Troponin I binds to actin. When Troponin T and Troponin I are present but calcium absent, tropomyosin blocks the attachment of myosin to actin. Troponin C combines with calcium, which changes the configuration of troponin. This change moves the tropomyosin away from the actin filament and allows the myosin head to attach to actin.

MYOSIN (thick) myofilaments are composed of many myosin molecules. Each molecule is composed of a tail of **light meromyosin** and a neck and head of **heavy meromyosin**. The coiled tail consists of two alpha helices. It is at the tail region where hundreds of myosin molecules connect to form a myosin myofilament. The tail attaches to the heavy meromyosin of the neck at the hinge region, and the neck attaches to two globular heads. The globular heads bind with actin to form **cross bridges** Each head contains a **pocket** and **cleft**. The pocket is where ATP binds to the globular head and where APT is hydrolyzed. The cleft is where the head attaches to actin. Opening of this cleft decreases the binding strength of the globular head to actin and closing of this cleft increases the head to actin binding strength. It is the formation and release of cross bridges that produce movement of the actin myofilaments during muscle contraction.

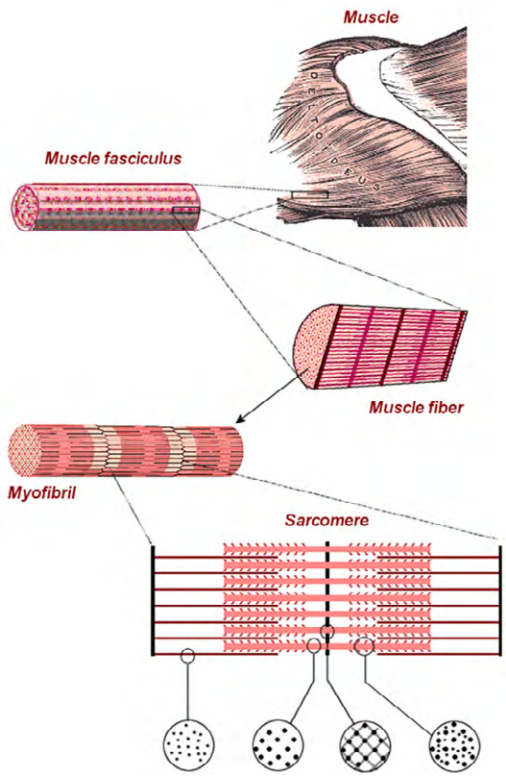


Diagram showing the structural components of skeletal muscle. A) Gross muscle. B) Fascicles containing many muscle fibers (cells). C) Muscle fibers containing myofibrils. D) Myofibrils containing myofilaments of actin and myosin. E) Arrangement of actin and myosin myofilaments forming the A-band, H-band, and I-band. (Modified from Fawcett 1986)

o MYOFILAMENT ARRANGEMENT

- Actin and myosin filaments are arranged in a series of repeated rows, producing light (I-bands) and dark (A-bands) strips or striations.
- A-bands are regions where actin and myosin filaments overlap and where cross bridges are formed.
- I-bands are regions that contain only actin filaments.
- In the center of each I-band is the dark Z line (or Z disc) that is the site where the abutting ends of actin filaments attach.
- Myosin filaments also attach to the Z line by thin strands composed of the protein titin that pass though the I-band from the myosin filament to the Z line.
- Z lines are connected to each other by intermediate filaments.
- Between two Z line is the sarcomere which is the structural and functional unit of skeletal muscle.

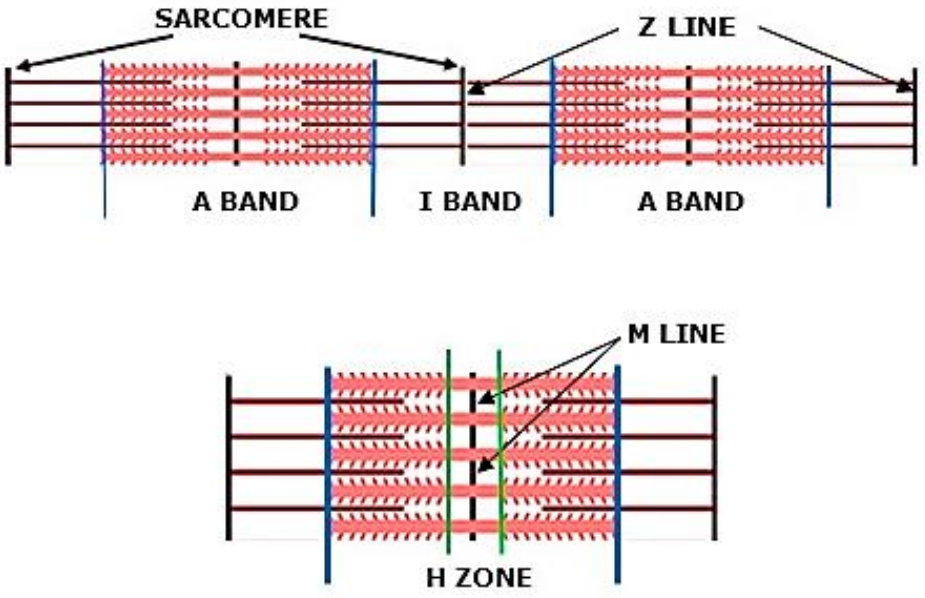


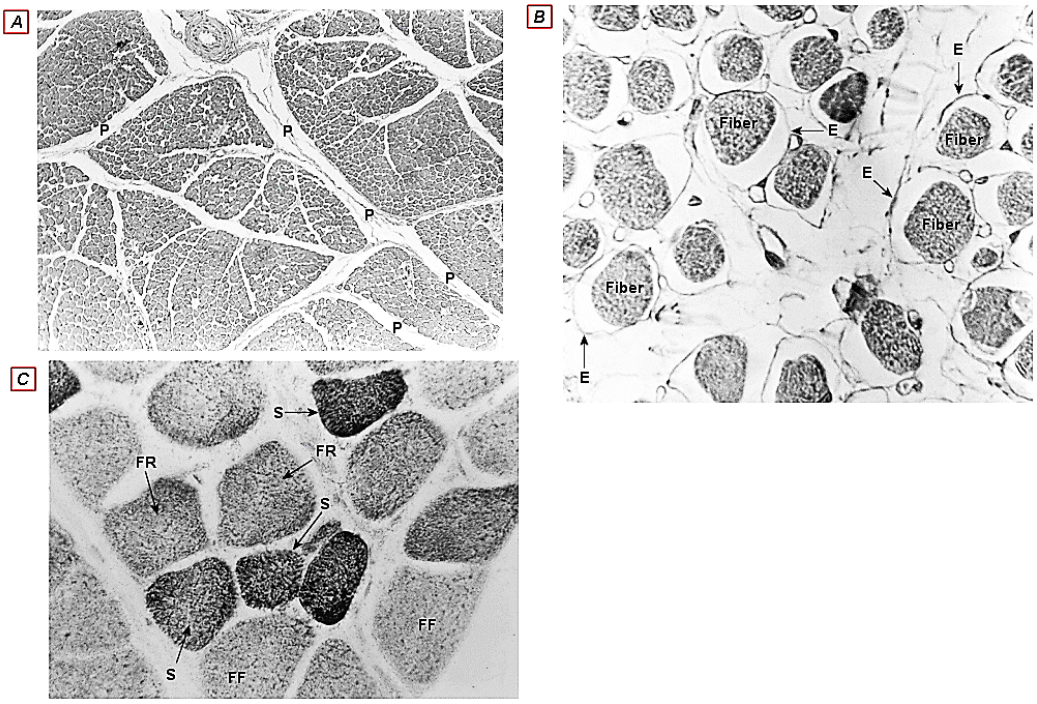
Diagram of the A and I bands, H Zone, M line, Z line and the sarcomere between the two Z lines.

- In relaxed muscle, the middle of the A-band shows a lightened H-zone which contains myosin but no overlapping actin.
- In the center of the H-zone is the dark M-line (region) that contains thin M-filaments that connect the ends of the myosin filaments and each other.

- TYPES OF SKELETAL MUSCLE FIBERS

  - The three main types of muscle cells are 1) slow twitch, 2) fast twitch fatigue resistant, and 3) fast twitch fatigable.
  - SLOW TWITCH FIBERS (S) produce the least amount of force (less than half the force produced by fast twitch fatigue resistant fibers), but are the most resistant to fatigue.
  - FAST TWITCH FATIGABLE FIBERS (FF) produce the greatest amount of force (two to three times the force produced by fast twitch fatigue resistant fibers), but are the least resistant to fatigue.
  - FAST TWITCH FATIGUE RESISTANT FIBERS (FR) produce forces that are greater than slow twitch fibers but less than fast twitch fatigable fiber, but these fibers are more resistant to fatigue than fast fatigable fibers but less fatigue resistant than slow twitch fibers.

	SLOW TWITCH FIBERS (S)	FAST FATIGUE RESISTANT FIBERS (FR)	FAST FATIGUABLE FIBERS (FF)
OTHER NAMES	Red, Slow Oxidative *(Type I)*	White, fast oxidative, *(Type IIA)*	White, fast, glycolytic *(Type IIB)*
TWITCH TIME	Slow	Fast	Fast
TWITCH TENSION	Low	Intermediate	High
FATIGUE RATE	Slow	Intermediate	Fast
OXIDATIVE CAPACITY	High	High	Low
FIBER DIAMETER	Small	Large	Large
MOTOR UNIT SIZE	Small	Large	Large
CAPILLARY DENSITY	High	High	Low
MITOCHONDRIA	Many	Many	Few



Photomicrographs of (A) a cross-section of skeletal muscle showing fascicles separated by perimysium (p). (B) high magnification of skeletal muscle fibers surrounded by endomysium (e) and capillaries (c). (C) high magnification of skeletal muscle fibers showing different types of fibers based on histochemical reactions. Slow twitch (S), fast fatigue resistant (FR), fast fatiguable (FF).

- In the body, muscles are composed of different percentages of each type of muscle cell with some muscles, such as those of the deep back, containing predominately slow twitch fibers, but other muscles, such as those of the limb, containing a mixture of all three types with fast twitch fibers predominating.
- Disuse or immobilization produces atrophy in non-athletes mainly of the slow twitch (type I) fibers.
  + In athletes involved in endurance type sports, it appears that the slow twitch (type I) and fast fatigable fiber (type IIB) are mainly effected.
  + In athletes involved in explosive type sports, it appears that the fast twitch (type II) are mainly effected.
  + Isometric exercises, commonly used during immobilization, help maintain the type II fast twitch but not the type I slow twitch.

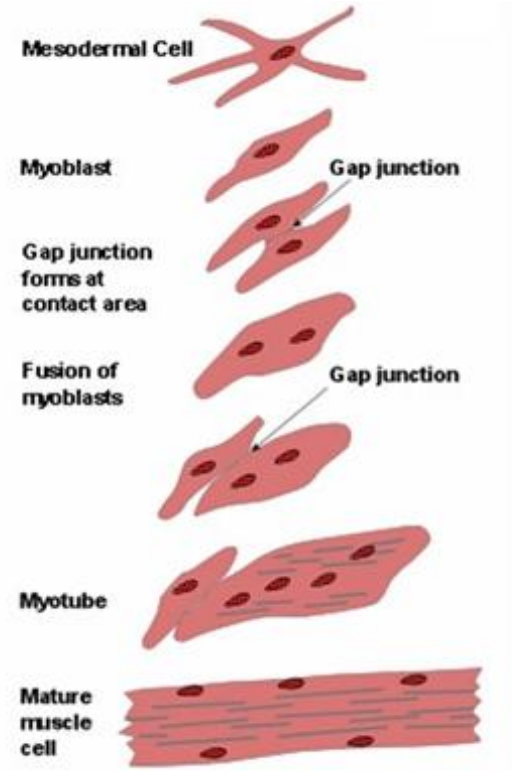
- SKELETAL MUSCLE CONNECTIVE TISSUE

  - EPIMYSIUM is a thin dense irregular connective tissue coat that covers the entire skeletal muscle.
  - PERIMYSIUM is a thin layer of dense irregular connective tissue that extends inwardly from of the epimysium to subdivide the whole muscle fascicles.
  - ENDOMYSIUM is a thin loose connective tissue that extends from the perimysium into the fascicle to surround the individual muscle cells.

- SKELETAL MUSCLE PRENATAL DEVELOPMENT

  - Skeletal muscle cells are derived from mesodermal cells (mesenchyme) which elongate to form myoblasts that are spindle shaped cells with a central nucleus.
  - The myoblasts divide and proliferate and neighboring myoblasts converge and align themselves so that the long axis of each cell lies in parallel.
  - Adjacent cell membranes adhere; the adhering cell membranes break down and the myoblasts fuse together producing a multinucleated, cylindrical myotube.

- Additional myoblasts are added to length of the myotubes, myofilaments form, myotubes enlarge, more myofilaments develop and are arranged to form bands of overlapping actin and myosin filaments.
- As the myofilaments arrange to form bands, the sarcoplasmic reticulum and T-tubules develop and the nuclei, which are initially in the center of the myotubes, move peripherally under the sarcolemma and the myotube transforms into a skeletal muscle cell.



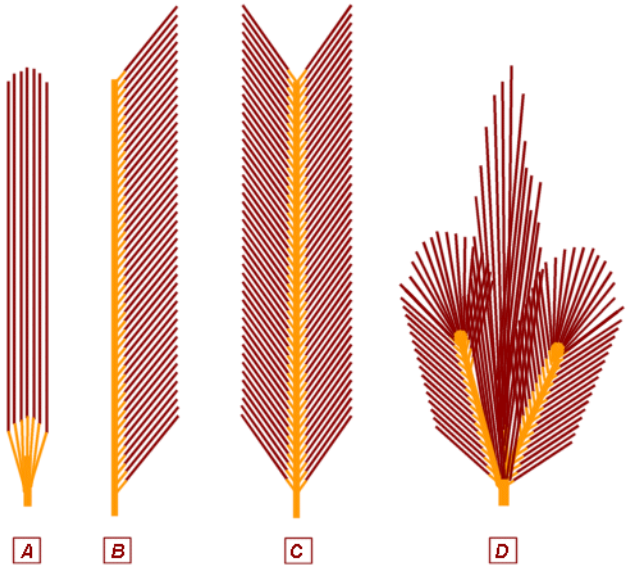
Drawing showing the development of skeletal muscle fiber from myoblasts and myotubes (modifies from McComas 1996).

o POSTNATAL DEVELOPMENT OF SKELETAL MUSCLE

- Skeletal muscle cells increase in length by adding sarcomeres at the ends of the myofibrils.
- Growth in the diameter of the muscle cell occurs by adding more myofilaments, which increases the force production capacity of the muscle.
- Exercise stimulates muscle cells to increase the contractile components to meet the demands of increased loading: muscle cells add actin and myosin myofilaments and the diameter of the cell increases (hypertrophy).
- Muscle disuse (immobilization, bed rest, and various nutritional deficiencies) results in the loss of myofilaments and myofibrils and muscle cells decrease in diameter (atrophy).

○ SKELETAL MUSCLE FIBER ARRANGEMENTS

- Muscle fiber lengths are approximately proportional to the shortening velocity of the muscle and to the amount of tendon excursion produced by the muscle.
- Muscle fibers can shorten 30–50% of the length of the fiber.
- The number of muscle fibers is reflected by the physiological cross-sectional area (PCSA) of muscle which is approximately proportional to the amount of force that muscle can produce.
- Muscles with short fibers and large PCSA are designed for force production.
- Muscles with long fibers and small PCSA are designed for producing large excursion and high velocity movements.
- Muscle fiber in a muscle can be arranged in several ways.



Drawings of common arrangement of muscle fibers in a muscle. (A) Parallel-fibered muscle, (B) Unipennate fibered muscle, (C) Bipennate fibered muscle, and (D) Multipennate fibered muscle.

- PARALLEL-FIBERED MUSCLES (A) have fibers that lie generally in parallel to each other and the tendon.
  + The amount of muscle fiber shortening in these muscles produces a similar amount of movement of the tendon. If a parallel fibered muscle shortens 3 cm, the tendon moves about 3 cm.
  + The magnitude of force from the contraction of these parallel muscle fibers produces a similar magnitude of force that is directly aligned with the pull of the tendon. If the muscle produces 1N of force, most of this force of contraction is applied to the tendon for motion.

- PENNATE-FIBERED MUSCLES have fibers that lie at an angle to the tendon.
  + There are unipennate (B), bipennate (C), or multipennate (D) based on the orientation of the fibers.
  + Because the fibers pull at an angle to the tendon, the amount of tendon movement and magnitude of muscle force are divided into X and Y components; the amount of muscle fiber movement and force aligned with the Y component are applied to the tendon for movement.
  + The smaller the degree of fiber angulation relative to the tendon, the greater the amount of applied force and the greater the amount of tendon movement.
  + The percentage (amount) of tendon movement is less than the percentage of muscle fiber shortening because of the angulation of the muscle fibers.
  + The resultant force on the tendon is also less than the magnitude of force produced by the muscle because of the angulation of the muscle fibers.

- TYPES OF MUSCLE CONTRACTIONS

  - ISOMETRIC contraction involves no movement and no mechanical work (work = force X distance); the tension developed is equal to the resistance of the external load; the length of the muscle is constant; and the energy of contraction is lost mainly as heat.
  - CONCENTRIC OR SHORTENING CONTRACTION requires that the tension generated by the muscle is greater than the resistance of the external load; the muscle shortens in length and there is joint movement in the direction of muscle shortening; mechanical work occurs.
  - ECCENTRIC OR LENGTHENING CONTRACTION occurs when muscle tension is not sufficient to resist the external load or when a shortened muscle is controlling the motion produced by an external load; the contracted (shortened) muscle lengthens and there is joint movement in the direction opposite to muscle action; mechanical work occurs.
  - Tension development is greatest with eccentric contraction, followed next by isometric contraction and least with concentric contraction.

4.4.2 FORCE DEVELOPMENT OF SKELEGAL MUSCLE

- **MODEL OF MUSCLE CONCENTRIC CONTRACTION**
  - Hill in the late 1800s published a model of muscle contraction which was later modified by Voight and still later by Maxwell and others.
  - Based on Hill's model, Maxwell published a model for muscle contraction with a contractile element, series elastic element and parallel elastic element.

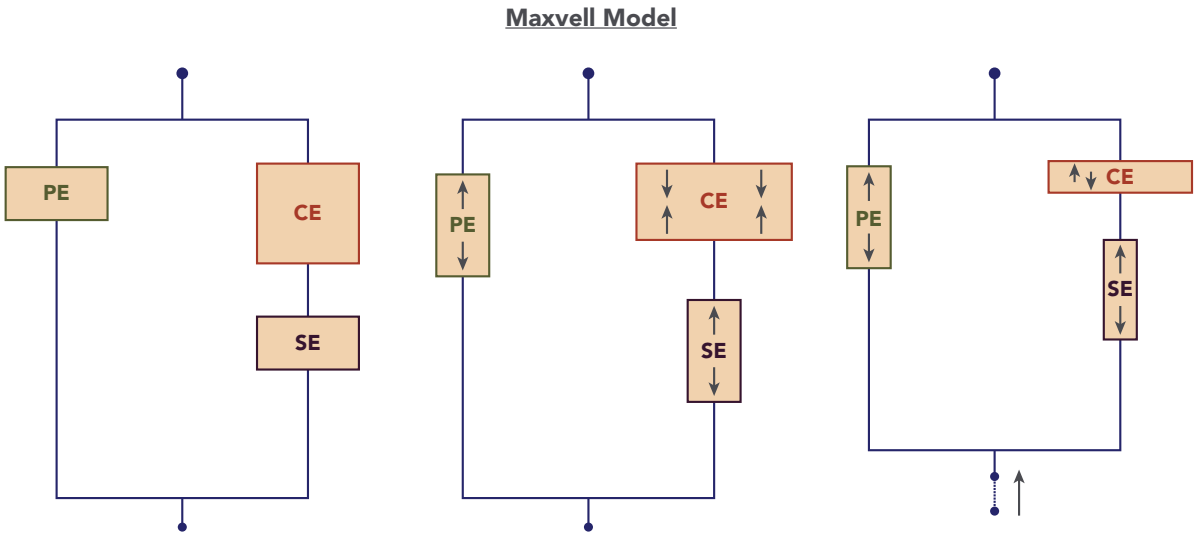


Diagram showing Maxwell's model in reference to muscle contraction. The LEFT schematic shows the elements with the muscle at rest. The Middle schematic shows the beginning of muscle contraction with the contractile element shorting and the series and parallel elastic elements tightening. Because the elastic elements are not taut, there is no movement. The RIGHT schematic shows further shortening of the contractile element. The series and parallel elastic element are now taut and movement occurs.

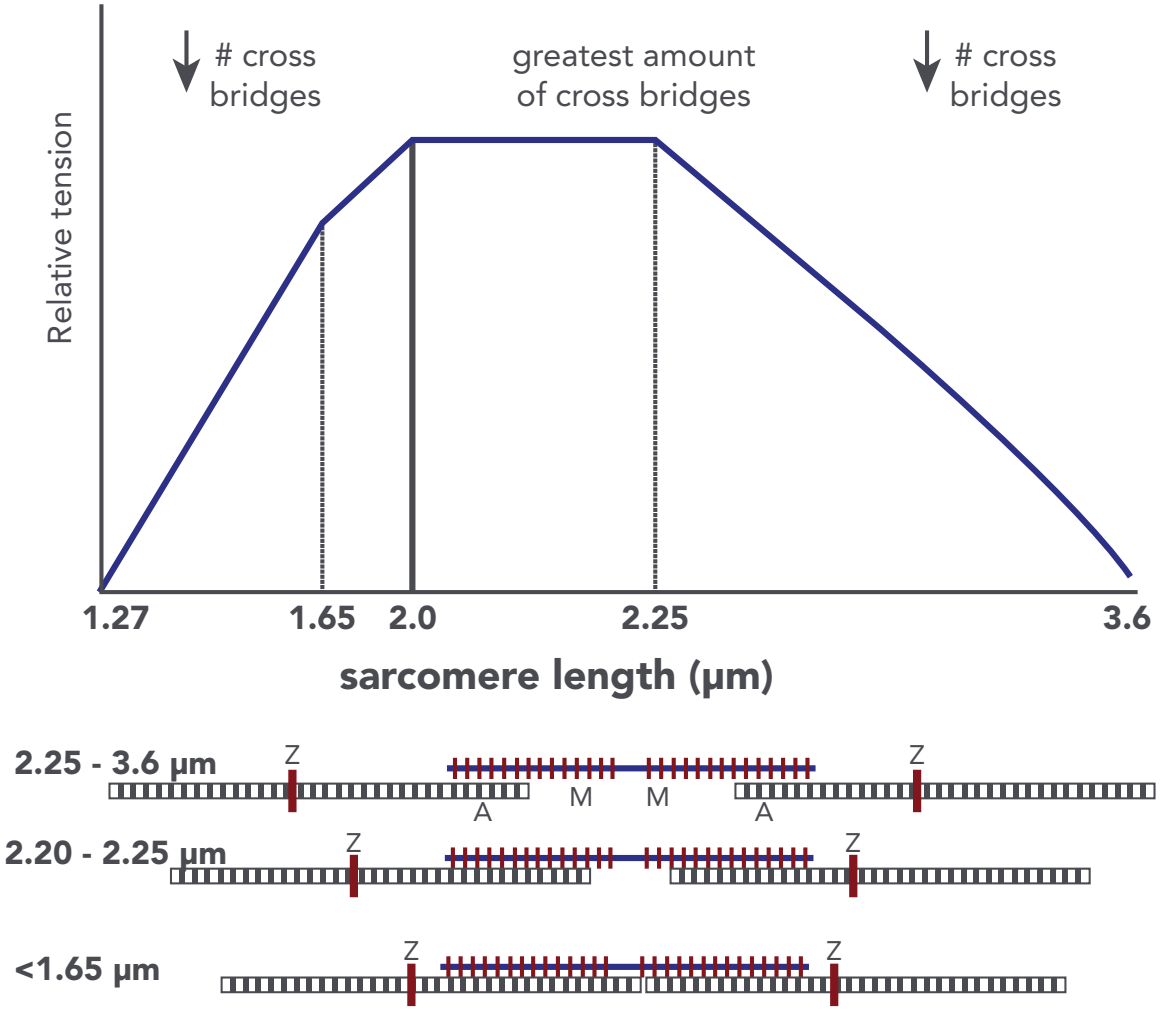
- The CONTRACTILE ELEMENT (CE) represents the actions of actin and myosin myofilaments.
- The SERIES ELASTIC ELEMENT (SE) represents the elasticity of the tendons.
- The PARALLEL ELASTIC ELEMENT (PE) represents the elasticity of the epimysium, perimysium and endomysium.
- Forces generated during shortening of the contractile element first tightens the parallel and series elastic elements.
- After the elastic elements are taut, then movement begins.
- Passive pre-stretching of a muscle before contraction acts to tense the elastic elements so that movement can occur with initial shortening of the contractile element.
- When a muscle relaxes, it is the elastic components that return the muscle to its resting length.

- **LENGTH – TENSION CURVE OF SKELETAL MUSCLE**

  - Sarcomere force production determines muscle fiber force production.
  - Sarcomere force is determined by the number of cross-bridges activated and the number of cross bridges activated is determined by the degree of overlap of the actin and myosin filaments.
    - When a large number of cross-bridges are activated, high forces are generated.
    - When a small number of bridges are activated, small forces are generated.
    - When muscle fibers are near resting length (2.0–2.25 um), the actin and myosin are positioned for maximal overlap and the peak production of force.

  - The figure below shows the relationship of force production and sarcomere length and as sarcomere length relates to actin and myosin overlap and it also relates to the degree of cross bridge formation.
    - The left side of figure shows the amount of tension generated when sarcomere lengths are short, the middle section shows tension when the sarcomeres are near the resting length and the right side shows tension when sarcomere lengths are long.

- When sarcomeres are short the filaments produce low tension because the close approximation of the actin filaments restricts further shortening and cross bridge formation.
- When the sarcomeres are long, the actin filaments barely overlap the myosin so few cross-bridges are formed and tension is low.
- When sarcomeres are near resting length, actin and myosin are positioned for optimal overlap which produces the largest number of cross bridges and the greatest tension.

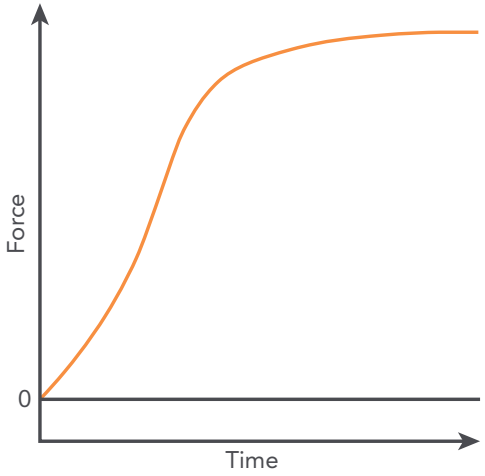


This diagram shows the length-tension curve for skeletal muscle. A = actin; M = myosin; Z = Z-line

- This relationship between sarcomere length and tension is important when considering the positions of the muscles and joints during muscle assessments, exercises, stretching or immobilization.

- If the position of a joint or limb places a muscle on slack, contraction of this slackened muscle must first take up the slack and tense the elastic elements before joint movement occurs. Because the contractile elements have shorted to take up the slack, it sacromeres lengths are shortened and its force capability reduced.
- If a muscle is weak and placed in a position where it is on slack, the muscle may only be able to remove the slack, resulting in no joint motion.
- A combination of movements, such as scapular and glenohumeral movements or pelvis and hip movements, may occur concurrently to maintain muscle length to maximize tension development.

- **ISOMETRIC FORCE – TIME CURVE**



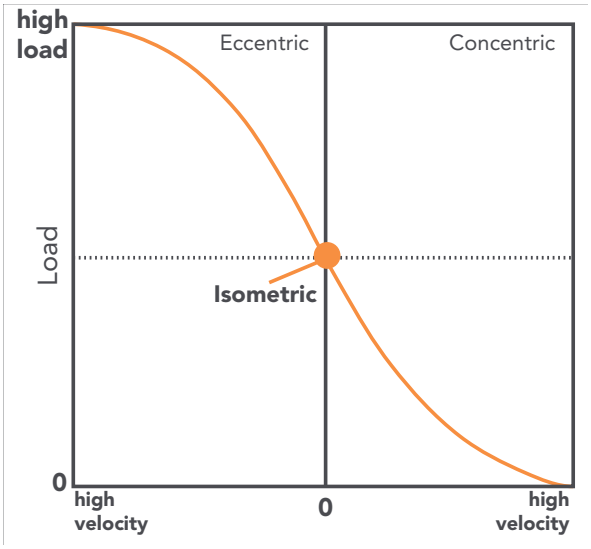
Graph showing the curve of isometric force relative to time.

- Early in an isometric contraction, the elastic elements of the muscle are stretched and become tense.
- After the elastic elements are tight, maximum isometric tension is reached.
- It takes about 300 ms to reach maximum tension.

- **FORCE – VELOCITY OF CONTRACTION RELATIONSHIP**

- With concentric contraction, muscle shortening velocity is inversely related to the magnitude of the load.
  - As the magnitude of the load increases, the velocity of muscle shortening decreases.
- With isometric contraction, there is no movement so the velocity of contraction is 0.
- With eccentric contraction, muscle lengthening velocity is directly related to the magnitude of the load.

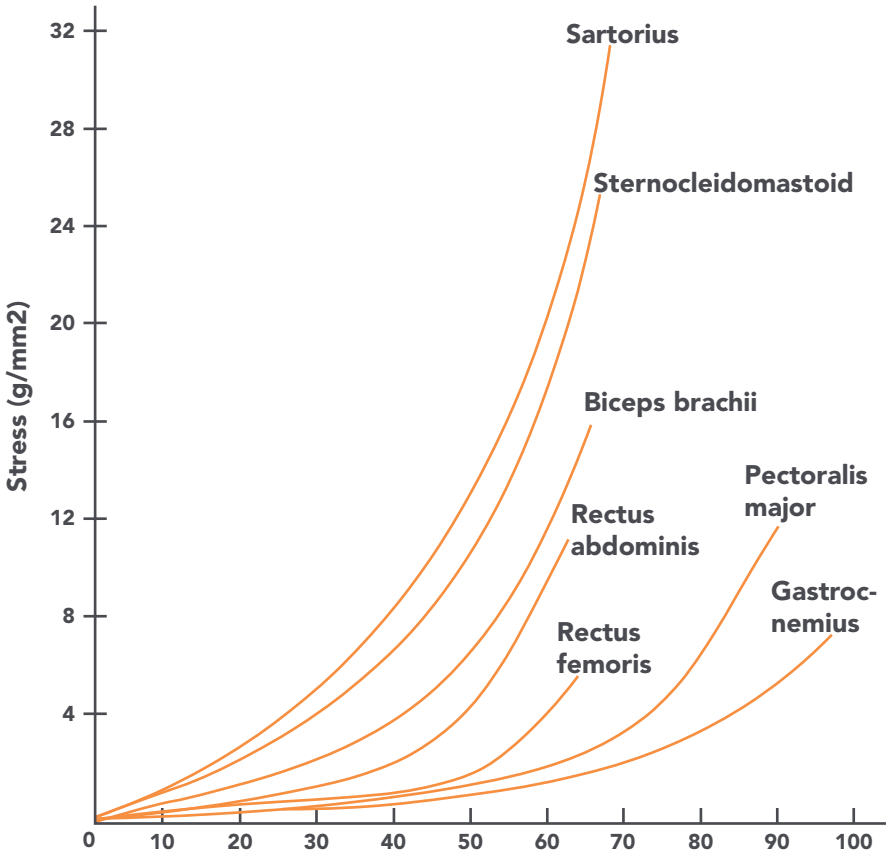
- As the magnitude of the load increases beyond the tension generating capacity of the muscle,the velocity of muscle lengthening increases.



Graph showing the load – velocity curve for concentric, isometric and eccentric muscle contractions.

- **MUSCLE TENSION – ELONGATION CURVE**

  - When a muscle is stretched beyond its resting length, the contractile element (actin and myosin) and the tightening of the series and parallel elastic elements (tendon, epimysium, perimysium, endomysium) resist the elongation.
  - This resistance to elongation will produce tension by the muscle.
  - This tension – elongation relationship varies among muscles.
    - For some muscles, elongation results in a rapid rise in tension which produces muscle stiffness and a high resistance to further elongation or stretching (see sartorius and sternocleidomastoid in graph below).
    - For other muscles, elongation results in a gradual increase in tension producing less muscle stiffness and less resistance to further elongation or stretching (see pectoralis major and gastrocnemius in graph below).
  - This tension – elongation relationship shows that the amount of elongation produced during muscle stretching varies considerably among muscles.
  - Simply stated, some muscles are easier to stretch than others.



Graph of tension stress-elongation curves for several muscles. (Yamada 1970)

- **EFFECTS OF TEMPERATURE ON MUSCLE FORCE**

  - Increasing muscle temperature increases enzyme activity within the muscle which is important in the activation and deactivation of cross-bridges.
  - Increasing muscle temperature increases force development and increase the blood flow to the muscle.
  - The heating of muscle or "warm-up" activities tend to improve muscle function but not the amount of muscle stretch.

4.4.3 SKELETAL MUSCLE AGING

- Skeletal muscle changes structurally and functionally with age, but the amount of change is highly variable among muscles and among individuals.
- Skeletal muscle size and strength increase from birth to peak between 20–30 years of age in males and females.
- Overall peak muscle strength and overall size are greater in males than females, but the strength per muscle cross-sectional area and the distribution of slow twitch and fast twitch fibers are similar between males and females.
- After age 30–40, there is a general decline in muscle mass and strength in both sexes with a decrease in the number of slow and fast twitch fibers, especially the fast twitch fibers.
- Loss of strength, as measured by isometric force production, begins slowly around 30–40 years old in males and females, but tends to accelerate after the age of 45 and correlates with the loss of fast twitch fibers and the loss of motor units.
- With age, the contraction velocity of skeletal muscle also decreases and appears to be associated with a longer release and uptake times of calcium by the sarcoplasmic reticulum.
- Although with age, skeletal muscle shows a decrease in muscle fiber number, a decrease in isometric strength, and a decrease in contraction velocity, skeletal muscle in males and females of advanced age (70–80 yo) will respond to resistive and endurance exercises and activities with an increase in strength, endurance, and muscle cross-sectional area.

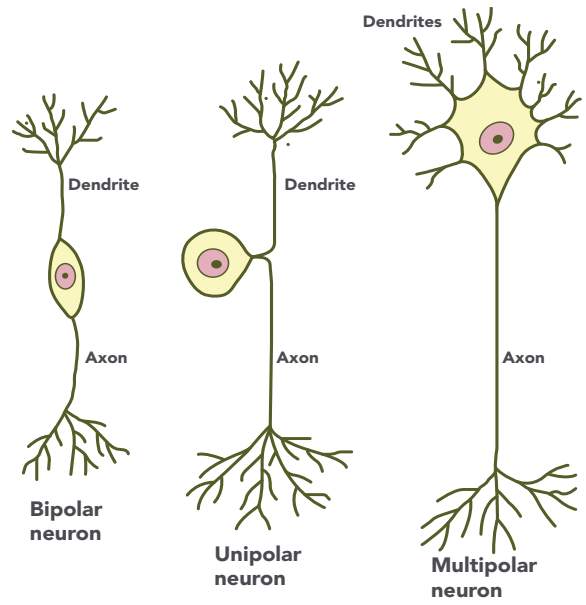
Study Questions (Muscle)

1. How are slow and fast twitch muscle fibers different in force production, fatigability, and response to immobilization?
2. How does the arrangement of muscles fibers in parallel differ in force production and displacement from muscle fiber in a pennate arrangement?
3. What is the Maxwell's model and how does it relate to muscle action and muscle weakness?
4. How does the length tension curve relate to exercise and Maxwell's model?
5. How does the force-velocity relationship differ between concentric and eccentric types of muscle contraction?
6. How is muscle affected by aging?

4.5 PERIPHERAL NERVES

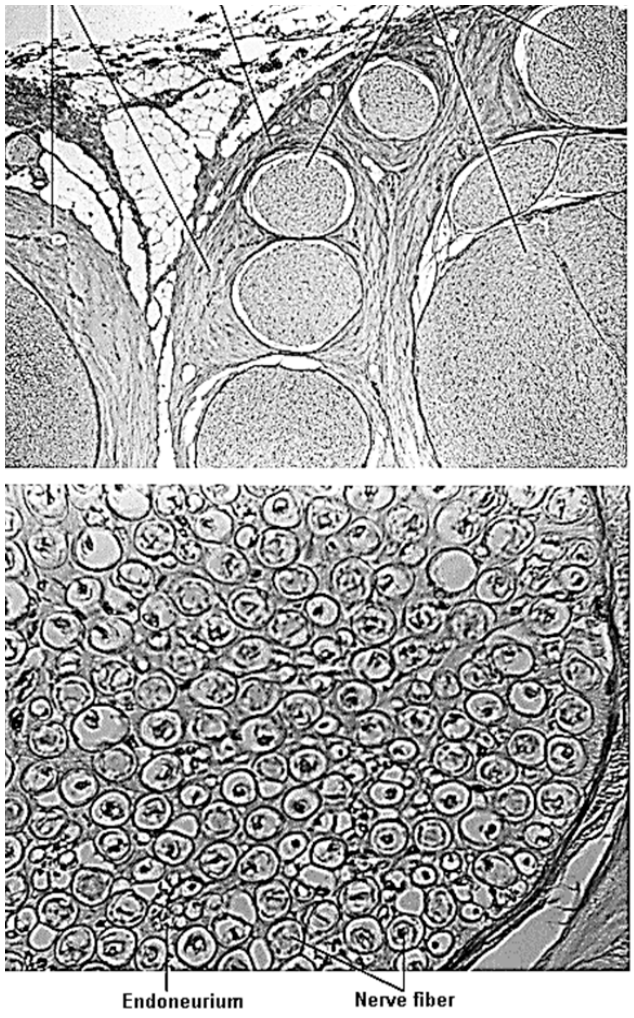
4.5.1 ANATOMY OF NERVES

- **NEURONS**
  - Neurons are the structural and functional unit of peripheral nerves.
  - Each neuron has a cell body, an axon and dendrites.
  - There are 3 basic types of neurons.
    - UNIPOLAR (PSEUDOUNIPOLAR) NEURONS are mainly sensory neurons.
    - BIPOLAR NEURONS are mainly interneurons, linking neurons.
    - MULTIPOLAR NEURONS are mainly motor neurons.



Drawing of bipolar, unipolar and multipolar neurons.

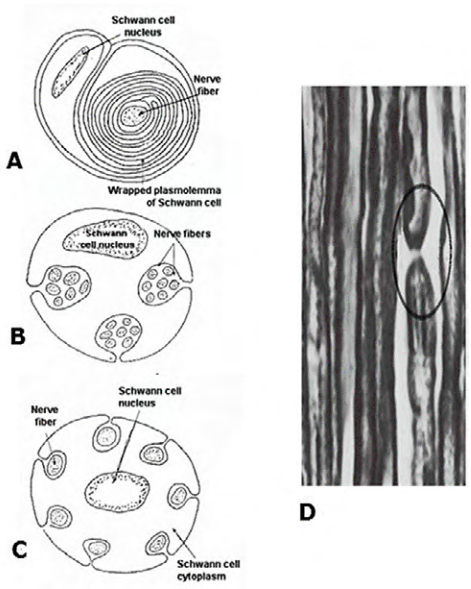
- **SUPPORTING TISSUE OF NERVE**
  - Peripheral nerves contain connective tissue supports and a Schwann cell support for each nerve fiber.
  - Peripheral nerves has an outer covering of connective tissue, septa of connective tissue that surrounds groups of nerve fibers in fascicles, and a fine and delicate array of connective tissue fibers that separate individual nerve fibers.
    - EPINEURIUM is a dense connective tissue layer that surrounds the entire peripheral nerve.
    - PERINEURIUM is a dense connective tissue layer that extends inwardly from the epineurium to surround bundles of nerve fibers called fascicles.
    - ENDONEURIUM is a delicate connective tissue layer that extends from the perineurium to loosely surround individual nerve fibers lying within each fascicle.



Photomicrographs of a cross-section of two peripheral nerves (A) showing its connective tissue jacket of epineurium and its perineurium lying between the nerve fascicles and (B) Higher magnification of a nerve showing individual nerve fibers separated by endoneurium.

- SCHWANN CELLS

  - These cells support nerve fibers and form the myelin sheath of myelinated nerves and engulf nerve fibers with cytoplasm in non-myelinated nerves

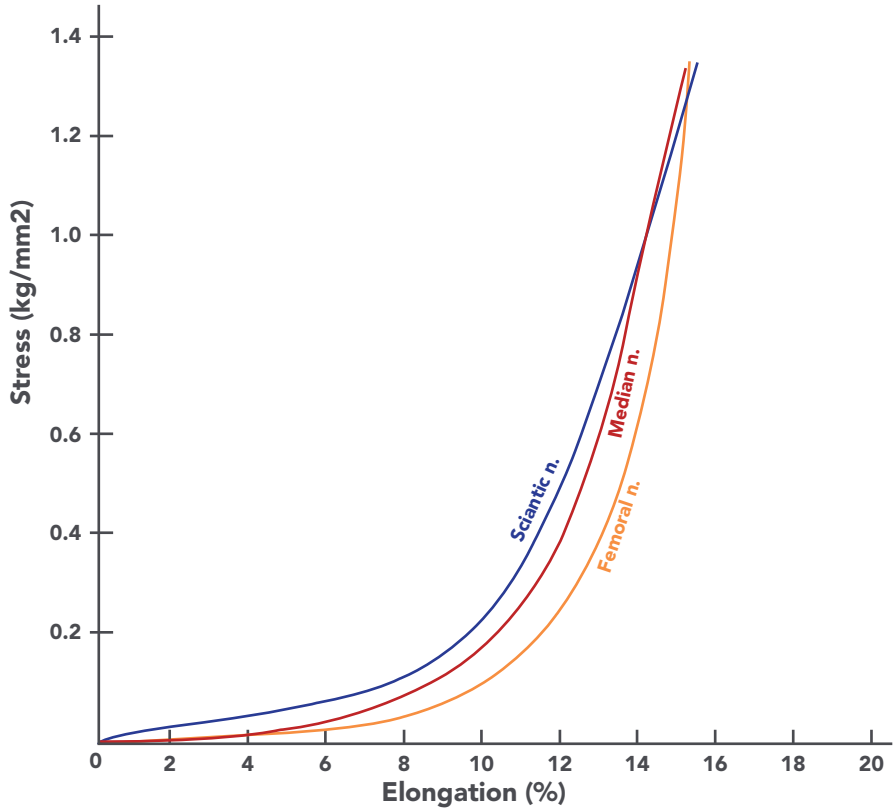


Drawing of a (A) Schwann forming the myelin sheath of a nerve fiber, (B) Schwann cytoplasm engulfing several nerve fibers of unmyelinated nerves, (C) Schwann cytoplasm engulfing single nerve fibers of unmyelinated nerves and (D) a photomicrograph of a longitudinal section of a nerve showing a Node of Ranvier (encircled).

- The myelin sheath covering myelinated nerves is produced by the wrapping of the plasma membrane of the Schwann cell multiple times around a small section of the nerve fiber.
- As the plasma membrane is wrapped, the cytoplasm between the layers of membrane is pushed aside and the membranes fuse, forming the lipid-rich myelin.
- As each Schwann cell wraps only a small section of the nerve, multiple Schwann cells are involved in producing the entire myelin sheath.
- The slit-like junction at which adjacent Schwann cells meet is called the Node of Ranvier which is void of myelin but the nerve fiber is still covered by Schwann cell cytoplasm.
- Myelination of a nerve permits the rapid conduction of signals along the nerve.
- Non-myelinated nerves are engulfed by the Schwann cell cytoplasm.
- As Schwann cells engulf only small sections of non-myelinated nerves, many Schwann intersect to cover the entire nerve.
- Non-myelinated nerves conduction velocities are much slower than for myelinated nerves.

4.5.2 NERVE TENSION-ELONGATION (STRESS-STRAIN) CURVE

- When tension is initially applied to a peripheral nerve, a small amount of force easily produces elongation as the slacked nerve itself and the slacked connective tissue component of the nerve gradually tighten.
- As the nerve and its connective tissue component tightens, the amount of force required to increase nerve elongation increases until the forces are high enough to rupture the connective tissue component and the nerve.
- As the nerve tightens, the hydrostatic force within the nerve increases and this increase in force will result in damage to the delicate nerve fibers before the nerve ruptures.



Graph tension stress – elongation strain curves for the sciatic, median, and femoral nerves. (Yamada 1970)

- As the nerve tightens, blood flow to the nerve will also be compromised (Rydevik et al., 2001).

    - In a relaxed peripheral nerve, the segmental blood vessels enter the peripheral nerve at an acute angle and are coiled.
    - With 5–8% nerve elongation, the blood vessels stretch and there is a reduction in blood flow to the nerve.
    - With 15–20% elongation, the blood vessels become taut, the lumen closes and blood flow can cease.
    - Reduced blood flow to nerves can also occur with arthrosclerosis, diabetes and with mechanical compression of the nerve or its blood supply by use of a tourniquet during surgery or by tumors, connective tissue scarring or excessive muscle tone in the surrounding tissues.

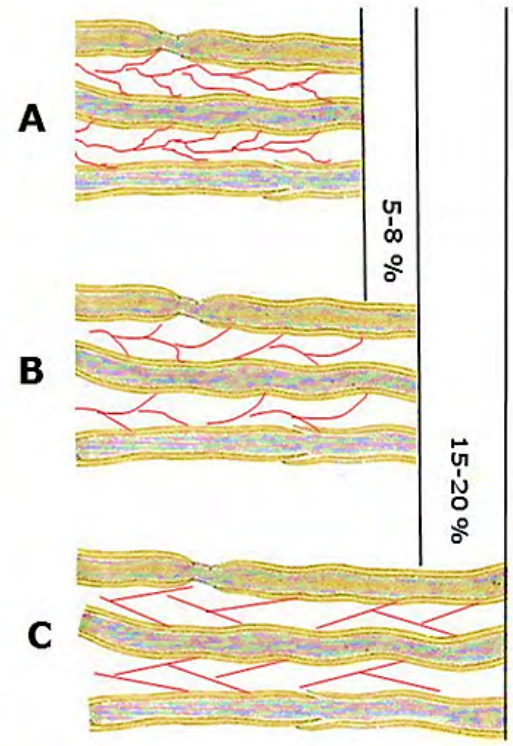


Diagram of (A) a nerve at rest with coiled blood vessels, (B) a nerve elongated 5–8% and its stretched blood vessels, and (C) a nerve elongated 15–20% with its taut blood vessels.

4.5.3 COMPRESSIONS ON PERIPHERAL NERVES

- **CIRCUMFERENTIAL NERVE COMPRESSION** (Rydevik et al, 2001)

  - In this type of compression, the entire circumference of a section of nerve is compressed; similar to a blood pressure cuff compressing the arm.
  - This type of compression mechanically damages mainly those nerve fibers along the circumferential contact area of the peripheral, producing an EDGE EFFECT.
  - Nerve damage may occur because of an increase in hydrostatic pressure within the peripheral nerve damaging the nerve directly and/or producing a decrease in blood flow.
  - Mechanical damage to the nerve may also reduce blood flow with this type of compression.
  - Examples of circumferential compression are carpal tunnel and ulnar nerve compression in the cubital tunnel.

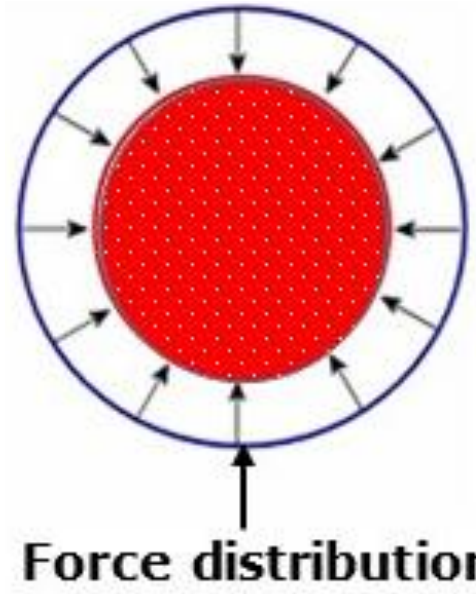


Diagram showing the force distribution and edge effect with circumferential nerve compression. (Modified from Rydevik et al, 2001)

- **LATERAL NERVE COMPRESSION** (Rydevik et al, 2001)

  - This type of nerve compression occurs when a nerve is compressed between two structures.
  - The nerve fibers mainly damaged are those directly under the contact areas.
  - Nerve damage may occur because of an increase in hydrostatic pressure within the peripheral nerve damaging the nerve fibers directly and/or producing a decrease in blood flow.

- Mechanical damage to the nerve may also reduce blood flow with this type of compassion.
- Examples of lateral nerve compression are compression between two bones, between a bone and dense fibrous structure, between two stiff fibrous structures, between a tumor mass and a hard or stiff structure.

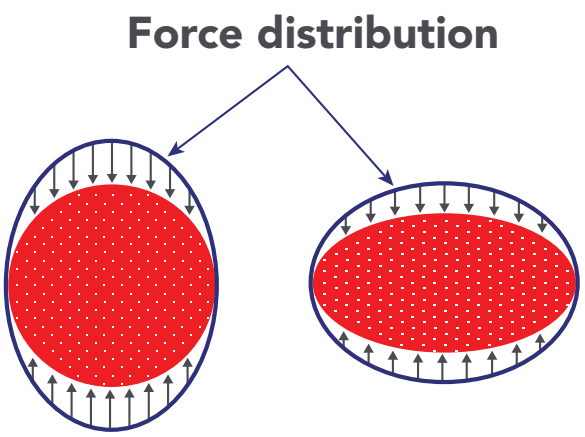


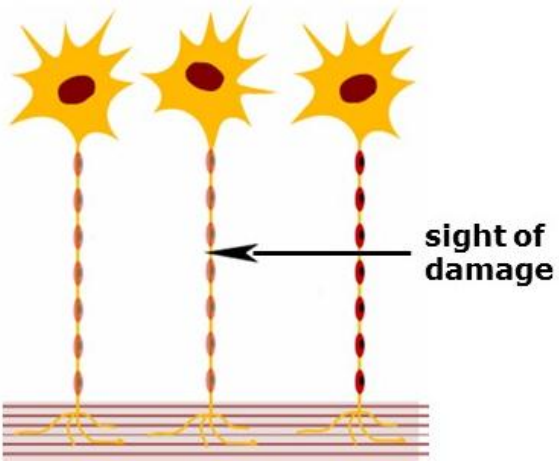
Diagram showing the force distribution with lateral nerve compression and the change from a round nerve cross-section (LEFT) to an oval shaped cross-section (RIGHT).
(Modified from Rydevik et al, 2001)

4.5.4 PROGNOSIS GUIDELINES FOR NERVE INJURY

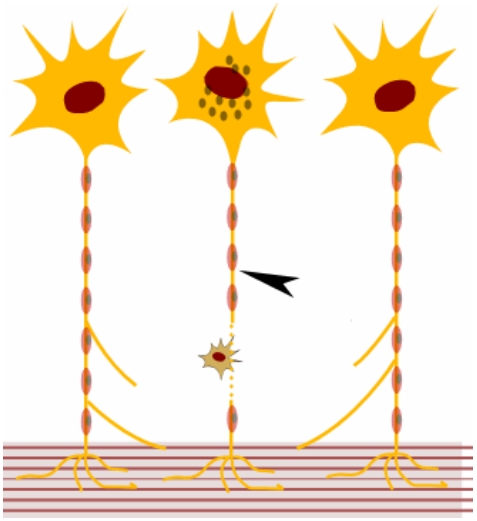
- The literature is not specific in stating guidelines for nerve injuries because of the variability of damage and recovery. However, there are some general guidelines that that may be clinically useful.
  - If the **force** was of **low** magnitude and for a **short duration,** then the prognosis should be **good.**
  - If the **force** was of **high** magnitude for a **short duration**, the prognosis should be **fair.**
  - If the **force** was of **low** magnitude but for a **long duration**, then the prognosis should be **fair.**
  - If the **force** was of **high** magnitude for a **long duration**, the prognosis should be **poor.**

4.5.5 PERIPHERAL NERVE REGENERATION

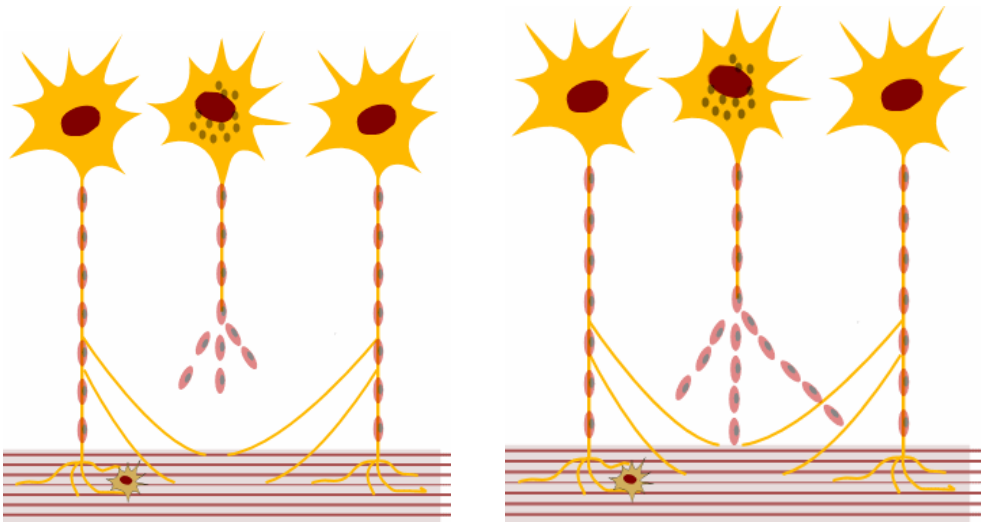
- Regeneration occurs at a rate of 1mm per day.
- The damaged nerve fibers degenerate distally and proximally. If the proximal degeneration does not kill the nerve cell body than regeneration distally is possible if Schwann cells and endoneurium are intact.



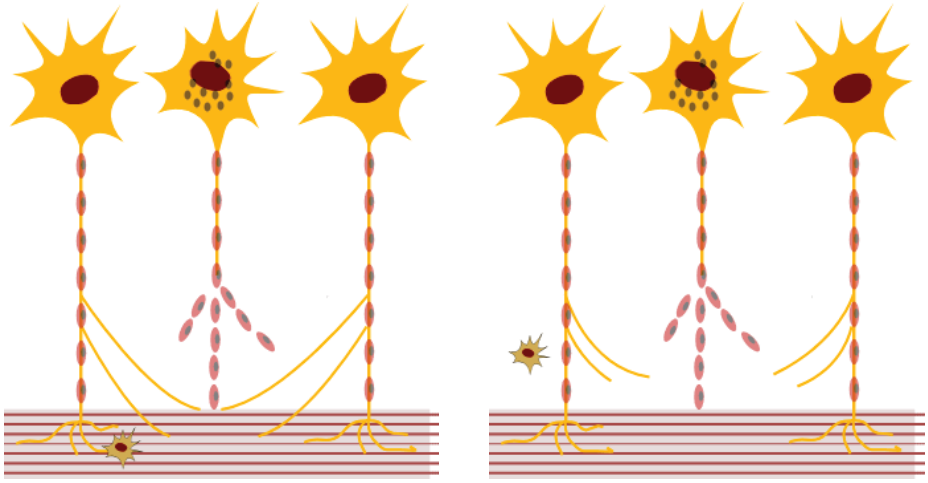
1. **Schwann cells and endoneurium are intact.**
2. **damaged nerve fibers dissolve**
3. **myelin sheet breaks down into fat droplets**
4. **macrophages remove the degenerated material.**



5. **Schwann cells then form water filled tubes with thick walls**
6. **very small opening for one nerve fiber**
7. **multiple sprouts grow outwardly**



8. **sprouts may find tube and grow to effector organ**
9. **sprouts not finding proper tube degenerates**



- **FACTORS AFFECTING REGENERATION**

  - TYPE OF TRAUMA
    - Crushed nerve injuries may have a better outcome than severed nerve injuries because crushing might damage fewer Schwann cells and endoneurium in the nerve allowing for more Schwann cell tubes to be formed.
    - Severed nerves tend to separate and twist which makes realigning of matching tubes to the nerve sprouts very difficult if the tubes even develop because of damage to the Schwann cells and endoneurium at the site of the cut.

  - TYPE OF NERVE
    - Mixed nerves carrying motor and sensory fibers would have a lower success rate of matching sensory to sensory and motor to motor tubes than a nerve that is predominately sensory or motor.
      + Because of the mixture of sensory and motor nerves fibers, realignments of matching tubes with the proper nerve sprout are difficult especially if the nerve is severed.
      + The probability of a severed mixed nerve regaining function is very low.
      + The probability of a severed mixed nerve regaining function is less than that of a crushed mixed nerve.
    - Predominantly sensory or motor nerves show a higher probability of regaining function than mixed nerves because the probability of matching the proper Schwann cell tubes with the proper type of nerve sprout is higher than in mixed nerves.
      + The probability of a crushed predominantly sensory or motor nerve regaining function is greater than a severed predominantly sensory or motor nerve.
      + The probability of a crushed or severed mixed nerve regaining function is less than with a predominantly sensory or motor nerve.

4.5.6 PERIPHERAL NERVE AGING

- There is very little change in the mechanical properties of peripheral nerves with age.
- Tensile strength and elasticity change very little after the age of 20.
- Nerve conduction velocities do decrease after the age of 60 but the decrease is less than 10 meters/sec (normal is 50–70 m/sec).
- Most changes in peripheral nerve function with age appear to be a result of trauma, decreased blood flow and disuse.

Study Questions (Nerve)

1. How can nerve fibers in a peripheral nerve be damaged when tensionis applied to the nerve?
2. How can a nerve be damaged in compression?
3. Why is nerve regeneration so often unsuccessful in a mixed peripheral nerve?
4. What are the effects of aging on non-pathological peripheral nerve?

5 BIOMECHANICS STUDY QUESTIONS

Study Questions (spine)

1. How are each of the following involved is motion of the spine:
    a. articular facet angulation
    b. annulus fibrosus
    c. nucleus pulposus
    d. cruciform ligament
    e. alar ligament
    f. anterior longitudinal ligament
    g. ligamentum flavum
    h. supraspinous ligament
    i. rib cage
2. How do movements of the facets in the lower cervical, thoracic and lumbar regions compare during the following:
    a. forward flexion
    b. side bending
    c. rotation
3. What occurs at the spine during the following vertebral movements:
    a. forward flexion
    b. backward extension
    c. side bending
4. What are the sequence of events that occur during the following:
    a. forward trunk flexion from upright standing
    b. side bending from upright standing
5. Which muscles extend, laterally bend and rotate vertebrae to the sameside?
6. Which muscles extend and rotate vertebrae to the opposite side?

ANSWERS

1. a. The angulation of the facet guides movement at the facet joints and thus vertebral movement.
   b. The physical properties of the annulus fibrosus enable it to stabilize the adjacent vertebral bodies but at the same time allow the annulus to deform so that the bodies can move in multiple direction. As the annulus deforms during movements of the vertebral bodies, the proteoglycans and collagen will limit the amount of annulus deformation and thus limit the movements of the vertebral bodies.

c. The spherical configuration of the nucleus pulposus, which is maintained by the annulus fibrosus, acts as a pivot point about which the adjacent vertebral bodies can tilt or rock in multiple directions. The direction of tilting is guided by the angulation of the facets and the extent of motion restricted by the physical properties of the annulus.
d. The cruciform ligament has 3 components. The transverse ligament of the atlas forms a sling around the dens to hold it in proper alignment at the median atlanto-axial joint so that the atlas and head can pivot smoothly about the dens. Upward or downward slippage of this sling is controlled by the superior and inferior longitudinal ligaments of the cruciform. Slack of these ligaments would lead to wobbling of the dens and uneven motion at the lateral atlanto-axial joints. This type of motion could affect the degree of rotation and accelerates joint wear. Further, slack of the cruciform may allow the dens to move posteriorly, decreasing the size of the spinal canal and potentially compress the spinal cord. Tightness of the cruciform may limit rotation by increasing compression forces at the median atlanto-axial joint.
e. The alar ligaments, like the cruciform are important in holding the dens in proper alignment and limiting any posterior movements of the dens toward the spinal canal. The alar ligaments also restrict rotation of the atlas and head about the dens. Slack of the alar ligaments may allow the dens to move posteriorly and increase the tension on the cruciform. This tension may in time stretch the cruciform and allow the dens to decrease the size of the spinal canal and potentially compress the spinal cord. Tightness of the alar ligaments would tend to restrict rotation of the atlas and head.
f. The anterior longitudinal ligament lies on the anterior surface of the spine and will limit extension of the spine. Slack in this ligament can allow vertebral bodies to slide anteriorly on each other. This slippage is most often seen in the lower lumbar spine and can be associated with fractures of the pars articularis. Anterior slippage of one vertebrae on another will decrease the size of the intervertebral foramen and increase compression of the facets. The increase in facet joint compression can limit movement in all three planes.

g. The ligamentum flavum is an elastic ligament that stretches with flexion and recoils with extension. Stretching of this ligament during flexion may control the rate of facet motion. An imbalance in the elastic properties between a paired set of ligaments may result in the facet on one side moving more rapidly than the other and segmental obstruction occurring during flexion. The recoil of this ligament assists in the re-positioning of the facets. Because it attaches to the joint capsule, the ligamentum flavum also prevents the facet capsule from being pinched between the facets as the capsule slackens during extension. Tightness of the flavum would restrict spinal flexion. Slackening of the ligamentum flavum could result in abnormal segmental movement, pinching of the facet capsule between the facets and a bulging of the slackened ligamentum flavum into the spinal canal. This bulging is known to occur in the cervical spine during extension and can result in compression of the spinal cord.

h. The supraspinous ligament becomes tight with flexion and slack with extension. Its tightness during flexion limits flexion but its slackness during extension allow extension.

i. The rib cage limits movements of the thoracic spine in flexion, extension, side bending and rotation. The lower rib cage can also limit side bending of the lumbar spine.

2. a. With flexion, the lower cervical, thoracic and lumbar facets slide up bilaterally but in the lower cervical region both facets also slide forward.
   b. With side bending, the facet on the side of the direction of bending slides downward and the opposite facet sides up in the lower cervical, thoracic and lumbar regions. In the lower cervical region, the downward sliding facet also slides backward and the upward sliding facet slides forward. This backward and forward sliding in the lower cervical region does not occur in the thoracic and lumbar regions.
   c. With rotation, facet movements in the lower cervical region are the same as for side bending. In the thoracic and lumbar regions, the facet on the side of the direction of rotation distracts and the opposite facet compress and act as a fulcrum for rotation.

3. a. During vertebral flexion, the body of the vertebra rocks anteriorly over the nucleus. This movement compresses and bulges the anterior disc, tenses the posterior disc and deforms the nucleus posteriorly. At the same time, 1) the intervertebral foramen increases in size, 2) the spinous processes separate, 3) compression on the facet joints decreases, 4) the supraspinous, interspinous, (ligamentum nuchae in neck), intertransverse, ligamentum flava and posterior longitudinal ligaments tense and 5) the anterior longitudinal ligament slackens.
   b. During backward extension, the body of the vertebra rocks posteriorly over the nucleus. This movement tenses the anterior disc, compresses and bulges the posterior disc and deforms the nucleus anteriorly. At the same time, 1) the intervertebral foramen decreases in size, 2) the spinous processes converge, 3) compression on the facet joints increases, 4) the supraspinous, interspinous, (ligamentum nuchae in neck), intertransverse, ligamentum flava and posterior longitudinal ligaments become slack and 5) the anterior longitudinal ligament tenses.
   c. During side bending, the body of the vertebra rocks laterally over the nucleus. This movement compresses and bulges the disc on the side toward the movement, tenses the disc on the side opposite the movement and deforms the nucleus to the side opposite the movement. At the same time, 1) the intervertebral foramen on the side of movement decrease, 2) the intervertebral foramen on the side opposite the movement increases, 3) compression increases at the facet joint on the side of movement and decreases at the facet joint on the side opposite the movement, and 4) the intertransverse ligament on the side of movement slackens but the intertransverse ligament on the opposite side tenses.

4. a. During forward trunk flexion, concentric contraction of the abdominal and iliopsoas muscles initially moves the trunk forward so that gravity can continue to flex the trunk forward. As gravity moves the trunk forward, the erector spinae eccentrically control the forward bending movement. At about 60 degrees of forward trunk flexion, the pelvis begins to rotate anteriorly at the hip joint and the gluteus maximus and hamstrings eccentrically control this anterior pelvic rotation as the trunk reaches full flexion.
   b. During trunk side bending, the quadratus lumborum, erector spinae and abdominal muscles on the side of the direction of bending concentrically contract to initiate trunk side bending. Gravity then continue to bend the trunk laterally while the quadratus lumborum, erector spinae and abdominal muscles on the side opposite the direction of bending eccentrically controls the rate and distance of side bending.

5. splenius cervicis, iliocostalis cervicis/thoracis/lumborum, longissimus cervicis/thoracis
6. semispinalis cervicis/thoracis, multifidus cervicis/thoracis/lumborum, trapezius

Study Questions (Posture)

1. What is the LOG moment for each of the following regions and what are the active and passive forces that counteract this moment to maintain good posture?
   a. lower cervical spine
   b. thoracic spine
   c. lumbar spine
2. What structures would be stretched and shortened for each of the following postural misalignments?
   a. excessive anterior pelvic tilt
   b. excessive thoracic kyphosis
   c. excessive lumbar lordosis

ANSWERS

1. a. lower cervical spine: extension moment; passive forces = anterior longitudinal ligament; active forces = ant. scalene, longus capitis and coli
   b. thoracic spine: flexion moment; passive forces = post. Longitudinal ligament, ligamentum flava, supraspinous ligament; active forces = back extensors
   c. lumbar spine: extension moment; passive forces = anterior Longitudinal ligament; active forces = rectus and oblique abdominal muscles

2. a. excessive anterior pelvic tilt: stretched = abdominal muscles; shortened = iliopsoas muscle
   b. excessive thoracic kyphosis: stretched = dorsal back and scapular muscles, posterior longitudinal ligament; shortened = anterior longitudinal ligament, anterior shoulder girdle muscles, upper abdominal muscles
   c. excessive lumbar lordosis: stretched = anterior longitudinal ligament; shortened = posterior longitudinal ligament, supraspinous ligament, interspinous ligament, ligamentum flava, lumbar extensor muscles

Study Questions (TMJ)

1. What occurs at the TMJ during stage 2 (terminal opening)?
2. What occurs at the TMJ during initial jaw closing?

ANSWERS

1. During stage 2 or terminal jaw opening the jaw opens from 11–25 mm of opening to 40–50 mm of opening. At this time, the mandibular condyles rotate and translate anteriorly over the articular eminence, the articular disc also translates anteriorly with the condyles over the eminence, the elastic superior lamina stretches and the fibrous inferior lamina tightens.
2. During initial jaw closing, the jaw move upward from its wide open position to 11–25 mm from full closure. At this time, the mandibular condyles rotate and translate posteriorly back into the mandibular fossa, the articular disc translates posteriorly as the stretched superior lamina recoils, the fibrous inferior lamina slackens, and the lateral pterygoid muscle contracts to control the rate of posterior movement of the articular disc.

Study Questions (General Tissue Mechanics)

1. What is the difference between the elastic and plastic regions of a tissue stress vs. strain curve?
2. What is Young's modulus and how does it relate to tissue stiffness?
3. What is the difference between resilience and toughness?
4. What is the difference between toughness and brittleness?

ANSWERS

1. An elastic tissue or a tissue in the elastic region of a stress vs. strain curve will return to its original starting position of zero strain (no deformation) when the stress is removed. If a tissue is strained into its plastic region then it is permanently deformed and it will not return to its original starting position when the stress is removed.
2. Young's modulus is the ratio of the amount of stress applied divided by the amount of strain (deformation). A material with a high Young's modulus means that for a lot of stress there is little strain and that the material is stiff. A material with a low Young's modulus means that the material shows a lot of strain with little stress. This material has low stiffness and is thereby more elastic.

3. Resilience is a factor of the amount of mechanical work lost during deformation while toughness is the amount of resistance a material absorbs before it mechanically fails. A highly resilient material will lose only a little mechanical work when it deforms. It will return to its original shape or position when the stress is removes. Articular cartilage is a highly resilient material. On the other hand, a poor resilient material will readily deform permanently when it stressed. Styrofoam is a poor resilient material. A resilient material can also be tough but so can a poor resilient material because toughness is a measure of a tissue ability to absorb the energy of mechanical work before breaking. Toughness is not a measure of mechanical work lost because once the material break, all mechanical work is lost.
4. Toughness is the amount of mechanical work a material can absorb before breaking whereas brittleness is a factor of the amount of strain that occurs before breaking. A brittle material does not deform very much before it breaks. Some brittle material materials are fragile and weak, such as chalk. However, some are strong and tough, such as a drill bit.

Study Questions (Connective Tissue Proper)

1. What are the functional benefits of Loose connective tissue and Dense Irregular connective tissue?
2. How do tendons/ligaments respond to slow and fast rates of tension and what are the functional implications of these responds?
3. How do tendons/ligaments respond to tension?
4. How does the tendon/ligament load vs. joint displacement (stress vs. strain) curve relate to tendon/ligament injury?
5. How does tension affect wound healing and when is the most effective time to obtain the best response and why?
6. How does the repair process for a tendon/ligament wound differ from that for a ligament replacement?
7. How are tendons/ligaments strengthened?
8. What are the effects of aging on the mechanical properties of tendons/ligaments?

ANSWERS

1. Because loose connective tissue contains few collagen fiber and is composed mainly of cells and the ground substance, it is weak but it permits structures surrounded by loose CT to move and change shape. The loose CT surrounding neurovascular bundles allow these bundles to move during limb and trunk movements without imposing large compressive and tensile stresses and strains on these structures that could damage them. The loose CT also allows vascular channels to dilate and constrict. Loose CT between muscles allows muscles to contract individually even though these muscles lie adjacent to each other. Large numbers of cells in loose CT are involved in the immune and inflammatory responses and contribute to localized infection control and localized repair of injured tissue. Dense irregular CT is composed mainly of strong Type I collagen with few cells and little ground substance. As collagen resists tension and is arranged to resist tension in several directions in dense irregular CT, this tissue is located where multidirectional tensile stresses and strain are applied. In the dermis of the skin, it limits tension that occurs when the skin is moved up/down, forward/back, and from side to side. In a joint capsule, different parts of the capsule are tensed with different movements and thus the collagen fibers need to be arranged in multiple directions to resist tension associated with movement in different directions.

2. With a slow rate of tension, low stress produces a large degree of elongation. With a fast rate of tension, the tendon stiffens rapidly resulting in high stress and much less elongation than when a slow rate is applied. For stretching tendons/ligaments, low stress applied slowly would be more beneficial for producing elongation than a fast rate of stretch. However, if a rapid movement is needed, the muscle must contract rapidly and this rapid muscle contraction would produce a fast rate of tension on the tendon. If the tendon does not stiffen rapidly with the fast application of tension but elongates, the resulting movement would be slowed rather than the rapid movement needed for desired function.
3. Tension on tendons/ligaments stimulates the production of collagen fibers and the formation of cross-links, strengthening the structure. Tension also aids in the repair of tendons/ligaments by accelerating healing, increasing the strength of the scar and orienting the direction of collagen with the lines of stress.
4. As the tensile load on tendons/ligaments increases during activities, microfailures occur in the tendon as some of the collagen fibers break. This results in pain and inflammation but no instability. When additional numbers of collagen fibers break, tendons/ligaments cross the yield point and are permanently deformed. In this case, there is pain and inflammation as well as instability. When all the collagen fibers break, there is complete failure. There may be no pain with complete failures but there is instability.
5. In the repairing of tendons/ligaments, tension activates the production of collagen, the production of large collagen bundles, controls the direction of fiber alignment, increases scar strength, and increase the rate and completeness of healing. The most effective time to apply tension is during the fibroplasia stage and maybe the early consolidation stage when there is active collagen synthesis and remodeling. It is during this time that tension will align the fibers to resist the appropriate stresses, strengthen the collagen, and increase the rate and completeness of healing.
6. When a ligament is replaced, it initially degenerates and weakens significantly so that tension is not advised for may be several weeks. As new collagen is produced so that tension will not damage the replacement, then and only then would tension be applied as the replacement is now in the fibroplasia stage of healing. Tension at this stage would have the same benefits as a damaged tendon/ligament.
7. Tendons/ligaments are strengthened by adding collagen, forming thick collagen bundles, and increasing cross-links.
8. The tensile strength and elongation of tendons/ligaments change little (5%) until after age 70 when there is a marked decrease (20%) in tensile strength but less of a decrease (10%) in elongation.

Study Questions (Cartilage)

1. How are tension and compression resisted in articular cartilage?
2. What are the functions of proteoglycans in articular cartilage?
3. What is the difference between creep and load (stress) relaxation and how are these properties similar?
4. What are the three types of joint lubrication and how do they differ?
5. What are the effects of aging on articular cartilage and fibrocartilage?

ANSWERS

1. Tension is resisted by the type II collagen fibers in the cartilage and compression by proteoglycans. Because the glycoaminoglycans forming a proteoglycan monomer give the monomer a negative charge, the compaction of the monomers during compression results in the negatively charges monomer repelling each other. This repelling force stiffens that ground substance and resists compression of the cartilage.
2. Proteoglycans resist compression forces applied to the cartilage and because they are hydrophilic, proteoglycans are important in maintaining the high water content of the cartilage and in its nutrition. As articular cartilage is avascular, it is the loss of water during compression that removes the waste products of chondrocyte metabolism and the hydrophilic property of the proteoglycans that returns water and nutrients to the cartilage.
3. Creep is a change in articular cartilage thickness that occurs in time with a constant load. After the cartilage is compressed and the water and metabolic waste products are exuded and the load on the cartilage continues at a constant level, the collagen and proteoglycan reorganize and the result is a further decrease in articular cartilage thickness or creep. Stress relaxation occurs when the thickness of the cartilage after compression remains constant. With stress (load) relaxation, the distribution of the forces from the surface to the deep zone of the cartilage is even prior to loading. When the cartilage is loaded and its thickness decreases because of the loss of fluid, the surfaces stresses increase and are larger superficially than deep. After the fluid loss, the thickness remains constant but the stresses are still larger in the surface of the cartilage than the bottom part. With time and the redistribution of proteoglycans and collagen, the stresses from top to bottom change so that the stresses at the surface and the stresses at the bottom are equivalent. This process reduces the surface stresses and thus these surface stresses are relaxed. With both creep and stress relaxation, there is a redistribution of collagen and proteoglycans to obtain the effects of creep and stress relaxation.

4. Boundary lubrication occurs with extreme loading and when the normal lubricating method for that joint fails. It is the extrusion of lubricant from the surface layer of the articular cartilage. Hydrodynamic and squeeze film lubrication involves the distribution of synovial fluid which lies within the joint cavity and both are normal mechanisms for joint lubrication. The difference between the two is determined by the shapes of the articulating surfaces. If the surfaces are not in parallel to each other than the hydrodynamic mechanism of lubrication is used, but if the surfaces are in parallel then the squeeze film mechanism is used. In some joints, both types of lubrication are used.
5. With articular cartilage, tensile and compression properties begin to show a noticeable change at about age 40 but decline even further after age 50. The decline is less in compression properties than in tensile properties. With fibrocartilage vertebral discs, tensile and torsional properties decrease slightly by age 40. After age 40, both of these properties continue to decrease with tensile properties decreasing slightly more (20–25%) than torsional properties (15–20%).

Study Questions (Bone)

1. What resists tensile and compressive forces in bone?
2. Why is it necessary to remodel bone even in old age?
3. What are the differences between spongy and compact bone and do these relate to the strength of long bones (i.e. radius, femur, etc.)?
4. How does the cross sectional area of a long bone relate to the mechanical properties of that bone?
5. How does aging affect the following:
   a. general properties of bone
   b. long bones
   c. vertebral bodies

ANSWERS

1. Tensile forces are resisted by the type I collagen found in bone especially in the laminae of the osteons where the collagen fibers in one lamina is arranges at an acute angle to the fibers in the adjacent lamina. Compression forces are resisted by the mineralized ground substance of the bone which contains mostly calcium phosphate and calcium carbonate.
2. The reasons to remove and lay down new bone throughout the lifespan are 1) repair damage to bone due to microfractures, 2) replenish osteocytes to maintain the organic and inorganic component of the bone, 3) adapt bone to changes in stress, 4) make calcium stored in bone available to the body and 5) remodeling of a bony callus formed during healing of a fracture.
3. Spongy bone is a highly branching network of thin spiny processes while compact bone is solid and consists of long columns of osteons. Spongy bone is concentrated at ends of long bones and compact bone along the shaft. Spongy bone is much weaker in compression and tension than compact bone. Thus areas rich in spongy bone but little compact bone as those at the distal and proximal ends of most long bones would be weaker than the shaft of the long bones which is mainly compact bone with little spongy bone.

4. The cross sectional area (thickness) is directly related to the strength of resistance to compression, bending and torsion of long bones. The larger the cross sectional area the strong the bone is to these forces. However, cross sectional area is inversely related to deformation (strain). The larger the cross sectional area, the less the bone will deform. The cross sectional area for tension differs from the above relationship in that those bones with small cross sectional areas are stronger in resisting tension than bones with large cross sectional areas. An explanation for this difference may be that bones with large cross sectional areas also have a hollow medullary center where as bones with small cross sectional areas are more like solid rods. When tension is applied, the compressive stresses acting at the hollowed out shaft may cause a collapse of this tubular structure resulting in bones with large cross sectional areas breaking.
5. a. In general, bones in males and females are at maximum strength between ages 20–29 but then strength slowly decreases after age 30. This decrease continues after age 40 as osteoblast activity decreases. Before age 50, the mechanical properties of bones are similar in males and females but after age 50, the strength and amount of trabecular bone decreases more rapidly in females than males.
   b. The tensile and bending properties of long bones decrease by about 25% after age 50 while the compressive and torsional properties decrease less (15%).
   c. The tensile properties of vertebral bodies decrease by less than 20% with age but compressive properties decrease markedly by 40–50%. Torsional properties show a large decrease in breaking moment (40%) but only about a 20% decrease in strength and angle of a twist.

Study Questions (Muscle)

1. How are slow and fast twitch muscle fibers different in force production, fatigability, and response to immobilization?
2. How does the arrangement of muscles fibers in parallel differ in force production and displacement from muscle fibers in a pennate arrangement?
3. What is the Maxwell's model and how does it relate to muscle action and muscle weakness?
4. How does the length tension curve relate to exercise and Maxwell's model?
5. How does the force-velocity relationship differ between concentric and eccentric types of muscle contraction?
6. How is muscle affected by aging?

ANSWERS

1. Slow twitch fibers have low force production and a low fatigue rate (can hold a contraction for a very long time). With non-athletes and endurance athletes, slow twitch fibers are mainly affected with immobilization. Fast fatigable fibers have very high force production and a high fatigue rate (fatigue rapidly). In athletes in explosive type movements, the fast fatigable fibers are the ones that a affected the most. Fast fatigue resistant fibers show a force production and fatigue rating between slow twitch and fast fatigable fibers. In endurance athletes, the fast fatigue resistant and slow twitch fibers are mainly affected.
2. With the fibers in parallel, the pull of the fibers is in line with the pull of the tendon. Thus, any shortening of the fibers will produce a force that is entirely directed along the line of action of the muscle and the distance the fibers shorten will be transmitted entirely to the tendon for movement. With fibers in a pennate arrangement, the pull of the fibers is at an angle to the tendon. In this arrangement, only part of the force produced by the shortening pinnate fibers is directed to the tendon for movement. Further, because of the angulations of the fibers, only part of the distance traveled by the fibers during contraction is directly applied to the tendon for movement.

3. Maxwell's model describes a relatively simple but very useful mechanism for muscle action. It contains three elements: 1) a contractile element which is the actin and myosin interaction, 2) a series elastic element which is the tendon, and 3) a parallel elastic element which is the connective tissue components of the muscle. The model shows that the initial shortening of the contractile element tenses the series and parallel elements but will not produce movement until these elastic elements are tight enough to transmit the force for movement. Thus, if a muscle is weak, it may be able to contract but only enough to tense the elastic elements and not enough to produce movement. This inability to produce movement can be worsened if the muscle is in a slackened position initially.
4. The amount of tension a muscle fiber can produce is related to its sarcomere length. A stretched sarcomere or a shortened sarcomere produces less tension than a sarcomere at rest with a length between 2.0–2.25 micrometers. If a muscle is in a slackened position, the shortening contraction of the contractile element must take up the slack, tightening the elastic components of the muscle. Once the elastic components are tight then move can occur. However, in this example, the shortening of the contractile element would shorten sarcomere length and reduce the amount of tension that the muscle could produce. If this is an already weak muscle, the process of taking up the slack and tensing the elastic components of the muscle may take up all the tension this muscle can produce. As a result, there is contraction, but no movement. By placing the muscle in a non-slack position so that the elastic elements and the muscle are tight, the tension produced by a weak muscle may result in movement rather than simply taking up slack.
5. With concentric contraction, the velocity of muscle shortening decreases as the load increases. So with light loads, muscle can concentrically contract more rapidly than with heavier loads. With eccentric contraction, the velocity of contraction increases as loads increase. So with heavy loads, the velocity of contraction is more rapid than with lighter loads.
6. After age 30–40, there is 1) a decrease in muscle fiber number, especially the number of fast twitch fibers, 2) a decrease in isometric strength, and 3) a decrease in muscle contraction velocity.

Study Questions (Nerve)

1. How can nerve fibers in a peripheral nerve be damaged when tension is applied to the nerve?
2. How can a nerve be damaged in compression?
3. Why is nerve regeneration so often unsuccessful in a mixed peripheral nerve?
4. What are the effects of aging on non-pathological peripheral nerve?

ANSWERS

1. Tension can damage nerve fibers in the following ways: a) A tensile load to a peripheral nerves produces compression, tensile and shear stresses and strains to nerve fibers which mechanically can cause physical damage to the fibers, b) A tensile load elongates the peripheral nerve which increases the hydrostatic pressure within the nerve causing compression and damage to nerve fibers, and c) A tensile load elongates the peripheral nerve and stretches the blood vessels to the nerve which can reduce and even stop blood flow to the nerves depending on the amount of elongation. As nervous tissue, including nerve fibers, are very sensitive to oxygen levels, a decrease in blood flow can damage these delicate nerve fibers.
2. Peripheral nerve compression most often results from either circumferential compression or lateral compression. With circumferential compression, the forces are applied to the circumference of the nerve, like a blood pressure cuff applies compression to the circumference of the arm. This type of compression mainly damages fibers circumferential along the periphery of the peripheral nerve producing what is called the "edge effect". With lateral compression, the forces are applied at opposite sides of the peripheral nerve, like squeezing or sandwiching the nerve between to structures. In lateral compression, nerve fiber damage is directly under the contact areas where the forces are applied.
3. A main factor that affects regeneration of all nerves is the availability of the Schwann cells to form tubes along the channels of degenerated nerve fibers to guide regenerating nerve sprouts back to the original motor or sensory innervation site. With mixed nerves, motor and sensory nerve fibers are intermingled together. This arrangement makes it more difficult for interconnecting a motor sprout with a tube from a degenerated motor nerve or a sensory sprout with a sensory tube. Nerve sprouts from a regenerating motor nerve fiber can just an easily enter a tube formed from a degenerated sensory nerve fiber as a sensory sprout can enter a tube formed from a motor nerve. If a regenerating sprout enters the incorrect motor or sensory tube, the sprout will degenerate and innervation lost.
4. The mechanical properties of peripheral nerve change little with age when there is no pathology present that affects peripheral nerve. However, nerve conduction velocity does decrease. Normal nerve conduction velocity is 50–70 m/sec. After age 60, there is usually a decrease of up to 10 m/sec.

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