FIFTH EDITION

DUTTON'S ORTHOPAEDIC

Examination, Evaluation, and Intervention

MARK DUTTON

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NOTICE

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Mark Dutton, PT

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For my parents,
Ron and Brenda, who have always helped, guided, and inspired me
and to my two daughters, Leah and Lauren, who provide me with such joy.

Your Legacy

Will you have earned the respect of your peers and the admiration of your critics?
Will you have acted humbly during success and gracefully in the face of adversity?
Will you be remembered for how often you brought smiles to the hearts of others?
Will you have looked for the very best, and done your utmost to build worth, in others?
Will you have left this world a better place by the life you have lived?

Modified from The Legacy You Leave ©2000 by Rick Beneteau

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Preface

The fifth edition of this book is an update of information and bibliography provided in the previous versions together with a reorganization of various chapters.

The 2017 Global Burden of Disease study revealed that musculoskeletal disorders are the second biggest contributor to disability worldwide.[1] The United States currently spends more money on healthcare per person than any other country in the world, with current projections indicating that the United States will spend 20% of the gross domestic product on healthcare by the year 2019.[1] As the population continues to age, the treatment of musculoskeletal conditions, and their subsequent expenses, will also increase. This financial burden will place an increasing pressure on the orthopaedic clinician to provide value for money—the achievement of a health outcome relative to the costs incurred. Gone are the days when a clinician can rely on an expensive shotgun approach to treatment. Instead, the emphasis must now be placed on outcomes such as patient satisfaction and accurate measures of clinical outcomes, for it is the consistent measurement and reporting of clinical outcomes that are the most powerful tools in moving toward a value-based system.[2]

The APTA's current vision statement, "Transforming society by optimizing movement to improve the human experience," highlights the fact that the "physical therapy profession will define and promote the movement system as the foundation for optimizing movement to improve the health of society."[2]

To that end, this book aims to provide the reader with a systematic and evidence-based approach to the examination and intervention of the orthopaedic patient from the viewpoint of an expert on the movement system. Such an approach must be eclectic because no single method works all of the time. Thus, this book attempts to incorporate the most reliable concepts currently available.

I hope that this book will be the best available textbook, guide, review, and reference for healthcare students and clinicians involved in the care of the orthopaedic population.

Mark Dutton, PT

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Comments about this book may be sent to me at pt@mcgraw-hill.com.

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- To the countless clinicians throughout the world who continually strive to improve their knowledge and clinical skills.

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Introduction

"The very first step towards success in any occupation is to become interested in it."

—Sir William Osler (1849–1919)

Until the beginning of the last century, knowledge about the mechanism of healing and the methods to decrease pain and suffering were extremely limited. Although we may scoff at many of the interventions used in the distant past, many of the interventions we use today, albeit less radical, have still to demonstrate much more in the way of effectiveness. That may soon change with the recent emphasis within many healthcare professions on evidence-based clinical practice. The process of evidence-based practice is outlined in Table I-1. When combining clinical expertise with the best available external clinical evidence, clinicians can make informed decisions regarding patient management, including the selection and interpretation of the most appropriate evaluation procedures. Also, intervention strategies based on the best available evidence will have a greater likelihood of success with the least associated risk.

TABLE I-1	The Process of Evidence-Based Practice
1.	Identify the patient problem. Derive a specific question.
2.	Search the literature.
3.	Appraise the literature.
4.	Integrate the appraisal of literature with your clinical expertise, experience, patient values, and unique circumstances.
5.	Implement the findings.
6.	Assess outcome and reappraise.

Data from Sackett DL, Strauss SE, Richardson WS, et al. *Evidence Based Medicine: How to Practice and Teach EBM.* 2nd ed. Edinburgh, Scotland: Churchill Livingstone; 2000.

The goal of every clinician should be to enhance patient/client satisfaction, increase efficiency, and decrease unproven treatment approaches. The management of the patient/client is a complex process involving an intricate blend of experience, knowledge, and interpersonal skills. Obtaining an accurate diagnosis requires a systematic and logical approach. Such an approach should be eclectic because no single method works all of the time. For any intervention to be successful, an accurate diagnosis must be followed by a carefully planned and specific rehabilitation program to both the affected area and its related structures. In this book, great emphasis is placed on the appropriate use of manual techniques and therapeutic exercise based on these considerations. Electrotherapeutic and thermal/cryotherapeutic modalities should be viewed as adjuncts to the rehabilitative process. Please go to www.accessphysiotherapy.com, for numerous video clips of manual techniques and therapeutic exercises, which the reader is encouraged to view. The following icon is used throughout the text to indicate when such clips are available. **[VIDEO]**

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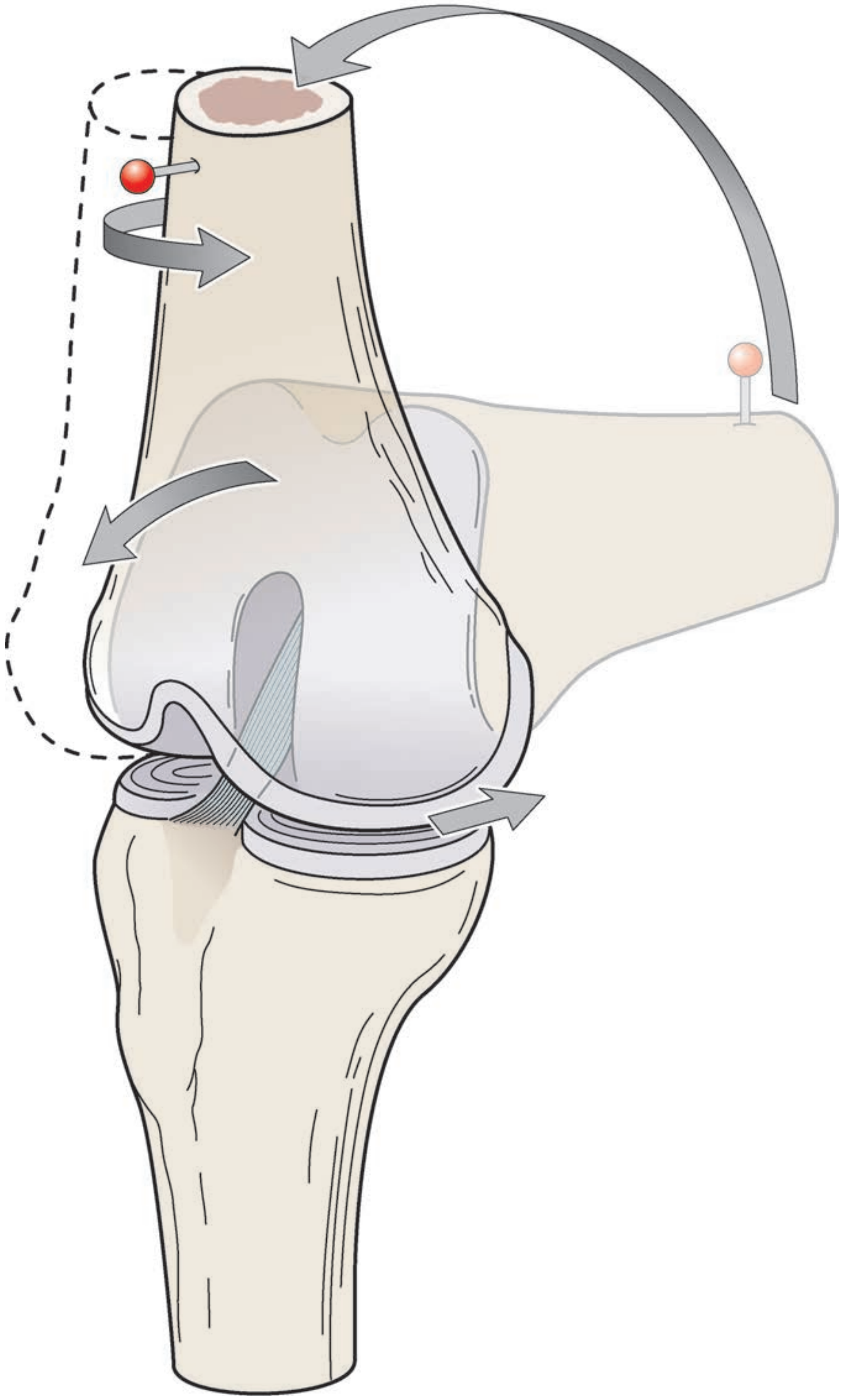
SECTION I ANATOMY



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CHAPTER 1

The Musculoskeletal System

CHAPTER OBJECTIVES

At the completion of this chapter, the reader will be able to:

1. Describe the various types of biological tissue of the musculoskeletal system.
2. Describe the tissue mechanics and structural differences and similarities between muscle, tendons, fascia, and ligaments.
3. Describe the different types of joints and their various characteristics.
4. Define the various terminologies used to describe the joint position, movements, and relationships.
5. Give definitions for commonly used biomechanical terms.
6. Describe the different planes of the body.
7. Define the body's center of gravity and its location.
8. Describe the different axes of the body and the motions that occur around them.
9. Define the terms *osteokinematic motion* and *arthrokinematic motion*.
10. Differentiate between the different types of motion that can occur at the joint surfaces.
11. Describe the basic biomechanics of joint motion in terms of their concave–convex relationships.
12. List the different types of levers found within the body and provide examples of each.
13. Describe the difference between a closed kinematic chain and an open kinematic chain and how each can influence an exercise prescription.
14. Define the terms *close-packed* and *open-packed* and the significance of each.

OVERVIEW

The correct embryonic development of the musculoskeletal system requires a coordinated morphogenesis of the fundamental tissues of the body. Throughout the human body, there are four major types of tissues:

- Epithelial. Epithelial tissue covers all internal and external body surfaces and includes structures such as the skin and the inner lining of the blood vessels.
- Connective. Connective tissue (CT) includes four different classes: CT proper, bone, cartilage, and blood tissue. In the embryo, muscle tissue and its fascia form as a differentiation of the paraxial mesoderm that divides into somites on either side of the neural tube and notochord. The cartilage and bone of the vertebral column and ribs develop from the sclerotome, which is the anterior (ventral) part of the somite.[1,2] The dermomyotome, which is the posterior (dorsal) part of the somite, gives rise to the overlying dermis of the back and the skeletal muscles of the body and limbs.[2] CT provides structural and metabolic support for other tissues and organs of the body.
- Muscle. Muscles are classified functionally as either voluntary or involuntary, and structurally as either smooth, striated (skeletal), or cardiac. There are approximately 430 skeletal muscles in the body, each of which can be considered anatomically as a separate organ. Of these 430 muscles, about 75 pairs provide the majority of body movements and postures.[2]
- Nervous. Nervous tissue provides a two-way communication system between the central nervous system (brain and spinal cord) and muscles, sensory organs, and various systems (see Chapter 3).

CONNECTIVE TISSUE

CT proper has a loose, flexible matrix, called *ground substance*. The most common cell within CT proper is the fibroblast. Fibroblasts produce collagen, elastin, and reticular fibers:

- Collagen is a group of naturally occurring proteins. The collagens are a family of extracellular matrix (ECM) proteins that play a dominant role in maintaining the structural integrity of various tissues and in providing tensile strength to tissues. The ECM is formed from glycosaminoglycan (GAG) subunits that are long polysaccharide chains containing amino sugars and are strongly hydrophilic to allow rapid diffusion of water-soluble molecules and the easy migration of cells. Proteoglycans, which are a major component of the ECM, are macromolecules that consist of a protein backbone to which the GAGs are attached. There are two types of GAGs: chondroitin sulfate and keratin sulfate.[2] Glycoproteins, another component of the ECM, consist of fibronectin and thrombospondin and function as adhesive structures for repair and regeneration.[2,3]
- Elastic fibers, as their name suggests, are composed of a protein called *elastin*, which provides elastic properties to the tissues in which it is situated.[4] Elastin fibers can stretch, but they normally return to their original length when the tension is released. Thus, the elastic fibers of elastin determine the patterns of distention and recoil in most organs, including the skin and lungs, blood vessels, and CT. Bundles of collagen and elastin combine to form a matrix of CT fascicles. This matrix is organized within the primary collagen bundles as well as between the bundles that surround them.[2]
- Reticular fibers are composed of a type of collagen that is secreted by reticular cells. These fibers crosslink to form a fine meshwork, called reticulin, which acts as a supporting mesh in bone marrow, the tissues and organs of the lymphatic system, and the liver.

The various characteristics of collagen differ depending on whether it is loose or dense collagen. The anatomic and functional characteristics of loose and dense collagen are summarized in Table 1-1. Collagenous and elastic fibers are sparse and irregularly arranged in loose CT but are tightly packed in dense CT.

The various types of CT, as they relate to the musculoskeletal system, are described in the following sections.

Fascia

Fascia, for example, the thoracolumbar fascia and the plantar fascia, is viewed as a loose CT that provides support and protection to a joint, and acts as an interconnection between tendons, aponeuroses, ligaments, capsules, nerves, and the intrinsic components of muscle.[2] Fascia may be categorized as fibrous or nonfibrous, with the fibrous components consisting mainly of collagen and elastin fibers, and the nonfibrous portion consisting of amorphous ground substance.[2] Three different types of fascia have been identified, namely, superficial, deep, and visceral. Various three-dimensional biomechanical models of the human fascial system have been developed, which correlate dysfunctional movement with various interrelated abnormal amounts of tension throughout the network of fascia. In particular, deep fascia has been implicated in being involved with the deep venous return, in having a possible role in proprioception, and responding to mechanical traction induced by muscular activity in different regions.[5] However, there is still little evidence to justify such claims. Histological studies of deep fascia in the limbs show that it consists of elastic fibers and undulated collagen fibers arranged in layers.[6] Each collagen layer is aligned in a different direction, and this permits a certain degree of stretch as well as a capacity to recoil.

Tendons

Tendons are dense, regularly arranged CTs that attach muscle to the bone at each end of the muscle. At first glance, tendons appear to be very simple rope-like structures. However, closer inspection reveals that the structure and material properties of tendons are not universal, and therefore, each tendon cannot be treated in the same manner as another. Medical imaging today allows clinicians and researchers to more precisely characterize the tendon structures that provide the tendon with its physiological capacity. The predominant cell type found in a tendon is the tenocyte, a structure that is sensitive to the mechanical loading environment and is capable of controlling tendon structure.[7]

The collagen-forming triple helices (tropocollagen) of the tendon pack together to form microfibrils, which interdigitate to form fibrils, which coalesce to form fibers, which combine to form fascicles, which in turn are bundled together to

TABLE 1-1 Loose and Dense Collagen

Joint Type	Anatomic Location	Fiber Orientation	Mechanical Specialization
Dense irregular connective tissue	Composes the external fibrous layer of the joint capsule, forms ligaments, bone, aponeuroses, and tendons	Parallel, tightly aligned fibers	Ligament: binds bones together and restrains unwanted movement at the joints; resists tension in several directions Tendon: attaches muscle to bone
Loose irregular connective tissue	Found in capsules, muscles, nerves, fascia, and skin	Random fiber orientation	Provides structural support

form a tendon.[8] Tendon accommodates a high-tensile loading environment through a multiscale structural design: polypeptide hydrogen bonds create the strong triple-helical structure of a single collagen molecule; covalent bonds cross-linking between collagen molecules (fibrils) allow collagen fibers to withstand enormous forces; collagen fibers are bundled together within an ECM (fascicles) that limits the extent of neurovascular infiltration and maximizes mechanical integrity; and bundling of fascicles into primary, secondary, and tertiary fiber bundles reduces the impact of local fibril failure on the whole tissue.[9,10] The position and length of tendons enable the muscle belly to be an optimal distance from the joint upon which it is acting. This creates space, but also allows the tendon to work like a lever arm (see Levers later), moving the point of action away from the center of rotation (COR), thereby reducing the forces required for movement.[7] Also, due to their design, tendons provide a graduated change in material characteristics, which minimizes the development of areas of stress concentration where failure would likely occur.

Tendons must be sufficiently stiff to enable efficient force transfer from the muscles to produce joint motion, but they must also incorporate a degree of elasticity to enable them to stretch and store elastic energy.[7] Other tendons must modulate muscle contraction with extreme precision to allow it to perform intricate activities such as writing.[7] The thickness of each tendon varies but is proportional to the size of the muscle from which it originates. Vascularity within the tendon is relatively sparse, but the extent of vascularity is not universally the same, and those tendons with less vascularity may be more vulnerable to both progressive degeneration and a reduced healing potential.[11] Within the fascicles of tendons, which are held together by loose CT called *endotenon*, the collagen components are oriented in a unidirectional way. Endotenon contains blood vessels, lymphatics, and nerves, and permits longitudinal movements of individual fascicles when tensile forces are applied to the structure. The CT surrounding groups of fascicles, or the entire structure, is called the *epitenon*. The epitenon contains the vascular, lymphatic, and nerve supplies to the tendon. A peritendinous sheath (paratenon), which is composed of loose areolar CT in addition to sensory and autonomic nerve fibers, surrounds the entire tendon.[12] This sheath consists of two layers: an inner (visceral) layer and an outer (parietal) layer with occasional connecting bridges (mesotenon). The paratenon is richly vascularized and is responsible for a significant portion of the blood supply to the tendon via a series of transverse vincula, which function as passageways for blood vessels to reach the tendon. In addition, the blood supply to the tendon comes from two other sources: the musculotendinous junction (MTJ) and the osseous insertion.

CLINICAL PEARL

Paratenon lined with synovial cells of a variable structure is called tenosynovium, while one with a double layer sheath without synovial cells is known as tenovagium.[2]

The mechanical properties of tendon come from its highly oriented structure. Normal tendons display viscoelastic mechanical properties that confer time- and rate-dependent effects on the tissue. Specifically, tendons are more elastic at lower strain rates and stiffer at higher rates of tensile loading (see Chapter 2). Tendons deform less than ligaments under an applied load and are thus able to transmit the load from muscle to bone.[7]

CLINICAL PEARL

- At low rates of loading, tendons are more viscous or ductile and, consequently, can absorb more energy compared to high loading rates.[13]
- At high rates of loading, tendons become more brittle and absorb less energy, but they are more effective at transferring loads.[13]

Therefore, tendon load can be increased in one of two ways when prescribing exercise: by the external load or by the speed of movement.[13]

Patients with tendinopathy display tendons that are thicker, but with reduced energy-storing capacity, meaning that for the same load, their tendons exhibit higher strains than those of healthy individuals.[14] Material and structural properties of the tendon increase from birth through maturity and then decrease from maturity through old age.[8] Although tendons withstand strong tensile forces well, they resist shear forces less well and provide little resistance to a compression force (see Chapter 2). In addition to the primary load-bearing part of the tendon, there is an extensive network of septae (endotendon), where the nerves and vessels are mainly located.[14]

A tendon can be divided into the following three main sections[15]:

- The bone–tendon junction. At most tendon–bone interfaces, the collagen fibers insert directly into the bone in a gradual transition of material composition. The physical junction of tendon and bone is referred to as an enthesis[16] and is an interface that is vulnerable to acute and chronic injury.[7,17] One role of the enthesis is to absorb and distribute the stress concentration that occurs at the junction over a broader area.
- The tendon midsubstance. Overuse tendon injuries can occur in the midsubstance of the tendon, but not as frequently as at the enthesis.
- MTJ. The MTJ is the site where the muscle and tendon meet. The MTJ comprises numerous interdigitations between muscle cells and tendon tissue, resembling interlocked fingers.

Ligaments

Skeletal ligaments are fibrous bands of dense CT that connect bones across joints. Ligaments can be named for the bones into which they insert (coracohumeral), their shape (deltoid of the ankle), or their relationships to each other (cruciate).[18] The gross structure of a ligament varies according to location (intra-articular or extra-articular, capsular) and function.[19] Ligaments, which appear as dense white bands or cords of

CT, are composed primarily of water (approximately 66%) and collagen (largely type I collagen [85%], but with small amounts of type III) making up most of the dry weight.[2] The collagen in ligaments has a less unidirectional organization than it does in tendons, but its structural framework still provides stiffness (resistance to deformation—see Chapter 2). Small amounts of elastin (1% of the dry weight) are present in ligaments, with the exception of the ligamentum flavum and the nuchal ligament of the spine, which contain more. The cellular organization of ligaments makes them ideal for sustaining tensile loads and for tightening or loosening in different joint positions. At the microscopic level, closely spaced collagen fibers (fascicles) are aligned along the long axis of the ligament and are arranged into a series of bundles that are delineated by a cellular layer, the endoligament, and the entire ligament is encased in a neurovascular biocellular layer referred to as the epiligament.[18] Ligaments contribute to the stability of joint function by preventing excessive motion, acting as guides or checkreins to direct motion, and providing proprioceptive information for joint function through sensory nerve endings (see Chapter 3) and as attachments to the joint capsule.[2] Many ligaments share functions. For example, while the anterior cruciate ligament of the knee is considered to be the primary restraint to anterior translation of the tibia relative to the femur, the collateral ligaments and the posterior capsule of the knee also help in this function (see Chapter 20).[18] The vascular and nerve distribution to ligaments is not homogenous. For example, the middle of the ligament is typically avascular, while the proximal and distal ends enjoy a rich blood supply. Similarly, the insertional ends of the ligaments are more highly innervated than the midsubstance.

Cartilage

Cartilage tissue exists in three forms: hyaline, elastic, and fibrocartilage.

- Hyaline cartilage, also referred to as articular cartilage, covers the ends of long bones and permits almost frictionless motion to occur between the articular surfaces of a diarthrodial (synovial) joint. Articular cartilage is a highly organized viscoelastic material composed of cartilage cells called *chondrocytes*, water, and an ECM.

CLINICAL PEARL

Chondrocytes are specialized cells that are responsible for the development of cartilage and the maintenance of the ECM. Chondrocytes produce aggrecan, link protein, and hyaluronan, all of which are extruded into the ECM, where they aggregate spontaneously.[2] The aggrecan forms a strong, porous-permeable, fiber-reinforced composite material with collagen. The chondrocytes sense mechanical changes in their surrounding matrix through intracytoplasmic filaments and short cilia on the surface of the cells.[19]

- Articular cartilage, the most abundant cartilage within the body, is devoid of any blood vessels, lymphatics, and nerves.[2] Most of the bones of the body form first as hyaline cartilage, and later become bone in a process called *endochondral ossification*. Articular cartilage functions to distribute the joint forces over a large contact area, thereby dissipating the forces associated with the load. This distribution of forces allows the articular cartilage to remain healthy and fully functional throughout decades of life. The normal thickness of articular cartilage is determined by the contact pressures across the joint—the higher the peak pressures, the thicker the cartilage.[19] For example, the patellar has the thickest articular cartilage in the body.
- Articular cartilage may be grossly subdivided into four distinct zones with differing cellular morphology, biomechanical composition, collagen orientation, and structural properties, as follows:
  - *The superficial zone.* The superficial zone, which lies adjacent to the joint cavity, comprises approximately 10–20% of the articular cartilage thickness and functions to protect deeper layers from shear stresses. The collagen fibers within this zone are packed tightly and aligned parallel to the articular surface. This zone is in contact with the synovial fluid and handles most of the tensile properties of cartilage.
  - *The middle (transitional) zone.* In the middle zone, which provides an anatomic and functional bridge between the superficial and deep zones, the collagen fibril orientation is obliquely organized. This zone comprises 40–60% of the total cartilage volume. Functionally, the middle zone is the first line of resistance to compressive forces.
  - *The deep or radial layer.* The deep layer comprises 30% of the matrix volume. It is characterized by radially aligned collagen fibers that are perpendicular to the surface of the joint and have a high proteoglycan content. Functionally the deep zone is responsible for providing the greatest resistance to compressive forces.
  - *The tidemark.* The tidemark distinguishes the deep zone from the calcified cartilage, the area that prevents the diffusion of nutrients from the bone tissue into the cartilage.
- Elastic (yellow) cartilage is a very specialized CT, primarily found in locations such as the outer ear and portions of the larynx.
- Fibrocartilage, also referred to as white cartilage, functions as a shock absorber in both weight-bearing and non–weight-bearing joints. Its large fiber content, reinforced with numerous collagen fibers, makes it ideal for bearing large stresses in all directions. Fibrocartilage is an avascular, alymphatic, and aneural tissue and derives its nutrition by a double-diffusion system.[2] Examples of fibrocartilage include the symphysis pubis, the intervertebral disk, and the menisci of the knee.

Bone

Bone is a highly vascular form of CT, composed of collagen, calcium phosphate, water, amorphous proteins, and cells. It is

TABLE 1-2	General Structure of Bone		
Site	**Comment**	**Conditions**	**Result**
Epiphysis	Mainly develops under pressure Apophysis forms under traction Forms bone ends Supports articular surface	Epiphyseal dysplasias Joint surface trauma Overuse injury Damaged blood supply	Distorted joints Degenerative changes Fragmented development Avascular necrosis
Physis	Epiphyseal or growth plate Responsive to growth and sex hormones Vulnerable prior to growth spurt Mechanically weak	Physeal dysplasia Trauma Slipped epiphysis	Short stature Deformed or angulated growth or growth arrest
Metaphysis	Remodeling expanded bone end Cancellous bone heals rapidly Vulnerable to osteomyelitis Affords ligament attachment	Osteomyelitis Tumors Metaphyseal dysplasia	Sequestrum formation Altered bone shape Distorted growth
Diaphysis	Forms shaft of bone Large surface for muscle origin Significant compact cortical bone Strong in compression	Fractures Diaphyseal dysplasias Healing slower than at metaphysis	Able to remodel angulation Cannot remodel rotation Involucrum with infection Dysplasia gives altered density and shape

Reproduced with permission from Reid DC. *Sports Injury Assessment and Rehabilitation.* New York, NY: Churchill Livingstone; 1991.

the most rigid of the CTs (Table 1-2). Despite its rigidity, bone is a dynamic tissue that undergoes constant metabolism and remodeling. The collagen of bone is produced in the same manner as that of ligament and tendon but by a different cell, the osteoblast. At the gross anatomical level, each bone has a distinct morphology comprising both cortical bone and cancellous bone. Cortical bone is found in the outer shell. Cancellous bone is found within the epiphyseal and metaphyseal regions of long bones, as well as throughout the interior of short bones. Skeletal development occurs in one of two ways:

- Intramembranous ossification. Mesenchymal stem cells within the mesenchyme or the medullary cavity of a bone initiate the process of intramembranous ossification. This type of ossification occurs in the cranium and facial bones and, in part, the ribs, clavicle, and mandible.
- Endochondral ossification. The first site of ossification occurs in the primary center of ossification, which is in the middle of the diaphysis (shaft). About the time of birth, a secondary ossification center appears in each epiphysis (end) of long bones. Between the bone formed by the primary and secondary ossification centers, cartilage persists as the epiphyseal (growth) plates between the diaphysis and the epiphysis of a long bone. This type of ossification occurs in the appendicular and axial bones.

The periosteum is formed when the perichondrium, which surrounds the cartilage, becomes the periosteum. Chondrocytes in the primary center of ossification begin to grow (hypertrophy) and begin secreting alkaline phosphatase, an enzyme essential for mineral deposition. Calcification of the matrix follows, and apoptosis (a type of cell death involving a programmed sequence of events that eliminates certain cells) of the hypertrophic chondrocytes occurs. This creates cavities within the bone. The exact mechanism of chondrocyte hypertrophy and apoptosis is currently unknown. The hypertrophic chondrocytes (before apoptosis) also secrete a substance called *vascular endothelial cell growth factor* that induces the sprouting of blood vessels from the perichondrium. Blood vessels forming the periosteal bud invade the cavity left by the chondrocytes and branch in opposite directions along the length of the shaft. The blood vessels carry osteoprogenitor cells and hemopoietic cells inside the cavity, the latter of which later form the bone marrow. Osteoblasts, differentiated from the osteoprogenitor cells that enter the cavity via the periosteal bud, use the calcified matrix as a scaffold and begin to secrete osteoid, which forms the trabecular bone. Osteoclasts, formed from macrophages, break down the spongy bone to form the medullary cavity (bone marrow).

The function of bone is to provide support, enhance leverage, protect vital structures, provide attachments for both tendons and ligaments, and store minerals, particularly calcium. Bones also may serve as useful landmarks during the palpation phase of the examination. The strength of bone is related directly to its density. Of importance to the clinician is the difference between maturing bone and mature bone. The epiphyseal plate or growth plate of a maturing bone can be divided into the following four distinct zones[20]:

- Reserve zone: It produces and stores matrix.
- Proliferative zone: It produces matrix and is the site for longitudinal bone cell growth.
- Hypertrophic zone: It is subdivided into the maturation zone, degenerative zone, and the zone of provisional calcification. It is within the hypertrophic zone that the matrix is prepared for calcification and is here that the matrix is ultimately calcified. The hypertrophic zone is the most susceptible of the zones to injury because of

the low volume of bone matrix and the high amounts of developing immature cells in this region.[2]

- Bone metaphysis: It is the part of the bone that grows during childhood.

Skeletal Muscle Tissue

Skeletal muscles constitute approximately 30–40% of total body mass and have many vital roles such as generation of movement, protection, breathing, thermal regulation, and metabolism.[21] The microstructure and composition of skeletal muscle have been studied extensively. The class of tissue labeled *skeletal muscle* consists of individual muscle cells that work together to produce the movement of bony levers. A single muscle cell is long and cylindrical and is called a *muscle fiber* or *myofiber*. The myofiber is the most important part of skeletal muscle composition,[22] and the integrity and function of a myofiber can be affected by different traumas such as strain, contusion, laceration, immobilization, eccentric-induced muscle damage, ischemia, and others (see Chapter 2).[21] Because the nuclei of the myofibers are terminally postmitotic (i.e., they cannot divide anymore), muscle regeneration is ensured by a population of adult muscle stem cells, named satellite cells.[21,23]

CLINICAL PEARL

Satellite cells are essential to muscle regeneration post injury, and they also contribute to muscle hypertrophy.[21]

All muscles, depending on their size, are made up of thousands and, in some cases, hundreds of thousands of muscle fibers, which are wrapped in a CT sheath called *epimysium* (**Fig. 1-1**). As muscle cells differentiate within the mesoderm, individual myofibers are wrapped in a CT envelope called *endomysium*. Bundles of myofibers, which form a whole muscle (fasciculus), are encased in the *perimysium* (Fig. 1-1). The perimysium is continuous with the deep fascia. This

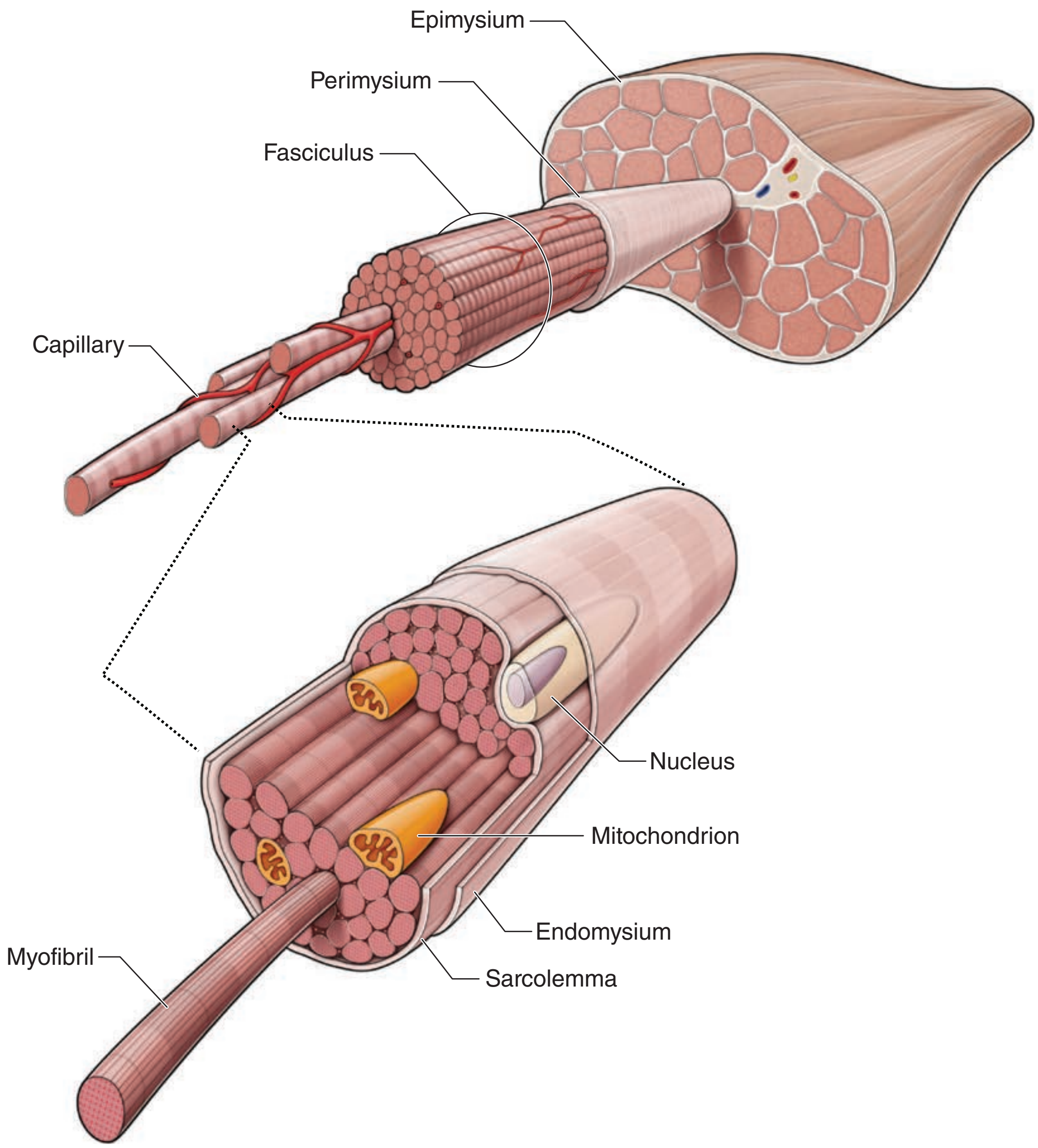

FIGURE 1-1 Microscopic structure of the muscle.

relationship allows the fascia to unite all of the fibers of a single motor unit and, therefore, adapt to variations in form and volume of each muscle according to muscular contraction and intramuscular modifications induced by joint movement.[6] Under an electron microscope, it can be seen that each of the myofibers consists of thousands of *myofibrils* (Fig. 1-1), which extend throughout its length. Myofibrils are composed of sarcomeres arranged in series.[2]

CLINICAL PEARL

The sarcomere (**Fig. 1-2**) is the contractile machinery of the muscle. The graded contractions of a whole muscle occur because the number of fibers participating in the contraction varies. Increasing the force of movement is achieved by recruiting more cells into cooperative action.

All skeletal muscles exhibit four characteristics:

1. Excitability, the ability to respond to stimulation from the nervous system.
2. Elasticity, the ability to change in length or stretch. The tension developed in skeletal muscle can occur passively (stretch) or actively (contraction). When an activated muscle develops tension, the amount of tension present is constant throughout the length of the muscle, in the tendons, and at the sites of the musculotendinous attachments to bone. The tensile force produced by the muscle pulls on the attached bones and creates torque at the joints crossed by the muscle. The magnitude of the tensile force is dependent on a number of factors.
3. Extensibility, the ability to shorten and return to normal length.
4. Contractility, the ability to shorten and contract in response to some neural command.

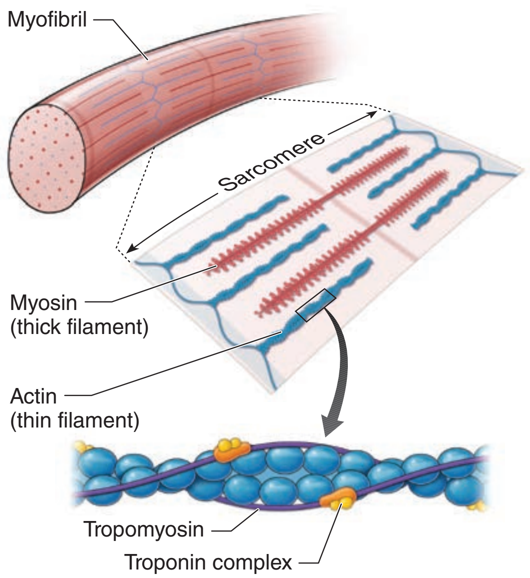

FIGURE 1-2 Troponin and tropomyosin action during a muscle contraction.

One of the most important roles of CT is to mechanically transmit the forces generated by the skeletal muscle cells to provide movement. Each of the myofibrils contains many fibers called *myofilaments*, which run parallel to the myofibril axis. The myofilaments are made up of two different proteins: actin (thin myofilaments) and myosin (thick myofilaments) that give skeletal muscle fibers their striated (striped) appearance (Fig. 1-2). The striations are produced by alternating dark (A) and light (I) bands that appear to span the width of the muscle fiber. The A bands are composed of myosin filaments, whereas the I bands are composed of actin filaments. The actin filaments of the I band overlap into the A band, giving the edges of the A band a darker appearance than the central region (H band), which contains only myosin. At the center of each I band is a thin, dark Z line. A *sarcomere* (Fig. 1-2) represents the distance between each Z line. Each muscle fiber is limited by a cell membrane called a *sarcolemma* (Fig. 1-1). The protein *dystrophin* plays an essential role in the mechanical strength and stability of the sarcolemma and is lacking in patients with Duchenne muscular dystrophy.[21]

CLINICAL PEARL

The sarcoplasm is the specialized cytoplasm of a muscle cell that contains the usual subcellular elements along with the Golgi apparatus, abundant myofibrils, a modified endoplasmic reticulum known as the sarcoplasmic reticulum (SR), myoglobin, and mitochondria. Transverse-tubules (T-tubules) invaginate the sarcolemma, allowing impulses to penetrate the cell and activate the SR.

Structures called *cross-bridges* serve to connect the actin and myosin filaments. Increased synthesis of actin and myosin stimulates new myofibrils that are added to the external layers of the preexisting myofibrils.[24] The myosin filaments contain two flexible, hinge-like regions, which allow the cross-bridges to attach and detach from the actin filament. During contraction, the cross-bridges attach and undergo power strokes, which provide the contractile force. During relaxation, the cross-bridges detach. This attaching and detaching is asynchronous, so that some are attaching while others are detaching. Thus, at each moment, some of the cross-bridges are pulling, while others are releasing.

The regulation of cross-bridge attachment and detachment is a function of two proteins found in the actin filaments: tropomyosin and troponin (Fig. 1-2). Tropomyosin attaches directly to the actin filament, whereas troponin is attached to the tropomyosin rather than directly to the actin filament.

CLINICAL PEARL

Tropomyosin and troponin function as the switch for muscle contraction and relaxation. In a relaxed state, the tropomyosin physically blocks the cross-bridges from binding to the actin. For contraction to take place, the tropomyosin must be moved.