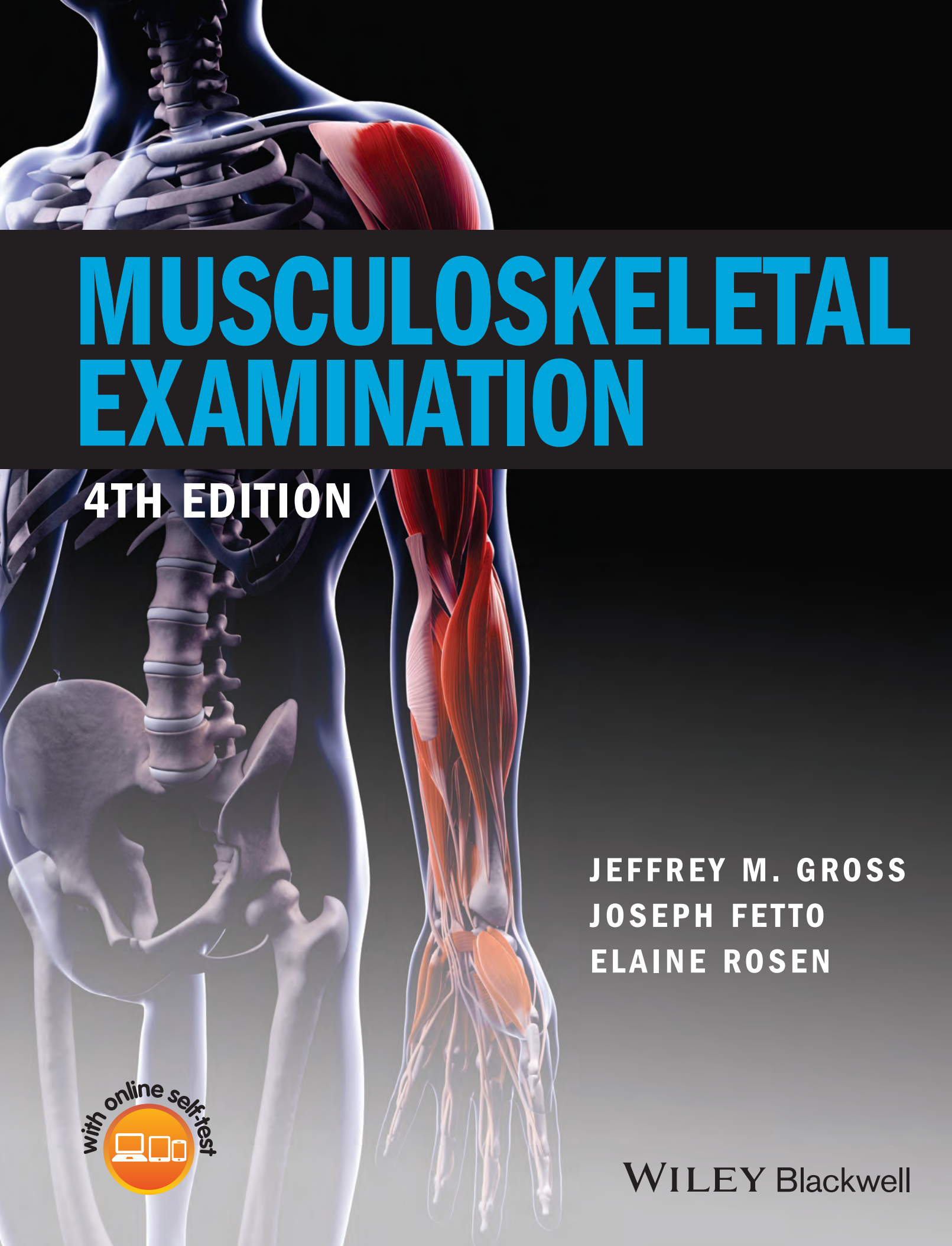

MUSCULOSKELETAL EXAMINATION

4TH EDITION

JEFFREY M. GROSS
JOSEPH FETTO
ELAINE ROSEN

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Musculoskeletal Examination

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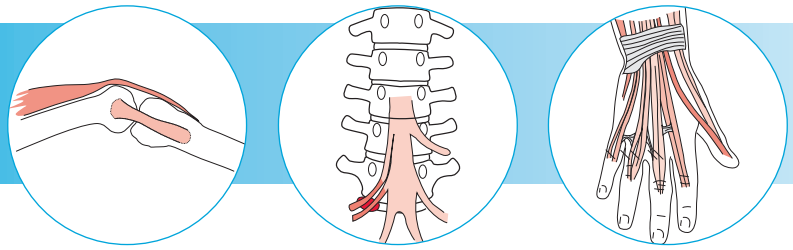

Musculoskeletal Examination

Fourth Edition

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Contents

How to Use This Book

Musculoskeletal Examination is to be used as both a teaching text and a general reference on the techniques of physical examination. This volume represents the joint authoring efforts of a physiatrist, an orthopedic surgeon, and a physical therapist and presents the information in a clear and concise format, free of any professional biases that reflect one specialty's preferences. The importance of this will be seen as we take you through each anatomical region and delineate the basic examination. Included in each chapter are the abnormalities most frequently encountered or noted while performing an examination.

The book is organized into regional anatomical sections including the spine and pelvis, the upper extremity, and the lower extremity. The book opens with two chapters that define the structures of the musculoskeletal system and discuss the basic concepts and parts of the musculoskeletal exam. A final chapter describes the examination of gait.

Each main chapter is organized in an identical manner:

- overview of the anatomical region
- observation of the patient
- subjective examination
- gentle palpation
- trigger points (where applicable)
- active movement testing
- passive movement testing
- physiological movements
- mobility testing
- resistive testing
- neurological examination
- referred pain patterns
- special tests
- radiological views

In Chapter 2, Basic concepts of the physical examination, we provide you with a framework for performing the examination, beginning with observation and ending with palpation. However, in each regional anatomy chapter, palpation follows observation and subjective examination and precedes all other sections. This is deliberate. For reasons of length, we felt it important to discuss each anatomical region and its own special anatomical structures as soon as possible in each chapter. This avoids repetition, gives you the anatomy early in each chapter, and then allows you to visualize each structure as you read the subsequent sections on testing. Hopefully, this will reinforce the anatomy and help you apply anatomy to function and function to the findings of your examination.

Each chapter includes a generous number of original line drawings, many of which are two-color. These provide clear snapshots of how to perform each examination technique. Thirty-two X-rays and MRIs have been included to help you with radiological anatomy. Paradigms and tables provide additional information that will help you understand the how and why of each examination technique.

By using *Musculoskeletal Examination* as a guide and reference, the reader will be able to perform the complete basic examination and understand common abnormalities and their pathological significance. We hope that our readers will gain an appreciation for the intimate relationship between the structure and function of the components of the musculoskeletal system. This understanding should then enable any reader to make a correct diagnosis and a successful treatment plan for each patient.

Acknowledgments

The writing of *Musculoskeletal Examination* would not have been possible without the overwhelming support and understanding of my wife, Elizabeth, and my sons, Tyler and Preston. I also want to thank my parents, Malcolm and Zelda Gross, for their guidance and efforts on my behalf.

J.G.

Thank you to my wife and family for their understanding, patience, support, and love.

J.F.

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To my business partner and friend, Sandy, for being there whenever I needed her.

To my family for their support, and to my many patients, colleagues, and friends who have helped me grow.

E.R.

About the Companion Website

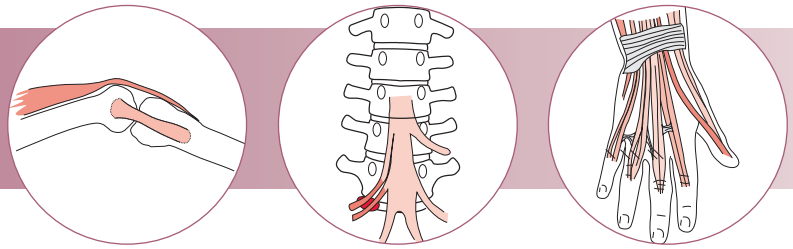
Don’t forget to visit the companion website for this book:

www.wiley.com/go/musculoskeletalexam

There you will find:
Hundred interactive multiple-choice questions to test your learning.
Links to the examination videos mentioned in the book.

Scan this QR code to visit the companion website

CHAPTER 1



Introduction

The intention of this book is to provide the reader with a thorough knowledge of regional anatomy and the techniques of physical examination. A second and equally important intention is to describe a method for the interpretation and logical application of the knowledge obtained from a physical examination.

What Is a Physical Examination?

The physical examination is the inspection, palpation, measurement, and auscultation of the body and its parts. It is the step that follows the taking of a patient history and precedes the ordering of laboratory tests and radiological evaluation in the process of reaching a diagnosis.

What Is the Purpose of the Physical Examination?

The physical examination has two distinct purposes. The first is to localize a complaint, that is, to associate a complaint with a specific region and, if possible, a specific anatomical structure. The second purpose of a physical examination is to qualify a patient's complaints. Qualifying a complaint involves describing its character (i.e., dull, sharp, etc.), quantifying its severity (i.e., visual analog scale; grade I, II, III), and defining its relationship to movement and function.

How Is the Physical Examination Useful?

By relating a patient's complaints to an anatomical structure, the physical examination brings meaning to a patient's history and symptoms.

This, however, presupposes that the clinician possesses a thorough knowledge of anatomy. It also requires a methodology for the logical analysis and application of the information obtained from the patient's history and physical examination. This methodology is derived from a clinical philosophy based on specific concepts. These concepts are as follows:

1. If one knows the structure of a system and understands its intended function, it is possible to predict how that system is vulnerable to breakdown and failure (injury).
2. A biological system is no different from an inorganic system in that it is subject to the same laws of nature (physics, mechanics, engineering, etc.). However, the biological system, unlike the inorganic system, has the potential not only to respond but also to adapt to changes in its environment.

Such concepts lay the foundation for understanding the information obtained on physical examination. They also lead to a rationale for the treatment and rehabilitation of injuries. A correlation of this type of analysis is that it becomes possible to anticipate injuries. This in turn permits proactive planning for the prevention of injuries.

How Does the Musculoskeletal System Work?

The musculoskeletal system, like any biological system, is not static. It is in a constant state of dynamic equilibrium. This equilibrium is termed homeostasis.

As such, when subjected to an external force or stress, a biological system will respond in a very specific manner. Unlike the inorganic system (i.e., an

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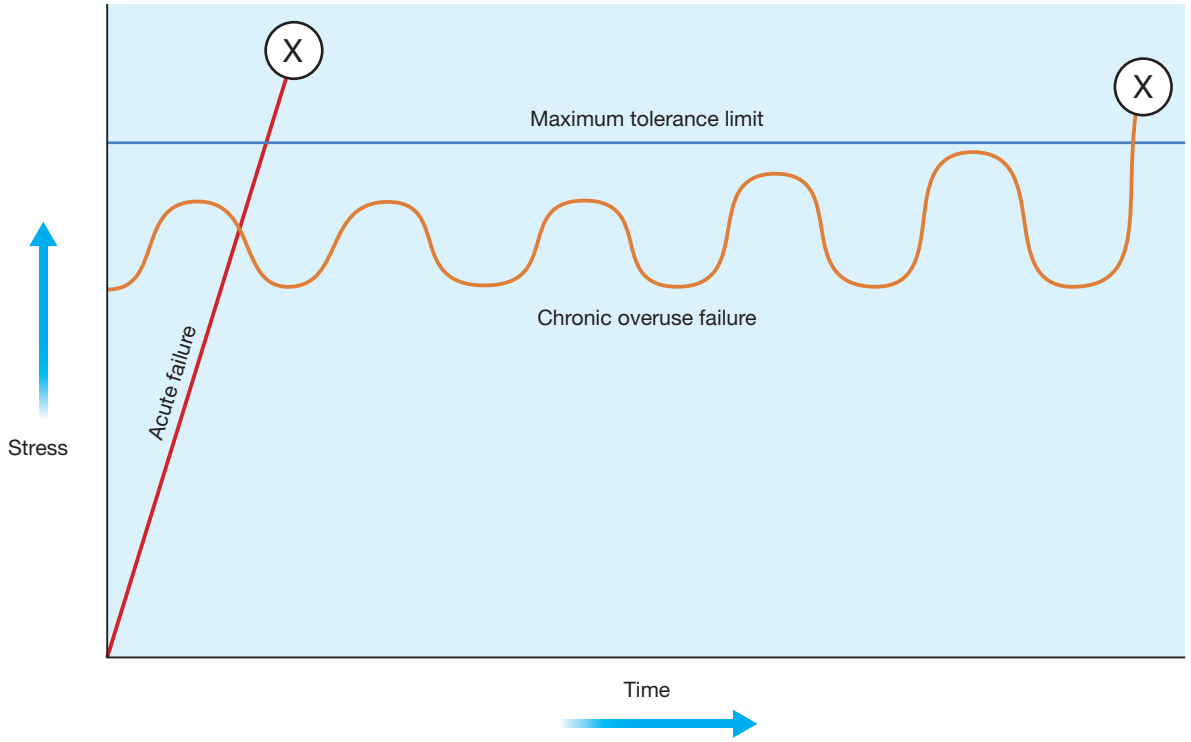


Figure 1.1 Biological systems, like inorganic systems, can fail under one of two modes: an acute single supramaximal stress or repetitive submaximal chronic loading.

airplane wing that is doomed to fail after a predictable number of cycles of load), the biological system will attempt to reestablish an equilibrium state in response to a change that has occurred in its environment. In doing so, the biological system will experience one of three possible scenarios: adaptation (successful establishment of a new equilibrium state without breakdown), temporary breakdown (injury), or ultimate breakdown (death). These scenarios can be expressed graphically. Any system can be stressed in one of the two modes: acute single supratolerance load or chronic repetitive submaximal tolerance load (Figure 1.1). In the first mode, the system that suffers acute failure is unable to resist the load applied. In the second mode, the system will function until some fatigue limit is reached, at which time failure will occur. In the biological system, either failure mode will initiate a protective-healing response, termed the inflammatory reaction. The inflammatory reaction is composed of cellular and humoral components, each of which initiates a complex series of neurological and cellular responses to the injury. An important consequence of the inflammatory reaction is the production of pain. The sole purpose of pain is to bring one's attention to the site of injury. Pain prevents further injury from occurring by causing protective guarding and limited use of the injured structure. The inflammatory response is also characterized by increased vascularity and swelling in the area of injury. These are the causes of the commonly observed physical signs (i.e., redness and warmth) associated with the site of injury.

However, the problem with pain is that although it brings protection to the area of injury (the conscious or unconscious removal of stress from the injured area), and permits healing to take place by removing dynamic stimuli from the biological system, this removal of stimuli (rest) promotes deterioration of a system's tolerance limit to a lower threshold. In this way, when the injury has resolved, the entire system, although "healed," may actually be more vulnerable to reinjury when "normal" stresses are applied to the recently repaired structures. This initiates the "vicious cycle of injury" (Figure 1.2).

Contrary to this scenario is one in which the biological system successfully adapts to its new environment before failure occurs. This situation represents conditioning of a biological system. The result is

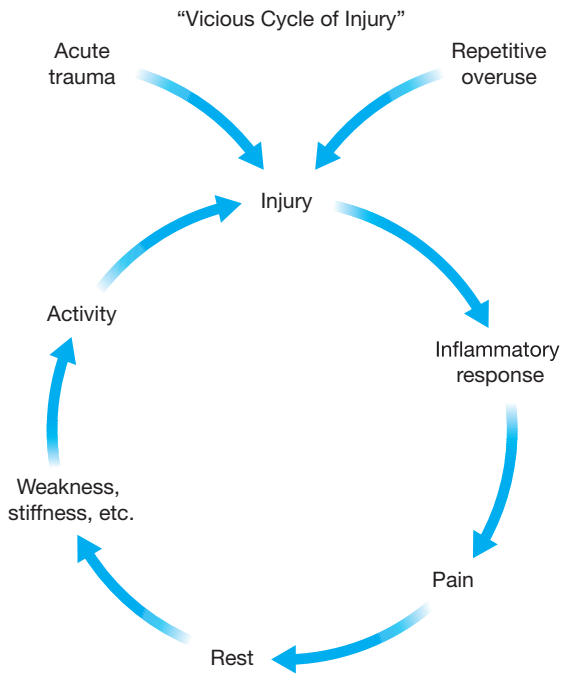


Figure 1.2 The "vicious cycle of injury" results from the reinjury of a vulnerable, recently traumatized system. This increased vulnerability occurs due to a diminishing of a system's tolerance limit as a result of adaptation to a lower level of demand during the period of rest necessitated by pain.

hypertrophy, enhanced function, and a consequent increase in the system's tolerance limit. The concept acting here is that the biological system's tolerance limit will adapt to increased demands if the demands are applied at a frequency, intensity, and duration within the system's ability to adapt (Figure 1.3).

Therefore, during the physical examination, asymmetry must be noted and analyzed as representing either adaptation or deconditioning of a given system. Any of these fundamental principles under which the musculoskeletal system functions makes it possible to organize the information obtained from a physical examination and history into general categories or pathological conditions (traumatic, inflammatory, metabolic, etc.), and the subsets of these conditions (tendinitis, ligamentous injuries, arthritis, infection, etc.). From such an approach, generalizations called paradigms can be formulated. These paradigms provide a holistic view of a patient's signs and symptoms. In this way, diagnoses are arrived at based on an analysis of the entire constellation of signs and symptoms with which a given patient presents. This method, relying on a multitude of factors and their interrelationships rather than on a single piece of information, such as the symptom of clicking or swelling, ensures a greater degree of accuracy in formulating a diagnosis.

What Are Paradigms?

Paradigms are snapshots of classic presentations of various disease categories. They are, as 19th-century clinicians would say, "augenblick," a blink-of-the-eye impression of a patient (Table 1.1). From such an impression, a comparison is made with an idealized patient, to evaluate for congruities or dissimilarities. Here is an example of a paradigm for osteoarthritis: a male patient who is a laborer, who is at least 50 years old, whose complaints are asymmetrical pain involving larger joints, and whose symptoms are in proportion to his activity. Another example might be that of rheumatoid arthritis. This paradigm would describe a female patient who is 20–40 years old, complaining of symmetrical morning stiffness involving the smaller joints of the hands, with swelling, possibly fever, and stiffness reducing with activity.

Paradigms may also be created for specific tissues (i.e., joints, tendons, muscles, etc.). The paradigm for a joint condition such as osteoarthritis would be well localized pain, swelling, stiffness on sedentary posturing, and pain increasing in proportion to use, whereas a paradigm for a mild tendon inflammation (tendinitis) may be painful stiffness after sedentary posturing that becomes alleviated with activity and gentle use. A paradigm for ligament injury would include a history of a specific traumatic event, together with the resultant loss of joint stability demonstrated on active and passive tensile loading of a joint.

The reader is encouraged to create his or her own paradigms for various conditions—paradigms that include the entire portrait of an injury or disease process with which a given patient or tissue may be compared. In this process, it will become obvious that it is not sufficient to limit one's expertise to the localization of complaints to an anatomical region. It is also necessary to be able to discriminate between the involvement of specific structures that may lie in close proximity within that region (i.e., bursae and tendons overlying a joint).

It can be concluded therefore that an accurate physical examination is as critical to the process of diagnosis as is a complete and accurate history of a patient's complaints. An accurate physical examination demands a thorough knowledge and familiarity with anatomy and function.

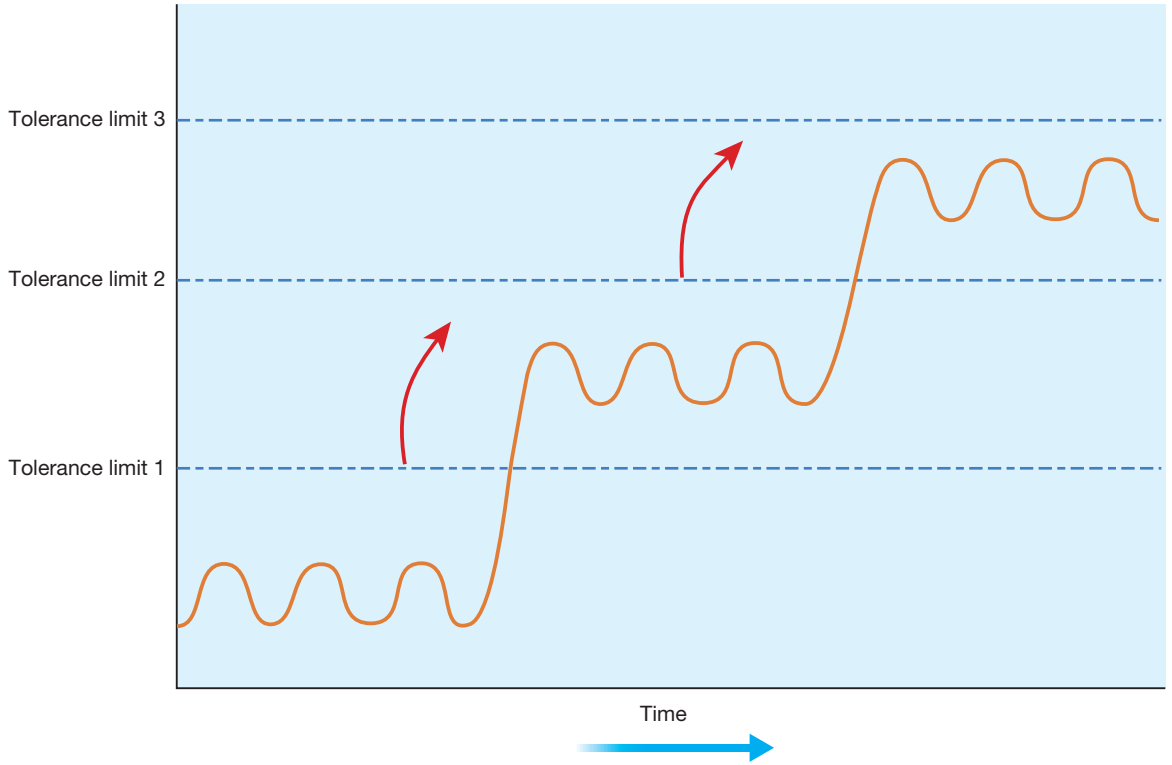


Figure 1.3 Conditioning is the adaptation of a biological system to the controlled application of increasing stress at a frequency, intensity, and duration within the system's tolerance limit, with a resultant increase in the system's tolerance limit.

What Are the Components of the Musculoskeletal System?

The musculoskeletal system is composed of bone, cartilage, ligaments, muscle, tendons, synovium, bursae, and fascia. This system is derived embryologically from the mesenchyme and is composed of soft and hard connective tissues. These tissues have evolved to serve two basic functions: structural integrity and stable mobility. The tissues are composite materials made up of cells lying within the extracellular matrix they produce.

Table 1.1 Paradigms for Osteoarthritis and Rheumatoid Arthritis

Paradigm for Osteoarthritis	Paradigm for Rheumatoid Arthritis
Male	Female
Laborer	20–40 years old
50+ years old	Symmetrical small joint involvement
Large joint involvement	Associated swelling, fever, rash, morning stiffness
Asymmetrical involvement	Abating with use
Pain in proportion to activity	

Collagen, a long linear protein (Figure 1.4a), is the most abundant of the extracellular materials found in connective tissues. The foundation of collagen is a repetitive sequence of amino acids that form polypeptide chains. Three such chains are then braided together to form a triple helical strand called tropocollagen. These strands join to make microfibrils; long linear structures specifically designed to resist tensile loading. The microfibrils are bonded together through chemical cross-linking to form collagen fibers. The degree of cross-linking determines the physical properties of a specific collagen fiber. The more cross-linking that exists, the stiffer the fiber will be. The degree of collagen cross-linking is in part genetically and in part metabolically determined. This explains why some people are much more flexible than others. Vitamin C is critical for the formation of cross-links. As such, scurvy, a clinical expression of vitamin deficiency, is characterized by "weak tissues." Hypermobility of joints (i.e., ability to extend the thumbs to the forearms, ability to hyperextend

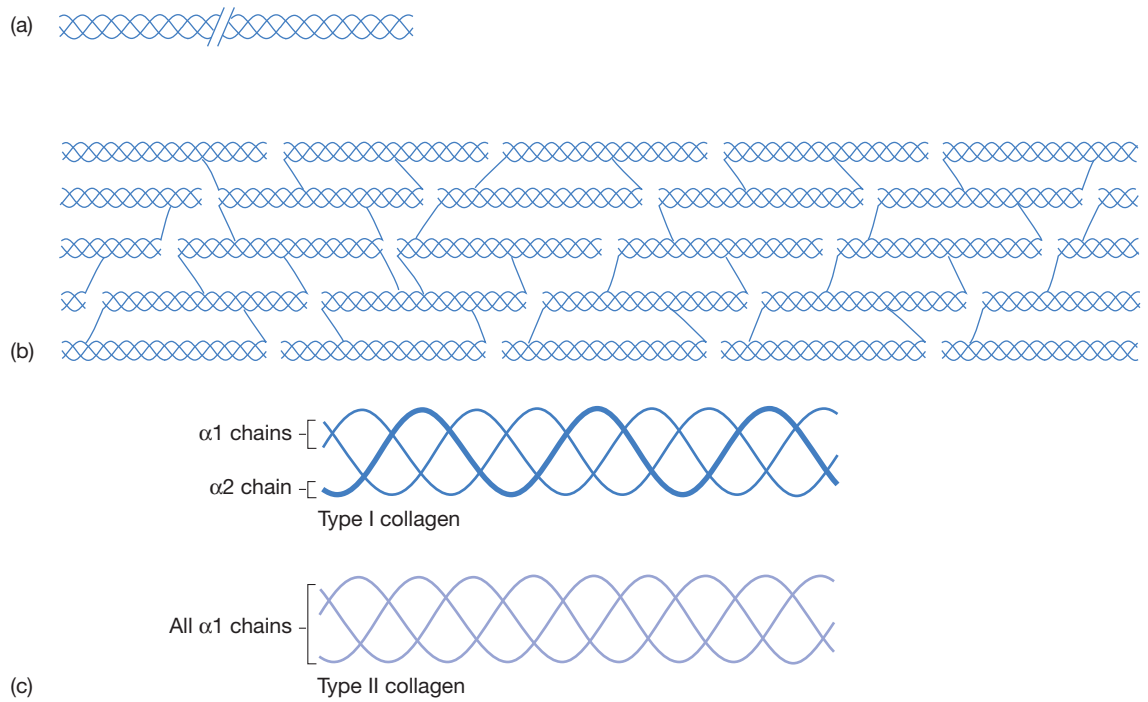


Figure 1.4 (a) Collagen is a linear protein made of α chains that wind into a triple-helix. (b) Collagen fibrils are formed by the cross-linking of collagen monomer proteins. (c) The different types of collagen are determined by the number of α1 and α2 collagen monomers that join to form a triple-helix collagen molecule. For example, two α1 chains and one α2 chain that join to form a triple-helix make type I collagen, which is found in bone, tendon, ligament, fascia, skin, arteries, and the uterus. Type II collagen, which is found in articular cartilage, contains three α1 chains. There are at least 12 different collagen types.

at the knees and elbows, excessive subtalar pronation with flat, splayed feet) is a clinical manifestation of genetically determined collagen cross-linking (Figure 1.4b).

Different types of collagen exist for different categories of tissues. These types are defined by the specific composition of the polypeptide chains that form the strands of the collagen molecules. Type I collagen is found in connective tissue such as bone, tendons, and ligaments. Type II is found uniquely in articular hyaline cartilage. Other collagen types exist as well (Figure 1.4c).

If collagen represents the fiber in the composite structure of connective tissue, ground substance represents the "filler" between the fibers. The main components of ground substance are aggregates of polyglycan macromolecules. An example of such a macromolecule is the proteoglycan hyaluronic acid, found in articular cartilage. Hyaluronic acid is a molecule of more than 1 million daltons. It is composed of a long central core from which are projected many protein side chains containing negatively charged sulfate radicals. It can best be visualized as a bristle brush from which many smaller bristle brushes are projected (Figure 1.5). These strongly negative sulfate radicals make the hyaluronic acid molecule highly hydrophilic (water attracting). This ability to attract and hold water allows the connective tissue ground substance to function as an excellent hydrostatic bearing surface that resists compression load.

Immobilization reduces the diffusion and migration of nutrients throughout the connective tissues. This in turn compromises cellular activity and upsets the normal homeostatic balance of collagen and ground substance turnover. The result is an atrophy of collagen fibers and a diminution of ground substance (Cantu and Grodin, 2011), with subsequent deterioration of the connective-tissue macrofunction (i.e., chondromalacia patellae).

Bone

Bone provides the structure of the body. It is the hardest of all connective tissues. One-third of bone is composed of collagen fibers and two-thirds mineral salts, primarily calcium hydroxyapatite. Bone is formed in response to stress. Although genetically

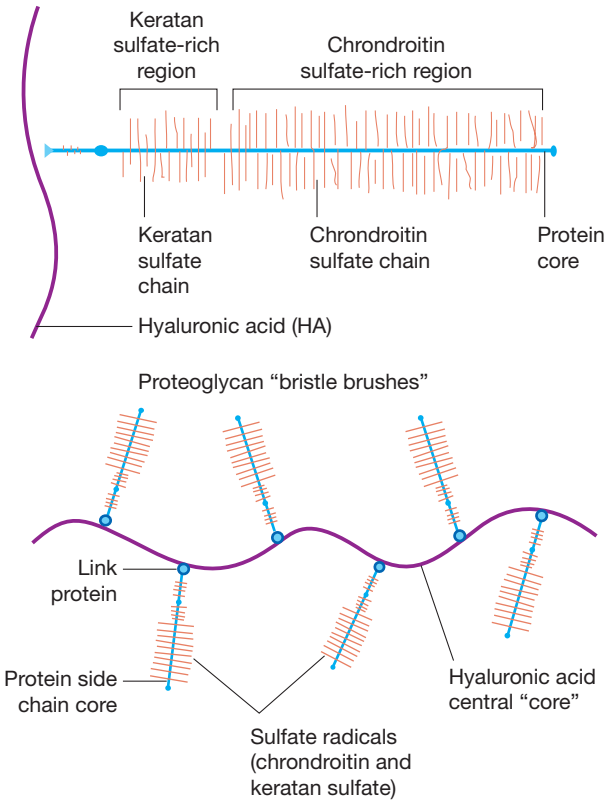


Figure 1.5 The proteoglycan aggregate is formed on a backbone of hyaluronic acid and has the appearance of a bristle brush.

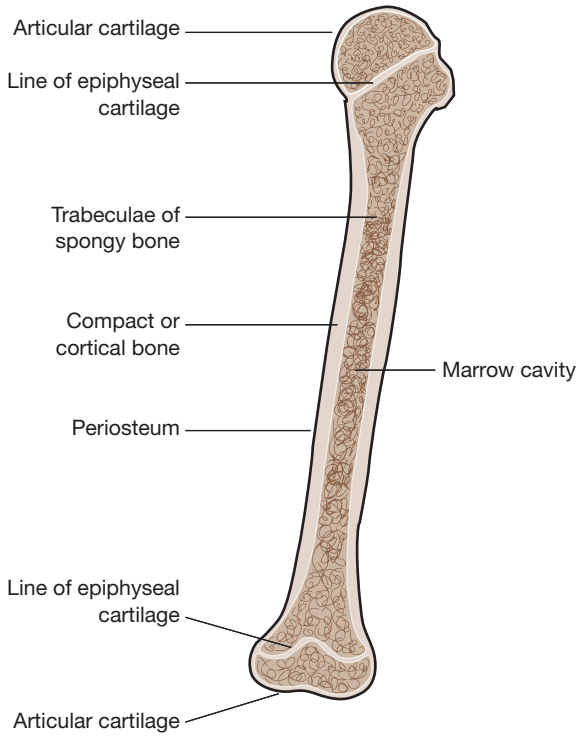


Figure 1.6 The structure of a typical long bone.

determined, the size and shape of a bone are dependent on environmental factors for its full expression. This response of bone to its loading history has been termed Wolff's law. There are two major types of bone: cortical and cancellous. All bones are covered by highly vascularized and innervated tissue called periosteum, except when they are within the synovial cavity of a joint (Figure 1.6).

Cortical bone is very dense, highly calcified, and uniquely constructed to resist compression loads. It can also resist tensile bending and torsional loads, but much more poorly. This is a direct function of cortical bone's ultrastructure, which is a composite of flexible collagen fibers and rigid mineral crystals. Cortical bone is usually found within the diaphysis of long bones. It has a hollow central cavity that is termed the medullary canal or marrow cavity.

At the end of long bones and at the sites of tendon and ligament attachments, bones tend to expand and cortical bone gives way to a more porous structure, termed cancellous or trabecular bone. The trabeculae of cancellous bones lie in the direction of transmitted loads. They act as conduits of load from the articular surface to the underlying diaphyseal cortical bone. Overload of the trabeculae will, on a microscopic scale, duplicate overload of an entire bone (i.e., fracture). This overload, because of the innervation that exists within a bone, will give rise to pain (arthritic discomfort due to mechanical overload secondary to joint deformity or erosion of articular cartilage). The resultant healing of these microfractures leads to increased calcium deposition, hence subchondral sclerosis noted around articular joints on x-ray films, and hypertrophy of stressed sites such as the midshaft of the tibia secondary to stress fractures occurring from overuse in distance running.

Cartilage

Cartilage is a connective tissue made of cells (chondroblasts and chondrocytes) that produce an extracellular matrix of proteoglycans and collagen fibers with a high water content. The tensile strength of cartilage is due to the collagen component. Its resistance to compression is due to the ability of proteoglycan to attract and hold water. Cartilage types include articular or hyaline cartilage (Figure 1.7); fibrocartilage,

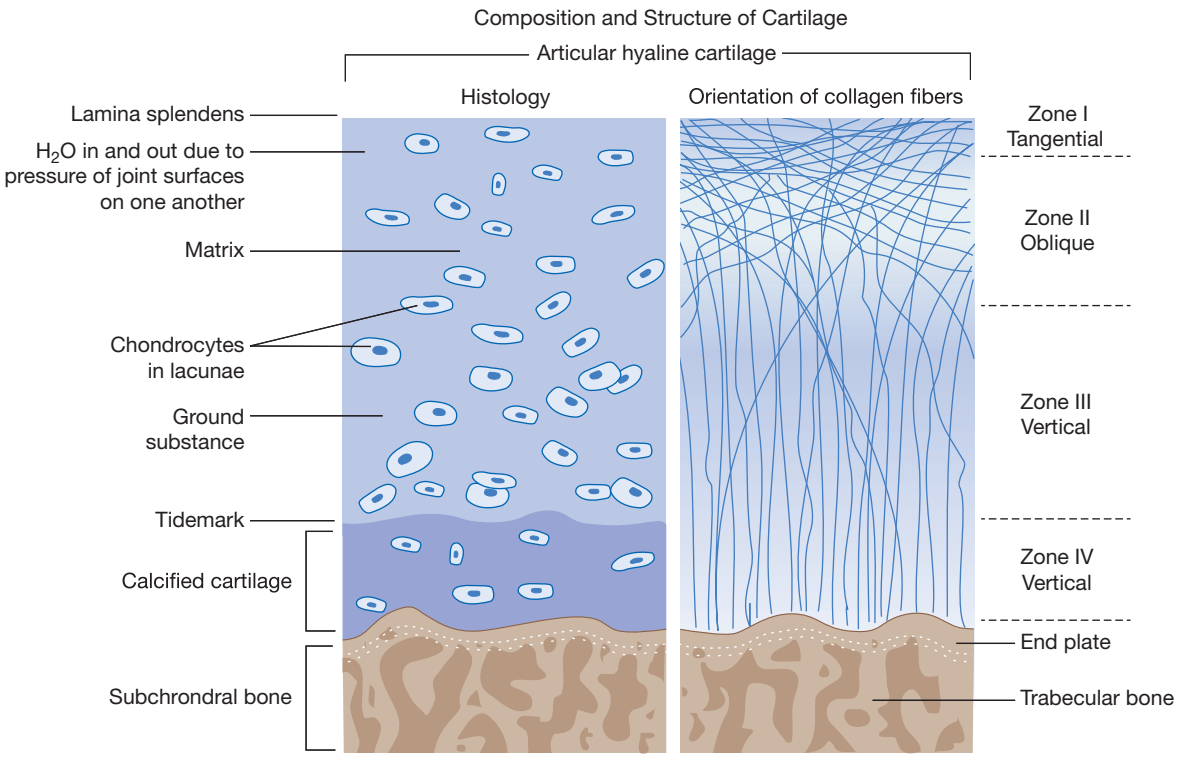


Figure 1.7 The composition and structure of articular hyaline cartilage. Water moves in and out of the cartilage due to the pressure of the joint surfaces on one another and attraction of the water by the ground substance. Note the orientation of the collagen fibers.

which exists at the attachment sites of ligaments, tendons, and bones; fibroelastic cartilage, found in menisci and intravertebral discs; and growth-plate cartilage, located in the physis of immature bones. With age, cartilage tends to decrease in water content and the number of cross-links among collagen molecules increases. The result is that cartilage tissue becomes more brittle, less supple, and less able to resist tensile, torsional, and compression loading. Hence, cartilage becomes more vulnerable to injury with age.

Articular cartilage lines the spaces in synovial joints. It is attached to the underlying bone by a complex interdigitation analogous to that of a jigsaw puzzle. Regeneration of this cartilage is slow and inconsistent in terms of restoration of articular integrity. It can be replaced by a less mechanically efficient fibrocartilage after injuries have occurred. There are no blood vessels within articular cartilage and nutrition is solely dependent on the loading and unloading of the joint, which allows water-soluble nutrients and waste products to enter and leave the cartilaginous matrix through a porous surface layer.

The fibroelastic cartilage of the intervertebral disc allows for very minimal movement between adjacent vertebrae while providing shock absorption. Because of the orientation of the fibers, they are more vulnerable to flexion and rotational forces. Fibroelastic cartilage is also present in the menisci of the knee. Here, it functions not only to absorb shock but also to increase the functional surface area of the joint, thereby providing additional stability. Because of its elastin content, fibroelastic cartilage is resilient and able to return to its prior shape following deformation.

Ligaments

Ligaments are the static stabilizers of joints. They connect bones to bones (Figure 1.8). Ligaments and other capsular structures of the joint are made of dense, organized connective tissue. Ligaments contain collagen and a variable amount of elastin. The collagen provides tensile strength to the ligaments and elastin provides suppleness. The fibers of collagen are arranged more or less parallel to the forces that the ligament is intended to resist. Most ligaments and capsular tissues enter the bone as a progression from collagen fibers to fibrocartilage to calcified cartilage

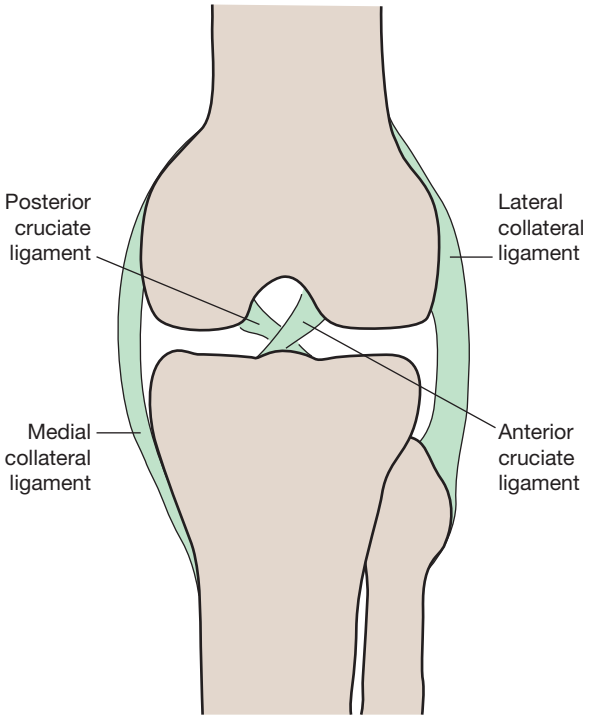


Figure 1.8 The ligaments of the knee. Because of the inherent instability of the joint, ligaments are necessary to prevent motion in all planes. They act as the primary stabilizers of the joint and are assisted by the muscles and other connective tissues.

and then finally bone. Some ligaments (and tendons) attach to the periosteum first, which then attaches to the bone. The site of ligament failure is a function of the load it experiences. Ligaments resist slow loading better than rapid loading. Therefore, rapid loading may produce an intraligamental lesion, whereas a slower pattern of loading will create injuries at or near the bone–ligament interface.

Elastin is a protein that permits elastic recoiling to occur in a tissue. Some ligaments, such as the cruciate ligament of the knee, contain almost no elastin. Other ligaments, such as the ligamentum flavum of the spine, contain large amounts of elastin. Figure 1.9 shows that because it contains more collagen than elastin, the anterior cruciate ligament can resist tensile loads with little elongation. In this way, the anterior cruciate ligament serves the knee well as a stabilizing structure. On the other hand, the ligamentum flavum of the spine, being composed mostly of elastin and little collagen, can be stretched a great deal before breaking, but can only resist very weak tensile loading.

Ligaments function to limit joint motion and to guide the bones as they move. Ligaments therefore usually have a dual internal structure, such that they may stabilize the joint at either extreme of motion.

Ligaments are most lax at midrange of joint motion. The capsule of a synovial joint is in fact a weak ligamentous structure. Disruption of a ligament can result in severe joint instability and increased frictional stresses to the articular surfaces of that joint. This will result in premature osteoarthritis. Conversely, a loss of normal capsular laxity from fibrosis following trauma will result in a severe restriction in joint motion (i.e., posttraumatic adhesive capsulitis of the shoulder).

Ligaments have very little vascularity; hence they heal poorly. However, they do have innervation, which may be useful to quantify the severity of a given ligamentous injury. When the structural integrity of a ligament has been completely compromised (grade III sprain), relatively little pain is produced on attempts to passively stretch the injured ligament. This is because no tension load can be created across a completely disrupted ligament. However, in a less severe partial tear (grade I sprain), severe and exquisite pain will be produced when tension is applied across the damaged structure. This paradoxical pain pattern (less pain equals a more severe sprain) can be a significant diagnostic clue obtained during the physical examination of a recently injured ligament. This also has dramatic import in defining a patient's prognosis and determining a treatment plan.

Muscle

Skeletal muscle is a contractile tissue made up of fibers that contain specialized proteins (Figures 1.10 and 1.11). A loose connective tissue known as endomysium fills the space between these fibers. This tissue attaches to a stronger connective tissue that surrounds the muscle vesiculae, known as perimysium. Perimysium is in turn connected to the epimysium, which encases the entire muscle. This in turn is anchored to the fascial tissues of the nearby structures. Muscles therefore are composed of two elements: contractile tissues and inert, noncontractile tissues. The forces generated by the muscles are extrinsically applied to the muscle and will affect both types of tissue.

Muscles exist in many shapes and sizes. Some of these are shown in Figure 1.12.

Muscles contain three different fiber types: I, IIa, and IIb. They are defined by the chemical machinery used to generate adenosine triphosphate (ATP). Genetic makeup, training, and neuromuscular disease

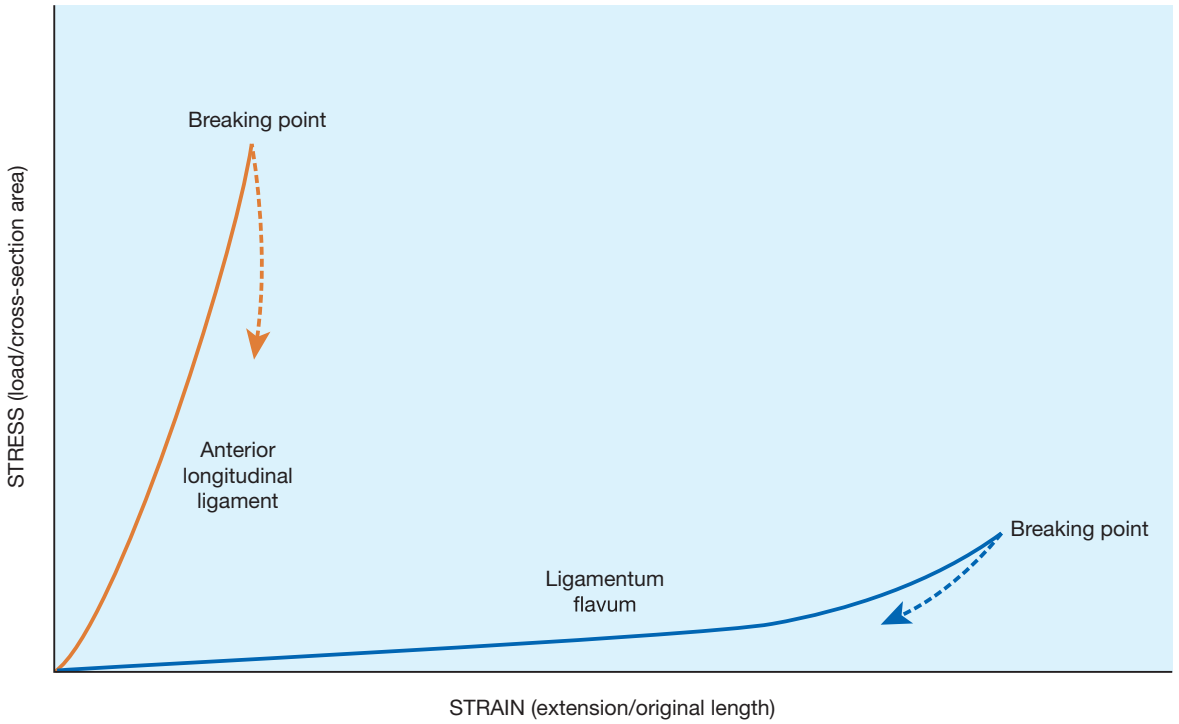


Figure 1.9 The mechanical response of stress and strain on the anterior longitudinal ligament and the ligamentum flavum. The anterior cruciate ligament, having more collagen than elastin, can handle a larger load but will only stretch a short amount before breaking. The ligamentum flava, having more elastin than collagen, cannot tolerate a very large load but can stretch a lot before breaking.

can affect the composition of a given muscle with respect to fiber type. Characteristics of these various fiber types are shown in Table 1.2.

Muscles act to move body parts or to stabilize a joint. As dynamic stabilizers of joints, muscles serve to duplicate the static stabilizing action of ligaments. Muscle fibers are capable of shortening to about 50% of their original length. The tension developed by a contracted muscle can be either active or passive. Active tension is due to the contractile components, namely, actin and myosin. Passive tension results from elastic properties of the contractile tissues within the muscle.

The strength of the muscle is proportional to its cross-sectional area and mass. The force of contraction of a muscle is related to many factors, including the length of the fibers, the velocity of contraction, and the direction in which the fiber is moving at the time of its contraction. Types of muscle contraction include concentric or shortening, eccentric or lengthening, and isometric, in which the muscle does not change length. Muscles are characterized by their function; agonists are prime movers, antagonists resist the action of prime movers, and synergists support the function of the agonists. For example, in ankle dorsiflexion, the anterior tibialis is the agonist. The extensor hallucis longus and extensor digitorum longus muscles assist the tibialis anterior muscle and therefore are synergists. The gastrocnemius and soleus and plantar flexors of the toes are antagonists of the tibialis anterior.

Muscles are described in anatomy texts as having origins and insertions. It is very important to recognize that this is an arbitrary distinction. A muscle that is referred to as a hip flexor because it brings the thigh toward the torso can function just as well to bring the torso over the thigh. In order for muscles to function normally, they must be both strong and flexible.

With respect to innervation of muscles, except for the deepest layers of the vertebral muscles, the exact innervation of the limb and trunk muscles is similar between individuals, with some variability. Tables listing segmental innervation differ from text to text.

Injuries to muscles are termed strains. Analogous to ligament injuries, they are classified by severity into three grades: grade I indicates minimal damage; grade II represents an intermediate amount of damage to the muscle structure; and grade III, complete disruption.

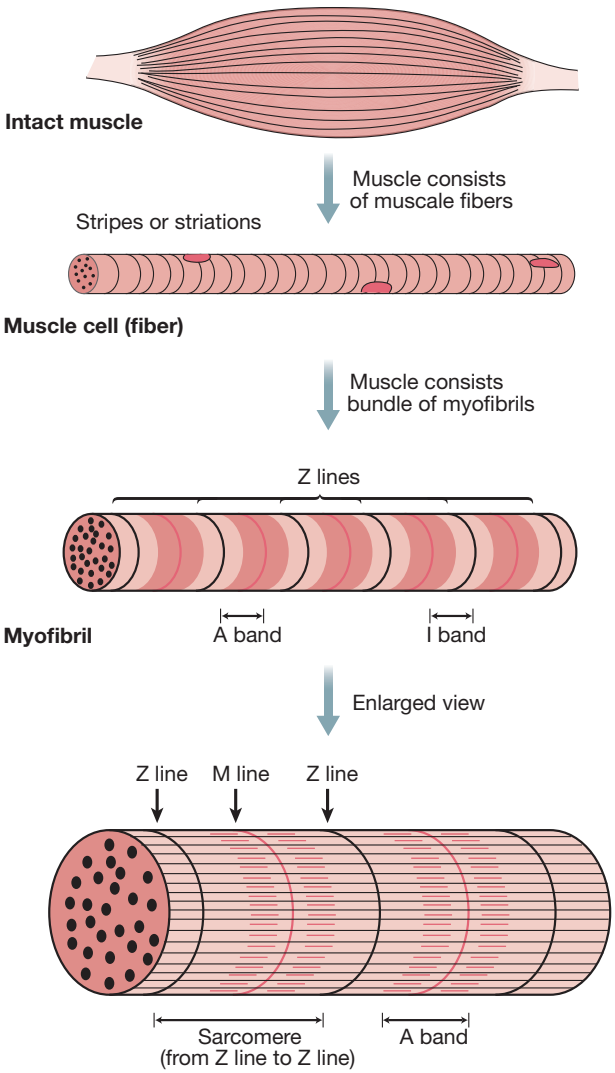


Figure 1.10 A microscopic view of muscle shows the repeated patterns of the sarcomeres and the fibrils.

Tendons

Tendons connect muscles to other structures (see Figure 1.13). Like ligaments, tendons are also composed of collagen, ground substance, and cells. The collagen of tendons is aligned in a very strict linear fashion and is always oriented in the line of the pull of the muscle. Tendons have been designed to transmit the force of the muscular contractile tissues to bone and other connective tissues, such as skin and ligaments, to which they are attached. Tendons are said to be able to withstand at least twice the maximum force that muscles can exert on them. The zone where the muscle blends into the tendinous tissues is called the musculotendinous junction. Muscle–tendon units represent tensile structures. As such, they may fail in the muscle, at the muscle–tendon junction, within the tendon, or at the tendon–bone insertion. Most commonly, however, failure occurs at the point of transition between two different materials (i.e., the musculotendinous junction). Some tendons are surrounded by a double-walled tubular covering, referred to as a tendon sheath or a peritendon (i.e., Achilles tendon or flexor tendons of the hand). This is lined with a synovial membrane. The sheath is used both to lubricate the tendon and to guide it toward the bony attachment. Tendon sheaths provide a pathway for the gliding movement of the tendon within the sheath. An inflamed tendon sheath can cause a locking or restricted movement, as in a trigger finger. Inflammation of the tendon structure is termed tendinitis.

Synovium and Bursae

Synovial tissue lies in the inner aspect of synovial joints and bursal sacs. It has two functions: to produce lubricating fluids and to phagocytize (remove) foreign debris. Synovium is highly vascularized and innervated. As such, when traumatized or inflamed, synovial tissue will rapidly enlarge and produce significant pain.

Bursal sacs serve to reduce friction. Therefore, they are located wherever there is need for movement between structures in close proximity. For example, the olecranon bursa lies between the olecranon process of the ulna and the skin overlying the posterior part of the elbow (see Figure 1.14). The subacromial bursa lies between the acromioclavicular arch above and the rotator cuff tendons below. Inflammation of synovial or bursal tissues due to trauma, inflammatory processes, or foreign materials is termed synovitis or bursitis.

Fascia

There are three kinds of fascial tissues: superficial, deep, and subserous. The fascia is composed of loose to dense connective tissue. Superficial fascia is under the skin; deep fascia is beneath the superficial and also envelops the head, trunk, and limbs. Subserous fascia surrounds organs in the thorax, abdomen, and pelvis.

Superficial fascia contains fat, blood vessels, and nerves. It is loose in consistency and very thin. It is attached to the undersurface of the skin.

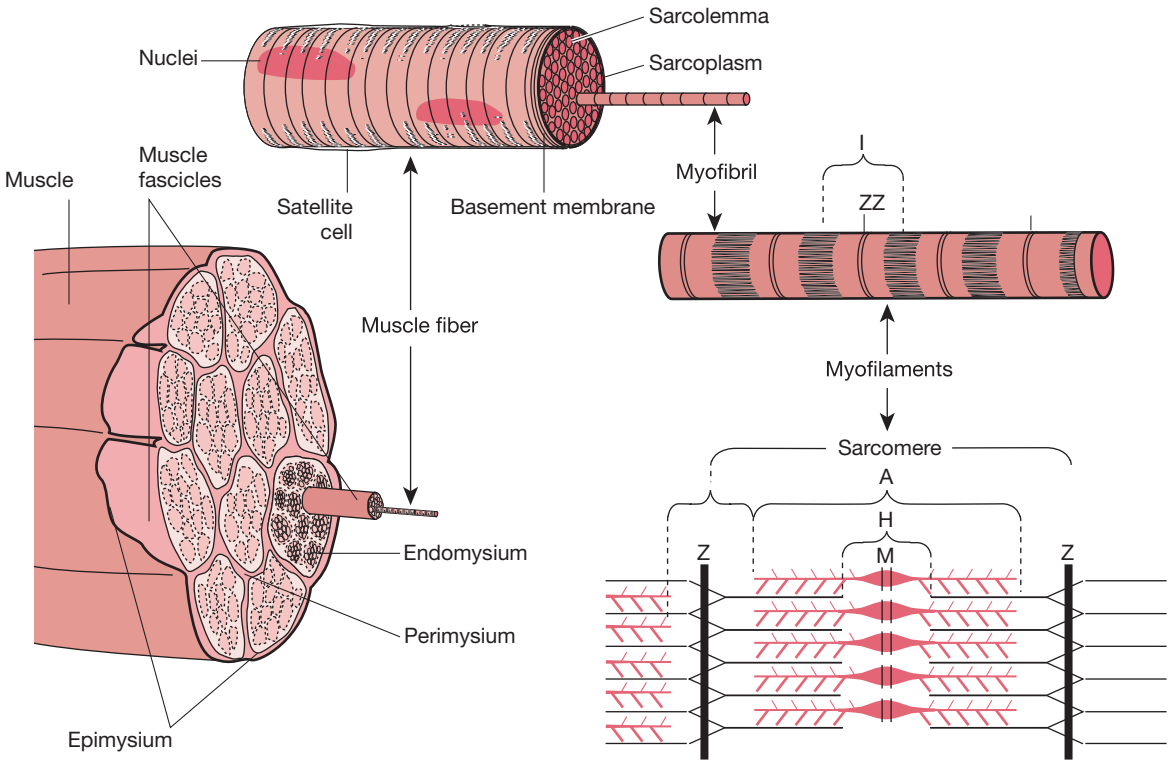


Figure 1.11 The organization of skeletal muscle tissue.

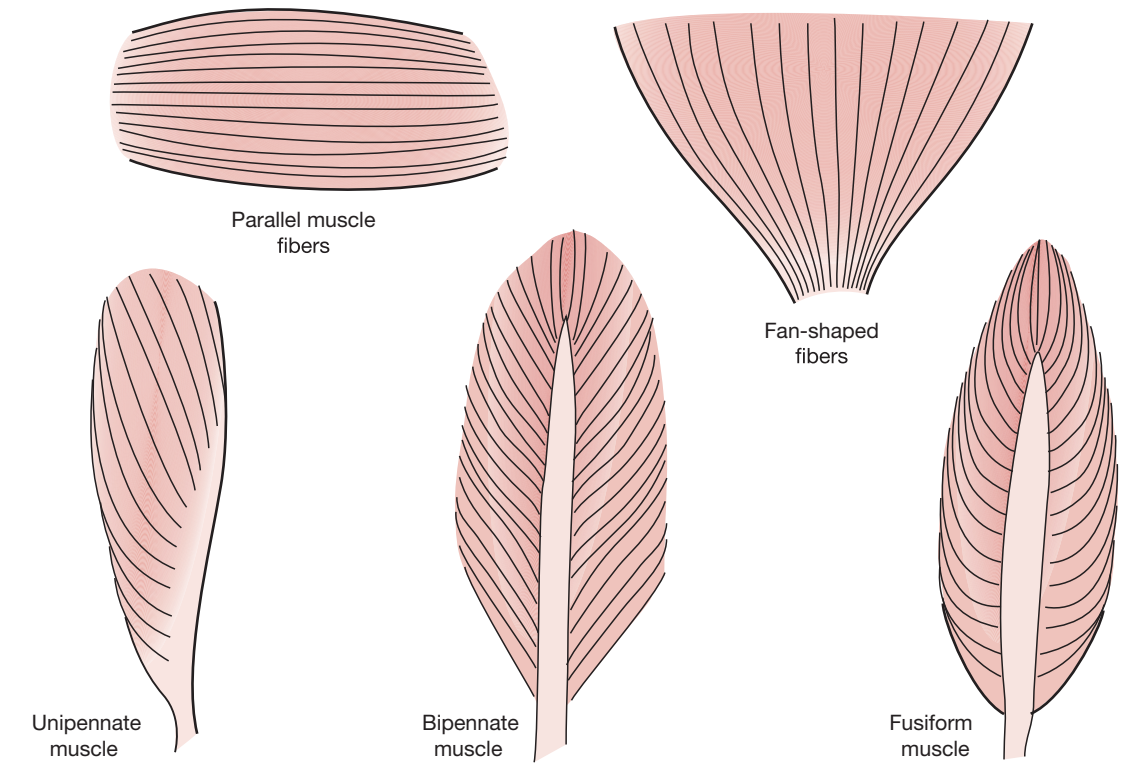


Figure 1.12 Different types of muscle–fascicle arrangements.

Table 1.2 Characteristics of Skeletal Muscle Fibers Based on Their Physical and Metabolic Properties

Property	Muscle Fiber Type Slow Twitch	Intermediate	Fast Twitch
Speed of contraction	Slow	Intermediate	Fast
Rate of fatigue	Slow	Intermediate	Fast
Other names used	Type I	Type IIB	Type IIA
	Slow oxidative	Fast oxidative/glycolytic	Fast glycolytic
Muscle fiber diameter	Small	Intermediate	Large
Color	Red	Red	White
Myoglobin content	High	High	Low
Mitochondria	Numerous	Numerous	Few
Oxidative enzymes	High	Intermediate	Low
Glycolytic enzymes	Low	Intermediate	High
Glycogen content	Low	Intermediate	High
Myosin ATPase activity	Low	High	High
Major source of ATP	Oxidative phosphorylation	Oxidative phosphorylation	Glycolysis

ATP, adenosine triphosphate.

Deep fascia is dense and tough and has two layers. It wraps around regions of the body and splits to envelop superficial muscles such as the sartorius and tensor fasciae latae. Periosteum, perimysium, and perichondrium are all elements of the deepest layer of the deep fascia. The deep fascia serves to interconnect the different muscle groups. By being continuous, it can provide tension at a distant site when pulled by a contracting muscle. Some muscles take their origin from the deep fascia. The fascia also separates groups of muscles with similar function, for example, the flexor and extensor groups of the leg. Because of the relative inelasticity of fascia, abnormally high pressure within a fascial compartment (i.e., due to injury or inflammation) can compromise the function of the nerves and blood vessels that course through that compartment. This may result in serious compromise of the tissues supplied by these nerves and vessels. Fascia may, as other tissues, experience an inflammatory reaction, fasciitis. This condition can be accompanied by moderate or even severe discomfort and scarring (fibrosis). Fibrosis can lead to stiffness and restricted movement.

The Interaction of Connective Tissues

In general, osteoarthrosis is a "wear and tear" condition. The body, although much more complex, is a machine and as such, it is subject to the same rules and laws of nature as our cars, etc. With use and in proportion to that use, it will "wear." Excluding secondary initiating causes, such as infection and inflammatory diseases, damage of an articulation (osteoarthrosis) which often becomes painful (osteoarthritis) is a manifestation of this wear and tear process. The development of osteoarthrosis should be predictable as a function of load per unit area (L/A^2) over time. However, this does not appear to be the case. For example,

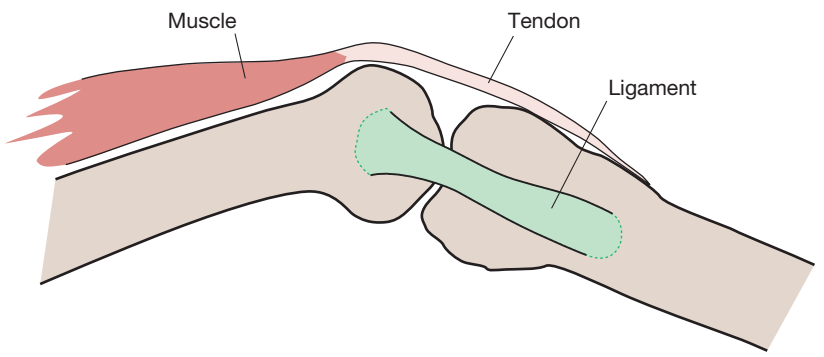


Figure 1.13 A tendon.

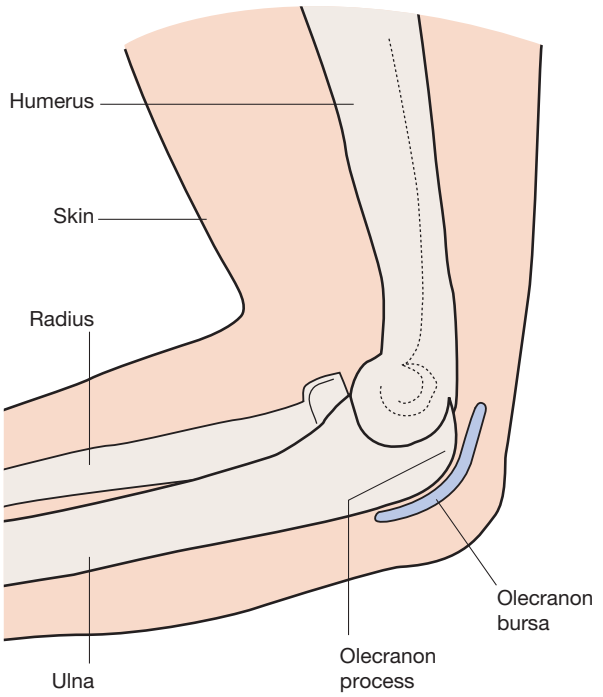


Figure 1.14 The olecranon bursa is between the skin and the olecranon process at the elbow.

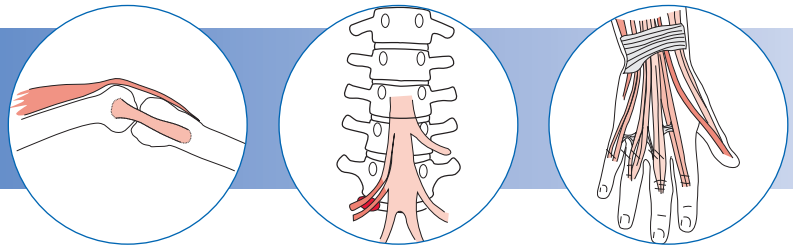
consider the ankle and the knee. The talus supports the same body weight as does the tibia. The ankle experiences an equal number of loading cycles during weightbearing throughout a lifetime as does the knee. Yet the ankle is far less often afflicted with primary osteoarthrosis, than is the knee!

Since the development of osteoarthrosis is related to the degeneration and deterioration of the articular cartilage which covers the surface of a bone, it would appear that any mechanical stress which is in excess of the physiologic tolerance of the cartilage may lead to damage of that material. In general, the surface of articular cartilage is a very well-structured alignment of collagen fibers with a microporosity permitting the diffusion of nutrients and water across this barrier. Any disruption of the integrity of the surface layer will permit the large macromolecule, proteoglycan, to escape through defects in the surface layer. This would be analogous to the stuffing of a pillow escaping through a tear in the fabric on the surface of the pillow. Once the proteoglycan has escaped from the articular cartilage matrix, the ability of articular cartilage to absorb and release water as a lubricant is compromised. The loss of this self-lubricating mechanism will increase articular surface frictional wear, causing further damage and deterioration of the entire articular cartilage structure. The inevitable consequence of this process is the erosion of the articular cartilage and the eventual exposure of the underlying subchondral bone leading to the classic "bone-on-bone" seen in end-stage osteoarthrosis.

From this model, it is easy to understand that excessive frictional or shear loads on the articular cartilage surface will lead to this inevitable demise of the joint. Stability therefore plays a critical role in the maintenance and protection of articular cartilage integrity. Stability of a specific articulation is determined by a combination of factors: its geometry and the soft tissue structures crossing that joint. The geometry of an articulation determines its degrees of freedom of movement in three dimensions. The soft tissue structures provide additional joint stability and are divided into two groups. The first is comprised of static structures (ligaments), each of which has a fixed length. The second group of soft tissue stabilizers is composed of the muscle–tendon units which cross the joint. Unlike ligaments, muscle–tendons have the dynamic capability to alter their length and tension. Therefore, the development of osteoarthrosis can be predicted by an assessment of a given articulation's inherent stability provided by its architecture or geometry, and the integrity of the soft tissues crossing that articulation.

In the case of the ankle, the mortise created by the medial malleolus, tibial plafond, and lateral malleolus, into which the talus is inset, creates a very rigid structure severely restricting and limiting the degrees of freedom of talar motion. While the knee, being composed of two large condylar surfaces resting on a relatively flat tibial plateau, has no inherent geometric stability and is therefore totally dependent upon soft tissues for its stability. It is easy to understand and predict therefore that the knee being solely dependent upon soft tissues, very vulnerable to injury, would be at a significantly greater risk of developing osteoarthrosis than would the talus. This is consistent with clinical observations. The ankle appears to be relatively immune to the spontaneous development of osteoarthrosis throughout an individual's lifetime. This is true except in the case of fracture involving the articular geometry of the ankle or damage of the syndesmosis ligament connecting the tibia and fibula causing a widening of the ankle mortise. In either of those cases, an ankle will rapidly develop osteoarthrosis. In this way, connective tissues, bone, ligaments, muscles, and tendons work in synergy to not only afford proper functioning of a given articulation but also provide a primary means of protection against "wear and tear" over the course of time.

CHAPTER 2



Basic Concepts of Physical Examination

Introduction

The ability to examine a joint completely and accurately is a critical part of the diagnostic process for the clinician evaluating an orthopedic problem. To accomplish this, the clinician must possess a thorough knowledge of anatomy, biomechanics, and kinesiology, as well as an understanding of the structure, purpose, and response of the various tissues. Information is obtained through observation and palpation. The clinician must be able to determine whether the patient's pathology is of musculoskeletal origin.

The examination process must be performed in a specific and logical order. This order will remain the same regardless of whether the clinician is examining the shoulder joint or the spine. It is important for the examiner to develop the habit of utilizing a set sequence in order to be as organized and efficient as possible and to avoid inadvertently omitting information.

Observation

The examination should begin in the waiting room before the patient is aware of being observed. Information regarding the degree of the patient's pain, disability, level of functioning, posture, and gait can be observed. The clinician should pay careful attention to the patient's facial expressions with regard to the degree of discomfort the patient reports that he or she is experiencing. Observing the patient sitting and coming to a standing position will provide insight into the patient's ability to tolerate flexion and to then go from flexion to extension. Observation of the patient's gait will provide information regarding the ability to bear weight, strength of push-off, balance in relationship to unilateral stance, and cadence. The information gathered in this short period could be very useful in creating a total picture of the patient's condition.

Subjective Examination (History)

The patient should be escorted to a private area to enable the clinician to begin the subjective portion of the examination. The patient will be much more comfortable and relaxed if he or she is allowed to remain dressed during this part of the examination. The clinician should pay close attention to the details of the present bout and all previous related bouts. The patient deserves and will appreciate the examiner's undivided attention, even if only for a short period. A skilled clinician must be able to listen politely while directing the interview. Concise and direct questions posed in a logical order will help to provide the appropriate information.

The clinician should begin the interview by determining the history of the present bout. Questions should include the following: When did the episode begin? What was the etiology (traumatic vs. insidious)? Are the symptoms the same or are they increasing? It is important to determine whether there were any previous episodes, and if there were, to determine when they occurred, what the etiology was, how long they lasted, and how they resolved (Box 2.1).

It is helpful to elicit whether the pain is constant or intermittent. Symptoms that are brought about by

Musculoskeletal Examination, Fourth Edition. Jeffrey M. Gross, Joseph Fetto and Elaine Rosen.

Companion website: www.wiley.com/go/musculoskeletalexam

Box 2.1 Typical Questions for the Subjective Examination

Where is the pain located?
How long have you had the pain?
How did the pain start? Was it traumatic or insidious? Is the pain constant or intermittent?
If it is intermittent, what makes it better or worse? How easy is it to bring on the complaint?
Describe the pain (nature of pain)?
What is the intensity of the pain (0–10)? Does the pain awaken you at night? What position do you sleep in?
What are your work and leisure activities? What type of mattress and pillow do you use? How many pillows do you sleep on?
Does the pain change as the day progresses?
Have you had a previous episode of this problem? If yes, how was it treated?

Past Medical History (PMH):
Thorough systems review.
Specific questions are beyond the scope of this text.

Medications:
Are you taking any medication?
For which problem (symptom) is the medication providing relief?

Special Questions:
Specific questions and concerns related to each joint are discussed in the individual chapters.

changing position may be mechanical in nature. If the symptoms remain unaltered regardless of position or activity, they may be chemical in nature, secondary to the release of noxious substances that are at a sufficient level to irritate the nerve endings. Constant pain that changes in intensity or quality is considered to be intermittent (Cyriax, 1982). It is also useful to determine what makes the symptoms better or worse and how long the symptoms remain following their onset. If a patient develops pain very quickly while performing an activity and the pain lasts a long time, the clinician would consider the patient's pain to be irritable (Maitland, 2014a,b). It would be beneficial to modify the physical portion of the examination so as not to exacerbate the symptoms. The pain can also be followed over a 24-hour period. Is the patient better or worse at times throughout the course of the day? If the patient is stiffer in the morning on arising, he or she may not be using a firm mattress, may be sleeping in an inappropriate position, or may have osteoarthritis, which presents with increased stiffness following prolonged inactivity. A pain scale (McGill Pain Scale; Melzack, 1975) may be used to gain a better understanding of the patient's perception of their pain. Easy to use visual and numerical scales exist.

To organize the information that is obtained, it is helpful to use a body chart (Figure 2.1). This chart allows information to be recorded graphically for observation and comparison. The chart also enables the recording of information concerning areas other than the one affected. If an area is examined and found to be asymptomatic (clear), a check mark can be placed over that area to indicate that it has been examined and found to be free of symptoms. For example, if the patient presents with pain in the right hip on the day of the initial examination but returns with pain in the left hip 2 weeks later, the clinician can quickly refer back to the diagram to confirm the history.

Information must be gathered regarding the primary area of the complaint and any related area(s). Areas of radiating pain, anesthesia, or paresthesia should be noted. This allows the clinician to develop a better total picture of the problem. It will also help to assess whether there is any relationship between the areas. For example, if the patient's major complaint is that of low back pain and pain in the right knee, there may or may not be a direct relationship. Perhaps the patient has radicular pain in an L3 dermatomal pattern, or perhaps that patient's injury was secondary to a fall in which the patient landed on the right knee at the same time the back was injured. The quality or description of the pain (stabbing, nagging) in the patient's own words must also be noted. If the patient complains of burning pain, the nerve root might be implicated, whereas a deep ache may be associated with muscle dysfunction.

Objective Examination

Dominant Eye

Accuracy in observation requires the use of visual discrimination. This can best be accomplished by using the dominant eye. Determination of the dominant eye is done as follows: the clinician extends both arms and uses the thumb and the index finger to make a small triangle. A distant object is then selected and aligned in the center of the triangle. The clinician then closes the left eye and checks if the object remains in the same position or if it moves. If it remains, the clinician is right-eye dominant. The procedure is repeated for the other eye. The dominant eye should be checked periodically since it may change. The dominant eye should be placed over the center of all structures as

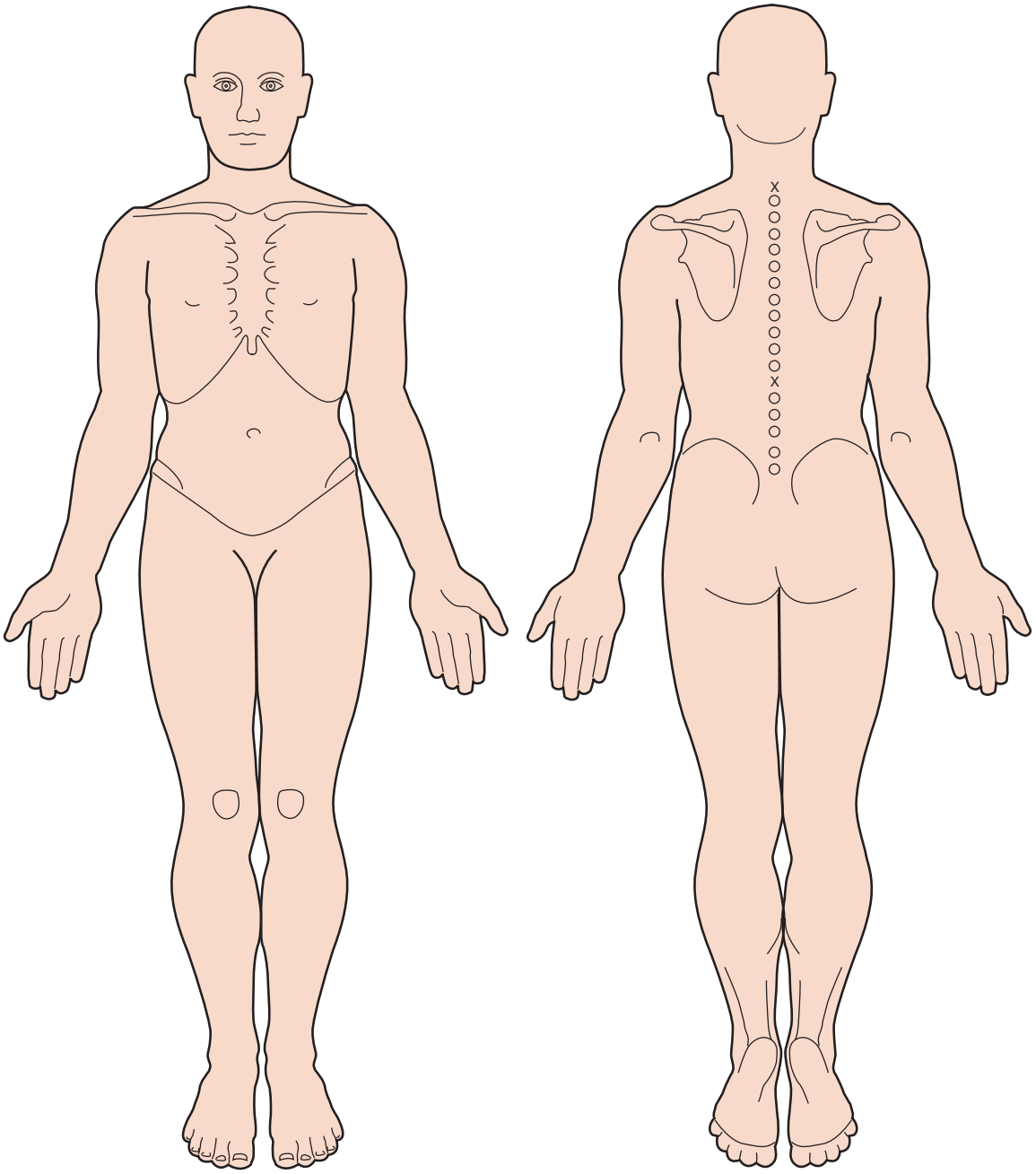


Figure 2.1 Body chart.

they are being examined to allow for more accuracy in visualization (Isaacs and Bookhout, 2002).

Structural Examination

The posture or structural examination is a static observation of the patient. This is an extremely important part of the total examination process. You can obtain a considerable amount of information regarding the patient on the basis of structure alone. Normal posture is maintained by balanced, strong, and flexible muscles, intact ligaments, freely moving fascia, healthy, properly functioning joints, a balanced line of gravity and good postural habits.

Changes in postural alignment may be secondary to structural malformation, joint degeneration, bone

deterioration, joint instability, a change in the center of gravity, poor postural habits, or pain. Faulty alignment creates unnecessary stress and strain on the individual, creating either excessive elongation or adaptive shortening of muscles. Muscle elongation or shortening results in decreased efficiency while performing even the easiest of activities. The structural examination will help you gain a better understanding of the patient's predisposition to overuse or to injury.

The structural examination allows you to integrate the structure and function of all the joints. Recognize that when a person develops elongated or shortened muscles, he or she may not develop symptoms immediately. It may take many years of stress and strain for problems to reach clinical recognition.

To begin the examination, the patient is asked to disrobe and is provided with an appropriate garment, which allows you to expose the areas that are being examined. It is important that the lighting in the room is equally distributed so there are not any shadows. The patient should be instructed to stand in the middle of the examining room with their feet approximately 6 in. apart so that you can observe him or her from the anterior, posterior, and lateral views. Note whether the patient is distributing the weight equally between both feet. Most examiners prefer to have the patient remove his or her shoes to observe the feet. If, however, the patient has a known leg length discrepancy and uses a lift or wears an orthotic device, have the patient wear the shoes with the lift or orthotic device in place. Observe the patient with and without inserts or lifts. Pay particular attention to symmetry of structure including bony landmarks, muscle tone, bulk, guarding, atrophy, and alignment of the joints. The optimal, most efficient posture is symmetrical and balanced. Recognizing that no one is perfectly symmetrical, minor variations are considered to be functional. Significant differences may be secondary to anatomical malposition which is either congenital or acquired; mechanical dysfunction whether hypomobile or hypermobile; or dysfunction of the soft tissue whether hypertrophied, atrophied, taut, or slack.

The examination is approached in a logical fashion, proceeding either in a cranial or caudal direction. Here, we describe the examination from the feet first on the basis of the assumption that the weight-bearing structures will influence the structures that rest on them. It is helpful to compare any affected joints to those on the "normal" opposite side. Information from this examination can be quickly recorded on a body chart for ease of documentation and recall.

Posterior View

Normal

In a normal individual the calcaneus is in neutral alignment with the Achilles tendon vertically aligned. The feet should show 8–10 degrees of toeing out. The medial malleoli should be of equal height on both sides. The tibias should be straight without any bowing or torsion. The popliteal fossae should be of equal heights and the knee joints should show 13–18 degrees of valgus. The greater trochanters and the gluteal folds should be of equal heights. The pelvis should be the same height on both sides, with the posterior superior iliac spines level on the horizontal plane. The spine should be straight without any lateral curves. The scapulae should be equidistant from the spine and flat against the thoracic cage. The levels of the inferior angles and the spines of the scapulae should be equal in height. The shoulders should be of equal height. Patients may demonstrate a hand dominance pattern where the dominant shoulder is lower and the corresponding hip higher (Kendall *et al.*, 2005). The head and neck should be straight without any lateral tilt or rotation (Figure 2.2).

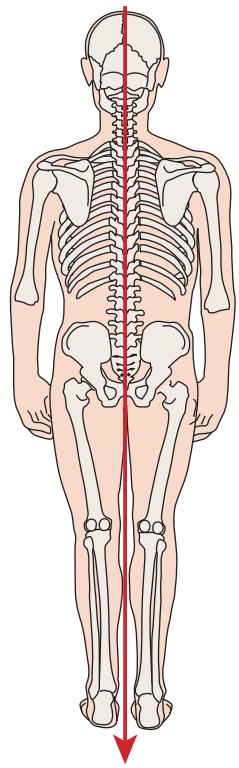


Figure 2.2 Normal posterior view.

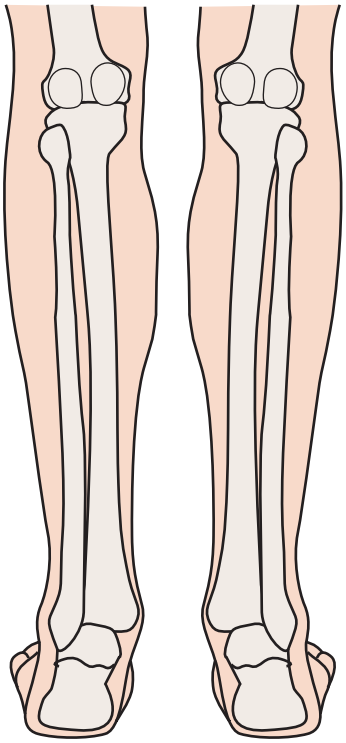


Figure 2.3 Calcaneal valgus deformity.

Possible Deviations from the Norm

Start by observing the patient's feet. Does the patient demonstrate pes planus or cavus and to what degree? Is the patient able to put the entire foot on the ground while not wearing shoes, or does he or she need a shoe with a heel because of an equinus deformity? What is the alignment of the calcaneus? Is there an excessive degree of varus or valgus (Figure 2.3)? Check the alignment of the Achilles tendon. Note the girth and symmetry of the calves. Is any atrophy or edema noted? Note the length of the leg. Does one tibia appear to be shorter than the other? Is there any bowing of the tibia or tibial torsion?

Check the alignment of the knee joints. From the posterior aspect you can observe genu recurvatum, varum, or valgum (Figure 2.4). Any of these deformities will cause a functional leg length difference unless they are symmetrical bilaterally. Note the height of the fibular heads. A difference in height may indicate an anatomical leg length difference in the tibia and fibula.

Note the alignment of the hip joint. Increased flexion may be present secondary to a hip flexion contracture (see pp. 317, 319–20, Figure 11.66). To confirm this, a Thomas test would have to be performed to test

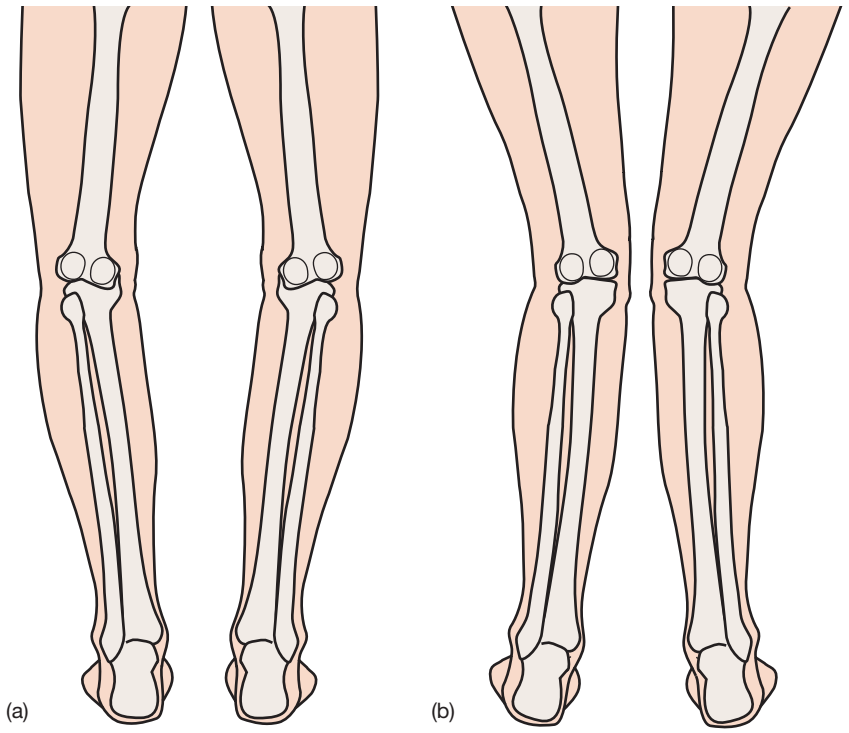


Figure 2.4 Genu varum (a) and valgum (b) deformities.

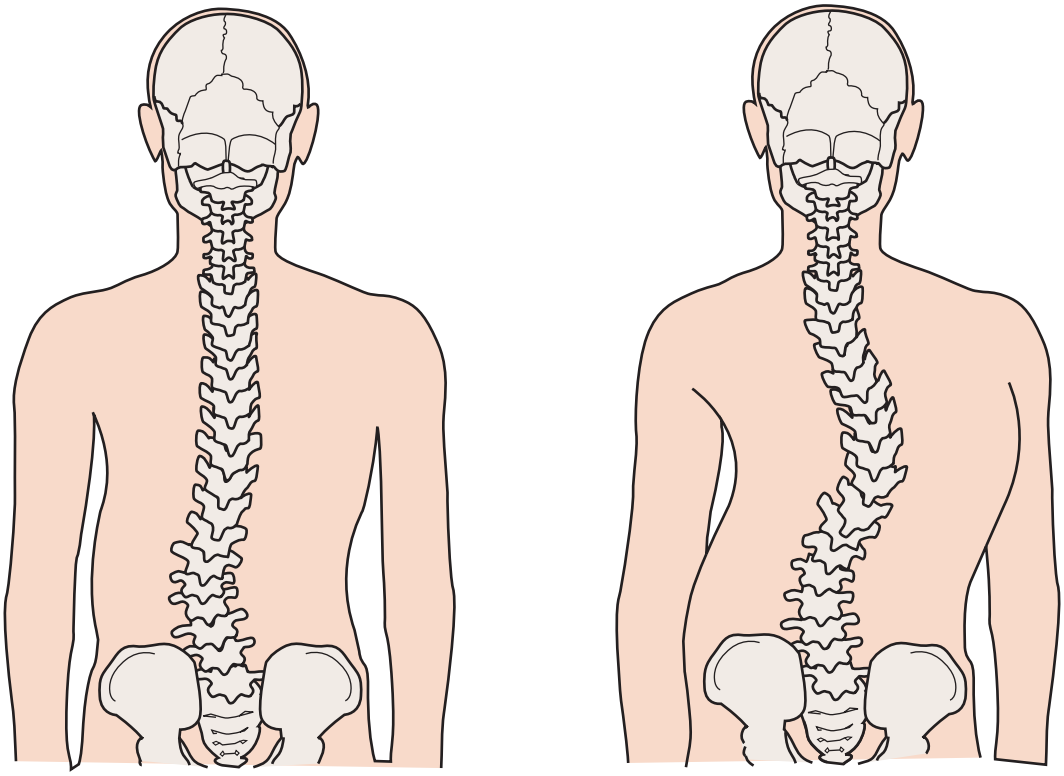


Figure 2.5 Scoliosis.

for hip flexor length. Is there excessive medial or lateral rotation? Check the relative heights of the greater trochanters. A difference in height may be secondary to a structural difference in the length of the femur.

Check the pelvis. Place your hands on the iliac crests and observe their relative heights. If one is higher than the other, it may be secondary to a pelvic torsion, a structural anomaly, or a structural or functional short leg. Place your hands on the posterior superior iliac crests and note their relative location. A change in height may be secondary to a pelvic rotation, a sacroiliac dysfunction, or a leg length discrepancy.

Observe the spine. First pay attention to the soft tissue. Are there any areas of muscle guarding or spasm? These may be secondary to a facilitated segment or surrounding an area of dysfunction. Note any differences in the skinfolds. This will allow you to better visualize lateral curves and spinal rotations. Note the alignment of the spinous processes. Is the back in straight alignment or does the patient present with a scoliosis (Figure 2.5) or kyphosis (Figure 2.6)? If scoliosis is present, note the rib cage, the degree of rotation, and the presence of any lateral humps. Is there symmetrical rib expansion both anteriorly/posteriorly and laterally? Is a lateral shift present? Is the patient

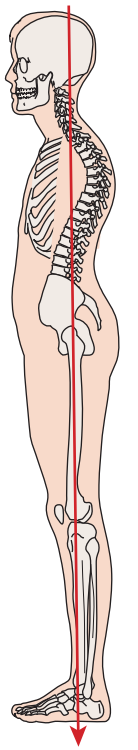


Figure 2.6 Rounded thoracic kyphosis.

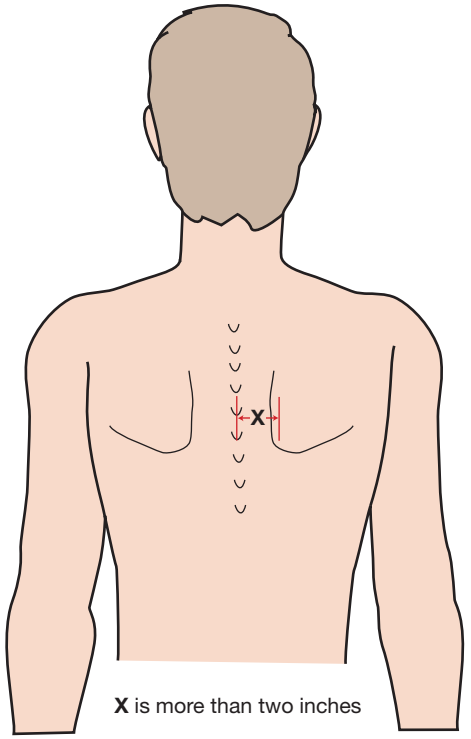


Figure 2.7 Abducted scapula.

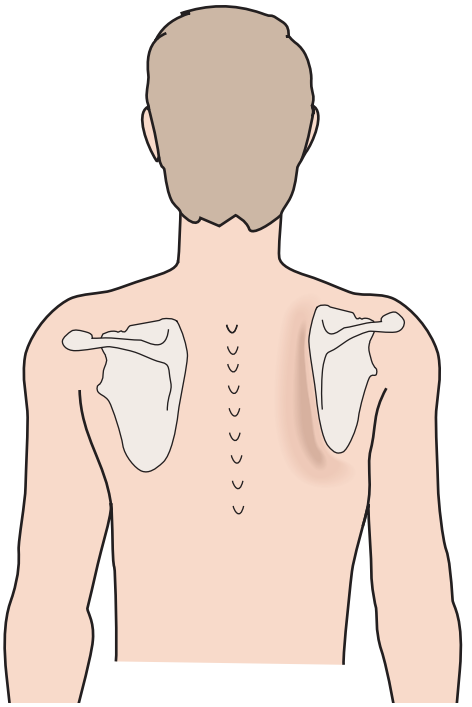


Figure 2.8 Winged scapula.

able to stand in the erect position or is he or she forward or laterally flexed?

Observe the scapulae. Are they equidistant from the spine? Are they of equal height? Are they overly abducted or adducted (Figure 2.7)? Is one side winged (Figure 2.8)? This may be secondary to weakness of the serratus anterior muscle or long thoracic nerve palsy. Is Sprengel's deformity present (Figure 2.9)? Note the muscle bellies of the infraspinatus, supraspinatus, and teres major and minor muscles over the scapula. Is there an area of atrophy? Disuse atrophy may occur in the supraspinatus or infraspinatus following a rotator cuff injury. Note the relative shoulder heights and position. Pay attention to the upper trapezius and note any hypertrophy or atrophy. Note the upper extremities. Does the patient position both arms in the same manner? Is one arm held farther away from the trunk or in either more internal or external rotation? This can be secondary to muscle shortening and imbalances or fascial restrictions.

Observe the position of the head and neck. Is the head in a forward, rotated, or laterally flexed posture? Can the patient hold the head up against gravity?

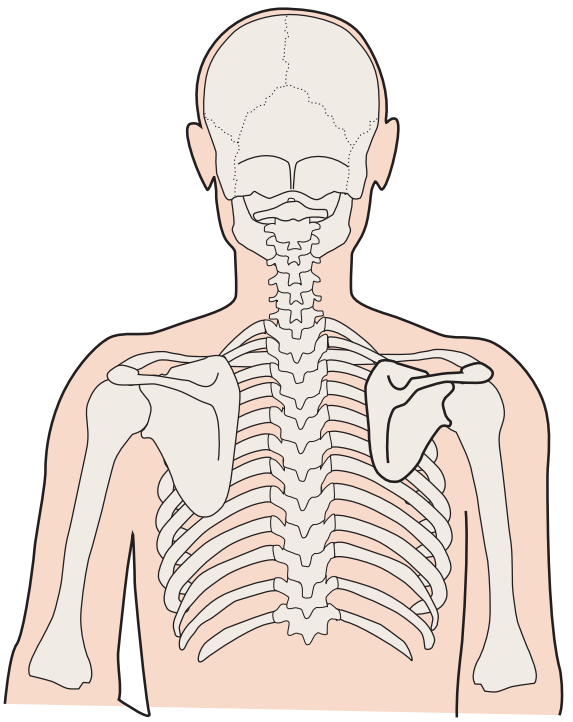


Figure 2.9 Sprengel's deformity.