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## **Tendons, Skeletal Muscles, and Ligaments in Health and Disease** ( 1-Jan-1985 )

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#### ANATOMY

##### CONNECTIVE TISSUE

Connective tissue has two components: cells and intra-cellular matrix.(20) The cells in unspecialized connective tissue consist of fibroblasts, macrophages, plasma cells, mast cells, fat cells, and pigment cells. Fibroblasts, which are concerned with the production of the intracellular matrix, are the most numerous. They are very active during wound repair and multiply to produce large numbers of cells embedded in granulation tissue, a mixture of fibrous tissue and capillaries. Mesenchymal cells also can give rise to new generations of fibroblasts, or fibroblasts themselves act as stem cells. A specialized contractile fibroblast, the myofibroblast, is responsible for contraction of granulation tissue in wound healing.(12)

The intracellular matrix has two components: amorphous ground substance and fibrous elements.(13) The ground substance is a viscous gel containing a high proportion of water, which is bound to carbohydrate molecules and carbohydrate-protein complexes and some tropocollagen. Therefore, the carbohydrate can take the form of glycosaminoglycans (mucopolysaccharides) or glycoproteins. The fibrous elements in the intracellular matrix consist of three fiber types: collagen, reticulin, and elastin. White collagen fibers are the most numerous. They are collected into bundles, which are oriented in a straight or sinuous path. Each fiber is composed of bundles of fibrils, each fibril 20 nm to 100 nm in diameter. A fibril can be broken up into its fibrillar units of tropocollagen, which in turn can be further subdivided into three polypeptide chains that are rich in the amino acids glycine, hydroxyproline, and hydroxylysine. The biosynthesis of collagen and the involvement of fibroblasts have been studied extensively.(13)

Reticulin fibers are produced by fibroblasts in most connective tissue and form the supporting framework for collagen fiber bundles. Elastin fibers rejoin freely and stretch easily with an almost perfect recoil. They are composed mainly of the protein elastin, with some glycoprotein, traces of collagen, ground substance, and lipid. (16)

Connective tissues vary considerably in appearance, consistency, and composition depending on function. The variances are related to the type of cell that is predominant; the concentration, arrangement, and types of fibers; and the nature of the ground substance. Ordinary connective tissue is classified as irregular or regular. The latter includes highly fibrous tissues, such as fascia, aponeuroses, ligaments, and tendons, in which the fibers are oriented regularly in relation to one another. The fibroblasts that secrete the fibers may eventually become trapped within the fibrous structure. Old and mature fibroblasts are often termed fibrocytes.

## BURSAE

True or anatomical bursae are connective tissue sacs lined by secretory endothelium, the synovial membrane, and containing a viscous fluid, synovia. They are interposed between moving parts and at points of unusual pressure. Thus, they are situated between tendons, ligaments and muscles, and many bony prominences, or occasionally between two tendons.

Bursae are usually flattened sacs of synovial membrane supported by dense irregular connective tissue. The apposed walls are separated only by a film of synovial fluid and are attached to structures that move in relation to one another, that is, periosteum, tendon, muscle, ligament. Bursae are therefore described as being subtendinous, submuscular, subfascial, and interligamentous. The majority of bursae are close to joints, and some communicate with the joint.

There are up to 11 bursae in the front legs of most dogs and 10 bursae in the hind leg. The anatomical sites are described elsewhere. (See Chapter 70.) Acquired or false bursae can develop in animals, usually between skin and bony prominences, as a result of chronic trauma. They begin as areas of hemorrhage and necrosis, which, as a result of the repeated trauma, cannot be absorbed by normal body mechanisms of repair. These areas instead become surrounded by a dense zone of fibrous tissue. The acquired bursae resemble closely the anatomical bursae; however, they are lined not by a synovial membrane but by a flattened layer of granulation tissue. Foci of hyaline cartilage can be found in the wall, which is composed mainly of dense collagenous tissue. The typical sites for acquired bursal formation are discussed elsewhere. (20)

Tendon sheaths, like bursae, are lined by a secretory endothelium on both the parietal and visceral surfaces. The wall of the parietal layer external to the endothelium is composed of a strong fibrous sheath. Some tendons have a more complex arrangement than bursae or sheaths, the presence of osseous or cartilaginous sesamoid bones. In general, these bones protect tendons at points at which they are bent around a bone surface. The apposed surfaces of the sesamoid and underlying bone are cartilage covered, and the whole is enveloped in a bursa or sheath. The tensile strength of tendon is similar to that of bone and is much in excess of ordinary demand. A tendon of cross-sectional area of 1 cm will support 600 kg to 1000 kg. Tendons have slightly extensible and elastic properties.

Tendons have a sparse but adequate blood supply from two main sources. Small arterioles enter the tendon from adjacent muscle and run longitudinally in the interfascicular areolar tissue, accompanied by veins and lymph vessels. These longitudinally situated vessels anastomose freely with small vessels from adjacent para-tendon tissue or synovial sheaths by means of the meso-tendon. There does not appear to be passage of vessels from bone to attached tendon.

The nerve supply of tendons appears to be almost entirely afferent, and specialized afferent receptors, neurotendinous endings, exist in tendons, especially near the muscle-tendon junction. The insertion of tendons and ligaments on bone has been studied. A clear progression from tendon to fibrocartilage to mineralized fibrocartilage to bone is apparent.(4)

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## APONEUROSES AND FASCIA

Aponeuroses are flat sheets of densely arranged collagen fibers and generally consist of several layers. These broad sheets of dense connective tissue are associated with the attachment of muscle. In some muscles, the whole attachment is aponeurotic (e.g., m. obliquus externus abdominis); in other muscles an attachment can be partly tendinous and partly aponeurotic. In most cases, the aponeurosis eventually is attached to bone, providing a wide and additional attachment of muscle to a bone or bones.

The term fascia is used to describe connective tissue that is collected into masses large enough to be visible grossly. Fascia occurs on the surface of muscles as investing fascia. Here it is in the form of loose areolar tissue that aids movement between structures and also allows passage of vessels and nerves. Superficial fascia occurs beneath the skin, where it facilitates movement between skin and underlying structures, allows passage of vessels and nerves, and acts as a thermal insulation. In animals, skin muscles such as platysma, in addition to varying amounts of fat, occur extensively in this fascia.

The superficial fascia is bound to underlying fibrous structures, the deep fascia. This fascia is normally more compact than superficial fascia and may be indistinguishable from aponeurotic tissue. The deep fascia can almost encircle the limbs, and many muscles arise from its inner layer. In other areas of the body, deep fascia exists as intermuscular septa, and in the case of flexor tendons, localized transverse thickenings of deep fascia form retinacular or annular ligaments that encircle tendons, hold them close to the bone, and thus prevent bowing of the tendon when its muscle contracts (Fig. 4-1).

In the dog, very important annular ligaments encircle the superficial and deep flexor tendons. The proximal one, Ig.

metacarpum transversum superficiale or the palmar annular ligament, lies at the metacarpophalangeal joint and runs between the borders of the sesamoid bones. The middle one, the proximal digital annular ligament, is situated on the middle of the first phalanx, and the distal one, the distal digital annular ligament, is situated immediately distal to the proximal interphalangeal joint. These annular ligaments are extremely important in repair of severed flexor tendons. They must be identified and the tendon must be retained within the encircling ligament.



FIG. 4-1 Muscles on the medial side of the left forearm and forepaw. (Evans HE, Christensen GC: Miller's Anatomy of the Dog, p 360. Philadelphia, WB Saunders, 1979)

In the lower limb, deep fascia can be a very important component of the mechanism of venous return by enclosing veins, together with muscles, in closed compartments. When muscles contract, pressure is exerted on veins, in which blood can flow only one way as a result of the action of venous valves.

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## LIGAMENTS

Ligaments are composed of regular connective tissue in which the fibers form a thick bundle. This regular connective tissue is predominantly collagenous, but some elastic components are present.

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## SKELETAL MUSCLES

**The Motor Unit and Innervation** The motor unit is the functional unit of the neuromuscular system and consists of (1) a motor neuron in the ventral horn of the spinal cord; (2) the axon of that neuron leaving the cord and terminating on the motor end-plate; and (3) the muscle fibers innervated by the motor neuron.(6,20)

Motor nerve fibers are admixed with sensory nerve fibers in individual peripheral nerves such that even in principally "motor" nerves about 50% of the nerve fibers are motor fibers. A single neuromuscular junction lies along the midpoint of the muscle fibers. The muscle fibers within a motor unit are supplied by a single motor neuron and its axon and constitute a field of randomly distributed cells sharing certain physiologic, biochemical, and histochemical features. The number of muscle fibers in a motor unit is thought to be related to the refinement of the movements of the muscle (e.g., extrinsic muscle of the eye has 10 muscle fibers per neuron; leg muscle up to 2000 fibers per neuron). The actual anatomical area of the muscle fibers in a single motor unit is usually round or oval, and there is considerable overlap between the area of distribution of muscle fibers of one unit and the area of another unit. These units are autonomous even though they overlap, and each unit can be stimulated with increasing voluntary effort, thus explaining the gradation of power of a muscle.

Each skeletal muscle receives one or more nerves of supply. In general, the nerve enters the deep surface of the muscle near its origin, where the muscle is relatively immobile. Blood vessels accompany the nerve to enter the muscle at the neurovascular hilum. Each nerve contains both motor and sensory fibers that supply motor end-plates, vascular smooth muscle (autonomic efferents), and various afferent sensory endings in muscle fibers, neurotendinous sensory endings, and endings in fascia.

When the nerve enters the muscle, it breaks up into a plexus that runs into the epimysium and perimysium before entering into the endomysium between fibers in a fasciculus. Each somatic motor axon terminates in a number of branches, each of which joins a muscle fiber at the motor endplate.

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## THE MUSCLE FIBER

The units of skeletal muscle are the muscle fibers or muscle cells, each having many hundreds of nuclei that are slender and oval. They are oriented parallel to the length of the fiber and lie peripherally in the zone immediately within the cell membrane or sarcolemma. Satellite cell nuclei lie between the basement membrane and the sarcolemma. They make up about 4% to 10% of all nuclei in the cells and may play an important role in muscle fiber regeneration.

The cytoplasm of the fiber or cell is divided into longitudinal threads of myofibrils, each about 1  $\mu$ m in diameter. In

longitudinal section, the myofibrils are seen to be traversed by striations, apparently continuous across the fiber and therefore involving many myofibrils at the same level. These cross striations are very complex and include isotropic or I bands, anisotropic or A bands, Z bands, and H bands,(6,20) further details of which will not be presented here. The number of myofibrils in each fiber or cell varies considerably depending on the size of the fiber, but on the average, each fiber contains hundreds. Myofibrils are composed of thousands of regularly arranged filaments of contractile proteins, the myosin filaments and the actin filaments (Fig. 4-2); they also contain myoglobins, glycogen, mitochondria, lysosomes, and fat globules. The physiology and biochemistry of normal muscular contraction have been reviewed.(8,17)



FIG. 4-2 Diagram showing successive levels of organization within a skeletal muscle, from whole muscle, through fasciculi, fibers, myofibrils, myofilaments, down to molecular dimensions. (Williams PL, Warwick R: Gray's Anatomy, 36th ed. Philadelphia, WB Saunders, 1980. Copyright 1980, Churchill Livingstone)

Muscle fibers vary in diameter from 10  $\mu\text{m}$  to 100  $\mu\text{m}$  depending on the function of the muscle, degree of exercise, and sex (males tend to have larger fibers than females). Each fiber is elongated, and most extend from one end of the muscle to the other, achieving a length of 30 cm in long muscles. Some fibers traverse only part of the length of a muscle and end in connective tissue within the muscle body.

In humans and experimental animals, two types of muscle fibers have been defined based on their physiochemical characteristics and speed of contraction. The two fiber types are intermingled within a muscle, and the predominance of one over the other depends on the function of the muscle. Type I fibers, or slow-twitch fibers, are known as red fibers and are capable of a slow twitch and sustained or weight-bearing action. Type II fibers, or fast-twitch fibers, are paler (white fibers) and are capable of sudden action.

Muscle fibers are arranged in bundles (fasciculi or myonemes) of various sizes and patterns. Connective tissue, or endomysium, fills the space between fibers in a fasciculus. Each fasciculus or bundle is surrounded by a strong connective tissue sheath, the perimysium. The strong connective tissue sheath, the epimysium, surrounds the muscle body and is continuous internally with the other perimysial septa and externally with the connective tissue of surrounding structures (Fig. 4-3).

#### MUSCLE FORM

Muscles vary widely in size, shape, and complexity (Fig. 4-4).(13) They may be grouped according to orientation of their fasciculi or bundles, which can be parallel, oblique, or spiral relative to the final direction of pull at their attachments.

Where the fasciculi are approximately parallel to the line of pull, muscle bodies vary from flat, short, and quadrilateral to long, straplike muscles in which the individual muscle fibers often pass from one end to the other. Some straplike muscles have transverse tendinous intersections at intervals along the muscle belly (m. rectus abdominus). In fusiform-shaped muscles, there are similar, approximately parallel fasciculi.

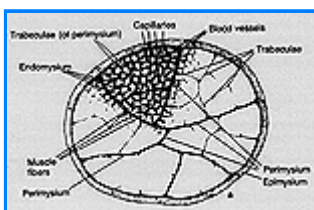


FIG. 4-3 Schematic drawing of connective tissue sheaths. (DeGirolami U, Smith, TW: Pathology of Skeletal Muscle Disease, Am J Pathol Teaching Monograph, 1982) pp 235-271

Where fasciculi are oblique to the line of pull, muscle shapes are triangular or pennate. The latter can be unipennate, bipennate, or multipennate. Because the line of force of muscle contraction is oblique to the line of pull of the tendons of insertion of the muscle, range of movement can be restricted, but considerable force of contraction can be developed.

Spiral muscles with fasciculi that are spiral relative to the final direction of pull very often tend to "despiral" when they contract.

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## MUSCLE ACTION

As stated previously, the rapid, repetitive synchronous stimulation of all, or even the majority of, motor units does not occur naturally, and physiological actions and movements are the result of a synchronous contraction of only a portion of available motor units. Therefore, at any one time during normal activity, the motor units in a muscle are in various physiological states, some relaxing, some undergoing stimulation, some contracting, and some quiescent. At the next point in time, the individual motor unit could well show a different activity. The proportion of units undergoing a particular activity, such as contraction, varies depending on the functional demand.

Kinesiology reveals that the action of muscles is exceedingly complex. In general, there is a "prime mover" in the initiation and maintenance of a particular movement, and an "antagonist" that can appose such a movement. In general, at the beginning of a movement, there is obviously activity of the prime mover, and there is a variable transient burst of activity in the antagonist. However, the antagonist then tends to remain quiescent until the final brief deceleration. However, the activity of prime movers is not unrestrained but is balanced against the various passive, inertial, and very important gravitational effects. One situation in which the events described above do not occur is seen in activities in which a part of the body is fixed in position. In this circumstance, obviously prime movers and antagonists can contract together. In some situations, a joint can be fixed by the interplay of prime movers and the force of gravity.



FIG. 4-4 The morphologic "types" of muscle based upon their general form and fascicular architecture. (Williams PL Warwick R: Gray's Anatomy, 36th ed. Philadelphia, WB Saunders, 1980. Copyright © 1980, Churchill Livingstone)

An important aspect of kinesiology is the prevention of unwanted movements by the action of synergic muscles, which can be seen to be partially antagonistic to the prime mover. For example, when a flexor muscle of the digits contracts, it can flex not only the digit (a desired activity), but also the carpus (usually an undesirable activity). Therefore, synergic muscles are activated to maintain the position of the carpus, or even to extend it.

Thus the majority of complex movements are the result of a finely integrated interplay of forces between prime movers, antagonists, synergics and fixators, and other forces such as inertia and gravity. The patterns of movement are continuously monitored by afferent nerve endings and continuously synchronized by analysis by and activity of the central nervous system. Even more complex kinesiological studies of joint and muscle mechanics have been described.(9-11)

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## BLOOD SUPPLY

Neighboring arteries provide muscular branches that usually enter the muscle together with the nerve at the neurovascular hilum. Additional arteries are usually present, and these enter the muscle at its ends. They branch into smaller arteries and arterioles, which spread into the perimysium, and these in turn give off capillaries that are situated in the endomysium. Although capillaries mainly parallel the muscle fibers, they form frequent transverse anastomoses, thus presenting a three-dimensional lattice. Numerous arteriovenous anastomoses are present in the epimysium and perimysium, and these circuits allow blood flow without passage into the endomysial capillary beds, passage that may be impeded during muscle contraction.(7)

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## HEALING MECHANISMS

### TENDON

**PHASES OF HEALING** Following surgical coaptation of a divided tendon, fibroblasts from surrounding connective tissue migrate into the wound, including the space between the tendon ends. The healing process follows the general pattern common to virtually all tissue, and the fibroblasts synthesize and discharge into the wound monomeric collagen and mucopolysaccharides needed for synthesis of mature scar. Rapid polymerization of monomeric subunits converts the initial fine reticular network immersed in a viscous ground substance into discernible fibrils and finally into a dense connective tissue scar.

At this early stage, the concept of a common wound throughout the healing area is an important one (Fig. 4-5).(14) The scar is uniform in consistency and physical properties in and around the severed tendon and throughout adjacent wounded tissue such as fascia, muscle, and even dermis. In long tendons, a gliding function will be restored by selective differentiation of various parts of the scar during secondary remodeling of scar tissue.

It is obvious that there are two aspects of tendon healing that distinguish healing of this tissue from healing of other tissues. The first is the tremendous tensile strength that must develop between the tendon ends. The second aspect is the development of a type of separation in the single scar such that the healed tendon can move or glide within the surrounding scar tissue.

Injury is followed by inflammation, a vascular and cellular response capable of defending the body against foreign substances, of disposing of devitalized tissue, and of initiating the mechanism responsible for repair. Although wound healing has been divided into processes, usually four, in fact the changes seen after surgery and in the healing process are the processes of inflammation. It is convenient to discuss a substrate phase, a repair phase, and a maturation phase.

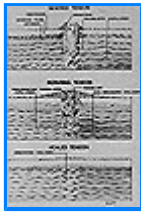


FIG. 4-5 The sequence of events following severing of a tendon. There is an initial hematoma between the tendon ends that is continuous with the general wound hematoma in injured surrounding tissue. (Rockwood CA, Green DP (eds): Fractures. Philadelphia, JB Lippincott, 1975) p 97-118

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#### SUBSTRATE PHASE (INFLAMMATORY AND DEBRIDEMENT PROCESSES).

- 1.) Immediately following injury, there is vasoconstriction of small vessels in the area and gradual vascular occlusion at the point of injury. This process tends to limit bleeding; however, the effect lasts for 5 to 10 minutes and is followed by active vasodilation.
  - 2.) At the injured site, blood flows into the gap created by the cutting instrument, fills the space, and clots, uniting the edges of the wound. Fibrinogen molecules from the blood quickly link up into interconnected strands of fibrin.
  - 3.) Almost immediately after injury, vascular, cellular, and fluid changes occur in the injured area. Small vessels dilate, leukocytes begin to adhere to endothelium, particularly of venules, and definite adhesion of erythrocytes and platelets occurs. Leakage of fluid from the venules begins through obvious "gaps" in the vessel walls. Leukocytes begin to move through the vessel wall by a process of diapedesis. This process carries nutrients, antibodies, and cells into the injured or healing area.
  - 4.) Beginning about 6 hours after injury, natural debridement commences. White blood cells migrate into the wound and remove and break down cellular debris, bacteria, and other foreign material. The first of these cells are the neutrophils, which can ingest organisms by phagocytosis. In a clean wound such as the one made by a surgeon, neutrophils have few bacteria to ingest. Under these circumstances, the cells degenerate and die. At this point, the outer membranes of the neutrophils rupture, and enzyme containing granules of various sizes pour into the wound. As the enzymes are released from the granules, they attack the extracellular debris at the site of injury. Within the first 12 hours after injury, monocytes begin to migrate into the wound. On entering the wound, monocytes become macrophages, phagocytic cells that remove most of the debris from the injured area by ingesting and then partially digesting this debris. Thus, inflammatory exudate, composed of escaping fluid from vessels, migrating leukocytes, and dead tissue, accumulates in the injured area, whether or not infection is present. The surgeon wants a minimal amount of this exudate in order that debridement can continue to remove all dead tissue, and proliferation can then proceed unhindered. The factors that determine whether the exudate is minimal or enough to constitute an abscess are the extent of injury to normal tissue, the extent of the cellular reaction, and the extent to which polymorphonuclear cells accumulate and die. Within limits, the inflammatory response shows a typical "dose-response" curve in relation to the severity of the trauma; therefore, it is desirable to minimize tissue injury. In addition, the state of the local tissue determines whether a healthy inflammatory reaction can occur and whether local tissues can effectively drain away accumulating fluid. The initiating factors in the inflammatory response are not clearly defined. It does appear that intracellular materials are released when cells are injured. These substances, such as histamine, serotonin, proteolytic enzymes, and biologically active peptides known as kinins, are responsible for the vascular dilatation, the increased permeability of venules, and the chemotactic effect on leukocytes.
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## REPAIR PHASE

Repair processes commence almost immediately after injury and proceed as fast as necrotic tissue, blood clots, and other barriers are removed from the injured area. In uncomplicated simple wounds, debris is usually removed by the third to fifth day, at which time fibroblast proliferation and capillary infiltration can commence in the wound area.

Many early workers postulated that wound fibroblasts arise from white blood cells. Current experiments have shown that the fibroblasts in a wound originate in undifferentiated mesenchymal cells in nearby connective tissue. The fibroblasts secrete the ground substances and collagen that form scar tissue, which eventually serves to replace the wound defect. The migrating fibroblasts appear to use the strands of fibrin as a scaffold, and the fibrin disappears coincidentally with collagen deposition. The removal of fibrin, as collagen is deposited, is due to the capillaries. The endothelial cells of new capillaries contain a plasminogen activator, such that fibrinolysis occurs and the fibrin network is broken down and removed as it is replaced by collagen. Initially, fibroblasts manufacture and secrete the protein-polysaccharides and various glycoproteins of the ground substance in the healing tissue. About the fourth or fifth day, collagen synthesis commences and continues at a very rapid rate. Thereafter a balance is reached until eventually collagen synthesis ceases. The fibroblastic phase of repair lasts 2 to 4 weeks, depending on the nature of the wound. At the end of this time, many capillaries regress and the number of synthesizing fibroblasts diminishes. The rate of total collagen synthesis decreases and eventually balances the rate of collagen destruction during the maturation phase of the wound healing.

New capillaries originate as budlike structures on nearby vessels, penetrate the wound, and flow into loops that ramify through the wound. The new tissue formed by the fibroblasts and the budlike capillaries constitute what is commonly referred to as granulation tissue. In the early stages of wound repair this network of capillaries provides large quantities of oxygen for the cells that are actively synthesizing protein in the wound. Before the new capillary network forms, there is a marked variation in the amount of oxygen within the wound, with the center of the wound being the most deficient. This gradient may be partially responsible for the branching of new vessels into the region. Once the continuity of the connective tissue has been reestablished, many of the new capillaries regress.

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## MATURATION PHASE

The final process in wound healing is maturation of the scar. In a healing wound there is normally an overproduction of collagen fibers, leading to a hypertrophied scar. As fibrogenesis proceeds, purposefully oriented fibers appear to become thicker, presumably because they accrue more collagen particles. Nonpurposefully oriented fibers seem to disappear at this time. This maturation and control appear to be the result of a delicate balance between collagen production and collagen destruction.

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## DEVELOPMENT OF STRENGTH IN A WOUND

In many descriptions of wound healing, the first stage is termed the "lag phase." It has been clearly shown that from a cellular and biochemical aspect, a lag phase does not exist; however, the term can be usefully preserved for the development of strength in a wound. In terms of overall healing, the term lag phase should be replaced by the term "production" or "substrate" phase.

During the lag phase of 4 to 6 days, wound strength does not increase appreciably. However, even in the first 24 hours after injury, a properly coapted wound has some strength. This is due to the formation of fibrin clot in the wound, and shortly afterwards, to some strength in the regenerating capillaries and in the newly formed ground substance, derived from serum glycoprotein that leaks into the wound from the circulation.

After 4 to 6 days, strength increases significantly to reach an early maximum strength at 14 to 16 days. This phase of wound healing is associated with the rapid period of fibroplasia and production of collagen. Hydroxy-proline, a measure of collagen concentration, increases rapidly beginning at day 4, with the highest rate between days 5 and 12, a lesser rate of increase between days 12 and 21, and a markedly lower rate from day 21 to day 60.

After the collagen content of a wound has stabilized, strength continues to increase as a result of cross-linking and reorientation of the already formed collagen fibers. The arrangement of collagen fibers in scar tissue is disorganized, and this is corrected to a degree during maturation; however, some disorganization persists. There is an almost imperceptible gain in strength for at least 2 years; however, the strength of the scar never reaches that of normal tissue. The development of strength in the healing tissue between tendon ends follows reorientation of the collagen fibers between the cut ends of the

tendon. It is obviously this same process that leads to restoration of the gliding mechanism. In remodeling of the scar tissue, collagen fibers surrounding the healing tendon will be subjected to less linear tension than the fibers between the tendon ends. Therefore, these fibers seem to disappear and be replaced by thinner and fewer fibers.

Throughout the history of tendon surgery, many methods have been used to prevent the formation of adhesions between healing tendon and surrounding healing tissue. These methods have centered on the use of artificial barriers between the tendon and surrounding tissue. Barriers have included blood vessels, fetal membranes, scar tissue, and several synthetic membranes including Millipore membrane. However, it has been shown conclusively that tendon healing is dependent upon migration of surrounding cells into the tendon defect, including fibroblasts, white cells, and cells forming capillary buds. In addition, oxygen and nutrients enter from surrounding tissue. Tendons contain longitudinally oriented blood vessels; however, these vessels are not capable of nourishing a healing tendon, and collateral flow is needed. Tendons contain some fibrocytes that appear to be inactive, and fibroblasts involved in healing come from the epitenon and paratenon and surrounding areolar tissue(15)

The most important factor in promoting an active gliding mechanism after tendon healing is not the prevention of adhesion formation between the tendon and surrounding tissue, but the reduction of scar tissue to the absolute minimum. This reduction requires minimal trauma, minimal hematoma and abscess formation, and rest to allow optimal healing to occur. It is not valid to consider the concept of "breaking down" adhesions as they form. This produces further inflammation and further scar tissue formation. A healed tendon with satisfactory gliding function is characterized not by the absence of adhesions but by the presence of adhesions that have been the source of fibroblasts and blood vessels and that now appear to have a length that is adequate to allow tendon gliding. There is no sudden breaking down of tissue around a healing tendon to allow gliding; rather, there is an almost imperceptible gradual gain in gliding motion as a result of slowly developing changes in the physical properties of the connective tissue scar. Among these changes, a change in length of bundles of collagenous fibers is probably the most important.

How a change in the length of fiber bundles occurs is not known, nor is it known whether longitudinal slippage occurs at a molecular, fibril, fiber, or fiber-bundle level. It seems more likely, for various reasons, that alteration occurs in the physical weave of relatively large subunits rather than in chemical cross-links. This aspect of tendon healing and maturation is discussed in depth by Peacock and Van Winkle.(14)

An important but unanswered question is why, during the healing and maturation process, new tissue between the tendon ends regains great physical strength and why new tissue that forms around this can be remodeled into loose tissue resembling and acting as paratenon. One suggestion has been the various linear forces that are applied to this developing scar. Peacock and Van Winkle have offered the suggestion that newly synthesized scar tissue remodels according to the architecture of the tissue that it adjoins. If this is so, it becomes clear why minimal trauma to the outside of tendons is a prerequisite for restoration of a gliding mechanism. Excessive trauma leads to destruction of paratenon tissue around the tendon, such that remodeling scar tissue is in juxtaposition with tendon tissue, not paratenon tissue. Healing of flexor tendons involves special features not found generally in healing of extensor tendons. First, many flexor tendons are surrounded by tendon sheaths and annular ligaments over part of their strength. Secondly, flexor tendons usually require more gliding function than extensor tendons. This aspect is much more important in humans than in animals. In the human hand, the amplitude of motion in the deep flexor tendon in the midpalm region is approximately 5 cm. In the dog the same tendon in the same site acts mainly as a weight-bearing structure with important but limited gliding function.

When tendon ends are left apart in loose connective tissue, the tendon stumps become atrophic and rounded. However, the ends are also embedded in the healing matrix filling the healing zone. At first this healing matrix is the gelatinous scar tissue, which eventually becomes fibrous with the production of collagen. Following production of this scar tissue, a remodeling of it occurs so that the portion between the tendon ends becomes longitudinally oriented and separates from the transversely oriented scar tissue. Thus a new tendinous structure appears to have formed in a new fibrous sheath, and it appears grossly that the bundles within the tendon have sprouted new tendon tissue in a manner similar to regeneration of a peripheral nerve. In truth this has not occurred. The new tendonlike tissue between the tendon ends is organized fibrous tissue that surrounds the rounded tendon ends and blends with the connective tissue surrounding the tendon and tendon bundles. As a result of an efficient secondary remodeling process of collagen fibrils between the tendon ends, the longitudinally oriented fibrils accrue more monomeric particles and become thicker and stronger, and fibrils that are not longitudinally oriented disappear. The collagen fibrils not between the tendon ends behave in a reverse fashion, such that transversely oriented fibrils become stronger and thicker. Thus a type of tendon sheath or paratenon is formed. The final product can be highly efficient reformation, not regeneration, of a tendon which, however, has little gliding function.

The reformed tendinous tissue does not in all instances rejoin severed ends of a tendon. In many cases, it will join the tendon ends to adjacent structures such as bone or joint capsule. A very efficient supporting structure can be formed that allows weight bearing without collapse of the foot. However, the flexor muscle is no longer connected to the digit or foot by a gliding tendon.

Tendons do not appear to contain cells with the potential to synthesize new collagen. Similarly, tendon sheaths do not contain these cells. When a tendon is ruptured within an intact sheath, an uncommon occurrence, the tendon ends become atrophic and rounded and do not rejoin. In the more common injury, both the tendon sheath and tendon are injured; therefore, the healing ends of the tendon are exposed to surrounding tissues that contain cells that can lead to reformation of the tendon, in the same manner as described above for tendon ends in loose connective tissue. Also, the tendon sheath can reform, and minimal gliding function can be present. The more common finding is limited gliding function, that is, many adhesions that can give good supporting function but no flexor function.

In human tendon surgery, restoration of gliding function in flexor tendons is achieved with great difficulty. In general, in the region of the flexor sheaths, a decision is first made between primary repair and secondary replacement, using a graft. Secondary repair is rarely done, since restoration of gliding function is rarely achieved by this means.

For primary repair to be successful in retaining gliding function, conditions must be ideal. These include a clean laceration, definitive surgical care within a few hours of injury, minimal contamination, superb facilities, and an experienced surgeon. These factors are rarely, if ever, present in animal surgery.

In human tendon surgery, it is agreed that the prognosis for restoration of gliding function in flexor tendons located within a tendon sheath can be excellent in a secondary replacement procedure. The accepted rule is to attempt to make the tendon suture line away from the injured surrounding tissue. This is the basis for secondary replacement rather than secondary repair. In the latter, the tendon repair zone is in the same plane or in the surrounding scar. In secondary replacement, the initial wound is allowed to heal. A new tendon is then found to bridge the scar area, and this tendon is placed in fresh tissue away from the scarred tissue. The anastomotic sites between graft and original tendon are away from the scarred area and, incidentally, away from the flexor sheaths.

Thus, the situation in humans is exceedingly complex owing to the importance of restoration of gliding function in flexor tendons. In animals the functional priorities are different, and, in general, supporting function is mandatory while gliding function has secondary significance.

As described previously, if two tendon ends are left apart in connective tissue, they are rejoined by reformed tendon that has little gliding function. Unfortunately, this "healed" tendon may also have little supporting function because it is now too long. Some contraction of the reformed tendon will, however, occur with scar maturation. Satisfactory supporting function and variable gliding function will be present if the tendon ends are sutured together or held together by an external splint that holds the leg in flexion. The end result will be a scar that actually joins together the tendon ends without tendon lengthening, or a scar that joins the distal tendon stump to adjacent bone or other firm structure. In either case, excellent supporting function can be preserved.

When a flexor tendon is divided within a tendon sheath in an animal, the situation should be assessed differently than in a human. It should be accepted that gliding function will be lost and that support is needed. Therefore, the choice of surgical repair is not between primary repair and secondary repair. Primary repair can involve surgical exploration with tendon suture or application of an external device to keep the severed tendon ends as close together as possible.

Unfortunately, primary repair that involves suturing is subject to the same restrictions in animals as in humans, including the need for a clean laceration with minimal contamination and an experienced surgeon. These factors are rarely present. Therefore, primary surgical repair of a tendon is almost always contraindicated in animals. Instead, the wound should be treated appropriately, closed or left open, and an external device applied with the limb in flexion. It is likely that successful primary surgical repair in animals can be attributed to the use of the supplementary splints or casts, and not to the skills of the surgeon.

Secondary repair of a tendon in animals is needed following failure of primary repair or when no primary care was given to the tendon. It is usually done in 2 to 4 weeks after the wound has healed. As stated previously, this is not recommended in

humans because gliding function is almost invariably lost. However, it is simple and direct and can be relied upon to give good supporting function. In general, the severed tendon ends are found embedded in the general scar tissue, which may already be showing some longitudinal orientation of collagen fibrils between the tendon ends. The tendon stumps are mobilized together with the interposed connecting scar tissue, and the tendon ends are brought together so that the original tendon length is restored. In some cases, the actual stump cannot be accurately approximated because of overall shortening of the muscle and tendon. The reformed tendon tissue can then be used for anastomosis. Tendon ends can be severed transversely and accurately apposed for tendon suturing, or they can be left long by using the reformed tendon tissue and overlapped for suturing. It must be remembered that since restoration of gliding function is usually a forlorn hope, neatness at the anastomotic site is secondary to strength. The ultimate goal is restoration of tendon length for support. If this involves restoration of gliding function, so much the better. Restoration of length for support is attained either by joining the distal tendon stump to adjoining firm structures, by new tendon tissue, or by a combination of both.

When primary repair by suturing is attempted, the surgeon should not attempt to repair the tendon sheath around the sutured tendon ends but should remove the fibrous sheath in the immediate vicinity of the tendon anastomosis.

One of the important considerations in tendon healing is management of the sutured tendon to produce maximum strength and minimum adhesions. Early post-surgical active motion has been advocated in an attempt to prevent formation of peritendinous adhesions and to form a type of bursal lining around the tendon and on the tissue adjacent to the tendon. However, this early active motion is not desirable. The healing tendon is not able to develop a blood supply from surrounding tissue, and dense collagenous adhesions are formed. Optimal healing is achieved by a 3-week rest period following surgery. The tendon ends receive a blood supply, healing takes place, and minimal scar tissue forms around the tendon. This peritendinous scar tissue is then remodeled when motion is resumed after 3 weeks. The remodeling process, as described previously, leads to the formation of paratenon-like tissue that allows gliding. The fundamental principle in postsurgery motion is not to rupture adhesions but to encourage secondary remodeling of scar tissue. After a 3-week period of absolute rest, a sutured tendon is not sufficiently strong that active motion and full weight bearing can be allowed. Increased collagen synthesis has been measured for as long as 35 days after tendon anastomosis. After 3 weeks of immobilization, the tendon should be supported by less vigorous external splintage for at least an additional 3 weeks. Continuous absolute immobilization beyond 3 weeks is probably not desirable. It can be shown that with continued immobilization there is not significant increase in tensile strength at 5 weeks as compared with that at 3 weeks. However, if active motion is allowed after 3 weeks of immobilization, the tensile strength at 5 weeks is three times as great as that at 3 weeks.(13)

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## SKELETAL MUSCLE

Wounds of skeletal muscle occur during surgical dissection and by lacerating wounds, spontaneous ruptures, and contusions. Muscle tears and ruptures are particularly common in racing greyhounds.(5,19)

Wounds of skeletal muscle can heal both by fibrosis and by regeneration of myofibrils. If the edges of the muscle wound are displaced, healing is by the usual fibrous protein synthesis and formation of scar tissue. This scar tissue has the ability to strangle the myofibrils and prevent regeneration. Between muscle ends the scar tissue can remodel and elongate to such an extent that muscle function is reduced.

If the edges of a muscle wound are carefully debrided of dead and devitalized tissue (in a fresh wound) or of scar tissue (in an old wound) and are approximated by sutures, optimal healing can be obtained by fibrous tissue formation and regeneration of myofibrils. Muscle does not heal by cell division but by myofibril regeneration across the defect. Fibers on each side of the defect break up into nucleated cylinders of cytoplasm. Macrophages remove dead material but leave the basement membrane intact. The muscle fiber cylinders now fuse and grow back inside the original basement membrane to form a myotube, until eventually the two growing undamaged ends fuse and fill this gap(1-3) The surviving portion of the muscle begins to form these fine outgrowths in 6 to 7 days, and cross striations can develop in 8 to 14 days. The rate of muscle regeneration by this type of sarcoplasmic outgrowth appears to be about 1.5 mm/day.

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## LIGAMENTS

The basic concepts of ligament healing resemble those of tendon healing; however, the process is simpler, since gliding is not involved.

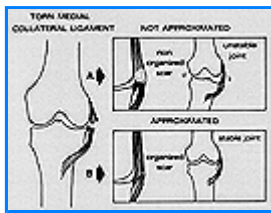


FIG.4-6 Surgical approximation of a severed ligament leads to a ligament that more closely approximates the surgical structure and usually gives better function. (Rockwood CA, Green DP (eds): Fractures. Philadelphia, JB Lippincott, 1975) p. 97-118

Under suitable conditions, injured ligaments have the ability to reform a structure that very closely approximates the original structure: connective tissue in which the fibers are regularly oriented with respect to one another to form a thick bundle. Healing is by formation of collagen and remodeling of this scar tissue. Fibroblastic activity comes from the ligament itself as well as from surrounding tissues. As in tendon healing, the "one wound" concept prevails, and healing proceeds in the wounded ligament and the wounds in surrounding structures as in a wound of a single structure.

If the ligament ends are separated, a gap of irregular fibrous tissue is formed between the ends. If the ends are sutured carefully, an organized scar develops in which collagen fibers are regularly oriented. The tensile strength of this organized scar exceeds that of the irregular fibrous tissue between separated ends of a ligament. In addition, if ligament ends are allowed to heal in a separated mode, the resulting healed ligament is not only weaker than a sutured ligament, it is also longer. This leads to instability of the joint supported by the ligament (Fig. 4-6).

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