Ligament Healing: Current Knowledge and Clinical Applications

Cyril B. Frank, MD, FRCS(C)

Abstract

The treatment of ligament injuries, particularly knee-ligament injuries, has occupied a substantial portion of the orthopaedic literature for several decades. It remains unclear, however, what orthopaedic surgeons can do to optimize the recovery of patients with ligament problems. In this review, the reasons for this lack of clarity are proposed, and the current state of laboratory knowledge about the response of isolated and multiple ligament injuries to various treatment modalities is reviewed for the ligaments that have been studied thus far (all of which are in the knee). In general, it appears that ligaments heal with scar tissue similar to that involved in skin-wound healing. The early controlled motion of stable (or surgically stabilized) joints appears to improve ligament scar behavior, but no treatment identified to date stimulates true ligament regeneration.

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Medically treated joint sprains and dislocations (in which, by definition, ligaments are either partially or completely torn) account for roughly 45% of all musculoskeletal injuries and annually affect 5% to 10% of persons up to the age of 65.[1] In North America, these data translate into the annual treatment by healthcare providers of more than 30 to 40 million ligament injuries. If the very prevalent back strains and sprains, in which ligament injuries are difficult to define, are excluded from these numbers, there remain in excess of 15 to 20 million reported injuries to other joints each year in the United States alone. Even according to these cost-related estimates, ligament injuries are extremely common. It is probable that far greater numbers of persons with minor ligament sprains never seek medical attention, since these injuries are considered to represent a relatively minimal risk of limiting future activity and appear to heal well.

Unfortunately, beyond the fact that for most joint sprains clinical symptoms resolve fairly quickly, it is not clear how well ligament injuries actually do heal. What is clear, however, is that some of the more major ligament injuries do not have benign clinical courses and cause a significant percentage of those patients who seek medical attention to require the care of an orthopaedic surgeon.[1] To the surgeons who deal with the latter group of patients, it remains somewhat mysterious why only a subset of patients with ligament injuries appear to be disabled, why some ligaments in particular fail to heal, and what clinical principles, if any, govern the success or failure of various clinical approaches. Specifically, it remains unclear what orthopaedic surgeons can do to optimize the recovery of their patients, with or without surgery.

In this review, I address part of the deficiency by summarizing what is known about both normal and healing ligaments from basic science and clinical perspectives. The complex topic of ligament grafting is not discussed; however, readers should be aware that many of the processes of graft healing appear to be similar to those described in this review.

Normal Ligament Function in Joints

An understanding of the role that uninjured ligaments play in joint function is important because there are a number of reasons that can explain why certain ligament injuries appear more clinically disabling than others. A discussion of three of these reasons will help put the subsequent discussion of ligament healing in an appropriate context.

Dr. Frank is Alberta Heritage Scientist and Professor of Surgery and Chief, Division of Orthopaedics, University of Calgary Faculty of Medicine, Alberta.

Reprint requests: Dr. Frank, McCaig Centre for Joint Injury and Arthritis Research, 3330 Hospital Drive NW, Calgary, Alberta, Canada T2N 4N1.

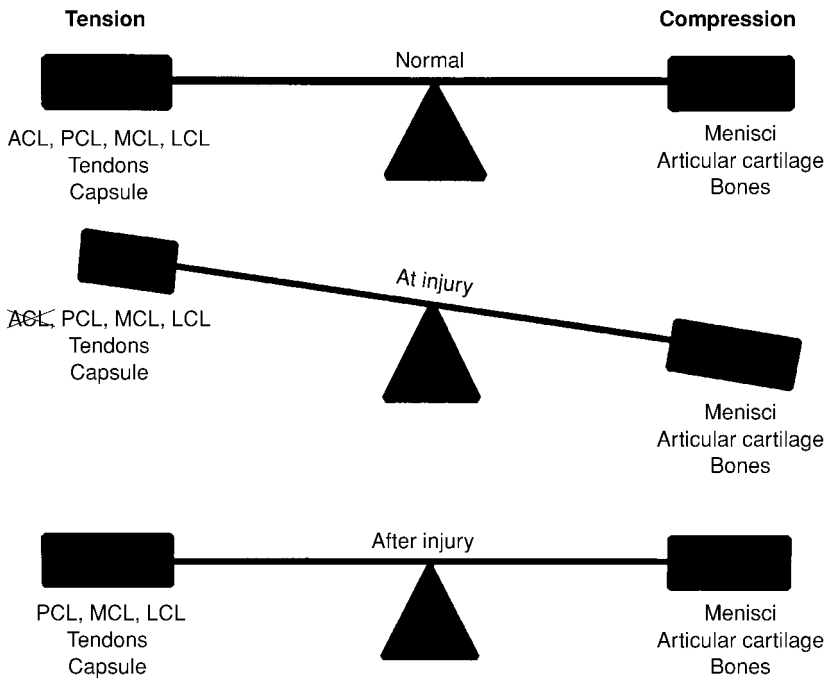

Fig. 1 The balance between the tension-bearing elements and the compression-bearing elements of a knee joint. **Top,** In the uninjured knee joint, the tensile and compressive elements are in equilibrium. **Center,** Immediately after failure of one or more structures, imbalance occurs instantly. **Bottom,** To restore joint function, the forces must be redistributed among the remaining structures, thus increasing the stresses on the remaining structures and predisposing some of them to subsequent failure. ACL = anterior cruciate ligament; LCL = lateral collateral ligament; MCL = medial collateral ligament; PCL = posterior collateral ligament.

First, a number of elements complement ligaments in the provision of normal joint stability. These elements include extrinsic and intrinsic loads, bone and cartilage geometry, bone and cartilage mechanical properties, joint pressures, central neural control of balance and muscle function, and feedback mechanisms between complex neuromuscular events. Problems with any of these elements, which are independent of the normal functioning and healing ability of ligaments, can predispose a joint to injury or reinjury. These factors can also have a compensatory role after injury and are at least indirectly involved in ligament healing.

A second factor involves ligaments, but not in the traditional sense. It has been speculated for many decades that ligaments (and other joint tissues) function as neurologic feedback sensors. This speculation is based on evidence that ligaments contain proprioceptive-type neural elements that appear to generate neurologic impulses on stretching.[2] However, there is mixed evidence concerning the possible loss of proprioceptive elements in joints after ligament injury. Furthermore, there has been only a preliminary investigation into the contribution of individual ligaments to proprioceptive losses and into neural regeneration in ligament healing. Therefore, it is not yet possible to comment definitively on the proprioceptive role of individual ligaments or on the role of proprioceptive abnormalities during the healing process.

Third, while we tend to assume that ligaments perform nearly independent roles as passive joint stabilizers, this is almost certainly not the case (Fig. 1). It is useful to think of diarthrodial joints in a state of relative mechanical equilibrium. To maintain equilibrium, there must, by definition, exist a balance between the compressive elements and the tensile elements in a joint. The tensile elements are primarily the ligaments and the tendons. During normal joint function, the tensile load is constantly redistributed among the tension-bearing structures as a function of muscle forces, extrinsic forces, and joint position.[3] If one or more of the many tensile elements that cross a joint become damaged, the tensile loads must be redistributed and carried by the remaining tensile structures. If the remaining tensile structures are able to carry the necessary load without failing in any way, the joint will remain functional; if not, the joint will not function.

For several reasons, these three concepts are critical to our understanding of normal ligament function, ligament healing, and joint rehabilitation. First, most ligaments appear to heal well because load-sharing and other dynamic mechanisms of compensation make it possible for a ligament-injured joint to function without symptoms despite the complete failure of true ligament healing. Therefore, it must be emphasized that asymptomatic joint function is not evidence of good ligament healing.

Second, the concept of load sharing suggests that immediately after a ligament injury, loads will be transferred to other structures. If the transferred loads are beyond the physical or biologic limit of those structures, they will ultimately fail. This explains the incidence of slow failure among the secondary stabilizers of various joints, with or without surgical repair or re-

construction. Similarly, load sharing helps explain why there is a higher incidence of failed healing of all ligaments in a joint when more than one has been damaged. Also explained is why the sooner a ligament resumes its load-carrying role (i.e., by healing or surgical replacement), the less chance there is that the joint system in question will fail.

Third, load sharing and load redistribution are significant because the aim of joint rehabilitation must be to stimulate the rapid recovery of the tensile properties of injured ligaments without overstressing the compensating structures. The concepts of load sharing and load redistribution also help explain the high incidence of low-grade failure in compensatory structures (tendons in particular) during the rehabilitation process.

Normal Joint–Ligament Complex Mechanical Function

It is well accepted that ligaments play a significant mechanical role in joints. Ligaments serve to stabilize joints throughout their range of motion and to guide the bones during that motion. There is now abundant evidence to suggest that ligaments work together in any given joint, with different components of each ligament functioning during particular portions of joint movements. While certain ligaments serve major functions, they do so with the assistance of other ligaments. For example, the anterior cruciate ligament (ACL) of the knee controls mainly anterior tibial translation relative to the femur. This concept has broad implications in terms of the mechanisms of ligament injury, combinations of ligament injury, compensation patterns, and healing. For example, ligaments that function fairly independently, and thus have fewer compensatory structures, will probably be more susceptible to isolated injuries and will heal less easily because they will be subjected to unrealistic compensatory conditions. The best example of this concept is clearly the ACL.

Given that it is critical for any healing response to fully restore at least the joint-stabilizing properties of each ligament, four aspects of ligament mechanical function—laxity, stiffness, strength, and viscoelasticity—must be considered major outcome measures of healing success. Researchers are just beginning to be able to quantify these properties accurately under very controlled laboratory conditions. While it is very difficult to use these properties quantitatively as measures of outcome success or failure in the clinical setting, clinicians should realize that these are nevertheless still the best measures available to establish the relative efficacy of any treatment. It is important to understand exactly what the four above-mentioned terms mean, in order to describe the specific mechanical characteristics in normal and healing ligaments.

"Laxity" refers to the displacement of the bones to which a ligament is attached from an anatomic position to a position in which the ligament takes up a tensile load (Fig. 2). Laxity is, therefore, a function of both joint position and direction of load. For example, a Lachman test is a test of anterior tibial translational displacement (anterior laxity) from an anatomic position at about 20 degrees of knee flexion. In a laboratory setting, these anatomic positions and displacements can be measured more accurately for isolated structures; however, the basic concept is the same in the clinical setting, in that forces are applied and displacements are measured, and vice versa. In the laboratory, the amount of displacement measured until an iso-

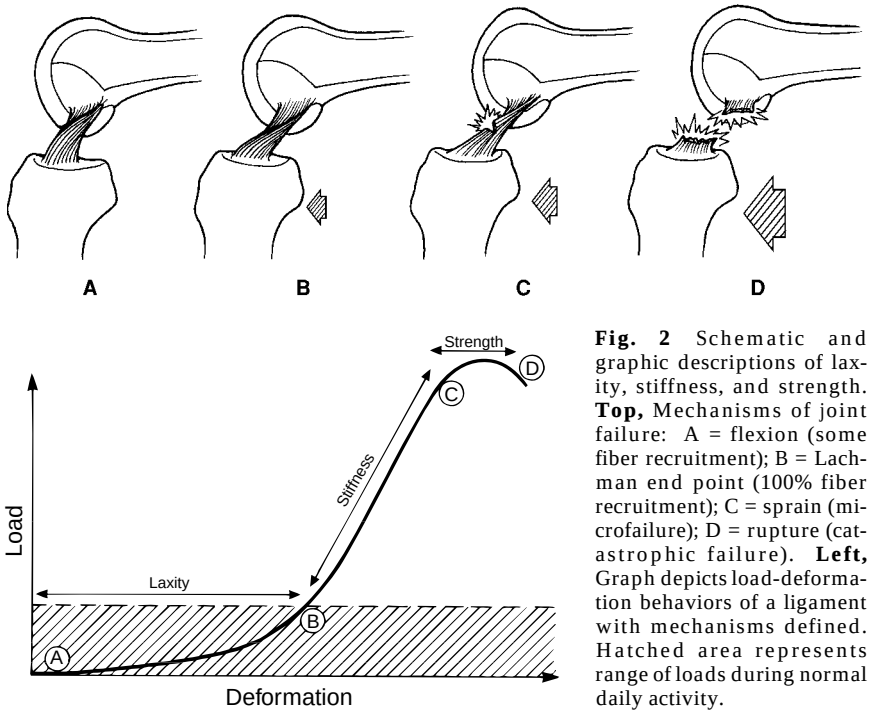

Fig. 2 Schematic and graphic descriptions of laxity, stiffness, and strength. **Top,** Mechanisms of joint failure: A = flexion (some fiber recruitment); B = Lachman end point (100% fiber recruitment); C = sprain (microfailure); D = rupture (catastrophic failure). **Left,** Graph depicts load-deformation behaviors of a ligament with mechanisms defined. Hatched area represents range of loads during normal daily activity.

lated ligament takes up a tensile load on a test machine has been defined as its laxity. From a structural point of view, the laxity of isolated ligaments is partly a function of collagen-fiber organization (as fibers are recruited to resist ligament displacement) and partly a function of the number of fibers recruited by a specific movement.

"Stiffness" refers to the amount of load required to displace the bones to which a ligament is attached in a particular direction. The more load required, the stiffer the ligament-joint complex. As with laxity, stiffness is a function of fiber recruitment; the more fibers recruited, the stiffer the ligament. The so-called end point of a Lachman test, for example, refers to a sudden change in ligament stiffness as fibers are recruited (Fig. 2). In a damaged ligament, either fibers are not recruited or the fibers that are recruited are not as stiff as normal ligament fibers. In either case, no end point would be felt during the clinical test. A lack of stiffness allows knee joints to subluxate under some load conditions, which potentially accounts for the symptom of instability.

"Strength" refers to the maximum tensile load that a bone-ligament-bone complex can withstand before it fails. As with ligament stiffness, failure load is a structural property. A large ligament would have greater structural strength than a small ligament. Failure load is a function of both the number of fibers that tighten within a ligament when it is stretched and the quality of those fibers. Failure load is also a function of the direction of tensile load in relation to the fibers. Load direction is thus a critical determinant of ligament strength. The material, or stress-strain, properties of ligaments (i.e., the mechanical qualities of the ligament tissue alone, as opposed to their structural properties) are normalized to the cross-sectional length and width of a ligament. A very large ligament, for example, would have essentially the same material properties as a very small ligament despite being much stronger structurally.

"Viscoelasticity" refers to the ability of tissues to respond to repetitive loading by altering length or load over time (Fig. 3). Viscoelastic variations can account for as much as 10% of changes in ligament length and up to 60% to 70% of changes in ligament loads under physiologic loading conditions. Although viscoelasticity has not been emphasized in the analysis of normal and healing ligaments, this mechanical property provides a fine-tuning mechanism for each ligament and for the joint as a whole, as loads and displacements become readjusted during loading.

Clinical Studies of Ligament Healing

In a review of current knowledge about ligament healing, it is impor-

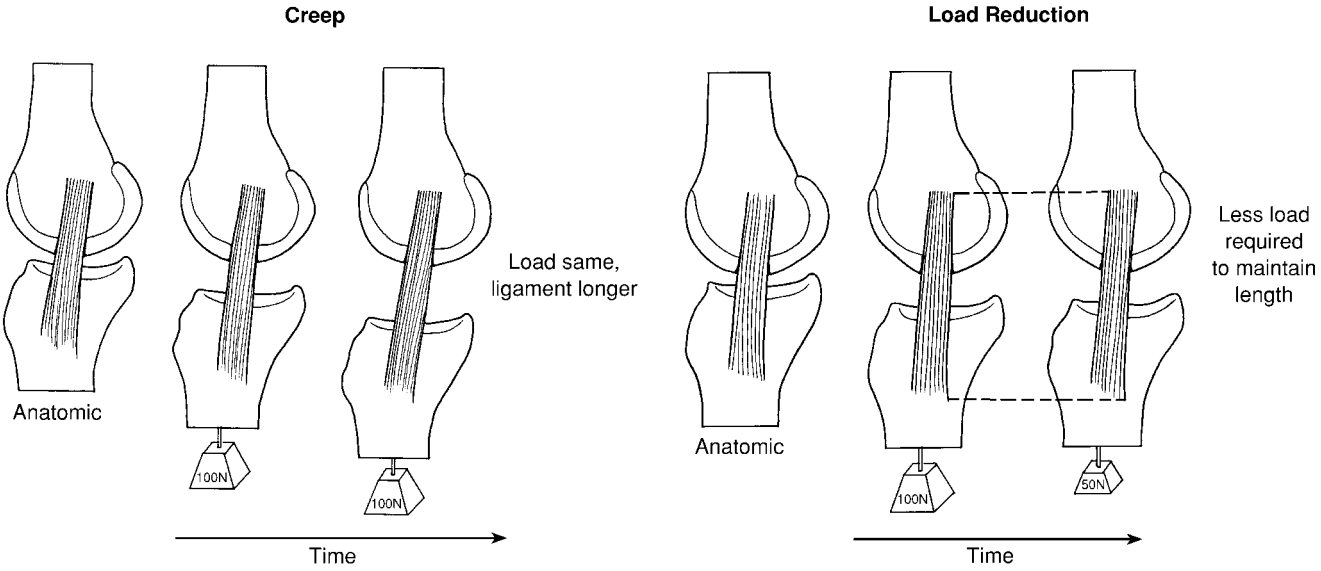

Fig. 3 The viscoelastic properties of ligaments. **Left,** Creep is the increase in the length of a ligament that occurs over time when a ligament is subjected to a constant load. **Right,** Load relaxation is the decrease in load that a ligament experiences over time when it is held at a specific deformation.

tant to note that there exists a vast volume of literature on virtually every aspect of ligament injury from the clinical perspective. Numerous articles review clinical experiences of ligament healing and provide important data on treatments that appear to work.[4-6] However, the literature does not discuss ligament healing itself, but rather focuses on those joints that, for a number of reasons, become functionally disabled by ligament injury and the forms of clinical treatment that can prevent or mitigate those disabilities. Perhaps most important, the literature provides strong evidence that decisions made by orthopaedic surgeons can and do alter the natural history of ligament injuries.[7] Further, it is clear that, with or without surgery, the early mobilization of many ligament-injured joints, combined with controlled rehabilitation, can restore joint motion, apparently without compromising joint stability.[6,8] Unfortunately, beyond these two major points, there are very few principles on which the clinician can depend. In truth, the natural history of untreated ligament injuries remains an enigma in the clinical setting, and definitive conclusions about surgical and rehabilitative treatments (e.g., splints, braces, and exercises) continue to be clouded by patient, injury, and treatment variables.

In the laboratory setting, some of these confounding variables have been controlled, and the effects of some treatment modalities have been tested on isolated healing ligament tissue. Although laboratory simulation of ligament healing entails its own problems, it has provided insights into what clinically relevant treatments can and cannot do at the tissue level.

Models of Healing

This section concentrates on mechanical elements of healing, because these can be related fairly easily to clinical perceptions of ligament repair. Information on the microstructural, biochemical, and metabolic factors involved in the healing responses of ligaments can be found elsewhere.[9]

The various animal models used to study ligament healing over the past 65 years have concentrated almost exclusively on the knee joint, specifically, on injuries of the medial collateral ligament (MCL), lateral collateral ligament, and the cruciate ligaments (mainly the ACL). These injuries have been studied alone and in combination, as well as with and without the use of various treatment modalities. Cruciate ligament healing has been regarded as unique, while collateral ligament healing has been considered more representative of extra-articular ligament healing in other joints. These assumptions, however, have not really been tested. In addition, animal studies of ligament healing almost universally involve either adolescent animals or young adults. Therefore, the effects on ligament healing of age and variations in metabolic rates have not been investigated. Furthermore, because of the differences in animal size, ligament-loading patterns, and rehabilitation techniques used in the various models, it is difficult to compare findings between studies. Thus, caution should be exercised before one extrapolates the following results directly to the clinical setting or, indeed, to all ligaments; rather, the information is offered to provide some understanding of the basic processes and principles of ligament healing.

Results

Isolated ACL Injuries

With or without primary surgical repair and with or without immobilization, severed ACLs in stifle joints in young adult quadrupeds have been shown to heal inadequately. Either the repaired ligaments rupture and the ruptured parts become resorbed, or they attenuate into structures that are lax, less stiff than normal, and unable to withstand more than a fraction (10% to 60%) of normal tensile loads. Why the ACL fails to heal more functionally has yet to be determined, but suggested reasons include both biomechanical factors (e.g., excessive stresses) and biologic factors (e.g., poor blood supply and synovial fluid inhibition).

It is important to note that cartilage abnormalities and osteophytes—changes similar to those that occur in human osteoarthritic conditions—develop in most ACL-deficient animal joints, irrespective of treatment modality. Indeed, the time it takes for these changes to occur in animals (weeks to months), compared with that in humans (years, if at all), indicates that quadrupedal animal models of ACL healing may represent a worst-case scenario for ligament healing. Interestingly, the one study of primary ACL repair in monkeys[10] indicated that healing was better in that species (63% of control strength after 4 months of healing); although some ACLs remained lax and some joints showed signs of degeneration, the data suggest a potentially important interspecies difference in ACL healing that requires further study.

Many reviews imply that ACLs do not heal without treatment, because it has been observed that many ACL injuries do not heal functionally. On the contrary, clinical and animal evidence indicates that, if the definition of healing is expanded to one in which a gap between the torn ends of a ligament fills with new tissue, a number of torn ACLs do "heal." In humans, many torn ACLs attach, by means of scarlike material, to the posterior

cruciate ligament. Although a number of these reattached ACLs are not functional, this may be due to their abnormal attachment site rather than to a failed healing response per se. Interestingly, the response of partially transected ACLs has recently been studied in some animal models,[11] and the results have confirmed that some healing, albeit inadequate, occurs.

Isolated MCL Injuries

In direct contrast to the findings in ACL injuries, both clinical experience and animal studies of MCL injuries have indicated a relatively good, but seldom perfect, healing response. In various animals, gaps between the cut or torn ends of MCLs have been shown to quickly, almost routinely, fill with what, on the basis of many descriptors, is considered to be scar tissue. These MCL scars undergo the same process as scars in healing wounds, namely, bleeding and clot formation (minutes to hours), inflammation and formation of granulation tissue (days to weeks), scar proliferation (weeks to months), and scar remodeling (months to years). A few early studies optimistically noted apparent ligament regeneration,[12] but subsequent studies have shown that, while normal ligament strength may nearly recover in some instances,[13] this is not due to the formation of true ligament tissue. Instead, normal strength is achieved by the accumulation of a larger than normal mass of inferior-quality scar tissue. Although the strength and stiffness of injured collateral ligaments have been restored to 40% to 90% of normal values in these animal studies, only about 30% to 70% of the material strength has returned. The reasons for scar inferiority include a variety of subtle but important factors (Table 1).

Other properties appear to recover somewhat better than strength and stiffness. Viscoelastic properties have not been studied in great detail but appear to return to 70% to 90% of normal values fairly quickly at low loads and displacements. Neither load relaxation nor creep has been studied thoroughly; however, it does appear likely that both measures remain abnormally high for a significant period of time after injury. In contrast, ligament laxity, under ideal circumstances (single injury, movement allowed), recovers completely over a period of several months. If scar mass is sufficient and subsequent joint loads are not excessive, it appears that ligament-scar contraction, analogous to skin-scar contraction, restores normal joint stability. If, in fact, ligament scars prove analogous to scars in skin, then all of the better-known principles of skin wound healing can be applied to the treatment of ligament injuries. In particular, the more aggressive scar response observed when the closure of wounds is delayed (the so-called secondary healing response) may have great significance in the clinical treatment of ligament injuries and arthrofibrosis. This speculation clearly requires further investigation.

Collectively, evidence suggests that, in an extra-articular environment, ligaments heal with scar material very similar, if not identical, to that described in the wound-healing literature. The scar, while not as good as normal ligament tissue in terms of high-load behavior, performs well under low loads. Unfortunately, evidence also shows the maturation and remodeling processes of ligament scars, even in young animals, to be slow, taking months to years. It also appears unlikely that ligament scars, even under optimal conditions, will ever match the exact quality of normal ligament tissue without major manipulation.

Multiple Ligament Injuries

Combined ligament injuries in animal models have shown a poorer functional ACL healing response than isolated ACL injuries. The ACL attenuation and failure rates are higher in joints with multiple ligament injuries.[14] Collateral ligaments also do not heal as well in joints with multiple ligament injuries. Measurements of high-load structural strength and stiffness are lower than in isolated injuries at comparable healing intervals. Furthermore, the material properties

Table 1
Differences Between Normal Ligaments and Scars

Normal (Uninjured) Ligaments	Ligament Scars
Collagen aligned	Collagen disorganized
Collagen densely packed	Defects between collagen fibers
Large collagen fibrils	Small collagen fibrils
Mature fiber cross-links	Immature cross-links
Primarily collagen type I (<10% type III)	More collagen type III
Small proteoglycans	Some large proteoglycans
Other components minor	Excesses of other components
Cell metabolism low	Cell metabolism high
Low cell density	Increased cell density
Low vascularity	Increased vascularity

of injured collateral ligaments in ACL-deficient knees are poorer than those in isolated injuries because the scars that form are generally larger. The increase in scar mass, however, does not compensate for collateral scar length, which is consistently greater in the unstable joint than in the stable joint. The MCLs that heal in ACL-deficient knees have also been shown to be more lax than those in joints with an intact ACL.[14,15] This looseness appears to correlate grossly with the rapid progression toward osteoarthritic-type changes in the joint.[14]

Effects of Various Treatments

Suture Repair

On the basis of available evidence in a model in which gap size was the only variable, it would appear that, in otherwise stable knees, the immediate closure of gaps between torn collateral ligament ends can increase their structural strength by 10% to 30%.[16] The improvement appears to be caused by local differences in scar remodeling, in that very large gaps become filled with more disorganized scar tissue, which contains a greater amount of material not found in uninjured ligaments (e.g., fat cells and blood vessels). The suturing of cut or torn ends serves to fill the gap with dense (old) ligament material, thus eliminating the need for the ends to be reconnected by scar. Unfortunately, no other mechanical improvement in ligament scars (including laxity and viscoelastic properties) has ever been attributed to suture repair.

The evidence from animal studies suggests that suture repair of collateral ligaments in ACL-deficient knees does not improve the structural, material, or viscoelastic properties of the tissue. The collateral ligaments remain relatively lax, but with good strength and stiffness. Repair, it seems, does not add enough stress resistance to the MCL in the ACL-deficient animal knee to make a significant difference in its healing behavior.

In contrast, it has been suggested that the suture repair of ACLs adds some structural or functional stability to some ACL-deficient knees, both in animal models and in humans. Although in a canine model the resorption of ACL stumps occurred almost universally in cut ACLs without suture repair,[17] more consistent integrity was found in repaired ACLs. Unfortunately, this integrity is not particularly substantial mechanically and does not prevent ACL laxity or attenuation. This observation appears to correspond with clinical experience, in which primary ACL repair has generally failed over time.[18] Nevertheless, a percentage of ACL repairs do result in clinical success.[19]

Collectively, these findings support the notion that the suture repair of ligaments does modify some of the local events involved in the healing process and that it has a beneficial role in the treatment of injured ligaments (Fig. 4). Unfortunately, the effects are generally not what some clinicians would perceive as being of major or consistent benefit in terms of the mechanical properties of any healing ligament studied thus far. In particular, suture repairs have not been shown to affect the laxity, stiffness, or strength of any ligament in more than moderate amounts. If this principle is transferable clinically, the risk-benefit ratio of these modest improvements must be considered carefully before recommending acute ligament repair.

Immobilization

Joint immobilization has a number of significant effects on both the joint and its healing ligaments. As documented elsewhere in the literature, the prolonged immobilization of any joint (normal or injured) can damage its articular surfaces, change the shape of the associated bones, and induce adhesions both within the joint and within the nonligamentous periarticular tissues. These changes can cause the joint to lose mobility and to become stiff. The immobilization of joints with combined ligament injuries also involves some of these early degenerative changes, particularly in the canine model.[14]

The immobilization of an injured joint has specific effects on healing ligaments. First, immobilization has been shown to protect some ligament repairs grossly, particularly ACL repairs in animal models. A larger number of ACLs have healed in continuity after primary repair plus immobilization in a canine model[17]; however, as previously noted, their mechanical quality has not been very good.

The second effect of immobilization is that it causes isolated ligament scars to be less stiff and significantly less strong than scars in joints that have been allowed to move.[13,15,20] This weakness is related to the duration of immobilization (longer immobilization is more inhibitory), with only a few weeks of immobilization soon after injury having permanent inhibitory effects on ligament strength.[20] Reasons have not been totally elucidated, but definitely include the inhibition of collagen production and the inhibition of matrix remodeling.

The third effect that immobilization has on healing ligaments is the potential minimization of scar length (and thus ligament laxity). In rabbit knee joints with combined MCL and ACL deficiencies, immobilization helps prevent the formation of MCL scars in lengthened positions, thus minimizing MCL laxity in the first few months of healing.[21] In the same model, immobilization at least temporarily protects the joint from the destructive changes of osteoarthritis. Interestingly, this is not the case with canine knees, in which the immobilization of an isolated

MCL injury for 3 to 6 weeks is associated with increased varus-valgus joint laxity, even after several months of remobilization. The immobilization of joints with combined MCL and ACL injuries results in more laxity and degenerative changes after 2 months of healing.[14]

The reasons for the effects of immobilization noted in these animal models remain unclear. However, there is no evidence to suggest any long-term benefit of immobilization (Fig. 4).

Movement

Joint movement can encompass a huge spectrum of loads, stresses, and load histories. At one extreme, there is minimal joint movement (thus, minimal stresses on healing structures); at the other extreme, there are clearly excessive and disruptive stresses. While certain implantable devices have been used successfully to document relative ligament forces or strains under various loading conditions,[22] it is not yet possible to accurately and reproducibly measure stresses on healing ligaments in vivo. Therefore, the safe-stress spectrum has not been defined for any model of ligament healing. Unfortunately, a quantitative commentary on the principles of movement governing ligament healing is still impossible.

There appears to be one major principle regarding joint movement during the ligament-healing process, namely, the application of controlled movement in which stresses on injured ligaments are low (Fig. 4). Controlled movement has been shown to stimulate improved scar stiffness and strength without compromising scar length.[23,24] This is particularly true in otherwise stable joints in which only one ligament has been injured or in which a number of other stabilizing elements exist. The principle is also theoretically true in joints with multiple ligament injuries, but in these joints it is much harder to minimize stresses during joint motion. In joints in which the kinematic properties have been restored by a reconstructive procedure (e.g., ACL reconstruction), joint motion can probably be initiated with slightly less concern, as grafts should carry some tensile load during motion, to help prevent abnormal stresses on the other ligaments.

The mechanisms by which movement stimulates ligament-scar formation remain debatable but probably include both local effects (on scar cells) and regional effects (e.g., blood flow and inflammation). Movement has been shown to stimulate collagen synthesis and certain aspects of ma-

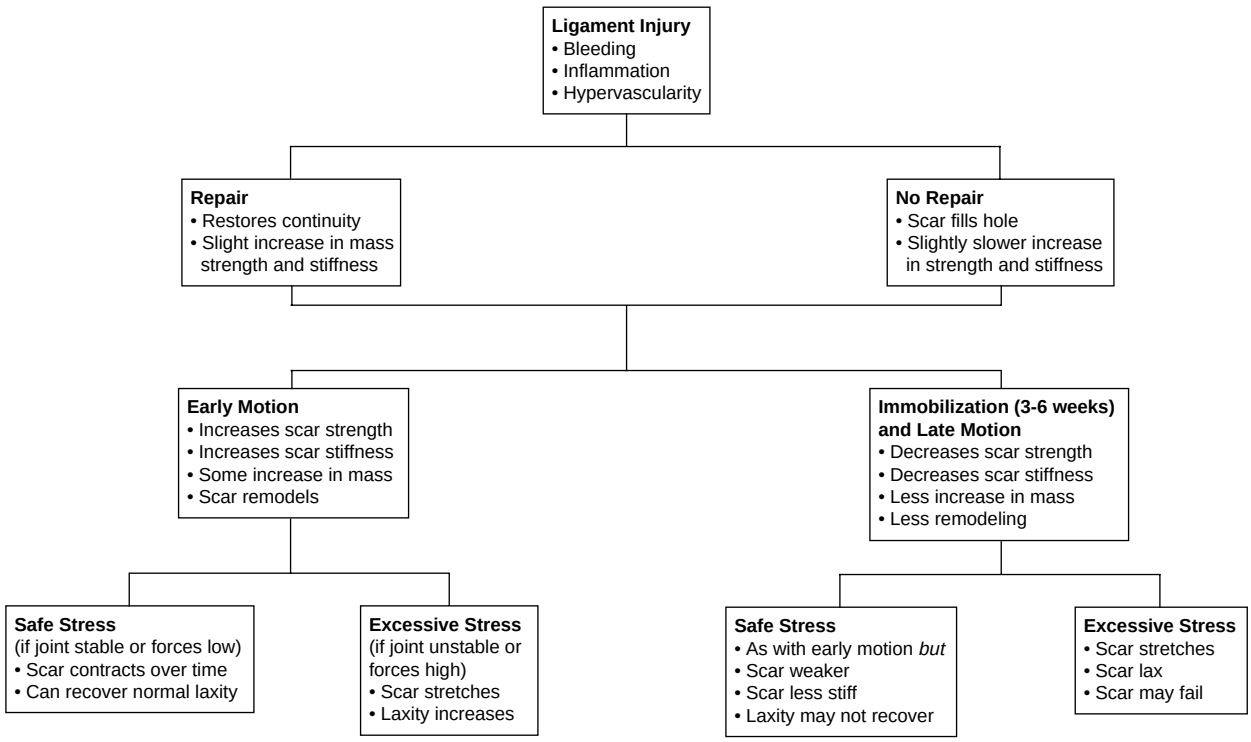

Fig. 4 Algorithm depicts the gross advantages and disadvantages of various treatment modalities.

trix remodeling and to produce scars of slightly better quality compared with those produced with immobilization. Movement also stimulates the production of increased scar mass and thus an increased amount of material to resist the tensile stresses involved. These mechanisms appear to be more active when early scars are mobilized (within weeks of injury) rather than when inflammation has begun to subside. The later introduction of movement, while still stimulatory to scar remodeling, does not appear to have the same potential to improve scar mass.[20]

Immobilization Followed by Movement

As already noted, movement initiated after a few weeks of immobilization does not have the same potential to increase structural strength or stiffness in scar tissue as movement started immediately.[20,24] Although immobilization confers temporary advantages in terms of reduction of ligament laxity, it appears that this benefit is lost over time. In terms of ligament healing, as opposed to the control of clinical symptoms (e.g., pain and swelling), there appears to be no advantage to a significant period of joint immobilization after a ligament injury in animal models (Fig. 4).

Summary

The ligaments are part of a joint system with very complex systemic and local control mechanisms. These mechanisms no doubt influence both the clinical symptoms and the mechanisms of ligament repair. Evidence from animal models of ligament-tissue healing supports current clinical trends. This evidence suggests that if a ligament-injured joint is not too unstable, early motion of the joint should promote scar formation and subsequent scar remodeling. If a ligament-injured joint is grossly unstable due to multiple ligament injuries, the joint may be reduced and partially stabilized by surgical repair, reconstruction, or both, followed by the careful application of controlled motion to stimulate the scarring process. Immobilization, while potentially benefiting ligament healing in the short term, appears to have long-term negative consequences. Unfortunately, an optimal formula for the repair and rehabilitation of injured ligaments has not yet been defined, and true ligament regeneration remains a challenge for the future.

References

1. Praemer A, Furner S, Rice DP: *Musculoskeletal Conditions in the United States*. Rosemont, Ill: American Academy of Orthopaedic Surgeons, 1992.
2. Johansson H, Sjölander P: Neurophysiology of joints, in Wright V, Radin EL (eds): *Mechanics of Human Joints: Physiology, Pathophysiology, and Treatment*. New York: Marcel Dekker, 1993, pp 243-290.
3. Markolf KL, Gorek JF, Kabo JM, et al: Direct measurement of resultant forces in the anterior cruciate ligament: An *in vitro* study performed with a new experimental technique. *J Bone Joint Surg Am* 1990;72:557-567.
4. Johnson RJ, Beynnon BD, Nichols CE, et al: The treatment of injuries of the anterior cruciate ligament. *J Bone Joint Surg Am* 1992;74:140-151.
5. Kannus P, Renström P: Treatment for acute tears of the lateral ligaments of the ankle: Operation, cast, or early controlled mobilization. *J Bone Joint Surg Am* 1991;73:305-312.
6. Shelbourne KD, Wilckens JH: Current concepts in anterior cruciate ligament rehabilitation. *Orthop Rev* 1990;19:957-964.
7. Fu FH, Jackson DW, Jamieson J, et al: Allograft reconstruction of the anterior cruciate ligament, in Jackson DW, Arnoczky SP, Woo SLY (eds): *The Anterior Cruciate Ligament: Current and Future Concepts*. New York: Raven Press, 1993, pp 325-338.
8. Kannus P: Long-term results of conservatively treated medial collateral ligament injuries of the knee joint. *Clin Orthop* 1988;226:103-112.
9. Frank CB, Bray RC, Hart DA, et al: Soft tissue healing, in Fu FH, Harner CD, Vince KG (eds): *Knee Surgery*. Baltimore: Williams & Wilkins, 1994, vol 1, pp 189-229.
10. Cabaud HE, Rodkey WG, Feagin JA: Experimental studies of acute anterior cruciate ligament injury and repair. *Am J Sports Med* 1979;7:18-22.
11. Hefti FL, Kress A, Fasel J, et al: Healing of the transected anterior cruciate ligament in the rabbit. *J Bone Joint Surg Am* 1991;73:373-383.
12. O'Donoghue DH, Rockwood CA Jr, Zaricznyj B, et al: Repair of knee ligaments in dogs: I. The lateral collateral ligament. *J Bone Joint Surg Am* 1961; 43:1167-1178.
13. Woo SLY, Inoue M, McGurk-Burleson E, et al: Treatment of the medial collateral ligament injury: II. Structure and function of canine knees in response to differing treatment regimens. *Am J Sports Med* 1987;15:22-29.
14. Piper TL, Whiteside LA: Early mobilization after knee ligament repair in dogs: An experimental study. *Clin Orthop* 1980;150:277-282.
15. Lechner CT, Dahners LE: Healing of the medial collateral ligament in unstable rat knees. *Am J Sports Med* 1991;19:508-512.
16. Chimich D, Frank C, Shrive N, et al: The effects of initial end contact on medial collateral ligament healing: A morphological and biomechanical study in a rabbit model. *J Orthop Res* 1991;9:37-47.
17. O'Donoghue DH, Rockwood CA Jr, Frank GR, et al: Repair of the anterior cruciate ligament in dogs. *J Bone Joint Surg Am* 1966;48:503-519.
18. Sherman MF, Lieber L, Bonamo JR, et al: The long-term followup of primary anterior cruciate ligament repair: Defining a rationale for augmentation. *Am J Sports Med* 1991;19:243-255.
19. Kaplan N, Wickiewicz TL, Warren RF: Primary surgical treatment of anterior cruciate ligament ruptures: A long-term follow-up study. *Am J Sports Med* 1990;18:354-358.

20. Inoue M, Woo SLY, Gomez MA, et al: Effects of surgical treatment and immobilization on the healing of the medial collateral ligament: A long-term multidisciplinary study. *Connect Tissue Res* 1990;25:13-26.
21. Bray RC, Shrive NG, Frank CB, et al: The early effects of joint immobilization on medial collateral ligament healing in an ACL-deficient knee: A gross anatomic and biomechanical investigation in the adult rabbit model. *J Orthop Res* 1992;10:157-166.
22. Beynnon BD, Johnson RJ, Fleming BC: The mechanics of anterior cruciate ligament reconstruction, in Jackson DW, Arnoczky SP, Frank CB, et al (eds): *The Anterior Cruciate Ligament: Current and Future Concepts*. New York: Raven Press, 1993, pp 259-272.
23. Hart DP, Dahners LE: Healing of the medial collateral ligament in rats: The effects of repair, motion, and secondary stabilizing ligaments. *J Bone Joint Surg Am* 1987;69:1194-1199.
24. Gomez MA, Woo SLY, Inoue M, et al: Medial collateral ligament healing subsequent to different treatment regimens. *J Appl Physiol* 1989;66: 245-252.