Injury location affects ligament healing

A morphologic and mechanical study of the healing rabbit medial collateral ligament

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Based on the heterogeneity of the rabbit medial collateral ligament (MCL) along its length, we tested the hypothesis that injury location would affect its healing response. The right MCL of 80 skeletally mature New Zealand white rabbits was sectioned adjacent to bone at the femoral end (40 rabbits) or the tibial end (40 rabbits) and reapposed with sutures. Animals were killed after 3, 6, 14, or 40 weeks of healing to examine wounds histologically (2 rabbits per healing interval) and mechanically (8 rabbits per healing interval). Results of the mechanical tests were compared to midsubstance MCL repairs (24 rabbits) and to uninjured normal MCLs (20 rabbits). The morphology of the near-insertion repairs was characterized by abnormal callus-like formation and patchy bone resorption, particularly at the tibial insertion. Mechanically, insertional injuries remodeled towards normal MCL low-load, viscoelastic and failure properties more slowly than midsubstance injuries at the early healing intervals. After 40 weeks of healing, few injury-specific differences persisted. All injured ligaments had ultimate strengths 15–35 percent short of normal at 40 weeks and the femorally-injured ligaments were weaker than normal at this time. These results suggest that rabbit MCLs, injured near either end, heal more slowly that those injured in their midsubstance and develop abnormal insertion morphology.

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Despite a long history of clinical and experimental investigation into the healing of knee ligaments with a wide spectrum of treatment regimens, it is not clear whether or how a variation in injury site affects the rate, quality or endpoint of healing. Variations in the site of injury to the medial collateral ligament (MCL) have been documented clinically (Palmer 1938, Wilkins 1980) and experimentally in animal models (Horwitz 1939, Jack 1950, Clayton and Weir 1959).

Recent studies have shown the normal rabbit MCL to be morphologically, biochemically and mechanically heterogeneous along its length (Cooper and Misol 1970, Woo et al. 1983, Frank et al. 1988, Woo et al. 1988, Matyas 1990, Matyas et al. 1990). Chimich et al. (1992) showed that the water content of the adult rabbit MCL was greatest near the femoral insertion and decreased distally, while collagen concentrations were greatest in the region just proximal to the tibial insertion and least near the femoral insertion. The stress states which exist at the two insertions have also been shown to differ from each other (Simbeya et al. 1993, Matyas et al. 1994, Wilson et al. 1994). These data suggest that the ends of the rabbit MCL differ both from the MCL midsubstance and from each other. Because MCL healing in animals has been characterized previously by the study of midsubstance healing alone, we designed a study to compare the morphologic and structural mechanical healing response of rabbit MCLs injured in their midsubstance or near their ends. We hypothesized that the healing responses of areas injured near ligament ends would differ both from each other and from midsubstance injuries.

Animals and methods

80 skeletally mature (12 months old), non-lactating, female New Zealand white rabbits (Rieman's Fur Ranch, St. Agatha, Ontario) were given injuries near either the tibial (40 rabbits) or the femoral (40 rabbits) end of their right MCL. 20 other rabbits were used as controls and a further 2 rabbits were used to verify the histologic location of the injuries. Injury models were compared directly with a similar, previously published (Chimich et al. 1991), midsubstance injury model in the right MCL (24 rabbits).

Sterile surgery was performed under general anesthesia by a single operator. The right lower limb of each rabbit was shaved and prepared with iodine. The

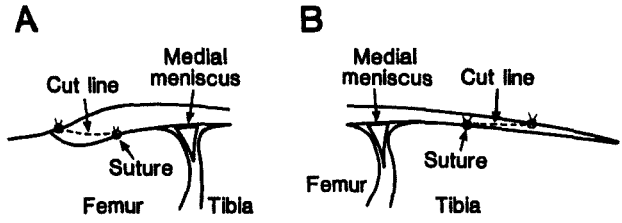

Figure 1. Schematic diagrams showing the anatomic location of the injuries near the femoral insertion (A) and the tibial insertion (B). The MCL was detached as close as possible to the bone.

MCL was exposed with a medial incision through the skin and fascia. A scalpel blade was used to detach the MCL from the medial meniscus, without damaging the ligament. The blade was then passed under and then through the MCL almost parallel to the bone near either the femoral insertion or the tibial insertion. The blade was guided so that the MCL was detached as close to the bone as possible near the insertion site, while maintaining some adjacent tissue to which the MCL could be reattached with sutures. One 6–0 nylon suture was placed at each of the 4 corners of the cut ends and sewn into the juxtainsertional periosteum in order to maintain the apposition of cut ends (Figure 1). In our previously published midsubstance injury model (Chimich et al. 1991), a Z-plasty was created at the joint line of each MCL, perpendicular to the long axis of the MCL. Cut ends were reapposed and contact was maintained by placing one 6-0 nylon suture at each corner. In all animals, after wounds had been reapposed, the overlying fascial flaps were replaced and skin wounds were closed with interrupted nylon sutures. The animals were allowed unrestricted postoperative activity and were weighed regularly as a general index of health. The rabbits were killed with an injection of sodium pentobarbital in groups of 6 (midsubstance) and 10 (near the tibial and femoral insertion) at postoperative intervals of 3, 6, 14, and 40 weeks.

The right femur-MCL-tibia complexes of 20 age-matched rabbits which did not undergo surgery were used as mechanical controls. These rabbits were cared for in the same way as the experimental animals and were killed in groups of 5 at the same intervals as the injured animals.

Histology

In order to document the histologic location of injuries near the ligament ends, 2 additional rabbits were killed and the stifle joints of each of the 4 hind limbs were harvested. Each MCL was removed by injuring the femoral and tibial insertions as described above, thus providing 8 time-zero insertion area samples (4 tibial and 4 femoral). The peri-insertional tissues were fixed in a paraformaldehyde-lysine-periodate solution prepared by the method of McLean and Nakame (1974) for 24–48 hours at 4 °C. The insertions were embedded in polymethyl methacrylate cement, sectioned at 5 μ with a tungsten carbide blade on a Jung K microtome, placed on slides which had been coated with Haupt's adhesive and dried under pressure at 60 °C. Slides were subsequently soaked in methyl acetate to remove the polymethyl methacrylate, and then hydrated, stained with hematoxylin and eosin and viewed with a microscope.

2 animals from each of the near-insertion healing groups were killed at the relevant healing intervals. These healing MCLs were processed and viewed as above.

Mechanical testing

The method used to test the MCLs mechanically has been described previously (Chimich et al. 1991). Each tibia and femur was mounted with polymethyl methacrylate cement in the custom-designed clamps of a materials-testing machine, with the stifle joint at an approximate flexion angle of 70°. Care was taken to ensure that ligament insertions remained several millimeters from the cement. After the cement had cured, each MCL was isolated by sequentially cutting and removing the menisci and all other ligaments. Two tension-compression cycles were performed between 2 N tension and 4 N compression (± 0.05 N) in order to establish ligament zero, the cross-head position at which the MCL first resisted the tensile load (0.05–0.10 N). Ligament laxity was recorded as the displacement between this zero load position and the position of initial joint compression. Each specimen was subsequently immersed in phosphate-buffered saline (pH 7.4) maintained at 35 °C.

The viscoelastic behavior of ligament complexes was assessed with cyclic and static load relaxation tests. For the cyclic load relaxation test, 30 consecutive loading cycles were applied (cross-head speed 10 mm/min) between ligament zero and 0.68 ± 0.05 mm of joint displacement. Cyclic load relaxation was measured as the peak load of the 10th cycle expressed as a percentage of the peak load of the first cycle. On the 31st cycle, the cross-head was stopped at 0.68 mm of joint displacement and maintained in that position for 1200 seconds for the static load relaxa-

tion test. Percent static load relaxation was calculated using the formula: $((L_i-L_f)/L_i)*100$, where L_i was the load at the start of load relaxation and L_f was the load at the end of load relaxation. Immediately following the static load relaxation test, complexes were loaded to failure at a displacement rate of 20 mm/min and failure sites were recorded.

Statistics

Each dependent variable (animal mass, MCL laxity, static and cyclic load relaxation and failure load) was examined for normality and homogeneity of variance. Since only 3 minor violations of homogeneity were found (laxity at 6 and 14 weeks, and failure load at 40 weeks), parametric statistics were used. A multivariate analysis of variance (MANOVA SPSS) was performed for each healing interval to evaluate the differences between groups (normal, midsubstance injury, near-tibial insertion injury and near-femoral insertion injury). Post hoc univariate analyses (Scheffe, SPSS) were performed to identify specific differences between groups at each interval. Statistical significance was determined at an overall level of $p < 0.05$.

Results

Animal mass did not differ among the normal (5.43 *0.53* kg, mean *SD*) and injury (5.25 *0.61* kg) groups at any interval and all animals remained healthy throughout the experiment. All repairs were successful upon gross examination.

Morphology

The time-zero injuries near the femoral insertion were found to be at bone proximally and distally, with a small amount of tissue in the central bony divot. Injuries near the tibial insertion were at the bone proximally, but away from the bone distally (Figure 1). None of these wounds cut solely through fibrocartilaginous insertion areas, but the injury sites were very close to the ends of MCLs, reproducible in both location and configuration.

A qualitative analysis of the near-insertion injuries revealed many findings typical of healing ligaments: early inflammation, scar formation and scar remodeling. No major differences were seen between scars from different sites at comparable healing intervals. Early scars were hypercellular and disorganized, while later scars became less cellular and more organized. Whereas the ligament collagen fibers near the uninjured insertions had a uniform, parallel arrangement, those near healing insertions were disorganized (Figure 2).

Changes in the bone morphology of all insertions were observed. The most striking finding was pitted bony resorption at ligament-tibia interfaces over time in the near-tibial insertion injury model, this feature being particularly marked at the 6-week healing interval. In addition, evidence of new bone production, similar in appearance to osteophyte formation, was observed in the metaphyseal bone. This phenomenon was slightly more pronounced in the near-femoral insertion injury model, while bone resorption was less noticeable (Figure 2).

Mechanics

Normal MCL laxity ranged from mean values of 0.22 to 0.62 mm (Table 1). The ligaments with midsubstance injuries did not differ from normal at any healing interval. After 3 weeks of healing, ligaments injured near the femoral insertion were more than twice as lax as the normal and midsubstance-injured ligaments (p 0.01), while those injured near the tibial insertion did not differ from normal or midsubstance repairs. By 6 weeks, the MCLs injured near the tibial insertion were more lax than normal (p 0.002), with values similar to the femorally injured ligaments. Both near-insertion injury groups were more lax than the normals and the midsubstance-injured MCLs at this interval (p 0.001). These differences were not significant at 14 weeks. By 40 weeks, all injured ligaments had laxities indistinguishable from normal.

Table 1. Ligament laxity. Mean, *SD* (mm)

Interval (wk)	Normal	Midsubstance	Near-tibial insertion	Near-femoral insertion
3	0.6 *0.3* [a,b]	0.6 *0.4* [a]	0.7 *0.6* [a,b]	1.4 *0.5* [b]
6	0.4 *0.3* [a,b]	0.2 *0.1* [a]	1.0 *0.5*	1.0 *0.5* [b]
14	0.3 *0.1*	0.5 *0.5*	1.3 *0.8*	1.2 *0.9*
40	0.4 *0.3*	0.3 *0.1*	0.4 *0.3*	0.4 *0.3*

Within each interval, significant differences were found between groups with different letters (a < b< c).

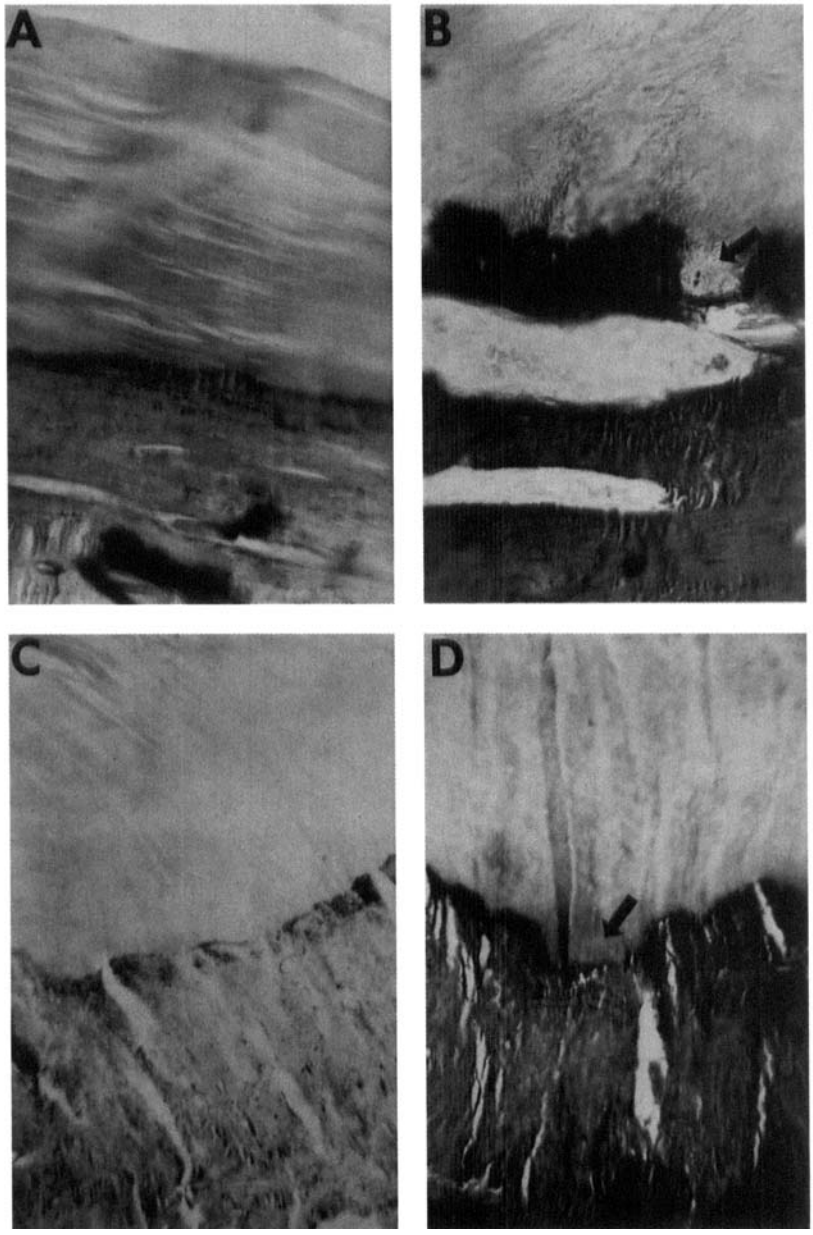

Figure 2. Photomicrographs showing a typical uninjured MCL tibial insertion (A), a 6-week healing ML tibial insertion (B), a typical uninjured MCL femoral insertion (C), and a 14-week healing femoral insertion (D). Note the pitted bony resorption (arrows) at the ligament-bone interfaces. Hematoxylin and eosin stain. Magnification ×160.

The cyclic load relaxation of all injury groups exceeded normal values at the 3-week interval by approximately 14 percent (Table 2), the midsubstance and femorally-injured ligaments having greater values (p 0.02). At 6 weeks, the midsubstance-injured ligaments were within approximately 7 percent of normal, while the ligaments injured near the femoral and tibial insertions relaxed approximately 11 percent and 15 percent more than normal, respectively (p 0.001). The near-tibial insertion-injured ligaments relaxed significantly more than the midsubstance-injured ligaments. This pattern continued at 14 weeks. By 40 weeks, however, all groups were indistinguishable from normal.

Table 2. Cyclic load relaxation (%). Mean, *SD*

Interval (wk)	Normal	Midsubstance insertion	Near-tibial insertion	Near-femoral
3	25 *4* [a]	40 *8* [b]	38 *7* [a,b]	39 *9* [b]
6	24 *4* [a]	31 *7* [a,b]	39 *3* [c]	35 *2* [b,c]
14	21 *4* [a,b]	29 *9* [a]	35 *6* [c]	30 *3* [b,c]
40	24 *2*	27 *3*	27 *6*	21 *4*

Within each interval, significant differences were found between groups with different letters (a < b< c).

Table 3. Static load relaxation (%). Mean, *SD*

Interval (wk)	Normal	Midsubstance	Near-tibial insertion	Near-femoral insertion
3	44 *8* [a]	76 *8* [b]	60 *12* [a,b]	60 *10* [b]
6	37 *8* [a]	60 *13* [b]	67 *6* [b]	64 *5* [b]
14	36 *5* [a]	56 *8* [b]	60 *9* [b]	64 *11* [b]
40	41 *3* [a]	60 *10* [b]	54 *11* [a,b]	43 *10* [a]

Within each interval, significant differences were found between groups with different letters (a < b< c).

Table 4. Load at failure (N). Mean, *SD* (mm)

Interval (wk)	Normal	Midsubstance	Near-tibial insertion	Near-femoral insertion
3	378 *31* [b]	92 *26* [a]	85 *24* [a]	76 *52* [a]
6	389 *43* [c]	169 *61* [b]	76 *37* [a]	114 *35* [a,b]
14	362 *47* [b]	207 *36* [a]	150 *38* [a]	158 *67* [a]
40	316 *36* [b]	271 *12* [a,b]	258 *43* [a,b]	207 *65* [a]

Within each interval, significant differences were found between groups with different letters (a < b< c).

Site-specific differences in static load relaxation occurred at the 3- and 40-week intervals (Table 3). 3 weeks after injury, ligaments injured in the midsubstance and near the femoral insertion relaxed significantly more than normal ligaments (p 0.001), while ligaments injured near the tibial insertion were indistinguishable from normal. By 6 weeks, and continuing to 14 weeks, the injury groups were similar to each other and relaxed significantly more than normal (p 0.01). 40 weeks after injury, only those ligaments injured in the midsubstance continued to relax significantly more than normal (p 0.006).

All injured ligaments failed at significantly lower loads than normal until the 40-week interval (p 0.001) (Table 4). At 40 weeks, ligaments injured in the midsubstance and near the tibial insertion failed at loads within 18 percent of normal, while ligaments injured near the femoral insertion failed at a mean of 34 percent smaller load than normal (p 0.004). Among the injury groups, the midsubstance-injured ligaments failed at higher loads than either of the near-insertion injury groups at all healing intervals. However, the difference between the midsubstance-injured and near-tibial insertion-injured ligaments was significant only at the 6-week interval (p 0.001). Failure load improved at every interval in the ligaments injured near the femoral insertion. In contrast, ligaments injured near the tibial insertion had a slightly decreased failure load between 3 and 6 weeks, before increasing to 57 percent between 6 and 14 weeks, and to 26 percent between 14 and 40 weeks.

In terms of failure site (Table 5), midsubstance injuries predominated in all but the tibial insertion injury group at 6, 14, and 40 weeks, where that injury site appeared to be the weak link in the system.

Table 5. Location of femur-MCL-tibia complex failure at each healing interval for normal MCLs, midsubstance-injured MCLs, MCLs injured near the femoral insertion) and MCLs injured near the tibial insertion.

Interval (wk)	Midsubstance	Femoral fracture	Femoral insertion	Tibial insertion	Other
Normal controls					
3	3	2			
6	3	1	1		
14	2	1	2		
40	2		2		1
Midsubstance-injured					
3	6				
6	3			1	1
14	5			1	
40	5				
Femoral end-injured					
3	5		2		1
6	7		1		
14	8				
40	4		4		
Tibial end-injured					
3	5			3	
6				8	
14	3			5	
40	3		2	3	

No significant association was detected between failure mode and either failure load or group.

Discussion

To our knowledge, this is the first study to demonstrate any site-specific healing response along the length of any ligament. We hypothesized that injuries created through areas of the MCL which have been shown to be biochemically (Frank et al. 1988) and morphologically (Matyas 1990) unique would have similarly unique healing responses. This, indeed, was the case: injuries near MCL insertion sites appeared generally to remodel mechanically more slowly than analogous injuries to the ligament midsubstance. The reasons for this difference are uncertain, but several possibilities exist.

We recognize that the gross stress states which would have occurred across the cut ligament surfaces of each injury model would probably have varied. The stress states would have varied because the angle at which the ligaments were sectioned relative to the direction of applied load during testing was different in each injury model. The midsubstance cross-sectional cut would have resisted direct tensile stresses when the ligament was distracted, while the tibial near-insertion injury cut would have resisted mainly shear stress (with some direct tension) and the femoral near-insertion injury would have been subject to a complex and varying combination of tension, shear and lateral compression (Matyas et al. 1994) (Figure 1). Remodeling to resist these stress states at the insertions may have been slower than for the simpler stress state in the midsubstance. It is also possible that the pattern of scar deposition could have created resistance to more direct tension at the near-insertion injury sites—for example, by extending the scar, and thereby shielding the load from the insertion. Such scar may unwittingly have been cut away in specimen preparation.

The morphologic destruction observed at the bone-ligament interfaces is an interesting phenomenon which appears to account for some of the mechanical differences between injury groups. One potential cause of this destruction could have been a peri-insertional inflammatory response initiated by the ligament disruption at that site. The fact that morphologic changes in bone persisted throughout the healing period studied, long after inflammation had subsided, is more suggestive of a disuse effect at the insertion sites. Similar disruptions at the femoral and tibial insertions following unrepaired midsubstance injuries in the adult rabbit MCL have been reported (Matyas and Frank 1990a, b).

The near-insertion-injured ligaments were more lax than the normal controls early in the healing process, while the midsubstance-injured MCLs were not. One possible reason for this pattern may have been technical: the cut ends of the near-insertion wounds

were harder to suture, due to the distal taper of the tissue. Another possibility is that stresses near the insertion sites could have been higher during the healing process and could subsequently have contributed to a temporary increase in laxity. However, since all injured ligaments recovered normal laxity values by the 40-week healing interval, regardless of injury site, it seems clear that all injuries eventually underwent the well-known process of scar contraction (Gabbiani and Montanden 1977, Rudolph 1980).

The results of the cyclic load relaxation tests were similar to those of the laxity tests, since the values for all injured MCLs returned to normal by 40 weeks. Differences, however, also existed among the 3 injury models, the ligaments injured near either end having a delayed recovery. Interestingly, this was not so clear with the static load relaxation tests, where only femoral injuries recovered to normal by 40 weeks.

Although the failure load of all the injured MCLs improved throughout the process of healing, site-specific responses were observed. The structural strength of ligaments injured near their insertions appeared to lag slightly behind that of ligaments injured in the midsubstance. The most probable explanation of this observation is that differences probably existed in the amount of scar tissue present in the different injury locations. Injuries near ligament ends may have been constrained by bone, causing scars to be smaller and scar stresses to be higher. In line with other studies of healing rabbit MCLs (Frank et al. 1983, 1985), with the exception of those ligaments injured at the femoral insertion, the weak link in the injured MCLs was in most cases at, or in close proximity to, the site of the scar. This was particularly true of injuries near the tibial insertion, which appeared to induce a substantial weakness at that bone interface. The near-femoral insertion-injured MCLs failed predominantly in the midsubstance. Although the femoral end injuries were anatomically only a few millimeters from the midsubstance and although failure modes were judged by gross observation during failure-testing, these modes of failure are quite distinct from one another. Thus, as in some of the near-tibial repairs, it appears that the midsubstance was weakened by the adjacent injury. This weakening may have been due to inflammation, load shielding, or both. Regardless of the mechanisms, our data support the concept that not only are normal rabbit MCLs heterogeneous along their length, but they appear to have subtle variations in their healing along their length as well. The clinical significance of these observations remains to be determined, but it could suggest some need to customize rehabilitation based on sites of ligament injury.

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