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THE IMPORTANCE OF CONTROLLED PASSIVE MOBILIZATION ON FLEXOR TENDON HEALING

A Biomechanical Study

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The effects of controlled passive motion on primary tendon repair were studied using the canine forepaw flexor apparatus as experimental model. The animals were divided into seven groups based on duration (3 to 12 weeks post repair) and mode of immobilization and partial mobilization. The repaired tendons were subjected to biomechanical evaluation of their gliding function and tensile strength characteristics. The results showed positive effects of controlled passive motion on tendon repair. The rate of tendon repair was significantly improved over those animals that were continuously immobilized. At 12 weeks, the repaired flexors from the motion group had regained over one-third of the ultimate tensile load as compared to their contralateral intact controls. Of equal importance is that these repaired tendons maintained good gliding function within the sheath during the repair process. The gliding function of these tendons was also significantly better than those subjected to continuous immobilization.

Key words: biomechanics; flexor tendons; primary repair, controlled passive motion, strength, gliding

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Complications relating to the clinical management of severed finger flexor tendons within the digital sheath are well known. The clinical results of tendon repair in this region have been unpredictable (Potenza 1970). The sutured tendon not only has to regain strength sufficient to resist high tensile loads, but must also be able to glide smoothly within the sheath. To date there has been no treatment regimen capable of fully avoiding the complications of tendon adherence, poor excursion and poor digital function.

Several authors have advised early motion following tendon repair in an attempt to overcome some of the problems associated with tendon adhesions (Lahey 1923, Harmer 1921, Murray 1960, Hernandez et al. 1967, Kessler & Nissim 1969, Furlow 1972, Kleinert et al. 1973, Strickland & Glogavac 1980 and Duran & Houser 1975). Mason & Allen (1941), in a study of flexor carpi ulnaris tendon repairs in dogs, showed that the tensile strength of tendons immobilized for 3 weeks and then mobilized was several times higher than those continuously immobilized. Their study suggested that mobilization may stimulate both the healing rate and strength of repaired flexor tendons.

Other authors have pointed out the deleterious effects of early stress applied across the tendon repair site, however. Ketchum et al. (1977) showed that early *active* mobilization did not improve tendon gliding, and, on the contrary, increased the tension across the suture line, which

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led to gap formation between the tendon ends, tendon ischemia, tenomalacia and tendon rupture. It is apparent that while early motion may be advantageous, it must be controlled so that appropriate levels of stress and strain are placed upon the tendon repair site.

The purpose of the present work is to systematically investigate the effects of controlled passive motion on flexor tendon repair. A comprehensive experimental program was designed to evaluate the potential advantages of this approach. The biomechanical testing procedures included the measurement of strength and gliding of the repaired tendon, with tendon healing evaluated as a function of the range of controlled passive motion at various stages following tendon repair.

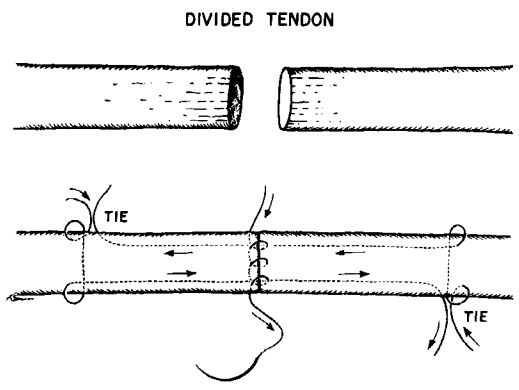

Figure 1. A schematic diagram illustrating the modified Kessler suture technique used in primary flexor tendon repair.

MATERIAL AND METHODS

Twenty-nine adult mongrel dogs with body weights ranging from 25 to 35 kg were used as experimental animals. Dogs were chosen because of the similarity of the flexor tendon apparatus to that of humans (Potenza 1962). The experimental animals were lightly anesthetized with an initial intravenous dose of sodium pentathol (0.15 cm^3/kg) and supplemented by intermittent injections (0.05 cm^3/kg) as required. The repairs were performed aseptically in an animal operating facility. The flexor tendons of the left second and fourth toes were approached through separate mid-lateral incisions. The sheath was incised at the proximal interphalangeal (IP) joint and the flexor superficialis decussation. The lacerated tendons were repaired in the manner described by Kessler & Nissim (1969) with 4-0 braided dacron suture under 3.5 times magnification. A continuous 6-0 nylon epitenon suture was inserted to invaginate free tendon ends (Figure 1). The sheaths were not repaired. Hemostasis was obtained with electrocautery. All limbs were initially immobilized in a polyurethane shoulder spica cast with the elbow and wrist held at 90° of flexion. The experimental animals were divided into seven groups based on the duration of rigid immobilization and partial mobilization prior to sacrifice. The groups are:

Group 1: Three weeks of immobilization with wrist at 90° flexion.
Group 2: Six weeks of immobilization with wrist at 90° flexion.
Group 3: Twelve weeks of immobilization with wrist at 90° flexion.
Group 4: Three weeks of immobilization and 3 weeks full weight-bearing mobilization (equivalent to active cage motion).
Group 5: Three weeks immobilization and 3 weeks of partial mobilization (wrist at 90°).
Group 6: Three weeks immobilization, 6 weeks of partial mobilization (3 weeks with wrist at 90° and 3 weeks at 45°).
Group 7: Three weeks immobilization, 9 weeks of partial immobilization (3 weeks with wrist at 90°, 3 weeks at 45°, and 3 weeks at neutral).

For partial mobilization the volar one-half of the forearm portion of the cast was cut with the remaining half of the cast serving as an extension block. The wrist and digits were manually flexed and extended to the limits of the dorsal extension block for 5 minutes daily. The extension block was gradually brought out from the 90° flexed position to 45°, and to neutral, at 3-week intervals. At sacrifice the second and fourth toes of both forepaws were disarticulated at the metacarpal phalangeal (MP) joints. The flexor digitorum profundus tendon was transected 2–3 cm proximal to the MP joints. The repair site was not approached surgically. Rupture of the tendon anastomosis could be detected by a lax accordian-like appearance of the proximal tendon stump. For Group 4 (3 weeks of immobilization and 3 weeks of full weight-bearing mobilization) five out of six repaired tendons ruptured *in vivo*. Additionally, there were nine repaired tendons which ruptured *in vivo*, i.e. two each from Groups 1 and 2, one each from Groups 3, 5 and 6, and two from Group 7. The remaining 44 successfully repaired tendon specimens with their distal bony attachments intact were first examined for their gliding function and then subjected to tensile test to failure. Care was taken in the specimen preparation so as not to disturb the tendon sheath around the repair site. Fifty-three contralateral control tendons from the experimental animals (only one control from Group 4) were similarly prepared and tested for comparison.

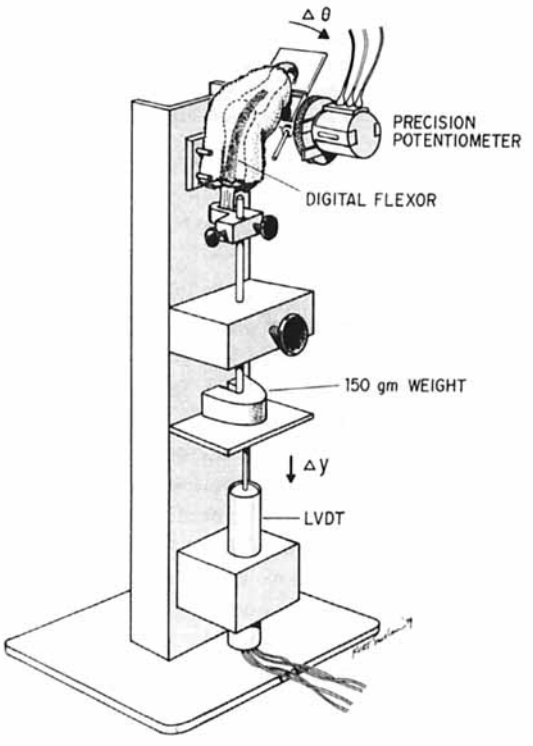

Figure 2. An apparatus specially designed to measure the excursion properties (gliding function) between the tendon and its surrounding sheath.

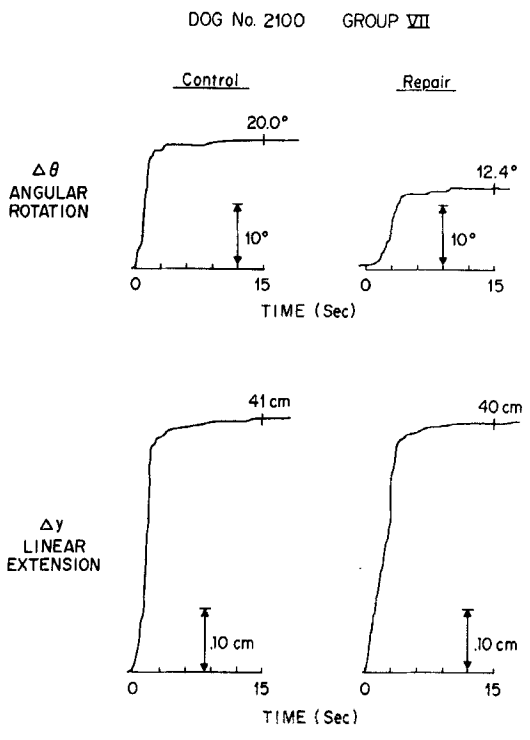

Figure 3. A typical recording of the linear extension (Δy) and angular rotation (Δθ) of the control and repaired tendons.

The excursion (gliding) function of the tendons was evaluated by means of an apparatus specially designed to simultaneously measure the angular rotation of the distal IP joint (Δθ) and the linear extension (Δy) of the tendon when a small load of 150 g was applied (Figure 2). All the control and repaired tendons, with the exception of those from Groups 1 and 4, were tested with this apparatus for evaluation of the effect of controlled passive motion on tendon gliding.

The experimental procedure consisted of first removing the proximal phalanx. The specimen was fastened onto the metal plate platform by means of a 1 mm diameter copper wire passing through the middle phalanx. The axis of rotation of the metal plate platform was connected to a precision potentiometer for measuring angular rotation (Δθ). The end of the transected tendon was clamped and connected to a weight platform. A linear voltage displacement transducer was attached directly beneath the weight platform to measure the vertical displacement (Δy). When a 150 g weight was applied, the linear extension of the tendon (Δy) could be measured simultaneously with the angular rotation (Δθ) of the digit. During the experiment, the weight was applied for 15 seconds and the values of angular rotation (Δθ) and linear extension (Δy) were recorded (Figure 3). After the excursion study, the middle phalanx was removed leaving the distal phalanx with tendon, called the "tendon-bone composite", as the test specimen. The specimen was then fastened by a set of specially designed clamps and mounted on an

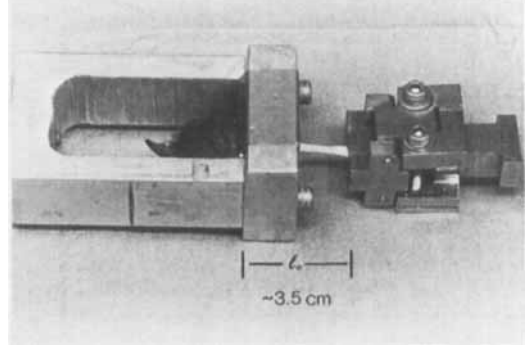

Figure 4. The clamping system used to test the tensile characteristics of the tendon-bone complex.

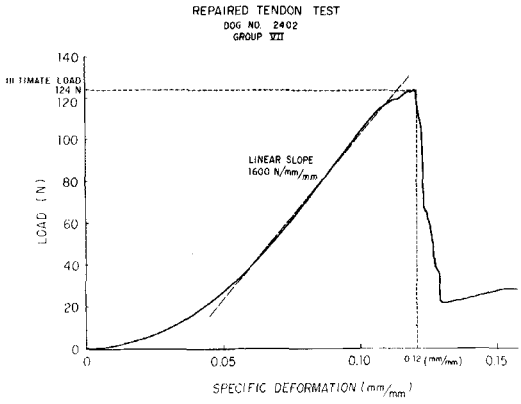

Figure 5. A typical tensile-load specific deformation (F vs. $\Delta l/l_0$) diagram of the tendon-bone complex tested to failure.

Instron apparatus for tensile testing to failure. The clamp for the distal phalanx (bone) end was designed so that only the phalanx was securely restrained while allowing the tensile load to be entirely sustained by the tendon and its insertion to bone. The clamp for the free (tendon substance) end had pivoted jaws to increase the clamping force as the tendon was being pulled (Figure 4). After clamping, the clamp to clamp distance of each specimen was measured and this initial length (l_0) for all specimens ranged from 3 to 4 cm. The test specimen was then immersed in a saline solution bath at 37°C for 15 minutes for temperature equalization prior to testing. An Instron crosshead rate of 2 cm/min was used.

The Instron crosshead (or tendon-bone composite) displacement was accurately monitored by a linear voltage displacement transducer (LVDT). A special load cell* was used to measure the tensile load. Hence, a tensile load-specific deformation (F vs $\Delta l/l_0$) diagram for each tendon was recorded using an X-Y recorder, and the strength characteristics, as represented by the ultimate load, the maximum specific displacement at failure and the linear slope, were determined (Figure 5).

RESULTS

For the control tendons, tensile failures occurred either by pull-out at the distal digit insertion, or within the pivoted clamp. It was not possible to obtain flexor tendon substance failure in spite of our special clamps (Woo et al. 1981). For the experimentally repaired tendons, however, all failures occurred at the repair site. The tensile strength characteristics of all control and repaired tendons are detailed in Table 1. It can be seen that the repaired tendons from the Group 1 animals (3 weeks immobilization) had ultimate loads at failure similar to those of the freshly repaired flexor tendons (Figure 6). Continuous immobilization to 6 weeks did not significantly change the repaired tendon strength ($P > 0.50$). But after immobilization for 12 weeks the strength increased significantly. The ultimate load at failure for Group 3 repaired tendons was more than threefold that of Groups 1 and 2 ($P < 0.001$,

* Gould Measurements System Division, Inc., Oxnard, Calif. USA.

Table 1. Tensile strength characteristics of control and repaired canine flexor tendons

Animal groups	No. of animals	Ultimate load (N)		Specific deformation ($x10^{-2}$ mm/mm)		Linear slope ($x10^2$ N/mm/mm)	
		C*	E*	C*	E*	C*	E*
1	4	413±31	22±2	21±2	29±2	27.8±0.6	1.5±0.3
2	6	371±12	21±4	25±2	20±4	22.3±0.9	2.1±0.4
3	3	327±25	70±9	25±1	9±1	24.6±0.9	11.6±1.9
4	3	334	84	22	32	23.7	6.3
5	3	334±18	31±1	20±2	24±6	26.4±1.4	3.3±0.6
6	4	358±28	71±12	23±1	12±2	21.3±0.3	9.9±1.3
7	6	327±13	113±5	20±2	11±1	25.6±1.1	15.5±0.9

* C Control tendons
E Experimentally repaired tendons
** Note: Data presented as mean ± standard error

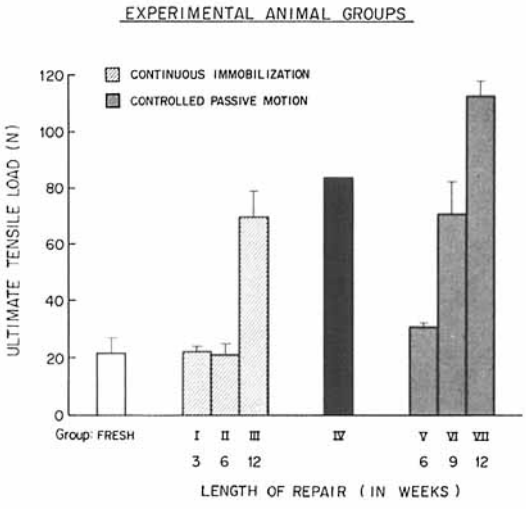

Figure 6. Comparison of the ultimate tensile load values of the experimental repaired tendons from all experimental animal groups.

$P < 0.001$, respectively). Similar results were obtained for the linear slope (Table 1).

Early unencumbered weight-bearing, in the case of the Group 4 animals, resulted in tendon rupture in five out of six cases. The one surviving tendon, however, had a substantially higher ultimate load than the 6-week continuously immobilized tendons, i.e. Group 2 animals. Its failure load of 84 Newtons was even higher than the averaged value for the Group 5 (9 weeks post repair) tendons (Figure 6).

With controlled passive motion, however, the repaired tendons had a high survival rate and the strength at the repair site also responded positively. The repaired flexors from this series of animals have a higher rate of increase in the ultimate load and the linear slope than those of the continuous immobilization group. For example, the averaged ultimate load and linear slope values for Group 6 (9 weeks post repair) were similar to those after 12 weeks of continuous immobilization, i.e. Group 3 tendons (Figure 6 and Table 1).

The changes in tensile properties of the repaired tendons from the controlled passive motion groups are detailed in Figure 7. Expressed as a percentage of the intact flexors from the contralateral control digits, the ultimate load and the linear slope of the Group 5 tendons showed uniform increases from the values of the Group 1 tendons. Further increase in range of motion appeared to accelerate the rate of repair as the large increases in the strength characteristics occurred during the 6th and 12th weeks post repair, i.e. from Group 5 to Group 6 and Group 7. Those flexors from Group 7 had achieved over one-third of the maximum strength properties of their contralateral intact controls. Correspondingly, the specific deformation reduced as a function of length of repair in these groups. Presumably, the majority of the tensile elongation of the repaired tendons came from the repaired site, and, therefore, as healing progressed, the specific deformation reduced as the strength of the repaired site increased.

The strength properties of the repaired tendons from the continuous immobilization groups and the controlled passive motion groups are shown in Figure 8. It can be seen that at 6 weeks post repair, Group 5 tendons had higher values in the ultimate load and linear slope than those of

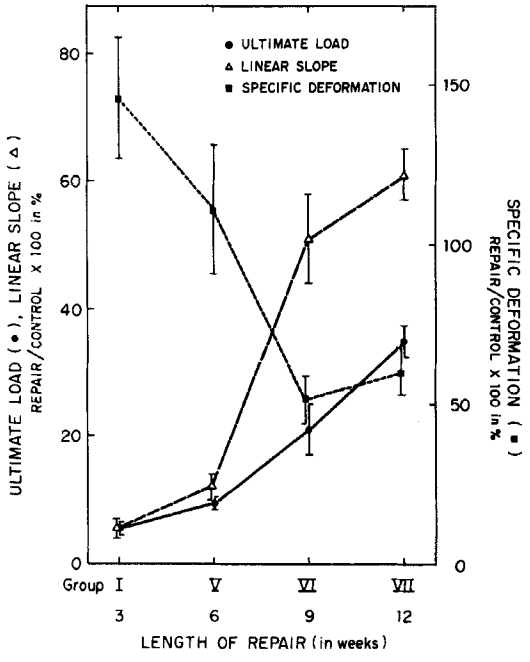

Figure 7. Comparison of the strength characteristics in tension of the repaired tendons from the animal groups subjected to controlled passive motion.

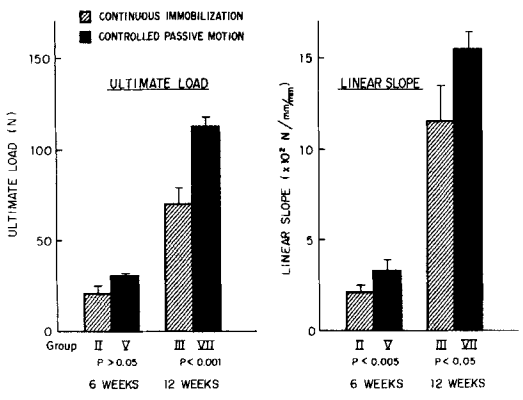

Figure 8. Histogram showing the differences in tensile characteristics of the repaired tendons from continuous immobilization and controlled passive motion groups following the same periods of post tendon repair.

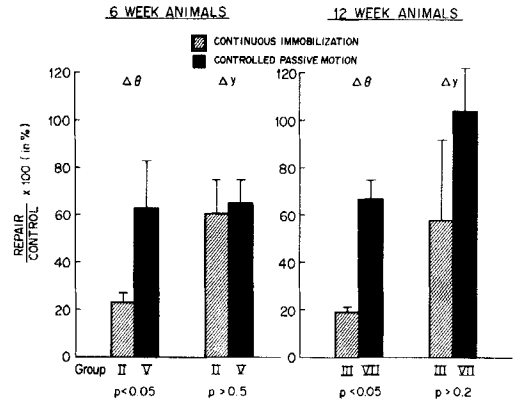

Figure 9. Histogram showing the difference in excursion properties of the repaired tendons from continuous immobilization and controlled passive motion groups following the same periods of post tendon repair.

Group 2. These results indicate that the initial 3 weeks of passive motion with 90° wrist block already had significant positive effects on the rate of tendon repair.

Additional periods of passive motion with larger ranges of motion resulted in even higher differences in the various strength characteristics (Figure 8). At 12 weeks post repair, the average values of ultimate load and linear slope of the repaired tendons following the controlled passive motion program were 61 percent and 34 percent, respectively, higher than those tendons from the continuously immobilized groups.

It appears that the controlled passive motion program also significantly minimized the adhesion formation between the repaired tendon and the surrounding sheath, thus providing better gliding function. With continuous immobilization, the angular rotation ($\Delta\theta$) and linear excursion (Δy) of the tendons at 6 weeks post repair had diminished to 21 ± 5 percent and 61 ± 14 percent, respectively, of that of the contralateral controls. These values were further reduced to 19 ± 2 percent and 58 ± 34 percent, respectively, at 12 weeks post repair (Table 2). On the other hand, controlled passive motion resulted in better tendon gliding function than the continuous immobilization groups (Figure 9). At 6 weeks post repair, the angular rotation ($\Delta\theta$) of the digit from the mobilized group was nearly threefold that of the continuously immobilized group. This difference increased to become over three and one-half fold at 12 weeks post repair. Furthermore, controlled passive motion prevented further reduction of the tendon gliding function with length of repair. The values of $\Delta\theta$ and Δy for the repaired tendons from Groups 5, 6 and 7 are detailed in Table 2. Analyses of variance of $\Delta\theta$ and Δy for these three groups showed no statistically significant difference.

Table 2. The excursion properties of the repaired canine flexors from various experimental groups

	Angular Rotation* $\Delta\theta$	Linear Excursion* Δy
1 – Continuous immobilization series		
Group 2	21±5	61±14
Group 3	19±2	61±34
2 – Controlled passive motion series		
Group 5	63±20	65±10
Group 6	58±18	87± 1
Group 7	67± 8	104±18

* Data presented as repaired/control in percent

DISCUSSION

Healing following primary tendon repair is a slow process. After 3 weeks of immobilization the

strength of the repaired tendons was only similar to those of the freshly sutured tendons. Doubling the length of time of immobilization to 6 weeks increased the repaired tendon strength minimally. Only after an additional 6 weeks immobilization did a significant increase in repair strength occur. Controlled passive mobilization places tensile forces and motion on the repair site which appears to accelerate the tendon repair strength, as well as to increase the tendon excursion. The small forces applied were well tolerated for the most part. The results on the increase in strength of the repair process support the early work of Mason & Allen (1941). These properties, as represented by the ultimate failure load and the linear slope, were significantly improved with controlled passive motion. The repaired tendons from the Group 7 animals (12 weeks post repair) had regained over one-third the strength of the intact control tendons. These strength properties may be sufficient to sustain the stresses of full weight-bearing motion of the animal with limited activities. This is possible because of the well known structural safety factor of normal flexor tendons which are "over built" with respect to strength requirements.

Of equal importance are the tendon excursion properties. After 6 weeks of immobilization the repaired tendon had diminished gliding properties. The ability of the distal interphalangeal joint to flex ($\Delta\theta$) was only 21 percent of that of the contralateral control. At 12 weeks of immobilization, the excursion capability was further decreased. The decreased range of motion was mainly due to scar formation between the repaired tendon and its surrounding sheath. In contrast, the tendons from Group 5 had significantly better excursion properties than those of Group 2, presumably due to less scar formation and early stretching of the scar. As passive motion continued, the good gliding function was maintained, while the strength of healing rose at a rapid rate. In our experimental program, the Group 7 tendons (12 weeks post repair) had a good range of motion, the $\Delta\theta$ and Δy values being 67 ± 8 percent and 104 ± 18 percent, respectively, of that of the contralateral control tendons.

The linear excursion (Δy) values were consistently higher than the angular deformation ($\Delta\theta$) values for all groups. The reason for the difference can be largely attributed to the deformation of the tendon repair site since this area is the weakest link of the tendon-toe composite. Therefore, functionally $\Delta\theta$ and Δy have different meanings. For example, while significantly higher values of $\Delta\theta$ were obtained for the passive motion groups, no statistical differences in Δy values were found between the two methods of treatment at 6 and 12 weeks post repair (Figure 9). Part of the reason for the high Δy values in the immobilization groups may be the weaker repair site, as the tensile study showed inferior strength characteristics for these tendons as compared to those in the passive motion groups.

Since tendon strength during the initial 3 weeks of immobilization did not increase, it should be interesting to examine a more aggressive controlled mobilization program. Reports by Duran & Houser (1975) and more recently by Strickland & Glogovac (1980) showed that passive motion can be effective in improving tendon excursion if begun within the first few days post repair. Thus, experiments are planned to include the effects of earlier passive mobilization using our experimental animal model. In addition, treadmill exercise to enhance the acceleration of strength recovery in the later stages of tendon repair will be examined. It is hoped the further investigation will yield useful information for the clinical management of primary flexor tendon repair.

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