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정적 및 반복하중 시의 주관절 Tendon의 파괴 물성치 측정

한정수·이관희*·유재영**

한성대학교 산업시스템공학부/안전과학기술연구소
아주대학교 의과대학교 정형외과*, 웨스트 버지니아 주립대학 정형외과**
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Failure Properties of Common Tendon Origins at the Human Elbow after Static and Repetitive Loading

J.S. Han, K.H. Lee*, J.Y. Ryu**

Center for Safety and Science Technology, Industrial and Systems Engineering,
HanSung University
*Department of Orthopedics, Ajou University
**Department of Orthopedics, West Virginia University, WV, U.S.A
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요 약 : 임상학적인 관찰에 따르면, 반복적인 하중에 의하여 뼈/건의 접합부분에 발생하는 부분적인 파손은 병리학적인 변화를 유발시킴으로 인하여 주관절의 상골과염(Epicondylitis)으로 발전시킬 수 있는 주요한 원인으로 간주되고 있다. 반복적인 하중이나 정적인 하중 하에서의 주관절에 위치한 신전건 및 굴곡건의 기계학적인 물성치와 파괴양상은 지금까지 잘 알려져 있지 않다. 본 연구에서는 상골과염과 직접적인 관계가 되는 신전건 및 굴곡건의 기계학적인 물성치인 파괴강도, 반복하중의 회수와 변형율(Strain)간의 연관관계 및 반복하중에 있어서의 생체조직학적 변화의 양상, 특히 파괴의 진행양상을 관찰하였다. 적용하중의 속도에 따르는 신전건 및 굴곡건의 파괴강도의 통계학적인 차이는 보이지 않고 있으나, 파괴강도에 있어서 신전건은 1199.0 N/cm²±388.8, 굴곡건 1922.0 N/cm²±764.4로, 굴곡건이 신전건에 비하여 1.6배 정도 크게 나타났으며, 상호간의 파괴강도에 있어서 통계학적인 차이가 있음을 보여주고 있다($p<0.05$). 조직학적 관찰에 의하면, 반복하중 하에서 뼈/건의 접합부분 특히 Uncalcified Fibrocartilage 부분에서 분리가 시작되었으며, 이는 상골과염을 발생시키는 주요생체조직부분이라는 것을 시사하고 있다.

Abstract : Based on clinical observations, it is suspected that the bone-tendon origin is the site where partial failure, leading to pathophysiological changes in the humeral epicondyle after repetitive loading, is initiated. Mechanical properties and failure patterns of the common extensor and flexor tendons of the humeral epicondyle under static and repetitive loading have not been well documented. Our goal was to determine mechanical properties of failure strength and strain changes, to correlate strain changes and the number of cyclic repetitions, and to identify the failure pattern of bone-tendon specimens of common extensor and flexor tendons of the humeral epicondyle.

Mechanical properties of human cadaver bone-tendon specimens of the common extensor and flexor tendons of the humeral epicondyle were tested under two different loading rates. No statistically significant difference in ultimate tensile strength was found between male and female specimens or between slow (10 mm/sec) and fast elongation (100 mm/sec) rates. However, a statistically significant difference in ultimate tensile strength between the common extensor (1199.0 N/cm² ± 388.8) and flexor (1922.0 N/cm² ± 764.4) tendons was found ($p<0.05$).

When loads of 25%, 33%, and 41% of the ultimate tensile strength of their contralateral sides were applied, the number of cycles required to reach 24% strain change for the common extensor and flexor tendons were approximately 8,893, 1,907, and 410, respectively.

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통신저자 : 한정수, (136-792) 서울시 성북구 삼선동2가 389, 한성대학교 산업시스템공학부/안전과학기술연구소,
Tel. (02)760-4323, Fax. (02)760-4358,

The relationship between cycles and loads was correlated (R^2=0.46)

Histological observation showed that complete or partial failure after tensile or cyclic loadings occurred at the transitional zone, which is the uncalcified fibrocartilage zone between tendon and bone of the humeral epicondyle. Sequential histological sections revealed that failure initiated at the upper, medial aspect of the extensor carpi radialis brevis tendon origin.

Biomechanical and histological data obtained in this study indicated that the uncalcified fibrocartilage zone at the bone-tendon origin of the common extensor and flexor tendons is the weak anatomical structure of the humeral epicondyle.

Key words : Humeral epicondyle, Bone-tendon Origin, Ultimate Tensile Strength, Complete or Partial Failure

INTRODUCTION

Cumulative trauma disorders of the upper extremity, including tendinitis, tenosynovitis, carpal tunnel syndrome and epicondylitis, are leading causes of lost-time and workers' compensation claims in the industrialized world[3,11,24,32].

Epicondylitis of the elbow, which involves pathologic alteration in the bone-tendon origin or musculotendinous junction at the lateral or medial epicondyle is a cumulative trauma disorder[18,25]. A consensus based on clinical and surgical evidence suspects microtear, microfailure or partial failure at the bone-tendon origin or musculotendinous junction, most often within the origin of the extensor carpi radialis brevis, as an initiator of epicondylitis[7,8,12,13,18,21, 25-27].

The general morphology of the attachment zones of tendons, ligaments, and related structures are well known at the many anatomical sites[2,6,37], however, little information is available for the common extensor and flexor tendons of the humeral epicondyle. According to Benjamin et al.[2], since histological parameters, such as the amount of calcified and uncalcified fibrocartilage at the bone-tendon junctions, vary between tendons and from one part of a tendon to another, the morphological information is important.

Many studies have been conducted to determine biomechanical properties, such as stress-relaxation, failure strain, modulus, stiffness, and strain energy density of soft tissue [4,5,9,14,16,17,19,20,22,23,28-30,33,35,36,38]. The medial collateral ligament[5,20,23,35], anterior cruciate ligament[5,9, 20,22,23,28,33,36,38], and patellar tendon[5,14,16,17,19,22] are the most frequently studied anatomical sites because of their importance and high incidence of injury. However, very little information is available about the biomechanical properties of bone-tendon specimens of the common extensor and flexor tendons of the humeral epicondyle.

A standardized method of tensile testing for soft tissues of humans and experimental animals has not yet been established and remains a challenge, primarily because they have very complicated structure and it is difficult to evaluate mechanically[36]. Furthermore, results, even from specimens at the same anatomical site, are very difficult to compare each other because of differences in the orientation and direction of the applied tensile load, strain rate, and etc.

The objectives of this study were 1) to determine ultimate tensile strength (UTS) of common extensor and flexor tendons under different loading rates, 2) to investigate strain changes in the common extensor and flexor tendons under cyclic loading with different loads magnitudes, and 3) to histologically evaluate the failure pattern at the bone-tendon junction of the humeral epicondyle, histologically.

MATERIAL AND METHODS

1. Experimental Apparatus

The custom testing fixture was designed and attached to an electro-hydraulic materials testing system (MTS). This device permitted a human cadaver elbow to be placed in 45°, 90°, or 135° of flexion (Fig. 1). This device further allowed tensile force applied across the elbow joint to be directed at any angle with respect to the humerus in the sagittal plane. The direction of applied load could thus be adjusted along the anatomical direction of the common extensor and flexor tendons of the humerus.

Anatomical direction of the tendons was determined by the origin and insertion site of the tendon. Accurate adjustment of the direction of force applied with respect to the humerus in all three linear axes was made to ensure proper bone-tendon test specimen alignment. The elbow position tested in this study was 45° in flexion. A 10-15 cm long section of the humerus was inserted into a thick-walled cylinder (electrical conduit 4 cm in diameter) with multiple K-wires(3.2mm in diameter), then secured with bone cement (polymethylemethacrylate).

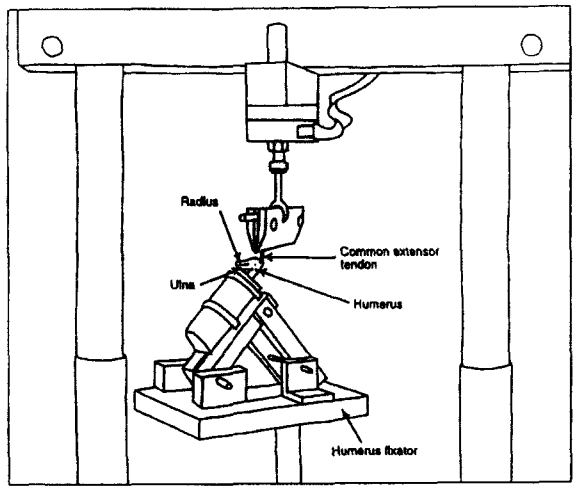

Fig. 1. Schematic diagram of the custom-designed experimental apparatus attached to the MTS machine

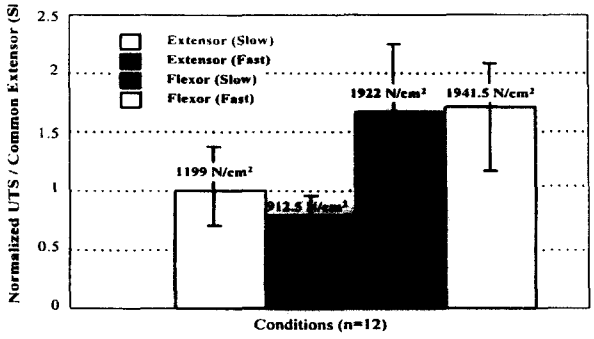

Fig. 2. The normalized ultimate tensile strength of common extensor and flexor tendons under two different loading rates: slow (10mm/sec) and fast (100mm/sec). The ultimate tensile strength was divided by the mean value of common extensor tendon (slow loading rate case) for normalization

2. Specimen Preparation

Fresh frozen human cadaver elbows were obtained and wrapped in saline soaked gauze, double wrapped in plastic bags and stored at -15℃. Prior to testing, a specimen was removed from the walk-in freezer and thoroughly thawed at room temperature. Anterior-posterior and lateral roentgenograms excluded specimens with any bony abnormality. All excessive muscle and soft tissue around the elbow joint were dissected from the upper extremity, leaving only the common and flexor tendons intact. The joint capsule was left intact to maintain anatomical and physiological joint movement. Approximately 4-5cm of the common extensor and flexor tendons distally from the origin remained after dissection. A custom-designed grip device held the tendon 1.5cm distal to its origin at the humeral epicondyle. This clamping device had serrated teeth that grasped the tendon, which was covered by a thin silicon tube, with minimal stress concentration and little damage to the gripped area. The clamp device was placed into the test fixture attached to the MTS machine with the distal part of the tendon at the selected angle of flexion. The loading fixture was oriented such that the direction of loading was along the anatomical position of elbow flexion used in this study. Specimens were kept moist with saline throughout preparation and testing.

3. Experimental Procedure

Three different tests were performed. Firstly, a tensile test was perfomed to measure the ultimate tensile strength of the bone-tendon specimen of the common extensor and flexor tendons under two different loading rates. In the second test, specimens were loaded cyclically with different load magnitudes to determine the number of cycles required to reach 25% strain change. We assumed that the 25% strain change was sufficient enough to damage the tissue so that microfailure and microdamage would be detectable from histological section. Lastly, specimens were also examined histologically to identify failure patterns at the bone-tendon junction.

Each bone-tendon specimen was preconditioned prior to mechanical testing. A pre-conditioning triangular wave form at a frequency of 2Hz was applied through the digital function generator to the hydraulic actuator using load control as feedback. By controlling the wave form amplitude, we kept a peak load of 10N applied to the specimen for 360 cycles (3 minutes). After pre-conditioning, a minimal tare load of 10N was applied as pre-loading to remove slack from the specimen.

Load and displacement were monitored with a precalibrated load cell and linear variable displacement transducer (LVDT), respectively. Resolution of the LVDT was approximately 0.025mm. Data from the load cell and the LVDT were collected simultaneously and sent to the data acquisition system through LabTech Notebook data acquisition software (Laboratory Technologies corporation, MA). Data sampling rate was 100Hz.

Tensile Testing : Twenty-four pairs of fresh cadaveric

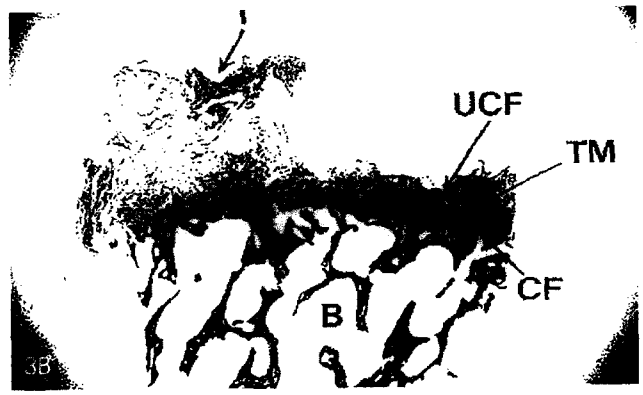

Fig. 3. A:Fross, B:Histological observation (H-E staining, ×50) of the failure site of the bone-tendon specimen of common extensor tendon after tensile load testing with 100mm/sec (UCF: Uncalcified fibrocartilage, CF:Calicified fibrocartilage, B:Bone, T: Tendon, TM:Tide mark)

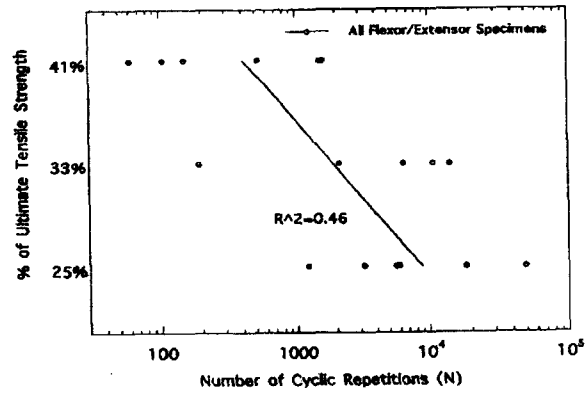

Fig. 4. The number of cycles for common extensor and flexor and flexor tendon specimens required to reach 25% strain change as load magnitude increased to 25, 33, and 41% UTS. UTS was obtained from contralateral side used as a control

human elbows (12 pairs of male and 12 pairs of female) were tested. Average age of the specimens was 72.2±9.4 for males and 80.7±10.4 for females. Twenty-four common extensor and twenty-four flexor tendon specimens for each gender were obtained and equally divided into two groups. These two groups were loaded at different loading rates.

Ultimate tensile strength of the bone-tendon specimens was measured at loading rates of 100mm/sec (fast rate) and 10mm/sec (slow rate) using the MTS machine. The mechanical failure pattern and site were observed histologically. Cross-sectional area of the bone-tendon specimens of the common extensor and flexor tendons at 1.5cm distal from the origin, where the grip device held the specimen, was measured by the area micrometer technique[10] using 0.1 Mpa compression for measurement before testing.

Number of cycles versus strain changes: Nine pairs of common extensor and eight pairs of common flexor tendons from nine pairs of fresh cadaveric human elbows were obtained to determine the number of cycles required to reach 25% strain change. Specimens were divided into three groups consisting of 6, 5, and 6 pairs of specimens. Each group was loaded cyclically (2Hz) with load magnitudes of 25%, 33% and 41% of UTS. Ultimate tensile strength was obtained from the contralateral side of each pair.

After specimens were preconditioned and preloaded, initial specimen length was monitored and measured when the load cell (500Lbs) attached to the MTS machine reached the pre-determined load magnitude (for example: 25% or 33% or 41% of UTS) under displacement control. Load and displacement were monitored simultaneously with a load cell and linear variable displacement transducer (LVDT), respectively. To avoid creep behavior during displacement control, specimens were loaded at 0.25 Hz. Based

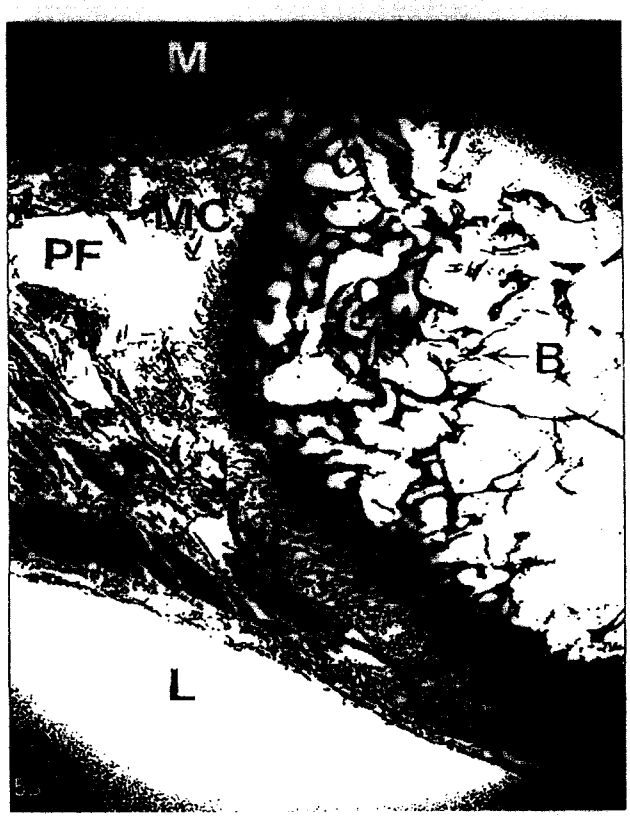

Fig. 5. Histological observation of A:Normal, B:Partial failure (×25), and c:magnified partial failure site (×100) at the bone-tendon junction of the common extensor tendon of humeral epecondyle (Masson-trichrome staining). (TM:tide mark, B; bone, T:tendon, PF:partial failure, MC:magnified area, L:lateral, M:medial, UCf:uncalcified fibrocartilage, CF:calcified fibrocartilage)

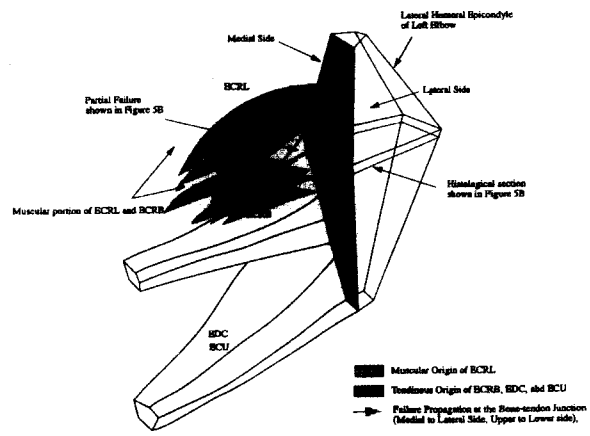

Fig. 6. Three-dimensional schematic diagram for histological section shown in Figure 5B demonstrates th location of the failure site and its failure propagation pattern

on the initial length of the specimen, displacement required to reach target strain changes (%) was precalculated as follows:

$$l_c = \frac{\varepsilon_c \cdot l_f}{100} + l_f$$

Where l_c is the required displacement under cyclic load over time to reach a target strain change 25%, ε_c is target strain change (%) under cyclic load with 25, 33, 41% of UTS, and l_f is a initial length required to reach a pre-determined load, such as 25, 33, 41% of UTS, under displacement control. After the required displacement was calculated, specimens were cyclically loaded until the required displacement was achieved under load control. After specimens reached target strain, the MTS machine was stopped manu-

Table 1. Ultimate tensile strength for common extensor and flexor tendons

	Extensor (N/cm^2)		Flexor (N/cm^2)	
	Slow	Fast	Slow	Fast
Male(n=12)	1163.4±365	888.0±195.8	1816.4±914.7	2107.5±703.3
Famale(n=12)	1234.5±412	937.0±171.9	2027.5±614.1	1776.6±394.8
Average(n=24)	1198.5±388	912.5±183.0	1921.5±764.0	1941.5±548.0

Table 2. The measured cross-sectional area of common extensor and flexor tendons

	Male (n=24)		Female (n=24)	
	Extensor	Flexor	Extensor	Flexor
Mean	0.7394	0.6555	0.5958	0.4936
*S.D.	0.1656	0.1297	0.1355	0.1280

Table 3. Average values of width, thickness, and cross-sectional area of one extensor and flexor tendon

	Extensor		
	Width (cm)	Thickness (cm)	*CSA (cm^2)
Mean	2.942	0.255	0.7474
*S.D.	0.108	0.020	0.0400
	Flexor		
Mean	2.576	0.170	0.4376
*S.D.	0.124	0.012	0.0210

ally. Simultaneously, the number of cycles was recorded by a window counter. Histological observations were made after the specimen had reached 25% strain change.

Histological Observation of Failure Pattern : Tested specimens were saved carefully and harvested for biopsy to examine the failure (or damage) pattern at the bone-tendon origin histologically. Each bone-tendon unit was marked for proximal-distal orientation and fixed in neutral buffered formalin. All specimens were decalcified with nitric acid, embedded in paraffin wax and cut on a microtome at 8um thickness after freezing the bed. Paraffin sections were stained with hematoxylineosin and Masson-trichrome.

Statistical Analysis: Data were analyzed using a two-way analysis of variance (ANOVA) to determine any statistically significant difference in ultimate tensile strength due to gender (male versus female), loading conditions (fast versus slow), and tendon specimens (flexor versus extensor). Significance was set at $p<0.05$. A power analysis using JMP™ software was performed to establish the credibility of a data set within a given sample size.

RESULTS

Tensile testing : Tables 1 and 2 show the biomechanical data for ultimate tensile strength as well as the cross-sectional area of tendon measured by area micrometer technique before testing. The values of ultimate tensile strength were 1198.5±388 N/cm^2 and 912.5±183 N/cm^2 for extensor and 1921.5±764 N/cm^2 and 1941.5±548 N/cm^2 for flexor tendon under slow and fast loading rates, respectively. Ultimate tensile strength was normalized with the mean value of ultimate tensile strength of the extensor tendon specimens (slow loading rate case) for a better comparison (Fig. 2).

Ten consecutive measurements were made to obtain the width and thickness of the bone-tendon specimens with an area micrometer technique, which showed high repeatability (Table 3). The cross-sectional area of prepared tendon was wide and thin, similar to flat sheets, 2-3mm in thickness and 25-30mm in width, while the bone-tendon origin was auricular shaped. Average cross-sectional areas for extensor and flexor tendons were 0.74 cm^2 and 0.66 cm^2 for male specimens and 0.60 cm^2 and 0.49 cm^2 for female specimens.

After tensile testing, two different modes of failure of the common extensor and flexor tendons were observed. Forty-six specimens under microscopic and histological examination showed bone-tendon origin failure without bony fragments or with minimally sized bony fragments, two specimens showed avulsion of bone at the bone-tendon origin. These examinations revealed that the failure site occurred mostly at the bone-tendon origin around the uncalcified fibrocartilage zone (Fig. 3).

The Number of Cycles versus Strain Changes : We found a correlation between 25% strain change and the number of cycles when load magnitudes were 25, 33, and 41% of UTS (Fig. 4). The number of cycles required to reach 25% strain change for common extensor and flexor specimens were approximately 8,853, 1,907 and 410 when 25, 33, and 41% of UTS were applied, respectively.

Failure Patterns : Histological observation of the bone-tendon specimens after cyclic and tensile loading in our study revealed that failure initiated at the upper and medial aspect of the extensor carpi radialis brevis from its origin and led to complete failure (Fig. 5). This complete failure propagated from the medial to the lateral aspect of the extensor carpi radialis brevis and simultaneously from the upper to the lower aspect of the extensor carpi radialis brevis toward the tendinous origin of the extensor digitorum communis and minimal part of the extensor carpi ulnaris (Fig. 6).

DISCUSSION

Our study was designed to 1) measure ultimate tensile strength of common extensor and flexor tendons of human humeral epicondyle, 2) correlate the number of cycles with strain change, and 3) identify failure patterns of specimens histologically after cyclic and tensile loadings

Statistical analysis showed no significant differences in ultimate tensile strength between slow and fast loading rates or between male and female specimens ($P<0.05$). This biomechanical data are compatible with clinical findings which showed that male and female prevalence rates of epicondylitis appear equal[15]. Mechanical failure strength was not loading rate sensitive, which coincides with results from other studies[31]. However, statistically significant differences in ultimate tensile strength between the common flexor tendons specimens were found ($p<0.05$). The common flexor tendons had 83% greater ultimate tensile strength than the common extensor tendons.

Failure from accumulated repetitive load can be explained by the "fatigue failure" phenomenon[1]. The more repetitions applied, the more strain changes will occur, thus leading from damage to failure. Additionally, the occurrence of damage seems proportional to the sum of energy absorbed; the more force applied, the fewer number of cycles required to create damage. We attempted to evaluate the threshold of failure by correlating strain change with the number of cycles. The correlation coefficient was $R^2=0.46$; its power analyses showed strong power (power=0.87).

Specimens had higher incidence of failure at the bone-tendon origin(96%) than avulsion(4%) under tensile testing. No midsubstance failure was observed regardless of differences in loading rates. Viscoelastic material properties change[37] rapidly at the bone-tendon junction zone, which is divided into four zones: tendon, uncalcified fibrocartilage, calcified fibrocartilage and lamellar bone[2,7,13,37]. The exact failure site in our specimens occurred around the uncalcified fibrocartilage zone (Fig. 3). This result agreed with our hypothesis and previous clinical observations[26,27, 34] that the weakest point in the bone-tendon unit of the lateral epicondyle is the transitional zone between bone and tendon.

The extensor carpi radialis brevis origin is often cited by researchers[7,8,25-27] as the primary site of abnormalities. This abnormal process may originate in either the extensor digitorum communis or extensor carpii radialis brevis tendon[18]. Nirschl and Pettrone[27] and Warhold et al.[34] observed during gross examination that the origins of the extensor carpi radialis brevis usually appear as grayish, immature scar tissue, which looks shiny, edematous, and friable at surgery. Soft-tissue calcification or bony exostoses are often seen later in the process of epicondylitis. They assumed that this calcification resulted from partial detachment of the tendon from its origin at the lateral epicondyle. Histologic evaluation of this tissue characteristically demonstrates an invasion of fibroblasts and granulation tissue, described as "angiofibroblastic hyperplasia" by Nirschl and Pettrone[27].

Histological findings from our study represent only mechanical failure patterns, which may not be the same as abnormalities observed clinically later in the process of epicondylitis, which usually involves pathophysiological change after trauma. Our study showed that the mechanical failure pattern can be initiated as the result of partial failure at the bone-tendon origin (especially, the uncalcified fibrocartilage zone) of the common extensor or flexor tendon of the humeral epicondyle.

Seventy-five percent of patients experience symptoms of lateral epicondylitis in their dominant arm[18]. However, we lacked information about the cadaveric specimens, and thus could not determine differences in ultimate tensile strength and strain response between dominant or non-dominant arms. Also, we revealed that two different loading rates used in this study may be limited to draw our results of UTS. Epicondylitis is reported to have a high incidence in the 40 to 50 year old age group[27], when its incidence is four times greater than at any other age[7]. Since there was limited availability of cadaver specimens, we were not able to address the effects of age (mid-40s), which has most prevalence in epicondylites, on the biome-

chanical properties.

This study was limited to biomechanical laboratory testing, which did not address clinical aspects of the etiology of epicondylitis. However, biomechanical data obtained in this study will be useful as we work toward an understanding of the etiology of epicondylitis. In the future, animal models should be developed to approach this problem more clinically.

REPERENCES

1. Anderson, T.L. *"Fatigue Crack Propagation"* Fracture Mechanics, Fundamentals and Application, Chapter 10, 597-651, CRC Press, Boca Raton, Florida, 1991.
2. Benjamin, M., Evans, E.J., Copp, L. *"The Histology of Tendon Attachment to Bone in Man"* J. of Anatomy, 149:89-100, 1986.
3. Bureau of Labor Statistics *"Occupational Injuries and Illness in the United States by Industry-1988 Bulletin 2366"* Washington D.C. Government Printing Office, 1990.
4. Butler, D.L., Grook, E.S., Noues, F.R., Zernicke, R.F., Brackett, K. *"Effects of Structure and Strain measurement Techique on the material properties of Young Human Tendons and fascia"* J Biomech 17:579-596, 1984.
5. Butler, D.L., Kay, M.D., and Stouffer, D.C. *"Comparison of Material Properties in Fascicle-Bone Units from Human patellar Tendon and Knee Ligaments"* J. Biomechanical 19:L425-432, 1986.
6. Cooper, R.R., and Misol, S. *"Tendon and Ligament Insertion"* Journal of Bone and Joint Surgery, 42A:1-20, 1970.
7. Coorad, R.W. and Hooper, W.R. *"Tennis Elbow: Its Course, Natural History, Conservative and Surgical Management"* J Bone Joint Surg, 55A:1177-1182, 1973
8. Cyriax, J.H. *"The pathology and treatment of tennis Elbow"* J Bone Joint Surg, 18:921-940, 1936.
9. Dorlot, J.M., Ait Ba Sidi, M., Trembly, G.M., and Drouin, G. *"Load Elongation Behavior of the canine Anterior Cruciate Ligament"* J. Biomech Engr 102:190-193, 1980.
10. Ellis, D.G. *"Cross-sectional Area Measurement for tendon Specimens: A Comparison of Several Methoks"* J Biomech 2:175-186, 1969.
11. Frederick, L.J. *"Cumulative Trauma Disorder"* AAOHN J., 40:3:113-116, 1992.
12. Garden, R.S. *"Tennis Elbow"* J Bone Joint Surg 43B: 100-106, 1961.
13. Goldie, I. *"Epicondylitis Lateralis Humeri: A Pathological Study"* Acta Chir Scand (Suppl) 34:339, 1964.
14. Graf, B.K., Ulm, M.J., Rogalski, R.P., Vanderby, R. *"Effect of Preconditioning on the Viscoelastic response of Primate Patellar teneon"* Trans Orthop Res Soc 17:147, 1992.
15. Gruchow, H.W. and Pelletier, D. *"An Epidemiologic Study of Tennis Elbow: Incidence, Recurrence, and Effectiveness of Prevention Strategies"* An J Sports Med., 1979;7: 234-238.
16. Haut, R.C., Powlison, A.C., Rutherford, O.W., and Kateley, J.R. *"Some Effects fo Donor Age and Sex on the mechanical Properties of Patellar Tendon Graft Tissues"* ASME, Adv. Bioeng 8:75-78, 1988.
17. Haut, R.C. and Powlison, A.C. *"The Effects of test Environment and Cyclic Stretching on the Failure Properties of Human Patellar Tendons"* J Orthop Res 8:532-540, 1990.
18. Jobe, F.W. and Ciccotti, M.G. *"Lateral and medial Epicondylitis of the Elbow"* J Am Acad of Ortho Surg 2:1-8, 1994.
19. Johnson, G.A., Tramaglini, D.M., Lenine, R.E., Ohno, K., Choi, N.Y., and Woo, S.L-Y. *"Tensile and Viscoelastic Properties of Human Patellar Tendon"* J. Orthp Res. 12: 796-803, 1994.
20. Kennedy, J.C., Hawkins, R.J., Willis, R.B., et al. *"Tension Studies of Human Knee aLigament. Yield Point, Ultimate Failure, and Disruption of the Cruciate and Tibial Collateral Ligaments"* J Bone Joint Surg 58A:350-355, 1976.
21. Leach, R.E., and Miller, J.K. *"Lateral and Medial Epicondylitis of the Elbow"* Clinics in Sports Med 6:259-272, 1987.
22. Lyon, R.M., Lin, H.C., Kwan, M.K., Hollis, J.M., Akeson, W.H., Woo, S. L-Y. *"Stress Relaxation of the Anterior cruciate Ligament(ACL) and the Patellar Tendon (PT)"* Trans Orthop res Soc 13:81, 1988.
23. Luon, R.M., woo, S. L-Y., Hollis et al. *"Comparison of the Mechanical Properties of the Medial Collateral and anterior Cruciate Ligaments of the Rabbit Knee"* ASME/ AMD 98:53-56, 1989.
24. Myers, M.I., Withers, P.C., and Johnson. J.A. *"Program of the National Institute for Occupational Safety and Health"* (DHHS Publication No 84-107), Washington, D. C.: U.S. Government Printing Office, 1984.

25. Nirschl, R.P. "*Tennis Elbow*" Ortho Clin North Am 4: 787-800, 1973.
26. Nirschl, R.P. "*Defining and Treating Tennis Elbow*" Contemp Surg 10-13-17, 1977.
27. Nirschl. R.P. and Pettrone, F.A. "*Tennis Elbow: The Surgical Treatment of Lateral Epicondylitis*" J Bone Joint Surg 61A: 832-839, 1979.
28. Noyes, F.R. and Frood, E.S. "*The Strength of the Anterior Cruciate Ligament in Humans and Rhesus Monkeys. Age Related and Species Changes*" J Bone Joint Surg 58A:1074-1082, 1976.
29. Noyes, F.R., Butler, D.L., Grood, E.S. et al, "*biocechanical Analysis of Human Ligament grafts used in Knee-ligament repairs and Reconstructions*" J Bone Joint Surg, 66A344-352, 1984.
30. Paulos, L.E., france, E.P., Resonberg, T.D., et al. "*Eompatative Material Properties of Allograft Tissues for Ligament Replacement. Effects of Type, Age, Sterilization and Preservation*" Trans Orthop Res Soc 33:129, 1987.
31. Peterson, R.H. and Woo, S. L-Y "*A New Methodology to Determine the mechanical Properties of Ligaments at High Strain Rate*" J of Biomechanical Engineering, 108:365-367, 1986.
32. Tanka, S., Seligman, P., Halperin, W., Thun, M., Timbrook, C.L., and Wasil, J.J. "*Use of Workers' Compensation Claims Data for Surveillance of Cumulative Trauma Disorders*" Occup. Medi., 30:6:488-492, 1988.
33. Viidk, A. "*Elasticity and tensile strength of the Anterior Cruciate Ligament in rabbits as Influenced by training*" Acta Orthop scad 74:372-380, 1968.
34. Walhold, L.G., Osterman, A.L., and Skirven, T.C. "*Lateral Epicondylitis: How to Treat it and Prevent Recurrence*" 1993.
35. Woo, S. L-Y., Gomez, M.A., Akeson, W.H. "*The Time and History Dependent Viscoelastic properties of the canine medial collateral Ligament*" J Biomech Eng 103:293-298, 1981.
36. Woo, S. L-Y., Hollis, J.M., Roux, R.D. et al. "*Effects of Knee flexion on the Structural Properties of the Rabbit Remut-Anterior Cruciate Ligament-tibia Complex (FATC)*" J Biomech 20:557-563, 1987.
37. Woo, S.L., Maynard, J., Butler, D., Lyon, R., Torzilli, P., Akezon, W., Cooper, R., and Oakes, B. "*Ligament, Tendon, and Joint Cpsule Insertions to Bone*" In Injury and Repair of the Musculoskeletal Soft Tissue (Eds: Woo, S.L. and Buckwltr,J.A.) Park Ridge, Illinois, American Academy of Orthopedic Surgenon, pp133-166, 1988.
38. Woo, S. L-Y., Hollis, J.M., Adams, D.J., Luon, R.M., and Takai, S. "*Tensile Properties of the Human Femur-Anterior Cruciate aligament-Tkbia Complex: The effects of Specimen Age and Orientation*" The American Journal of Sports Medicine, Vol 19:3:217-225, 1991.