THE ROLE OF FATIGUE IN SUSCEPTIBILITY TO MUSCLE STRAIN INJURY. Scott D. Mair, Anthony V. Seaber, Richard R. Glisson, William E. Garrett Jr. Department of Surgery, Duke University Medical Center, Durham, North Carolina.

It is thought that fatigue may be a predisposing factor in the occurrence of muscle strain injuries. A previous study developed a model for an acute and reversible state of fatigue in skeletal muscle, and showed that extreme fatigue results in decreased peak force, length change, and energy absorbed prior to failure (1). The purpose of the present study was to extend a similar model toward investigation of less severe and more precisely defined levels of fatigue, while also examining the interrelation of fatigue and rate of strain. In addition, an attempt was made to elucidate the mechanism by which fatigue influences muscle failure properties.

METHODS: This study was divided into two parts. In Part I, the extensor digitorum longus (EDL) muscles of 48 rabbits were surgically exposed and the distal tendon ligated and clamped to an Instron machine. After preconditioning, peak isometric tetanic force (Fm) and the muscle length at which it was generated (Lm) were determined by plotting sequentially active and passive length-tension curves. Stimulation was via the common peroneal nerve at 50 Hz and 5 times threshold voltage. One limb's EDL (fatigue side) was set at Lm and received cycles of 5 sec of tetanic stimulation alternating with 1 sec rest periods, continued until the muscle's ability to generate contractile force had been reduced from Fm by a predetermined amount (25 or 50%). The muscle was then immediately stretched to failure while stimulated. The contralateral EDL was fatigued in the same manner, allowed to rest until Fm had been restored, then activated and pulled to failure. Each rabbit was assigned to one of three strain rate groups (1,10,50 cm/s). Thus, there were two levels of fatigue and three strain rates, resulting in six groups, each with n=8. From the load-deformation curve of each muscle, the following parameters were calculated: 1) force to rupture, 2) percent length change to rupture, 3) energy absorbed prior to rupture. Statistical evaluation of fatigue vs. control muscles was by paired Student's t-test, and comparison of strain rates by factorial ANOVA. Part II of the study compared fatigue to submaximal stimulation, with 8 rabbits tested. One limb was mobilized, fatigued, and stretched to failure exactly as in Part I. The contralateral EDL was stimulated at low frequency, and this frequency was gradually increased until the force generated was equal to that of the fatigue side, before stretching to failure.

Figure 1

Figure 2

RESULTS: Part I. Force was reduced on the fatigued leg in all groups, ranging from 93.0 to 97.4% of controls. Statistical significance was found only in the 50% fatigue groups at the 1 and 10 cm/s strain rates. Percent length change was not different from controls in any fatigue group. Energy absorbed (Fig. 1) was significantly reduced in the fatigued limb in all groups, with the difference more pronounced at the 50% fatigue level. In addition, the effect of fatigue was found to be significantly greater at a slower rate of stretch. Figure 1 demonstrates the load-deformation curves of representative control and fatigue muscles. Most of the change in energy absorption occurs at lower deformations, early in stretch.

Part II. Failure properties were as follows:

	Force(N)	Length Change(%)	Energy Abs (J)
Fatigue	94.0±4	17.1±2	1.12±.07
Submax stim	94.5±5	17.0±2	1.13±.06

None of these values are significantly different.

DISCUSSION: Muscles which are activated and stretched are absorbing energy. These data suggest that muscle strain injury is more likely in a fatigued muscle, as it is unable to absorb as much energy prior to failure. Most of this effect of fatigue occurs early in stretch, so in order to absorb the same amount of energy, fatigued muscle must stretch further than non-fatigued muscle. Energy absorption (Fig. 2) was divided into two regions at 70% length increase to rupture, because previous studies have shown that it is at approximately this point where histologic damage begins to occur (2). Thus, the difference in energy absorption is more pronounced in the part of the stretch where it would be expected to be most important in preventing injury. These findings related to fatigue appear to be true for any factor which diminishes contractile ability of muscle, as maximally stimulated fatigued muscle showed no difference in failure properties when compared to submaximally stimulated non-fatigued muscle. This indicates that muscle strain injuries may also be more likely in settings where muscle is placed under stretch and central activation is diminished, e.g. uncoordinated or unexpected movements. These data also suggest that proper conditioning to avoid fatigue, as well as exercise modification in the event of fatigue, can help prevent acute muscle strain injuries.

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IN-VIVO ANALYSIS OF CANINE INTERVERTEBRAL AND FACET MOTION. K. Wood, M. Schendel, G. Butterman, J. Lewis, D. Bradford, Biomechanics Lab, Univ. of Minnesota, Minn., MN 55455

INTRODUCTION: Changes in joint motion may be directly related to the development of primary osteoarthritis. Animal models developed to study the early stages of the disease reveal that joint laxity initiates a series of events leading to articular degeneration. Van Sickle has suggested that the laxity accompanying aging joints allows traumatization of the articular surfaces during normal ambulation. Present models, however, usually involve capsular intervention with damage to the synovium which may release catabolic factors. Kinzel developed an instrumented spatial linkage (ISL) to measure the motion between the articular surfaces of the canine shoulder during locomotion. An ISL is a device constructed of 6 potentiometers to measure the 6° of freedom during motion. Analog signals from the potentiometers are fed into an analog to digital converter and processed with a computerized data aquisition system. Points on each bony surface are digitized, local coordinate systems defined, and by means of transformation matrices determined by the ISL, the coordinates of one surface are transformed into the coordinate system of the other. The location of any point on one body segment relative to the other, at any instant, can be determined. The canine intervetebral joint complex is an excellent model for the study of early osteoarthritis. Previous studies have

demonstrated histologic changes at the canine facets consistent with early osteoarthritis following chemonucleolysis-induced disc space narrowing. To investigate whether this arises from abnormal motion we developed a method to study the motion of the canine facet surfaces during locomotion.

MATERIALS AND METHODS: 5 adult mongrel dogs, 25-30 kg., were selected for the study. 4 4mm threaded stainless steel pins, 3.8 cm. in length, were placed transpedicularly into L2 and L3, via 2 small paralumbar incisions. 2 cms. of pin protruded into the paralumbar musculature. 3 to 4 days later the dogs were given Atropine and Surital intravenously to induce a brief anesthesia. The original incisions were reopened, complimentary pins 11 cm. in length, with a 2 cm threaded sleeve were firmly attached to each implanted pin. Lightweight aluminum cross bars were attached to each set of pins to provide further rigidity. A short acting local anesthetic was injected all about the incision, and the skin was loosely approximated about the protruding pins. Once the dog recovered, an ISL was mounted. Each dog was then put through a series of maneuvers: induced walking, bending, moving from sitting to walking, and moving from a 4 leg stance to a hind leg position. Voltage data was collected through-out. When testing was complete, the spine was harvested. The 2 vertebral body motion segment, with the pins and ISL still in place was potted at each end in methylmethacrylate and subjected to a series of 100Nm bending and torsion loads. The transverse, and spinous processes of each vertebral body were digitized using a stationary probe to determine local coordinate systems, which had origins within the neural canal ventral to the facet articular surfaces. In addition, opposing surfaces of the rt. articular facet joint were digitized as were the crosshairs at each base of the ISL. The ISL was removed and calibrated. Data was reduced to the sagittal, axial and coronal plane vertebral body and facet position changes of L2 relative to L3, as well as rotation changes in these same planes.

RESULTS: Optimization of the ISL revealed an accuracy of .5mm and .6°. Only the walking tests including sit to walk, and up onto hind legs produced consistent data between animals. With the canine lumbar facets primarily aligned in the sagittal plane, it appears that in normal locomotion, although from dog to dog the ranges of facet motion during walking were wide, within each animal it was much less varied as the facet surfaces primarily traverse a course of 3.4mm on each other with a ventral to dorsal slope.

DISCUSSION: We present a new method whereby motion at the canine facet level can be measured in-vivo during normal locomotion. The information provided accurately traces the path of the facet articular surfaces during the gait cycle. The information obtained is a valuable adjunct to the study of early osteoarthritis. An advantage to this method is that one can perform long term measurements. To test the hyposthesis that increased joint motion may be an etiology of early joint degradation, we have applied this technique to dogs whose discs have been injected with chymopapain. This study is in progress.