INTRODUCTION
The interactions of biological and mechanical factors in the pathogenesis of secondary osteoarthritis are not well understood. We have previously reported alterations in joint kinematics due to ligament deficiency in an in vivo ovine stifle joint model of osteoarthritis [1]. These kinematic changes were associated with severe cartilage degeneration 20 weeks after ligament transection.

Tribological studies of synovial joints suggest that both boundary and fluid film lubrication mechanisms are important in the lubrication of synovial joints [2]. In both forms of lubrication, surface velocity is an important determinant of the coefficient of friction at the joint. We hypothesize that changes in joint surface velocity after ligament transection may alter the joint lubrication mechanism and play a role in joint degeneration.

This study uses the ovine stifle joint model of osteoarthritis to investigate the hypothesis that the in vivo kinematics at the joint surface are altered following ligament transection.

METHODS
Ovine stifle joint model
Six skeletally mature trained Suffolk sheep were studied while walking at 2mph on a standard treadmill. The in vivo kinematics were measured for thirty strides with the joint intact, and at 2 and 20 weeks following combined transection of the anterior cruciate and medial collateral ligaments.

In vivo kinematic measurement
A rigid surgically implanted bone marker system was used to define the 3D positions of the tibia and femur during the in vivo motion. The 3D spatial positions of the bone markers during gait were recorded using a 4-camera high-speed (120Hz) video based motion analysis system (Expert Vision, Motion Analysis Corporation).

In vitro geometry measurement
Following euthenization, a 3D digitizer (FaroArm, Faro Technologies Inc) was used to define anatomically based coordinate systems within the tibia and femur (accuracy±0.05mm). The bone markers positions were then digitized relative to these anatomical coordinate systems for registration with the in vivo motion analysis data. The geometry of the articular surfaces of the tibia and femur were digitized by taking scans (resolution 0.05mm) over a 3mm grid projected onto the surfaces. Prior to digitizing, the bones were soaked in full strength bleach to remove remaining articular cartilage.

Reconstruction of joint surface geometry
The clouds of points obtained from digitizing were imported into 3D modeling software (CATIA, Dassault Systemes) and nurb surfaces were created through the point clouds (average deviation<0.15mm). A 4mm grid was projected onto the surfaces and the points of intersection were sampled for kinematic analysis.

Analysis of kinematic data
The 3D marker coordinates were tracked and smoothed (quintic spline; cutoff 6Hz), and then differentiated to obtain velocity and acceleration data in the global coordinate system. The method of Verstraete [3] was used to calculate the angular velocities of the tibia and femur at each time point. The transformation matrices between the global and anatomical coordinate systems were calculated using singular value decomposition [4]. Tibial joint angles and translational positions were described using a joint coordinate system. The positions of the anatomical origins were derived from the transformation matrices and then differentiated to obtain their velocities. The velocities of each point on the tibial surface were then calculated as points on the tibia and the corresponding points on the femur ($v_p = v_o + w x r_p/o$). The relative velocities between the tibia and the femur were then calculated and expressed relative to the tibial anatomical coordinate system. All data were normalized to percentages of the gait cycle between successive hoofstrikes.

For each specimen, the in vivo kinematics of the transected joint were compared with those of the intact condition. The pooled data were assessed using a standard t-test (p<0.05).
RESULTS AND DISCUSSION
The velocities of the tibia with respect to the femur, measured at the tibial insertion the anterior cruciate ligament, were significantly altered at both 2 and 20 weeks after ligament transection (Figure 2). At 2 weeks after transection, the anterior tibial velocity at hoofstrike and early stance (0-10%) was significantly increased. Prior to and during the flexion phase of swing (62-75%), the tibial velocity in the posterior direction was significantly increased. At the final extension phase of swing (99-100%) the tibial velocity in the anterior direction was significantly increased. Significant changes persisted and were further increased at 20 weeks after transection.

Figure 1. Velocity in the anteroposterior direction at hoofstrike calculated at 33 points on the lateral tibial plateau.

Ligament transection caused dramatic increases in both the magnitude and direction of the tibial velocities at hoofstrike (0%). The velocities at hoofstrike are shown for 33 points on the lateral tibial plateau of a representative subject (Figure 1). Quiver plots are superimposed over contour plots of the tibial surface geometry to show the surface velocities at the grid points (Figure 2). The vectors are scaled to $1/10^6$.

SUMMARY
Ligament transection causes significant changes in the in vivo kinematics at the joint surface. Further analysis of this data will investigate the significance of these changes throughout the gait cycle, and assess the relationship between these changes and the degenerative changes observed in these joints. The results of this investigation lend support to the hypothesis that changes in joint surface velocity are an important factor in the development of osteoarthritis in unstable joints.

REFERENCES

Figure 2. Velocity vectors on the tibial surface at hoofstrike (0%): A) intact joint   B) 2 weeks after ligament transection C) 20 weeks after ligament transection. The colorbar indicates the z-coordinates of the surface.

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