The effect of nerve injury on medial collateral ligament healing

T.J. Ivie, R.C. Bray, P.T. Salo,
Department of Surgery
University of Calgary, Calgary/ Alberta

INTRODUCTION

The role of innervation was examined to determine if it regulates medial collateral ligament (MCL) perfusion and repair. After lesioning innervation remotely, the physiological importance of neuronal populations responsible was found by observing functional deficits in injured MCL's. The MCL heals relatively well, however, specific contributions of the nervous system to the repair of the MCL are unknown despite the prevalence of these injuries. By lesioning innervation remotely, the contribution of MCL neuronal populations to healing can be determined. By lesioning the femoral nerve distal to the inguinal ligament, all known MCL innervation should be ablated. We hypothesize that denervated MCL's will exhibit inferior vascular response as well as inferior material and structural biomechanical properties than those with intact innervation.

METHODS

24 female adult (1 year) New Zealand white rabbits were used. All underwent transection of the right MCL at the midsubstance and half additionally underwent lesioning of the right femoral nerve without immobilization or repair. At 6 weeks the injured ligaments were assessed for blood flow using laser Doppler imaging and were harvested (half denervated and half intact innervation). Quality of repair was assessed with biomechanical tests for low load viscoelastic properties and ultimate failure.

RESULTS

At 6 weeks blood flow had decreased in the midsubstance by 64% from 239.77 +/− 39.05 perfusion units in innervated MCL's to 87.12 +/− 38.51 perfusion units in denervated MCL's, p< 0.05 (Fig.1). For healing tissue at 6 weeks the force required for ultimate failure was found to be significantly elevated by 34% in innervated MCL's: 153.14 +/− 20.71 N versus denervated MCL's: 101.29 +/− 17.88 N, p< 0.05 (Fig. 2). Total creep was significantly elevated by 45% in denervated MCL's: 5.29 +/− 0.62% than in innervated MCL's 3.64 +/− 0.31%, p<0.05 (Fig. 3). Static Creep was also significantly elevated by 66% in denervated MCL's: 2.83 +/− 0.45% than in innervated MCL's: 1.70 +/− 0.12%, p< 0.05 (Fig. 4). No significant difference was found in any physiological or biomechanical properties between contralateral tissues.
Fig. 1 Blood Flow

Fig. 2 Force at Failure

Fig. 3 Total Creep

Fig. 4 Static Creep
DISCUSSION

Our hypothesis that denervated MCL's will exhibit an inferior vascular response than those with intact innervation has been validated. In healing tissue, a vascular response is very important to ensure a quick healing time. Increased blood flow may remove heat by convection, add or remove regulatory substances, deliver oxygen, remove wastes, and even alter viscoelastic properties. Our hypothesis that denervated MCL's will exhibit inferior low load material and structural biomechanical properties than those with intact innervation has also been validated. The ultimate force at failure was significantly lowered in MCL's that had been denervated. It is unclear what neuronally derived mechanism has caused this weakness. However, since ligaments require sufficient strength for functional normality, further studies in neuronal contributions to MCL healing should be made.

The material properties (Creep) of denervated MCL's were found significantly elevated. No histology was performed to determine any differences in collagen deposition or fibroblast recruitment in healing tissue. Therefore, it is not certain what specific tissue changes occurred as a result of the denervation.

By further determining how denervation affects blood flow in healing MCL, we may understand why biomechanical properties change when healing tissue is denervated.

ACKNOWLEDGEMENTS

This work was supported by the Arthritis Society of Canada. Dr. Salo is a Clinical Investigator and Dr. Bray a Scholar of the Alberta Heritage Foundation for Medical Research. We thank Catherine Leonard, Kent Paulson, Ruth Seerattan, and Craig Sutherland for their expert technical assistance.