AN IN VIVO MODEL FOR ENTRAPMENT NEUROPATHY DUE TO REPEATED FINGER LOADING

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Introduction

Carpal tunnel syndrome is the most common entrapment neuropathy and accounts for high levels of disability in the workplace. Carpal tunnel syndrome may be caused by repetitive, forceful fingertip loading; however, the mechanisms of injury and the relationships between force-duration and injury are not fully known (1). Knowledge of the injurious mechanical factors (e.g., load rate, number of repetition cycles, peak load, etc.) would be useful in developing primary and secondary preventive measures. However, these mechanistic details will primarily emerge with the development of an appropriate animal model.

The purpose of this study was to develop and validate a rabbit model of median neuropathy at the wrist due to repetitive loading of the flexor digitorum profundus (FDP) muscle. The rabbit forearm, carpal tunnel, fingers and nerve anatomy are roughly similar to the human.

Methods

Five female, New Zealand White rabbits weighing 2.6 kg (± 0.3) were studied. Under general anesthesia the left FDP muscle was repetitively loaded for 2 hours per day, 3 days per week, for 7 weeks (21 days of loading). The contralateral limb served as the control. The study was approved by the University's Committee on Animal Research.

After inducing anesthesia with isoflurane, the rabbit was placed on the back with the forearms loosely secured to supports. The muscle stimulation needle (33G) was inserted subcutaneously in the mid-forearm region over the central region of the FDP and the needle tip was pushed back through the skin. A brass fingertip glove was slipped over digit 3 and connected to a load cell to measure flexion force of the finger about the metacarpalphalangeal joint. The muscle was stimulated (Grass) with a train of pulses at 1 Hz, with a train duration of 200 ms and a pulse rate of 100 pulses per second. The stimulation voltage was adjusted [6-12 V] to maintain a peak fingertip force of 0.36 N (15% of P_0). Fingertip force data was sampled every hour. After 2 hours of repetitive loading the stimulation electrode and fingertip glove were removed. The procedure was repeated 3 days per week for 7 weeks.

The distal motor latency (DML) of the median nerve across the carpal tunnel was measured at the beginning and end of the study in both forepaws using a clinical nerve conduction machine (TECA 200). Under general anesthesia the rabbit was laid on its back with the forearms resting on a support. The palm temperature in all cases was greater than 35 C. The recording needle electrode was placed into the first dorsal interosseus muscle (innervated by the median nerve). The stimulating electrode was inserted subcutaneously 25 mm proximal to the recording electrode over the median nerve. The stimulation current was set to 1.5 times the stimulation threshold and the DML to the takeoff was measured 3 times.

Results

Daily examinations of the wrist, forearm and elbow revealed no limping, reduction in gross claw flexion strength, skin breaks, or reduced finger or wrist motion. Mean rabbit weight at the end of the study was 3.2 kg (± 0.2).

At the start of the study the mean DML was 1.36 ms (± 0.10) on the loaded side and 1.37 ms (± 0.08) on the control side [N=5]. At the end the DML was prolonged on the loaded side. Pre-post change in DML for the loaded side was -0.17 ms (± 0.15) and for the control side was 0.03 ms (± 0.03). This difference between sides was borderline significant [p=0.06, paired t-test].

Necropsy evaluation of the subcutaneous region of the stimulation needle insertion site revealed minimal scar tissue localized within 5 mm of the insertion site; the scar tissue did not extend to the median nerve, which in this region, is deep to the flexor carpi radialis.

Discussion

In this rabbit model 1 Hz cyclical loading of the finger flexor at 15% of P_0 was produced for 2 hours without a decline in fingertip force. The loading regimen was well tolerated when applied for 3 days per week for 7 weeks. This loading regimen produced an electrophysiologic decrement in nerve function that was borderline statistically significant. The change can be considered important given the small sample size.

Previous models of entrapment neuropathy have involved direct compression of the nerve with a balloon or tube (2,3). As far as we are aware this is the first model of entrapment neuropathy associated with repeated finger loading. The loading rate, peak fingertip force, and force duration can be tightly controlled in the model.

In the future the model will incorporate examination of the histologic and biochemical changes of the median nerve at the carpal tunnel to evaluate the correlation of structural changes (e.g., nerve fiber count, myelin thickness) to decrement in nerve function. Ultimately, the model may be used to investigate pathophysiologic mechanisms, exposure-disorder relationships, and treatments for entrapment neuropathy associated with repeated finger loading.

Acknowledgement

The study was partially funded by NIH grant R03 OHAR03664.

References

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