EXPERIMENTAL INDUCTION OF CARPAL TUNNEL SYNDROME: A NEW ANIMAL MODEL

INTRODUCTION
Carpal tunnel syndrome (CTS) results from median nerve compression at the level of the transverse carpal ligament within the distal forearm. Peripheral neuropathy and pain ensue. Although it is generally recognized that CTS results from increased pressure within the carpal tunnel,1-3 the mechanical and biologic mechanisms through which this occurs is poorly understood.4 An understanding of these mechanisms would serve as a foundation for improved prevention and treatment strategies. Our group has developed a rabbit model for CTS in which coronary angioplasty balloon catheters are placed within the carpal tunnel and inflated to different magnitudes for varying lengths of time.5,6 Our preliminary data suggest that some key clinical hallmarks of CTS—delayed motor nerve latency, decreased conduction velocity, and histologic axon dropout—are related to the magnitude and duration of increased carpal tunnel pressure. Through this model, we expect to correlate carpal tunnel pressure with vascular events and resultant changes in both nerve and connective tissue histology. Our goal in this study was to establish whether CTS can be induced by excessive mechanical loading at the carpal canal in an animal model and that mechanical loading causes CTS in a dose-dependent manner. METHODS: 1st Experiment: in adult New Zealand White rabbits (2.5 to 2.8 kg wt), an inflatable angioplasty balloon catheter was inserted into the carpal tunnel so that with balloon inflation the contents of the carpal canal would be compressed. An uninflated balloon catheter was inserted into the carpal tunnel of the opposite side as the control. Balloon pressures of 40 mm Hg were tested. The pressure inside the carpal tunnel was measured by a monitor (PROPAC 104 software v. 6.0) at the time of surgery, at daily intervals during the first 7 days, and at weekly intervals until the animals were sacrificed. Nerve conduction EMG studies were performed on the animals using the Dantec EMG machine (Model 1500) at weekly intervals under general anesthesia. Distal motor latency was calculated by stimulating the nerve proximal to the muscle, and recording the motor potential in the thenar muscles with a concentric needle electrode. The nerve conduction velocity was determined by stimulating the nerve distal to the muscle and recording the motor potential. The conduction velocity was calculated by dividing the distance between the stimulating and recording electrodes by the latency. Latent axon dropout was assessed using histologic analysis with the use of osmium and 0.05% cresyl violet. Statistical analysis: The thickness of the endoneurium and the perineurium increased with nerve compression; the population of nerve fibers per unit area decreased, the diameter of the average fiber was reduced, and the average thickness of myelin was reduced as well. DISCUSSION AND CONCLUSION: Graded compression of the median nerve by a balloon catheter results in histologic and electrophysiologic changes to the median nerve. There is a definite dose-response relationship between pressure and time and resultant delays in motor latency. With increasing pressure, there is a decrease in the time required to cause CTS. There is also the suggestion that severity of CTS (% latency delay) is related to the amount of pressure applied to the median nerve. With an increase in pressure, there is a greater percentage delay in motor latency at the time that CTS is established. This is the first report of an animal model for graded carpal tunnel syndrome. In the future, this model will allow further study on the effect of compression and reversal of compression in nerve compression disorders and the effect of co-factors and treatment methods. REFERENCES 1. Marie P, et al. Revue Neurologie 1913; 26:647-650. 2. Gelberman RH, et al. J Bone Joint Surg 1981; 63A:380-383. 3. Landborg G, et al. J Hand Surg 1982; 7A:252-259. 4. Mackinnon SE, et al. Annals of Plastic Surgery 1984; 12:112-120. 5. Olinmark K, et al. Spine 1991;16:61-69. 6. Rydevik B, et al. J Hand Surg 1981; 6A:3-13.