Introduction
A rabbit model for Carpal Tunnel Syndrome has been developed by our group in which balloon angioplasty catheters are placed in the carpal tunnel and inflated. Previous studies have described changes in nerve function in response to compression. (1)

The present study investigates the cellular changes that occur as myelinated fibers undergo degeneration in response to compression using immunohistochemistry to characterize local Schwann cell, macrophage and axon populations.

Materials and Methods
Inflatable angioplasty catheters were inserted into the carpal tunnel of 17 adult male New Zealand white rabbits, and 80mm Hg of pressure was applied. An uninflated catheter in the contralateral limb served as a sham. Nerve conduction studies were performed weekly, and animals were sacrificed when the distal motor latency increased 15% for 2 consecutive weeks. Either the median nerve alone (15 rabbits), or the entire soft tissue bundle surrounding the nerve (7 rabbits) was removed for histological studies. Isolated nerves were imbedded in GMA resin, and 3 micron sections were stained with Giemsa or hematoxylin and eosin. The soft tissue bundles were embedded in paraffin for immunohistochemical studies. The following mouse monoclonal antibodies were used: S100 for Schwann cells associated with myelinated fibers, GFAP for Schwann cells associated with nonmyelinated fibers, NF200 for axons, and both Ram11 and Mac387 for macrophages. Staining results were assessed qualitatively and compared to normal, unoperated median nerves (4 rabbits).

Results
All compressed nerves achieved a 15% increase in latency after two weeks of compression, and the rabbits were sacrificed. None of the shams showed a significant increase in latency.
An extensive inflammatory response around the catheter was evident in the intact specimens of both sham and compressed nerves. Mac 387 and Ram 11 positive cells were prevalent immediately around the catheter, but scarce in most fascicles. (Fig.1)
NF200 staining of myelinated axons showed shrinkage and dropout of axons in degenerating fascicles. (Fig 2) Axon degeneration occurred in more fascicles of compressed nerves than in sham nerves, but significant variations in axon degeneration were seen in both sham and compressed nerves.
S100 staining revealed a variable response. One animal showed a decrease in S100 positive cells in some fascicles of both the compressed and sham nerves, and another showed an increase in S100 positive cells in some fascicles in the compressed nerve only. There was not an obvious change in the remaining 5 animals.
GFAP staining revealed a more definitive response. The number of GFAP positive cells increased in some fascicles in 6 of 7 sham nerves and 6 of 7 compressed nerves. (Fig 3)
Vasculitic changes, including degradation of elastin, thickening of the adventitia and intima, and narrowing or occlusion of the lumen were severe in 10 of 12 compressed nerves. (Fig.4) Mild vasculitic changes were seen in only 3 of 15 sham nerves.

Conclusions
♦ Epineurial and endoneural vasculitic changes were closely associated with the median nerve’s response to compression.
♦ The increase in the GFAP positive cell population that occurred in most sham and compressed nerves appears to be a response to the catheter rather than to compression of the nerve.
♦ NF200 staining showed shrinkage, decreased staining and dropout of axons in degenerating fascicles.
♦ Ram 11 and Mac387 positive macrophages are not a significant factor in the medians nerve’s response to compression.

Discussion
These findings indicate that important cellular events related to nerve compression can be demonstrated in our model that may have clinical ramifications in terms of future treatment strategies.

References:
Diao E, Shao F., Liebenberg E, Rempel D, Lotz JC., Carpal Tunnel Pressure alters nerve function in a dose dependent manner: a rabbit model for carpal tunnel syndrome. J Orthop Res., 2005 Jan, 23(1): 218-23

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