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Title (type in CAPITAL LETTERS): **CORRELATION BETWEEN THE STRUCTURE AND FUNCTION OF THE URINARY BLADDER FOLLOWING PARTIAL OUTLET OBSTRUCTION**

Aims of Study: Prior studies have shown that partial outlet obstruction of the rabbit bladder caused a progressive increase in bladder mass, a progressive decrease in the contractile response to different forms of stimulation, progressive denervation, and a selective decrease in the activity of mitochondrial and sarcoplasmic reticular enzyme activities. In this investigation, the effect of partial outlet obstruction on the physiological responses to field stimulation (nerve mediated contraction) and carbachol (receptor mediated contraction) were correlated with the structure and innervation of the detrusor smooth muscle in each animal.

Methods: A total of 28 rabbits were subjected to partial outlet obstruction which lasted from 1 to 70 days following surgery. Sham operated rabbits were euthanized at 7, 14, 28, and 70 days. At each time period, the rabbits were anesthetized and isolated strips of bladder body were mounted in individual baths and the contractile response to field stimulation and carbachol determined. Three additional strips from each bladder were fixed for electron microscopic analysis.

Results: The results demonstrated that: 1) Bladder mass increased rapidly during the first 7 days, was constant for the next 7 days, and then continued to increase gradually over the 70-day study period. 2) Dysfunction in the contractile response to field stimulation was noted at 14 days following partial outlet obstruction, and progressively increased over the 70-day study period. The dysfucntion in the response to field stimulation (neurogenic contraction) increased at a significantly faster rate than the dysfunction in the contractile response to carbachol. By 70 days, the bladders were severely decompensated.

EM studies demonstrated that detrusor muscle at 1 day after surgery revealed dilation of sarcoplasmic reticulum in some cells while at 3 and 7 days the majority of smooth muscle cells displayed the characteristics of hypertrophy, being extensively corrugated in outline, having increased length of subsarcolemmal dense bands and reduced numbers of caveolae. Perinuclear sarcoplasmic reticulum was increased in amount in many smooth muscle cells and small electron lucent regions within the myofilament-packed sacroplasm were commonly observed. At 14 days after surgery many smooth muscle cell profiles were vacuolated while others contained swollen mitochondria and dilated cisternae of sarcoplasmic reticulum. At 28

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days some smooth muscle cells displayed, in addition to swollen mitochondria, a loosely packed arrangement of myofilaments and an irregular distribution of sarcoplasmic dense bodies, the majority occurring towards the cell periphery. This change was widespread at 43 days when most smooth muscle cells contained grossly swollen mitochondria.

At 43 other cell types in the bladder wall (vascular smooth muscle, endothelial cells and fibroblasts) were first observed to contain occasional swollen mitochondria. At 70 days swollen mitochondria were present in all cell types of the bladder wall. Intercellular spaces between smooth muscle cells gradually increased with increasing time afer partial obstruction, becoming filled with strands of basal lamina-like material in addition to increased deposits of collagen and elastic fibers. Evidence of axonal degeneration was first observed at 7 days and became more extensive at later times after operation. Throughout the period of obstruction the presence of mitotic figures, nerve growth cones or regenerating smooth muscles cells was not observed.

Conclusions: The present results demonstrated that significant structural changes accompany the progression of decompensation, and parallel the severity of both the biochemical and contractile dysfunctions. These results are interpreted as evidence of ischaemic changes in the bladder wall induced by partial outflow obstruction.