connective tissue bindings to a different muscle bundle at one end, in two of which there was an accretion of elastin fibres. The remaining three fascicles merged into separate muscle bundles at each end. Cholinergic nerves were observed crossing the connective tissue endings of these fascicles, but were not seen crossing other connective tissue planes between bundles. Interstitial cells were present at the connective tissue endings and their processes were seen to cross to either side of the attachment.

Conclusions: The semi-automated technique of 3-D reconstruction described successfully facilitates comparison of muscle bundle arrangement and stereological parameters at a microscopic level. Detrusor muscle bundles are structurally independent, but attached through connective tissue elements which may contain elastin accretions. Subpopulations of fascicles are present which appear potentially to subserve a communication function, since nerve fibres and interstitial cells are present, and presumably the fascicle is able to exert physical force. Putatively, such an arrangement is consistent with the facility to generate asynchronous, non-propagated "micro-motions" [3] along with the general synchronous contractions of voiding. The findings are of interest in the study of detrusor instability: the myogenic hypothesis suggests abnormal dissemination of excitation as a result of changes in smooth muscle properties. Presence of nerves and interstitial cells at the interface between bundles suggests means by which activity might spread.

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REGIONAL DIFFERENCES IN BLADDER BLOOD FLOW AND MICROCIRCULATION RESISTANCE AT REST AND DURING FILLING

Aims of Study: Most studies have suggested that increased intravesical pressure during distention produces compressive forces on the bladder microcirculation that lead to decreased bladder blood flow. In the rabbit model, we found that the normally compliant bladder acts as a low pressure reservoir allowing little increase in intravesical pressure during filling. Intravesical pressure during filling was 20 mm Hg or less and thus always less than the mean arteriolar pressure in bladder wall (25-30 mm Hg). This would suggest the role of factors other than intravesical pressure in regulating the hemodynamics of bladder wall circulation. In this study we examined the hypothesis that changes in bladder microcirculation resistance independent of intravesical pressure play a role in regulating bladder blood flow during filling.

Methods: In surgically exposed bladders of anesthetized male New Zealand white rabbits (3.5-4 kg, n=16), bladder blood flow was measured with a laser Doppler flowmeter. The laser Doppler probes were placed directly into the bladder wall at the dome and at the base. An 18 gauge angiocatheter was inserted through the bladder wall for measurement of intravesical pressure. A 3 F catheter placed through the urethra was used to fill with normal saline. Simultaneous measurements of arterial pressure, bladder wall blood flow at the bladder dome and base and intravesical pressure were obtained at rest and at intravesical volumes of 25 and 50 ml. Changes in bladder microcirculation resistance (MCR, mm Hg/ml/min) were calculated from the ratio of mean arterial blood pressure (BP, mm Hg) to blood flow (Q, ml/min/100 g tissue) (MCR = BP/Q).

Results: With the bladder empty, bladder wall blood flow was significantly greater at the base (11.5 ± 0.4) than at the dome (8.6 ± 0.2). Filling caused an initial increase in blood flow at the base (12.5 ± 0.3), which

was sustained up to 25 ml intravesical volume. In contrast, at the dome the initial increase in blood flow was observed only up to 10 ml intravesical volume. At intravesical volumes of more than 10 ml, blood flow at the dome began to decrease significantly while intravesical pressure remained constant. Microcirculation resistance to flow (mm Hg/ml/min) was significantly greater at the dome (10.1 ± 0.4) than at the base (7.6 ± 0.3) in the empty bladder. After filling the bladder to 25 ml, MCR decreased significantly at the base (6.8 ± 0.2) but increased at the dome (12.0 ± 0.6) when compared with empty bladder. Filling the bladder to 50 ml resulted in slightly increased MCR at the base (8.9 ± 0.4) but markedly increased MCR at the dome (19.4 ± 1.0).

Discussion: According to Poiseuille's law, resistance to flow (R) is directly proportional to the fluid viscosity (η) and length of the vessel (l) and indirectly proportional to the fourth power of the vessel radius (r) {$R \propto \eta l/r^4$}. Assuming η to be constant, the remaining variables are vessel length and radius. If we treat the urinary bladder as a sphere with changing volume, changes in vessel length are proportional to the changes in bladder radius (a), which may be calculated for each bladder volume (V) from the equation: $a = .62\ V^{1/3}$. This equation predicts a 26% increase in vessel length if volume increases from 25 ml to 50 ml. Interestingly, we found that MCR at the bladder base increased by 30% as volume increased from 25 ml to 50 ml. This suggests that increased MCR at the bladder base may be explained almost entirely by an increase in the length of vessels supplying the area. In contrast, the increase in MCR at the bladder dome was equal to 61% suggesting that there were additional factors influencing blood flow. Since Poiseuille's Law states that MCR is inversely proportional to the fourth power of the vessel radius, even a small decrease in vessel radius would significantly increase resistance to flow. This suggests that the additional increase in MCR seen at the dome may be due to the decreased radius of the vessels secondary to excessive stretching and/or compression due to increasing bladder wall tension. According to the Laplace equation, bladder wall tension is directly proportional to the intravesical pressure and the bladder radius and inversely proportional to the bladder wall thickness. Since the dome is noticeably thinner than the base, increased tension at the dome may contribute to higher MCR.

Conclusion: Generally speaking, the pattern of blood flow during the filling phase in normally compliant bladder is biphasic. The first phase is characterized by a slight initial increase in blood flow and fall in MCR. Since the arterioles have a serpentine course at rest, this may be due to a slight straightening of these vessels during the initial phase of filling. Since fluid traveling through a serpentine blood vessel loses much more kinetic energy than through a straight vessel, straightening of the vessels might be expected to lead to decreased MCR and increased blood flow. Neurohumoral mechanisms are also possible explanations for this phenomenon. In the second phase of filling, decreased blood flow and increased MCR is caused by elongation and narrowing of the microvasculature. Since the bladder is supplied by vascular pedicles entering near the bladder base, the differences observed between bladder base and dome might be explained by the greater length of vessels traveling to the dome. The net effect is to make the dome much more susceptible to ischemia during distention than the base.