EDITORIAL

Urodynamics of Post-Prostatectomy Incontinence

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The opportunity to evaluate and treat a group of 150 cases of post-prostatectomy incontinence in one centre is unique. The aim of this editorial is to show how the application of urodynamic techniques that have evolved from this experience, has added to the understanding of the pathophysiology of this condition.

There are two forces operating in lower urinary tract function: those of expulsion and resistance. The bladder contraction and the intra-abdominal pressures are expulsive forces. The forces of resistance in the male can be divided into a) the proximal urethral sphincter (P. U.S.) composed of the bladder neck and the prostatic urethra above the verumontanum; and b) the distal urethral sphincter (D. U. S.) consisting of the fibro-elastic and smooth muscle tissue of the urethra below the verumontanum and the external sphincter musculature. Actual continence is achieved in the male by the P. U.S. mechanism; the D. U. S. mechanism acts only as a secondary or complementary force. However, when the proximal mechanism is surgically excised, the distal mechanism must assume the continence effort. The effectiveness of this short segment of urethra (2-3 cm long), located immediately above and in the area of the external sphincter, will depend on the ability to maintain a pressure (at any time: rest or stress, supine or erect) above the intravesical pressures.

Three types of tissue are responsible for this delicate task after prostatectomy: the fibroelastic, the smooth muscle below the verumontanum, and the skeletal musculature. The fibroelastic tissue provides this short segment of distal urethra with the ability to effectively coapt the walls of the urethra and simultaneously to easily distend and open during the act of voiding. Although this tissue is

important, no simple objective or reliable method of evaluating its function is available. The smooth muscle with its two layers of internal longitudinal fibers and external circular fibres is probably the most important factor in maintaining continence after prostatectomy. The external sphincter has two functions in providing resistance to the loss of urine: a) active (voluntary) contraction; and b) basic sphincter tonus (involuntary). Active contraction of the sphincter is clinically evaluated by the ability of the patient to stop and start the stream of urine voluntarily, and by having the patient contract the sphincter during cystoscopic inspection. A better objective assessment of voluntary sphincter function can be achieved by urethral pressure profiles. Most patients with post-prostatectomy incontinence will show ability to voluntarily contract the external sphincter and to show momentary increases in the closing pressure of the urethra. The other property of the external sphincter in providing continence is the inherent tonus of the muscle, which is attributable to the action of a stretch reflex (gamma loop) integrated in the S_2 - S_4 area of the spinal cord. Indirect estimation of this function is possible by studying the effect of general anaesthesia with curare-like agents, spinal anaesthesia, or pudendal block.

Blockade of the external sphincter function by spinal anaesthesia in patients following prostatectomy revealed that 30% to 50% of the closing pressure of the D. U.S. mechanism is related to external sphincter function. The remaining closing pressure of the urethra after anaesthesia depends on the residual smooth muscle activity in the D. U.S. All patients with post-prostatectomy incontinence have some damage to the smooth muscle but only a small

number of patients will have damage to the external sphincter or to both components (smooth and striated muscle).

Post-prostatectomy incontinence is a distressing and humiliating condition produced by varying degrees of damage to the D. U. S. mechanism. The findings indicate a shortening in the functional length of the urethra and a lowering (to differing extents) of the closing pressure of the urethra in the D. U. S. area. Not only is the passive closing pressure of the urethra impaired, but the ability of the distal sphincteric mechanism to compensate in the standing position or under stress is also affected, resulting in urinary incontinence.

We have observed three types of patients in post-prostatectomy incontinence: a) the most common group maintains some degree of closing pressure in the D. U.S. area (but lower than normal) and anaesthesia produces a flattening of the pressure curve (intraurethral pressures equal to intravesical pressures). This fact suggests that the main injury was done to the smooth muscle fibres in the area of continence. b) The rarest type of patient was seen only in a small number of cases following radical

perineal prostatectomy. Closing pressure of the urethra was lower than normal, but under anaesthesia, minimal change was observed, indicating that in these cases the main damage involved the skeletal musculature and not the smooth muscle. c) The third type of patient, with the worst clinical symptoms, represents the second most common group - those in whom the damage to the D. U. S. area has been extensive. The closing pressure of the urethra is similar in these cases to the intravesical pressures and anaesthesia does not affect the urethral pressure studies, indicating that both smooth and skeletal muscle components have been destroyed. Active continence is absent.

Proper understanding and a physiological approach to this disease are crucial before embarking on the treatment of patients with post-prostatectomy incontinence.

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