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Micturition

Micturition involves the simultaneous coordinated contraction of the bladder detrusor muscle, which is controlled by parasympathetic (cholinergic) nerves, and the relaxation of the bladder neck and sphincter, which are controlled by sympathetic (α -adrenergic) nerves. Micturition involves bladder filling and bladder emptying. Bladder filling requires (i) accommodation of increasing volumes at low intravesical pressure (compliance) and appropriate sensation, (ii) the bladder outlet to be closed at rest and during increases in intra-abdominal pressure, and (iii) the absence of involuntary bladder contractions. Bladder emptying requires (i) coordinated contraction of the bladder of adequate magnitude and duration, (ii) lowering of resistance at sphincters, and (iii) the absence of obstruction (e.g., enlargement of the prostate gland in the aging male may result in bladder outlet obstruction, precluding efficient micturition). Bladder dysfunction can then be clinically identified as a problem of filling or emptying or a combination of both, and the site of dysfunction may be the bladder, the urethra, or both.

The normal function of the bladder is to store urine until it has reached capacity and until it is socially acceptable to evacuate urine. Urine storage is accomplished at low pressures, measured as compliance. Compliance is calculated as the change in volume over the change in pressure. Bladder compliance is a result of the viscoelastic properties of the bladder. The bladder wall contains elastin, which allows it to stretch without a subsequent increase in pressure. Typical adult bladder capacity is approximately 350–450 ml. Compliance is also facilitated by sympathetic discharge primarily mediated through the β -adrenergic receptors within the bladder wall. This sympathetic

tone operates directly at the level of the bladder musculature to facilitate storage. There is also sympathetic discharge at the level of the autonomic ganglia, which has an inhibitory effect on the parasympathetic postganglionic neurons, thus preventing detrusor contraction and facilitating urine storage. Loss of compliance may lead to renal insufficiency.

Continence is maintained through the action of the urinary sphincters. The internal sphincter, or the bladder neck, is richly innervated with α -adrenergic receptors. During bladder filling this structure remains closed through constant sympathetic discharge via the hypogastric plexus. The external sphincter, composed of striated muscle, also maintains a resting tone to maintain continence. It is believed that the fibers of the external sphincter are primarily of the slow twitch variety and thus can maintain tension for long periods of time. With rapid increases in intra-abdominal pressure, fast twitch fibers are recruited to contract and further increase the urethral resistance to avoid urinary leakage.

As the bladder fills, its visceral afferents travel through the peripheral nerves, ascending through the spinal cord to the pontine micturition center. It is at this level that a detrusor contraction is initiated. However, there are inhibitory signals from suprapontine centers (e.g., prefrontal cortex and basal ganglia) that prevent the generation of a detrusor contraction until the bladder is full. At normal bladder capacity (350–450 ml), sensations of fullness are transmitted through detrusor afferents, nerves that provide reflex excitation through the central nervous system to the motor innervation to the detrusor. The cortex releases its inhibition of the pontine micturition center.