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CONTROL OF THE URINARY BLADDER

The urinary bladder and its sphincters are supplied by parasympathetic, sympathetic, somatic motor, and visceral afferent fibers (Fig. 18–10).

Several groups of visceral afferent fibers supply the urinary bladder. Pain and temperature impulses from the mucosa of the fundus travel with the sympathetic nerves and reach the spinal cord via the dorsal roots of T12 and L1. From the mucosa at the neck of the bladder, pain and temperature impulses travel with the sacral parasympathetic nerves to S2,S3,S4. The spinothalamic tract then transmits impulses of both groups of pain and temperature fibers to higher centers.

Fullness of the bladder is detected by mechanoreceptors in the bladder wall that send impulses to the spinal cord via the sacral parasympathetic route. The spinothalamic tracts carry "fullness" impulses to higher centers in the thalamus and cerebral cortex. The sensation that micturition is imminent arises from mechanoreceptors in the trigone of the bladder; these visceral afferent impulses travel with the sacral parasympathetic nerves to S2,S3,S4 and ascend in the dorsal column–medial lemniscus system.

Parasympathetic visceromotor neurons located in S2,S3,S4 give rise to preganglionic fibers that travel in the pelvic nerve to the hy-

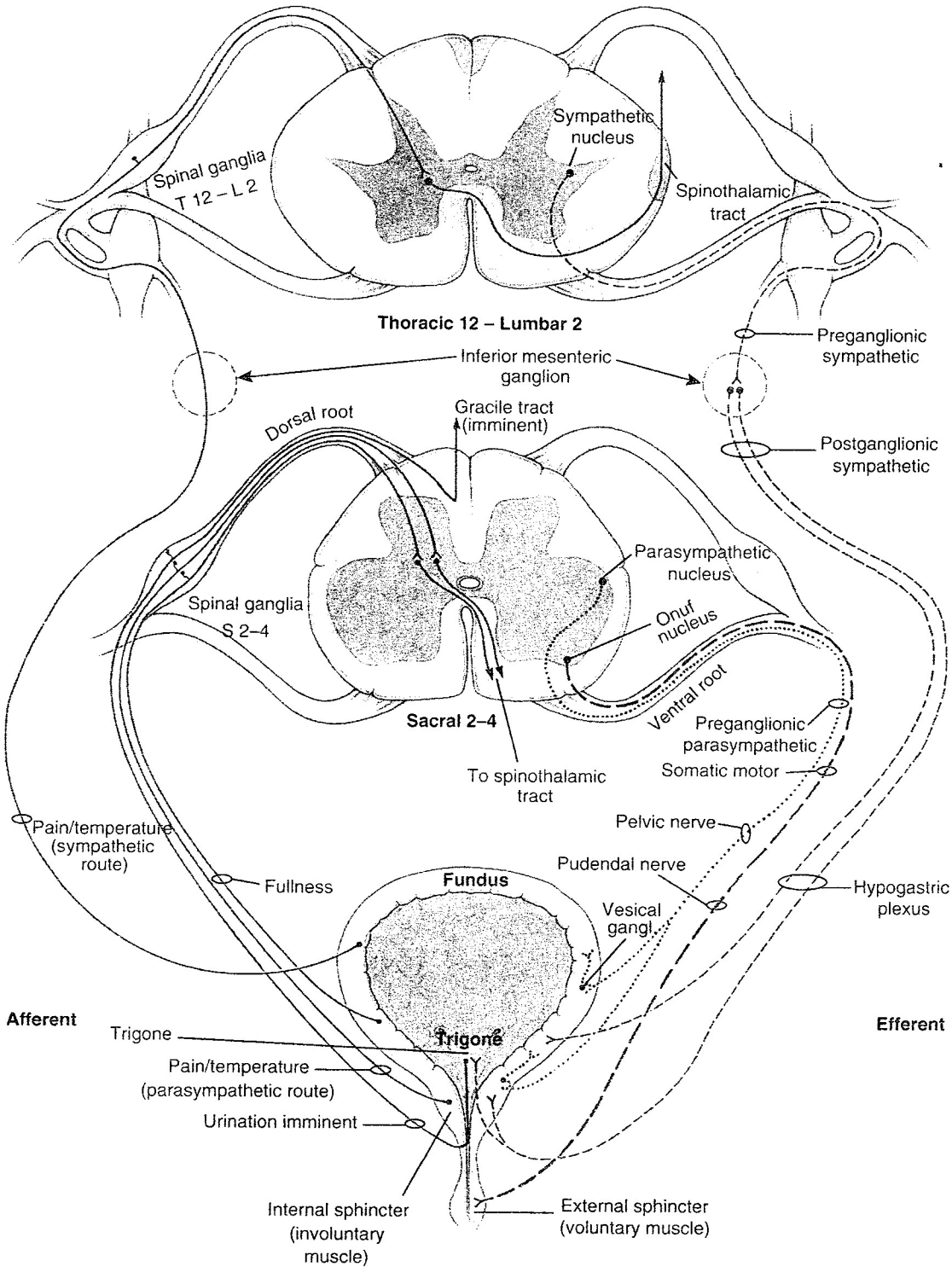

FIGURE 18–10. Schematic diagram showing the innervation of the urinary bladder.

pogastric and then to the vesical plexuses. Vesical ganglion cells give postganglionic parasympathetic fibers that supply the **detrusor muscle** that, on contraction, empties the bladder.

Sympathetic visceromotor neurons in spinal cord segments T11–L2 give preganglionic fibers that travel in the lumbar splanchnic nerves to the inferior mesenteric ganglion. Postganglionic sympathetic fibers from the inferior mesenteric ganglion reach the bladder via the hypogastric and vesicle plexuses and supply the internal urethral sphincter. During bladder filling, the sympathetic fibers relax the detrusor muscle directly and also indirectly by inhibiting the parasympathetic cells in the vesical ganglia. The sympathetic fibers elicit contraction of the internal urethral sphincter.

Lower motor neurons that make up the Onuf nucleus in S2,S3,S4 send axons via the internal pudendal nerve and its perineal branch to the skeletal muscle that forms the external urethral sphincter.

Micturition centers are located in the brainstem and cerebral cortex. A cortical center for voluntary control of the initiation and cessation of micturition is located in the superior frontal gyrus on the medial surface of the hemisphere. Two micturition centers are located in the pons. One pontine micturition center sends excitatory impulses to the sacral parasympathetic neurons that elicit contraction of the detrusor muscle. A second pontine micturition center sends excitatory impulses to the lower motoneurons of the Onuf nucleus that supply the external urethral sphincter. During micturition the pontine parasympathetic excitatory center inhibits the other pontine center. Thus, the external urethral sphincter relaxes when the detrusor muscle contracts, and emptying of the bladder occurs.

Reflex bladder control is initiated by visceral afferent impulses from volume and tension receptors in the bladder wall. At low levels of bladder distension, these visceral afferent fibers stimulate the lower motor neurons of the Onuf nucleus, resulting in contraction of the external sphincter. At high levels of bladder distension, visceral afferent impulses stimulate pontine micturition center neurons that inhibit sympathetic and Onuf somatic neurons, resulting in relaxation of the internal and external sphincters, respectively, and elicit parasympathetic activity resulting in contraction of the detrusor and emptying of the bladder. Thus, micturition is controlled by spinopontospinal reflex mechanisms.

Interruption of this reflex results in the so-called **neurogenic bladder.** Two types of neurogenic bladders exist: reflex and nonreflex (Fig. 18–11). The **reflex neurogenic bladder** is of upper motor neuron type; the nonreflex bladder is of lower motor neuron type. The reflex neurogenic bladder may be uninhibited or automatic. The **uninhibited reflex bladder,** which is incontinent but empties fully, results from bilateral lesions of the micturition centers in the frontal lobe. Emptying of the bladder is normal because reflex control of the pontine micturition centers are intact. The **automatic reflex bladder,** which is incontinent and does not empty fully, results from bilateral spinal cord lesions above sacral levels. Emptying of the bladder is incomplete because the spinal reflex pathways that trigger the pontine micturition centers are interrupted. The **nonreflex neurogenic bladder,** which is characterized by severe urinary retention and incontinence, results from bilateral lesions of the sacral spinal cord or the spinal nerve roots in the caudal equina (Fig. 18–11).

CONTROL OF THE SEX ORGANS

The sex organs are innervated by parasympathetic, sympathetic, and visceral afferent fibers. Visceral afferent fibers from the female and male sex organs pass to the spinal cord via sympathetic and sacral parasympathetic routes and have their cell bodies located in the dorsal root ganglia of T10–L2 and S2-S4, respectively. An exception to the rule that visceral pain fibers follow the sympathetic nerves occurs in the case of pain from the cervix of the uterus and the prostate. In both cases, pain travels with the parasympathetic nerves and enters the spinal cord at S2-S4.

The preganglionic parasympathetic fibers arise from S2-S4, enter the pelvic cavity via the pelvic nerve, and synapse on ganglia in the hypogastric and the uterovaginal or prostatic plexuses. Postganglionic parasympathetic fibers

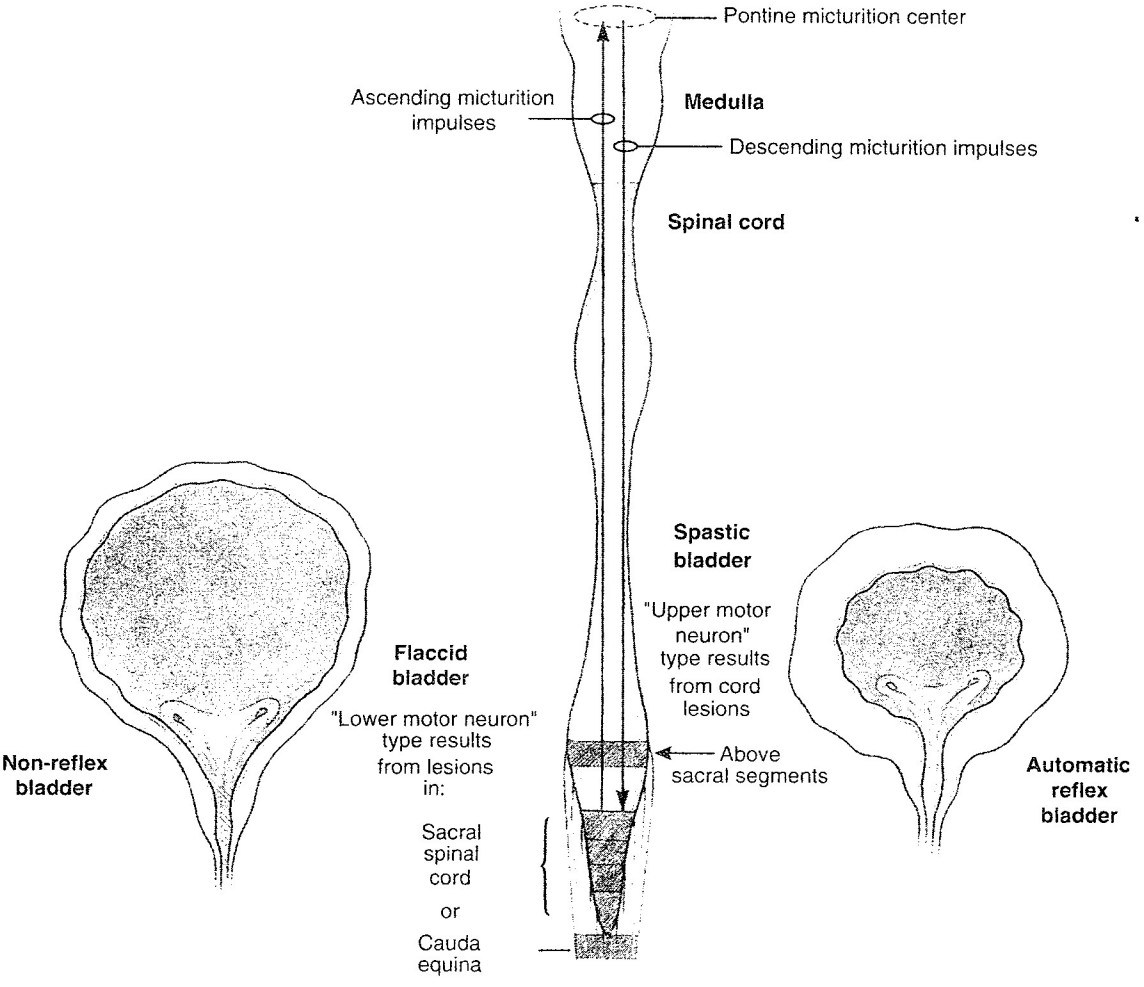

FIGURE 18–11. Locations of lesions resulting in flaccid and spastic neurogenic bladders.

Two commonly encountered abnormalities associated with the sympathetic system are Horner syndrome and **acute sympathetic shock syndrome.** Horner syndrome is characterized by miosis, ptosis, and anhidrosis (absence of sweating) and may occur as the result of unilateral peripheral or central lesions. The peripheral lesions involve (*a*) preganglionic fibers chiefly in spinal nerve T1 or in the cervical sympathetic trunk or (*b*) postganglionic neurons and fibers in the superior cervical ganglion. Central lesions producing Horner syndrome occur chiefly as the result of (*a*) interrupting the pupillodilator path in the dorsolateral part of the medullary reticular formation or in the cervical spinal cord or (*b*) destruction of the ciliospinal center in the sympathetic nucleus at C8 and T1.

Acute sympathetic shock syndrome is characterized by bradycardia, hypotension, bilateral Horner syndrome, and difficulties in adjusting to a warm environment because sweating and cutaneous vasodilation cannot be elicited. This syndrome occurs in acute bilateral cervical spinal cord injuries due to the interruption of the descending impulses to the sympathetic nuclei. The signs usually subside after several days when reflex regulation of sympathetic activities returns.

from the uterovaginal ganglia in the female innervate the vaginal glands and erectile tissue of the clitoris. In the male, the postganglionic parasympathetic fibers arise from the cavernous and prostatic ganglia and supply the cavernous or erectile tissue of the penis.

Sympathetic preganglionic fibers arise from T10–L2 and synapse chiefly in the inferior mesenteric ganglion. Postganglionic sympathetic fibers in the female supply the blood ves-

sels and smooth muscle of the uterus and vagina, whereas in the male sympathetic postganglionic fibers supply the ductus deferens, prostate gland, and seminal vesicle.

Parasympathetic activity in women produces secretion of vaginal glands and clitoral engorgement; in men parasympathetic impulses are necessary for penile erection. Sympathetic activity in women produces rhythmical contractions of the vagina; in men the sympathetic nerves are necessary for ejaculation.