

Loss of bladder function results from disruption of neural pathways between the bladder and the reflex voiding center at the S2 to S4 level (i.e., an LMN lesion) or between the reflex voiding center and higher brain centers for communication and coordinated sphincter control (i.e., a UMN lesion). Bowel elimination is a coordinated function involving the enteric nervous system, the autonomic nervous system, and the CNS. Persons with SCI above S2 to S4 develop spastic functioning of the defecation reflex and loss of voluntary control of the external anal sphincter. In LMN lesions or flaccid bladder dysfunction, lack of awareness of bladder filling and lack of bladder tone render the person unable to void voluntarily or involuntarily. Even though the enteric nervous system innervation of the bowel remains intact, without the defecation reflex, peristaltic movements are ineffective in evacuating stool. People with UMN lesions or spastic bladders lack awareness of bladder filling (i.e., storage) and voluntary control of voiding (i.e., evacuation). Damage to the cord at the S2 to S4 level causes flaccid functioning of the defecation reflex and loss of anal sphincter tone.