Anatomy and Neurophysiology of Micturition

Article Details	Relevant Anatomic Structures and Background Information	Neuronal Innervation	Process of Micturition	Process of Incontinence and Complications
Title: Continence and Micturition: An Anatomical Basis[1] **Year:** 2014 **Authors:** Ardarsh P. Shah, Amit Mecvha, Daniel Wilby, Anton Alatsatianos, John C. Hardman, Steven Jacques, Joanne C. Wilton **Article Type:** Review	**Endopelvic Fascia[1]:** - Endopelvic fascia is continuous with transversalis fascia of abdomen and parietal pelvic fascia that surrounds obturator internus, piriformis, levator anni, and coccygeus muscles - Serves to suspend urinary bladder neck and urethra on anterior vaginal wall in females and stabilizes organs above levator ani **Pelvic Floor Muscles[1]:** - Levator ani and coccygeus form pelvic diaphragm - Levator ani consists of iliococcygeus, pubococcygeus, and puborectalis - Urethra and urethra/vagina pass through opening in levator ani called urogenital hiatus in	**Autonomic[1]:** - Sympathetic innervation of bladder and IUS originates in T10 – L2 - Preganglionic neurons → superior hypogastric plexus via aortic plexus (T12) or inferior mesenteric plexus (L1-L2) → hypogastric nerve into inferior hypogastric plexi - Parasympathetic fibers course from rami of S2-S4 and unite with sympathetic nerves to create inferior hypogastric plexi - Inferior hypogastric plexi gives rise to other plexuses that innervate the bladder, prostate, uterus, vagina, and rectum - Afferent fibers are within hypogastric and pelvic nerves and provide input into reflex circuits that control bladder filling	**Storage Phase** – bladder is a reservoir to collect urine[1] **Voiding phase** – occurs once maximum filling has been reached[1] Phases controlled by reflexes within autonomic and somatic nervous system[1]: - During bladder filling, mechanoreceptors in bladder wall are activated via stretch - Pelvic nerves relay message initiating parasympathetic innervation of the detrusor to be inhibited - Supraspinal input allows micturition to remain under voluntary control b/c decision to void is based on emotional, social, and visceral sensation - To avoid leakage, there is simultaneous stimulation of	**Incontinence** – involuntary loss of urine that is a social or hygienic problem[1] **Stress incontinence** – intra-vesicle pressures exceed maximal urethral pressure w/o detrusor activity[1] - Pubococcygeus can be stretched during vaginal delivery - Puborectalis can also experience stretch related muscular deficits from vaginal birth - Pudendal nerve damage can lead to Levator ani atrophy **Detrusor overactivity**[1] – a bladder that contracts, spontaneously or with provocation, during bladder filling while attempting to inhibit micturition - An intact spinobulbospinal reflex w/o regulation from higher centers can lead

	men and women, respectively - Levator ani activity closes the urogenital hiatus which compresses urethra and distal vagina - Mid urethra, distal vagina, and rectum are closed with max contraction of puborectalis and pubococcygei muscles **Perineal Body**[1]**:** - Connective structure where levator ani, superficial transverse perineal muscles, and perineal membrane attach - Functions: 1. Anchors the anorectum 2. Supports the terminal vagina 3. Demarcates the urogenital triangle from anal triangle 4. Anatomical landmark for episiotomy	and emptying, and are the source of non-painful bladder fullness **Somatic**[1]**:** - Cholinergic motor innervation to striated muscle of EUS is from pudendal nerve (S2-S4) - Pudendal nerve is formed at upper border of sacrotuberous ligament, leaves pelvis via greater sciatic foramen to enter gluteal region by crossing sacrospinous ligament and ischial spine - Once in the gluteal region, pudendal nerve is susceptible to compression especially during childbirth in females - Also supplies sensory and motor input to transverse perinei, bulbospongiosus, ischiocavernosus, anterior part of external anal sphincter and levator ani - External anal sphincter is mainly innervated by	pudendal nerve at EUS and sympathetic activity to bladder neck and IUS (hypogastric nerve) - Maintaining continence during filling = guarding reflex (closes IUS, EUS, and prevents bladder contraction) - Sensory information is relayed to periaqueductal gray matter which then relays info to higher centers such as thalamus, insula, anterior cingulate gyrus, and prefrontal cortices - Periaqueductal gray matter is interface between afferent and efferent bladder control - When bladder is full, afferent activity in the periaqueductal gray matter sends signals to the pontine micturition center which leads to spinobulbospinal reflex (voiding)	to involuntary leakage during filling - Damage to suprapontine, etiology affecting frontal brain, or degeneration of dopaminergic neurons leads to a loss of inhibitory control of pontine micturition center Spinal cord injuries rostral to lumbosacral level leads to a blocking of the voluntary and supraspinal control of micturition (can lead to urinary retention in areflexic bladder) [1] This can lead to automatic micturition and neurogenic detrusor overactivity mediated by spinal cord reflex pathways → detrusor sphincter dyssynergia and low bladder compliance[1] **Low bladder compliance**[1] – large increases in pressure with small volumes of urine from fibrosis and decreased bladder wall elasticity Autonomic dysreflexia is often seen in patients with lesions above T6 (noxious stimuli

	5. Helps maintain urinary continence **Bladder**[1]: - When empty, rests within pelvis - When filled, rises anterosuperiorly into abdominal cavity - Sup. Surface covered by peritoneum that extends into retrovesical pouch in males - Sup. Surface covered by peritoneum and then reflects posteriorly to uterus to form vesicouterine pouch and rectouterine pouch of Douglass - Inf/lat surface of bladder relates anteriorly to pubis and puboprostatic ligaments in males and pubis/pubovesical ligaments in females - Urothelial cells detect both physical and chemical stimuli	inferior rectal nerve for motor supply	- Without the input from higher centers during bladder filling, spinobulbospinal reflex would act whenever filling reached a critical level leading to incontinence such as with suprapontine cerebral lesions or thoracolumbar cord lesions - Prefrontal cortex has an executive role in determining when micturition should occur	(bladder distension) leads to an unopposed sympathetic response with cardiovascular symptoms) [1] Conus/cauda equine lesions lead to more of a LMN presentation with areflexic and acontractile bladder with sphincter weakness → stress and overflow UI[1]

	- Stretching of bladder from filling causes release of chemical mediators which activates afferent nerves and myofibroblasts relaying the sensation of fullness **Internal Urethral Sphincter (IUS)** [1]**:** - Surrounds proximal urethra and is a continuation of the detrusor smooth muscle - Smooth muscle fibers arranged in horse-shoe shape - Striated muscle fibers in circular configuration and surround smooth muscle layer in mid-portion of urethra - Innervated by sympathetic autonomic nervous system (involuntary control) **External Urethral Sphincter (EUS)** [1]**:** - Derived from skeletal muscle of levator ani			

	- In males it is at level of prostate, with fibers in a horse-shoe shape w/o fixture to levator ani muscle (implies voluntary function via pudendal nerve) - In females it is at level of inferior bladder and includes sphincter urethrae muscle, compressor urethrae muscle, and urethrovaginal sphincter - In females, contraction of levator ani compresses vagina and simultaneous contraction of EUS and levator ani bends mid-urethra. Inferior portion of EUS contracts and applies force on urethra.			
Title: Central Control of Micturition in Women: Brain-Bladder Pathways in Continence and	Urinary bladder and urethral sphincters are controlled by complex network of neurons that allow for urine storage and micturition when socially appropriate.[2]	Detrusor and internal sphincter are innervated by sympathetic and parasympathetic nerves of autonomic nervous system.[2] Sympathetic nerves originate from T10 – L2 segments. Also	Storing of Urine[2]: - Primarily under sympathetic control with inhibited parasympathetic control	Not applicable.

Urinary Incontinence[2] **Year:** 2017 **Authors:** Nisha G. Arya, Steven J. Weissbart **Article Type:** Narrative Review	Bladder function controlled by autonomic and somatic nervous systems.[2] Detrusor and internal sphincter are under involuntary control.[2] External sphincter is under voluntary control.[2] Damage to sympathetic nervous system (SNS) or parasympathetic nervous system (PNS) can lead to a range of voiding dysfunction symptoms such as urgency incontinence, retention of urine, and/or stress incontinence.[2] Parasympathetic nerve ganglia involved in micturition are housed in intermediate gray matter of sacral segments.[2] Sympathetic nerve ganglia are housed in intermediate gray matter of lumbar segments.[2] Somatic nerves originate in Onuf's nucleus.[2]	called "nerves of filling" b/c they cause relaxation of detrusor muscle and contraction of urethral sphincter.[2] Short Preganglionic fibers synapse in sympathetic chain.[2] Postganglionic fibers relay in the superior hypogastric plexus and travel in hypogastric nerves to detrusor muscle and smooth muscle internal sphincter via adrenergic receptors.[2] Parasympathetic nerves that supply detrusor muscle originate from S2 – S4 of spinal cord.[2] Long preganglionic fibers travel through the pelvic bladder and synapse with short postganglionic fibers in pelvic plexus.[2] These postganglionic fibers go to detrusor via muscarinic receptors.[2] External sphincter is innervated by pudendal nerve (S2-S4 i.e., Onuf's nucleus).[2] Slow twitch fibers contribute to continence at rest.[2] Fast twitch fibers close urethral lumen and is an additional mechanism to maintain continence.[2]	Voiding[2]: - Primarily due to parasympathetic excitation which leads to detrusor contraction and internal/external sphincter relaxation from sympathetic and pudendal n. inhibition **Micturition Control[2]:** Parasympathetic Nervous System (PNS): - Responsible for micturition - Pelvic nerve from sacral spine, releases acetylcholine at muscarinic receptor to contract bladder Sympathetic Nervous System (SNS): - Responsible for storage - Hypogastric nerve from thoracolumbar spine releases norepinephrine at beta adrenergic receptors for detrusor relaxation - Also releases norepinephrine at alpha adrenergic	

	Afferent info. is carried by two fibers: myelinated which respond to bladder distension/contraction & non-myelinated which respond to noxious stimuli.[2] **Pontine Micturition Center (PMC)** [2]**:** direct connection to the sacral spinal cord, relaxation of striated urethral sphincter, initiation of detrusor contraction **Anterior Cingulate Cortex (ACC)** [2]**:** sensation of bladder fullness and micturition control, PFM contraction, emotional and motivational aspects of micturition, decision making executive function **Insula**[2]: response with increasing bladder filling, mapping visceral nociceptive sensations, integration of limbic and autonomic responses, co-activation of insula and ACC **Thalamus**[2]: gateway of all sensory pathways, relay station to cerebral cortex, activated during bladder filling		receptor to contract internal sphincter Somatic Nervous System: - Pudendal nerve releases acetylcholine at nicotinic receptor for voluntary pelvic floor muscle and sphincter contraction **During Empty Bladder**[2]**:** Parasympathetic Nervous System: - Afferent pelvic n. originate in bladder sends slow impulses about volume - Pelvic n. acts at muscarinic receptor with acetylcholine reduction which inhibits the contraction of the bladder Sympathetic nervous system: - Hypogastric n. release norepinephrine at beta adrenergic receptor for detrusor relaxation - Hypogastric n. releases NE at alpha adrenergic receptor	

	Hypothalamus[2]**:** inhibitory function, safe signal for voiding, sends afferent signals to the PMC during storage or voiding **Amygdala**[2]**:** emotional processes (fear), involved in response to normal bladder filling, suppression of urgency in overactive bladder during filling **Basal ganglia**[2]**:** inhibition of micturition (voiding reflex), activation occurs during micturition **Prefrontal cortex**[2]**:** receive sensory signals and interprets them into sensation from bladder **Pudendal nerve**[2]**:** S2 – S4 from sacral plexus. Innervate deep pelvic floor (levator ani)		to contract internal sphincter Somatic Nervous System: - Pudendal n. originates from sacral spine and releases Ach at nicotinic receptor = voluntary PFM and sphincter contraction **Full Bladder**[2] PNS: - Sensory Pelvic n. originates in bladder and sends rapid impulses about higher urine volumes - Efferent pelvic n. act at muscarinic receptor with Ach increase → facilitate contraction of bladder SNS: - Hypogastric n reduces release of NE at beta adrenergic receptor → no detrusor relaxation - Hypogastric n. reduces release of NE at Alpha adrenergic receptor → no contraction of internal sphincter Somatic:	

			- Pudendal n. reduces release of Ach at nicotinic receptor → facilitates voluntary PFM and sphincter relaxation **Voiding Reflex**[2] PNS: - Sensory pelvic n. continues to send rapid signals to interneuron in sacral cord - Efferent pelvic n. increase Ach release at muscarinic receptor → facilitate reflex loop for continued contraction of bladder SNS: - Continues to be inhibited Somatic: Continues to be inhibited	
Title: Neurogenic bladder in spinal cord injury patients[3] **Year**: 2015	Most common urologic complications following SCI are urinary tract infection (UTI), upper and lower urinary tract deterioration, and bladder or renal stones.[3] Important first step is bladder management, but	Parasympathetic efferents from sacral cord (S2-S4) via pelvic nerves provide excitatory input to bladder.[3] Sympathetic efferents originate from intermediolateral gray column Tll-L2 and supply bladder and urethra. They	Requires coordination of neuronal circuit between the brain and spinal cord and bladder and urethra.[3] Bladder contraction occurs via muscarinic receptor stimulation.[3]	**Spinal Shock**[3]**:** Spinal shock after SCI can last up to 3 months. In this period. Autonomic activation of bladder via parasympathetic nerves is inactive.

Author: Waleed Al Taweel, Raouf Seyam **Type:** Narrative Review	this is influenced by sex, lifestyle, hand dexterity, and access to health care.[3] Sacral micturition center (S2-S4), Pontine micturition center, & Cerebral cortex facilitate and inhibit voiding.[3]	inhibit input to bladder via hypogastric nerve.[3]	External sphincter inhibited by pudendal nerve.[3] Sympathetic stimulation causes bladder relaxation via beta receptor stimulation and contraction of sphincter via alpha receptor stimulation.[3]	Bladder is atonic and there is no conscious awareness of filling. Interruption below the pons due to SCI eliminates the micturition reflex causing urine retention. Managed with clean intermittent catheterization or an indwelling catheter. **Suprasacral lesion[3]:** After spinal shock following an SCI above S1, reflex bladder function will occur. Although conscious bladder filling may still be present but voluntary inhibition of micturition reflex is lost. Can lead to detrusor overactivity and dyssynergia which if left untreated can result in upper tract deterioration and renal failure. **Sacral lesion[3]:** SCI at sacral level leads to parasympathetic decentralization of bladder and denervation of sphincter. In complete lesions, conscious bladder filling is lost, and micturition reflex is gone. Will lead to acontractile bladders with nonrelaxing smooth and

				striated sphincters that retain some tone. **Complications of Neurogenic bladder[3]:** Urinary Tract Infection: - symptoms include fever, foul smelling urine, and/or hematuria - Hydrophilic coated catheter may reduce risk of UTI - Antibiotics can be used but have only found success in males versus females (concern over drug resistance) Urethritis and prostatitis - Should use clean intermittent catheterization and avoid indwelling catheters Epididymitis and epididymo-orchitis - Catheter related complications - Presents with swelling and skin redness - Treated with antibiotic therapy and occasionally abscess draining

				Bladder Stones - Primary formed due to infection, indwelling catheter use, and high residual urine levels - Can cause irritation and hematuria Renal Stone - Most common is struvite stones - Occurs 3.5% in neurogenic bladder Reflux and renal insufficiency: - Occur > 20% in neurogenic bladder - More common with suprasacral lesions - High detrusor pressure and reflex cause renal damage/failure Bladder Cancer - Risk is 20x higher in SCI patients - Risk factors: UTI, bladder stones, and indwelling catheters Autonomic Dysreflexia: - Triad of high BP, bradycardia, sweating (hot flashes and headaches)

				- With patients with SCI at or above T6 level - Risk factor for intracerebral hemorrhage - Afferent stimulation below SCI provokes this (distension of bladder and rectum is a common cause but can also be from a UTI, bladder stone, GI disease)
Title: Neurogenic Bladder, Neurogenic Bowel, and Sexual Dysfunction in People with Spinal Cord Injury[4] **Year:** 2002 **Authors:** Barbara T Benevento, Marca L Sipski **Type:** Narrative Review	Coordination center is in pontine mesencephalic reticular formation.[4]	- Parasympathetic efferent supply originates from S2 – S4 and travels to bladder via pelvic nerve (causes detrusor contraction)[4] - Sympathetic efferent nerve originates at T11 – T12 travels to bladder and urethra via hypogastric nerve (increases bladder storage)[4] - Beta adrenergic receptors in bladder cause relaxation of smooth muscles[4] - Alpha receptors at base of bladder and urethra cause contraction of smooth muscles[4]	- Intact pathway between pontine and sacral micturition center allows for coordinated voiding (relaxation of urethral sphincter and contraction of detrusor)[4] - Volitional micturition is controlled by medial frontal lobes and corpus callosum.[4]	- Suprapontine lesions allow for micturition to occur, but it is involuntary[4] - Central lesions can disrupt pontine and sacral micturition centers.[4] - Peripheral lesions can affect parasympathetic, sympathetic, or somatic supply.[4] - SCI patients often do not lose cortical control (pontine center) unless accompanying head injury occurs.[4] - Two categories to describe dysfunction[4]: failure to store & failure to empty - **Failure to store[4]:** detrusor hyperreflexia

		- Somatic efferents originate from sacral segments at S1-S4 and travel through pudendal nerve to innervate external urethral sphincter.[4]		(uninhibited bladder contraction), areflexic bladder outlet - **Failure to empty[4]:** areflexic bladder, sphincter unable to relax - Detrusor-sphincter dyssynergia is common in SCI patients **Goals of management[4]:** prevent urinary tract complications (hydronephrosis, renal calculi, bladder calculi, vesicoureteral reflux) **Bladder management considerations include** type of voiding dysfunction; level of injury; ability to self-catheterize, dress, and transfer[4] Detrusor activity usually occurs after spinal shock (resolved by 3 months) and is indicated by incontinence[4]
Title: Neurogenic Bladder in Spinal Cord Injury[5] **Year:** 2007	The bladder has two main functions: storage of urine and release of urine in a controlled manner at an appropriate time.[5]	**Peripheral innervation[5]:** 1. Autonomic – Parasympathetic and Sympathetic Parasympathetic excites smooth muscle of bladder and	Bladder outlet relaxes and bladder smooth muscle contracts during voiding.[5] Bladder outlet contracts (bladder neck) and detrusor relaxes during storage.[5]	A spinal cord injury disrupts the neural input to the lower urinary tract.[5] **Goals of bladder management are to[5]:** ensure social continence for community integration, improve low-

Authors: Gregory Samson, Diana D. Cardenas **Article Type:** Narrative Review	Lower urinary tract includes fundus, trigone, neck of bladder, pelvic diaphragm and urethra.[5] Bladder outlet includes bladder neck, urethral smooth and striated muscles[5] Bladder has four layers that is primarily smooth muscle.[5] Fundus, bladder neck muscles, urethral smooth muscles, striated sphincter muscles, and striated pelvic muscles contribute to bladder performance.[5]	inhibits urethral sphincter smooth muscles.[5] Fibers originate from sacral level S2-S4.[5] Travel via pelvic nerve to pelvic plexus. Bladder wall is mediated primarily via acetylcholine at muscarinic receptors.[5] Urethral inhibitory input is mediated by nitric oxide.[5] Sympathetic inhibits smooth bladder muscle and excites bladder neck.[5] Originate from T10 – L2 level and travel via hypogastric nerve and pelvic nerve.[5] Inhibition at bladder wall is mediated by noradrenaline to adrenergic receptors.[5] Excitatory input to bladder neck and urethra is mediated by noradrenaline to adrenergic receptors.[5] 2. Somatic Onuf nucleus – anteriolateral horn of spinal cord (S2-S3)[5] Excite striated muscle of urethral sphincter[5] Travel via pudendal nerve and excitatory input is mediated by acetylcholine at nicotinic receptors.[5]	Controlled by voluntary and reflex mechanisms.[5] At a certain bladder filling level, the sacral reflex centers at S2-S4 are stimulated and send afferent info to PMC and frontal cortex.[5] Brain then sends efferent signals to contract bladder, relax bladder neck, and close ureteral valves (sensation to void).[5] If it is an appropriate time to void, voluntary control via frontal cortex sends impulse to external sphincter via pudendal nerve (corticospinal tract).[5] The bladder wall will contract, and the internal and external sphincters will relax.[5] If it is an inappropriate time to void, then voluntary tightening of external sphincter will occur with accompanied bladder wall relaxation and internal sphincter contraction.[5] During storage, there is a sympathetic reflex via sacrolumbar reflex pathway that assist with storage of bladder, increases bladder	pressure storage and bladder emptying at low detrusor pressures, avoid stretch injury from distension, prevent upper and lower urinary tract complications, prevent recurrent urinary tract infection. **Lower motor neuron lesion[5]:** One below or at conus medullaris. Will impact efferent, afferent, or both portions of sacral-arc pathway. Typically present with areflexic detrusor with normal or underactive external sphincter. Coordination will be impacted leading to detrusor-external sphincter dyssynergia. If only motor neurons are impacted, will be able to maintain sense of fullness but this may be lost with recurrent overdistension and subsequent damage. With strictly afferent lesions, patients can void but have altered sensation which may impact emptying and distension.

		Sensory impulses sent to central nervous system from bladder and urethra via pelvic nerves.[5] **Central nervous system (CNS) [5]:** Fibers ascend to pontine micturition center (PMC), periaqueductal gray matter, or ventral posterior nucleus of thalamus and then ascend to cerebral cortex. Efferent information can come from suprapontine regions such as frontal cortex, periaqueductal gray matter and then project to PMC (integration site).[5] Efferent neurons from PMC project to motor neurons that innervate sphincter from Onuf's nucleus, sacral parasympathetic fibers, and rostral lumbar sympathetic fibers.[5]	capacity, and decreases amplitude and frequency of bladder contractions.[5] When bladder pressure reaches a certain threshold, PMC suppresses vesicosympathetic reflex allowing for micturition.[5] Coordinated relationship between bladder walls and sphincter is maintained via pons (bulbospinal pathways).[5]	Lesions involving both lead to a mixture of symptoms. **Upper motor neuron injury[5]:** Can occur intracranial (suprapontine) where PMC is intact but inhibition of detrusor contractility is lost. Can also occur in spinal region (suprasacral or infrapontine). This lesions spare sacral reflex arc. Modulation of detrusor and sphincter activity from PMC is disrupted leading to detrusor-internal sphincter dyssynergia (lesions above T6). Since sacral reflexes are still present, bladder contraction will be uninhibited leading to urinary incontinence w/ no sensation of bladder filling or urge. Can cause upper urinary tract deterioration due to elevated pressure. **Lower urinary tract conditions associated with neurogenic bladder[5]:** Detrusor overactivity[5]: suprasacral lesions (sacral reflex intact, but pontine modulation disrupted)

				Bladder wall compliance[5]: ratio of change in bladder volume to change in pressure (can be high or low, typically high in lower motor neuron injuries, low compliance associated with upper tract deterioration) High leak-point detrusor pressure (DLPP) [5]: max detrusor storage pressure where leakage occurs from bladder during passive filling. Associated with upper tract deterioration. Vesicoureteral reflex[5]: from high DLPP and low bladder compliance. Associated with urinary tract infection and upper tract deterioration. Detrusor-external sphincter dyssynergia[5]: involuntary contraction of urethral sphincter during detrusor contraction (common in suprasacral spinal cord lesions) Acute Bladder Management[5]: - In acute setting, SCI patients experience spinal shock and areflexive bladder so urinary retention lasts from 6 – 12 weeks

				- Goal is to preserve urinary tract - Usually achieved via indwelling Foley catheter followed by clean intermittent catheterization (CIC) when medically stable - CIC usually performed every 4 hours (may be adjusted based on fluid intake and urine volume output) - Ideal to teach CIC early on to patients with sufficient hand function Long term bladder Management[5]: - Depends on level/completeness of injury, hand function, sex, and motivation - Intermittent catheterization considered best/safest option - Chronic indwelling catheters associated with high rates of urinary tract infection, bladder stones, prostatitis, bladder cancer, etc. - Can also use anticholinergic agents

				for those with incontinecen between catheterizations from a hyperreflexic bladder **Credé and Valsalva maneuver[5]:** For lower motor neuron injuries to facilitate bladder emptying by increasing intraabdominal pressure or direct pressure to suprapubic area. **Reflex voiding[5]:** Suprasacral lesions maintain sacral reflex arc. Reflex voiding using suprapubic tapping may assist. Better suited for male patients who lack hand function for CIC. Typically used with an external collecting devise and a transurethral sphincterotomy. **Pharmacologic management[5]:** - Anticholinergics: associated with dry mouth and impaired GI secretion/motility - Tricyclic antidepressants - Antispasmodic drugs - Botulinum toxin type A injections into detrusor **Surgical[5]:**

				- Electrical stimulation and posterior sacral roto rhizotomy - Augmentation cystoplasty (increase total bladder capacity), cutaneous conduits, urinary diversions - Transurethral sphincterotomy **Special Considerations[5]:** Women - CIC is preferred method for bladder emptying if they have adequate hand function or consistent caregiver assistance - Often experience more difficulty with CIC esp. in tetraplegia. - Not a suitable external incontinence devise for females which leads to a higher use of indwelling catheters which is associated with labial and urethral erosion, leakage, and other skin conditions. Pediatric - Need to consider developmental process,

				psychomotor skills, and cognitive limitations as a result of age or brain injury - CIC is preferred method for children and participation is encouraged - Use of latex free catheters and gloves are recommended especially in children with spina bifida

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