Renal system

PHYSIOLOGY

Very important

Extra information

References :

- GUYTON AND HALL 12th edition
- LINDA 5th edition

* Guyton corners, anything that is colored with grey is EXTRA explanation

Physiology of Micturition

Objectives :

- Identify and describe the Functional Anatomy of Urinary Bladder.
- Describe the mechanism of filling and emptying of the urinary bladder.
- Cystometrogram.
- Appreciate neurogenic control of the mechanism of micturition and its disorders.

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Urinary Bladder

*For better understanding, Study **Urinary bladder Anatomy** First*

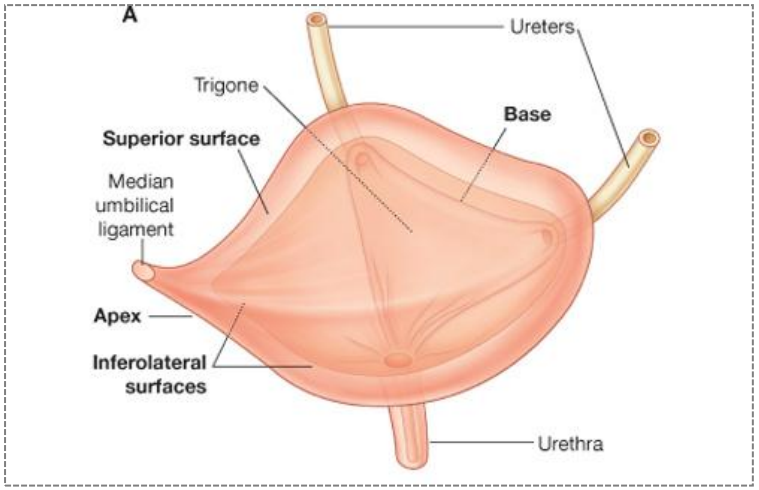
- The bladder is located, in the pelvic cavity when it becomes empty, but it *expands* superiorly to the *abdominal cavity* when it becomes full.

- **Site of urinary bladder at early age :**

  The urinary bladder is an *abdominal organ at birth*, move into the lower abdominal wall. Around the 5th or 6th year of age then bladder gradually descends into the area of the true (minor) pelvis.

Urinary bladder is composed of 4 parts :

- Apex
- Base
- Superior surface
- Inferolateral surfaces



Urinary Bladder

*For better understanding, Study **Urinary bladder Anatomy** First*

Internal structure of urinary bladder composed of :

Mucosa

- Is a membrane that lines cavities of bladder
- Mucosa is *folded* and attached to the wall bladder.
- When the bladder is empty it's numerous folds called rugae and when the urine increase it will flatten out with change in intravesical pressure
- Change in pressure about 10cm and will cause increase the compliance about 400 ml.

Muscle layer

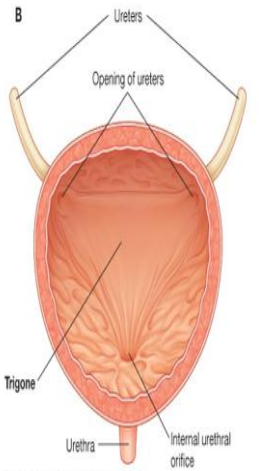
- It is composed if smooth muscle. *(detrusor muscle)*
- It consists of mixture of spiral and longitudinal fibers.
- It can increase the pressure in the bladder up to 40-60 mmHg.
- Will form Triangular area between the openings of the ureters and urethra on the inside of the bladder which known as *Trigone.*

Bladder Neck

- The muscle fibers arranged in 3 layers: inner longitudinal, middle circular, and outer longitudinal.
- The tone of the bladder neck muscles, prevents emptying of the bladder until the appropriate time.
- The *external urethral sphincter* (under voluntary control) used to prevent or interrupt urination.

Submucosa

It supports the mucous membrane.



Urinary Bladder

*For better understanding, Study **Urinary bladder Anatomy** First*

- Contraction of *Detrusor muscle* → emptying of bladder during micturition.

- **We have 2 sphincters:**
  - *Internal urethral sphincters* (IUS) in either side of urethra, made of *smooth muscle.*
  - *External urethral sphincter* (EUS), made of *skeletal muscle.*

- Internal urethral sphincters →Involuntary autonomic supply (sympathetic and parasympathetic)
- External urethral sphincter→ Voluntary (somatic).

• Guyton corner :

The urinary bladder, shown in Figure 26-6, is a smooth muscle chamber composed of two main parts:

(1) the *body,* which is the major part of the bladder in which urine collects.

(2) the *neck,* which is a funnel-shaped extension of the body, passing inferiorly and anteriorly into the urogenital triangle and connecting with the urethra. The lower part of the bladder neck is also called the *posterior urethra* because of its relation to the urethra.

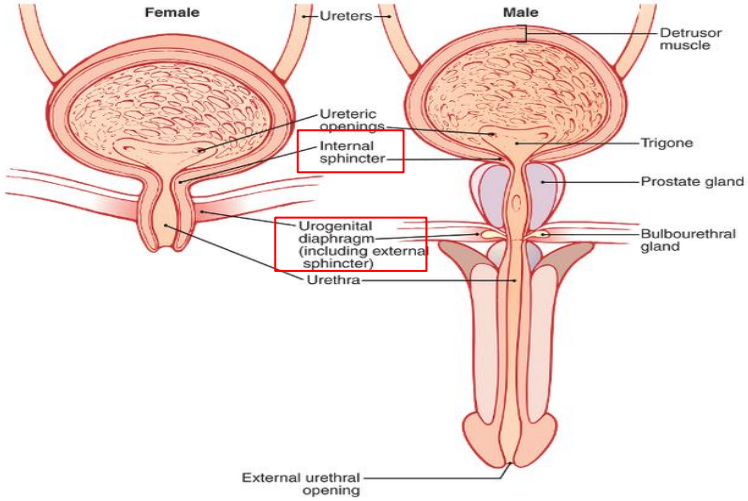


Figure 26-6

Urinary Bladder

*For better understanding, Study **Urinary bladder Anatomy** First*

- **Guyton corner :**

The smooth muscle of the bladder is called the *detrusor muscle.* Its muscle fibers extend in all directions and, when contracted, can increase the pressure in the bladder to 40 to 60 mm Hg. Thus, *contraction of the detrusor muscle is a major step in emptying the bladder*. Smooth muscle cells of the detrusor muscle fuse with one another so that low-resistance electrical pathways exist from one muscle cell to the other. Therefore, an action potential can spread throughout the detrusor muscle, from one muscle cell to the next, to cause contraction of the entire bladder at once. (IMPORTANT)

On the posterior wall of the bladder, lying immediately above the bladder neck, is a small triangular area called the *trigone.* At the lowermost apex of the trigone, the bladder neck opens into the *posterior urethra* and the two ureters enter the bladder at the uppermost angles of the trigone. The trigone can be identified by the fact that its *mucosa,* the inner lining of the bladder, is smooth, in contrast to the remaining bladder mucosa, which is folded to form *rugae.*

Each ureter, as it enters the bladder, courses obliquely through the detrusor muscle and then passes another 1 to 2 centimeters beneath the bladder mucosa before emptying into the bladder.

The bladder neck (posterior urethra) is 2 to 3 centimeters long, and its wall is composed of detrusor muscle interlaced with a large amount of elastic tissue. The muscle in this area is called the *internal sphincter.* Its natural tone normally keeps the bladder neck and posterior urethra empty of urine and, therefore, prevents emptying of the bladder until the pressure in the main part of the bladder rises above a critical threshold.

Beyond the posterior urethra, the urethra passes through the *urogenital diaphragm,* which contains a layer of muscle called the *external sphincter* of the bladder. This muscle is a voluntary skeletal muscle, in contrast to the muscle of the bladder body and bladder neck, which is entirely smooth muscle. The external sphincter muscle is under voluntary control of the nervous system and can be used to consciously prevent urination even when involuntary controls are attempting to empty the bladder.

Urinary Bladder

*For better understanding, Study **Urinary bladder Anatomy** First*

- **Guyton corner :**

✓ Transport of Urine from the Kidney Through the Ureters and into the Bladder :

Urine that is expelled from the bladder has essentially the same composition as fluid flowing out of the collecting ducts; there are no significant changes in the composition of urine as it flows through the renal calyces and ureters to the bladder.

Urine flowing from the collecting ducts into the renal calyces stretches the calyces and increases their inherent pacemaker activity, which in turn initiates peristaltic contractions that spread to the renal pelvis and then downward along the length of the ureter, thereby forcing urine from the renal pelvis toward the bladder. In adults, the ureters are normally 25 to 35 centimeters (10 to 14 inches) long.

The walls of the ureters contain smooth muscle and are innervated by both sympathetic and parasympathetic nerves, as well as by an intramural plexus of neurons and nerve fibers that extends along the entire length of the ureters. As with other visceral smooth muscle, *peristaltic contractions in the ureter are enhanced by parasympathetic stimulation and inhibited by sympathetic stimulation.*

The ureters enter the bladder through the *detrusor muscle* in the trigone region of the bladder.

Normally, the ureters course obliquely for several centimeters through the bladder wall. The normal tone of the detrusor muscle in the bladder wall tends to compress the ureter, thereby preventing backflow (reflux) of urine from the bladder when pressure builds up in the bladder during micturition or bladder compression. Each peristaltic wave along the ureter increases the pressure within the ureter so that the region passing through the bladder wall opens and allows urine to flow into the bladder. In some people, the distance that the ureter courses through the bladder wall is less than normal, so contraction of the bladder during micturition does not always lead to complete occlusion of the ureter. As a result, some of the urine in the bladder is propelled backward into the ureter, a condition called *vesicoureteral reflux.* Such reflux can lead to enlargement of the ureters and, if severe, can increase the pressure in the renal calyces and structures of the renal medulla, causing damage to these regions.

- **Pain Sensation in the Ureters, and the Ureterorenal Reflex :**

The ureters are well supplied with pain nerve fibers. When a ureter becomes blocked (e.g., by a ureteral stone), intense reflex constriction occurs, associated with severe pain. Also, the pain impulses cause a sympathetic reflex back to the kidney to constrict the renal arterioles, thereby decreasing urine output from the kidney. This effect is called the *ureterorenal reflex* and is important for preventing excessive flow of fluid into the pelvis of a kidney with a blocked ureter.

Innervation of Urinary bladder

IMPORTANT

- **Guyton corner :**

✓ **Innervation of the Bladder :**

The principal nerve supply of the bladder is by way of the *pelvic nerves,* which connect with the spinal cord through the *sacral plexus,* mainly connecting with cord segments **S2** and **S3** (Figure 26-7). Coursing through the pelvic nerves are both *sensory nerve fibers* and *motor nerve fibers.* The sensory fibers detect the degree of stretch in the bladder wall. Stretch signals from the posterior urethra are especially strong and are mainly responsible for initiating the reflexes that cause bladder emptying.

The motor nerves transmitted in the pelvic nerves are *parasympathetic fibers.* These terminate on ganglion cells located in the wall of the bladder. Short postganglionic nerves then innervate the detrusor muscle. In addition to the pelvic nerves, two other types of innervation are important in bladder function. Most important are the *skeletal motor fibers* transmitted through the *pudendal nerve* to the external bladder sphincter. These are *somatic nerve fibers* that innervate and control the voluntary skeletal muscle of the sphincter. Also, the bladder receives *sympathetic innervation* from the sympathetic chain through the *hypogastric nerves,* connecting mainly with the **L2** segment of the spinal cord. These sympathetic fibers stimulate mainly the blood vessels and have little to do with bladder contraction. Some sensory nerve fibers also pass by way of the sympathetic nerves and may be important in the sensation of fullness and, in some instances, pain.

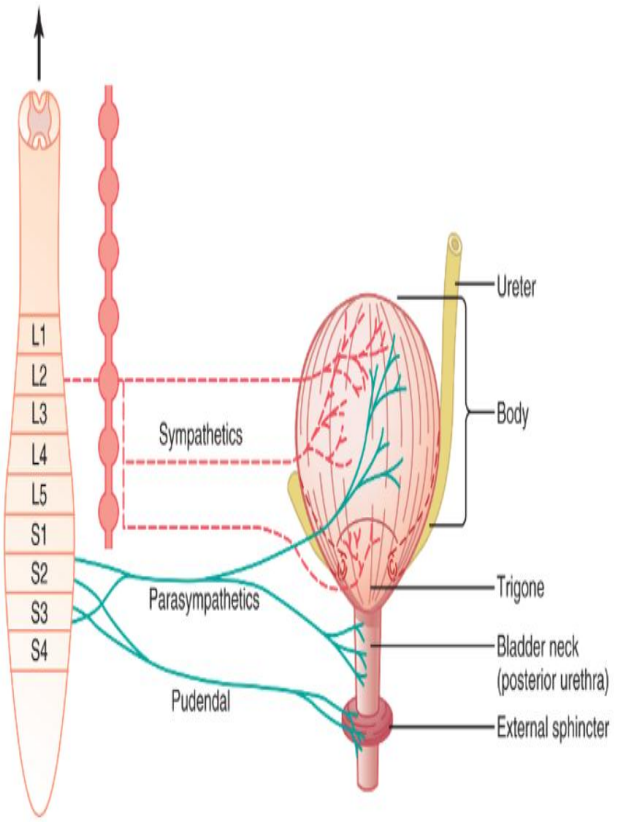


Figure 26-7

Innervation of the bladder

Nerve	Afferent supply	Afferent supply	Function
Hypogastric nerve *"sympathetic"*	• It transmit impulses from the pain receptors to the upper lumbar segment. • Detect bladder fullness • resulting pain sensation from the urethra & bladder. **e.g.** *severe bladder distension & inflammation.*	• Inhibitory to the bladder wall (detrusor muscle). • Motor to the internal urethral sphincter • Motor to the seminal vesicle, ejaculatory duct & prostatic musculature.	Stimulate mainly the blood vessels and have little to do with bladder contraction. Sensory nerve fibers of the sympathetic nerves also mediate the sensation of fullness and pain.
Pelvic nerve *"Parasympathetic"*	• Carry input from stretch receptors in the bladder neck. • transmit impulses from the tension & pain receptors in the wall of U.B. to the sacral region of spinal cord. • Resulting in both reflex micturition , sensation of bladder fullness and temperature sensation	• Motor to the bladder wall (detrusor muscle). • Inhibitory to the internal urethral sphincter " inhibitory = relaxation"	*Contraction* of bladder The sensory fibers detect the degree of stretch in the bladder wall
Pudendal nerve *"Somatic Nerve"*	• It transmit impulses for the sensation of Distension of the urethra and Passage of urine through the urethra.	• Motor to the external urethral sphincter	Fibers that innervate and control the *voluntary* skeletal muscle of the sphincter

Autonomic Innervation of the bladder

- **Sympathetic** supply to the bladder cause *storage* of urine.
- **Parasympathetic** supply leads to the *Passage* of urine.

	Parasympathetic	Sympathetic
Nerve	*Pelvic nerve*	*Hypogastric nerve*
Origin	**LHCs** of the *S2,S3, S4*	**LHCs** of the *L1,L2, L3.*
Supply	Body and neck of the bladder	Bladder neck

- Autonomic nerves always originate from lateral horn cells of the spinal cord while the somatic nerves are from the anterior horn cells.
- Sympathetic supply arises from lateral horn cells of L1-L2 and then it goes to supply the UB causing relaxation of detrusor muscle and contraction of sphincter for example during stressful conditions like exams you feel that you want to urinate but you can't this is due to sympathetic action - remember sympathetic =storage helps in urine storage.
- Urinary bladder contains stretch receptors which stimulate afferent nerves (sensory) to feel the fullness of UB this afferent nerve is part of pelvic nerve which originate from s2, s3, s4 and the other part (efferent) goes back to urinary bladder causing contraction of detrusor muscles and relaxation of internal sphincter.
- The most important reflex is parasympathetic reflex and its center located in the sacral plexus lateral horn cell

LHCs : Lateral horn cells

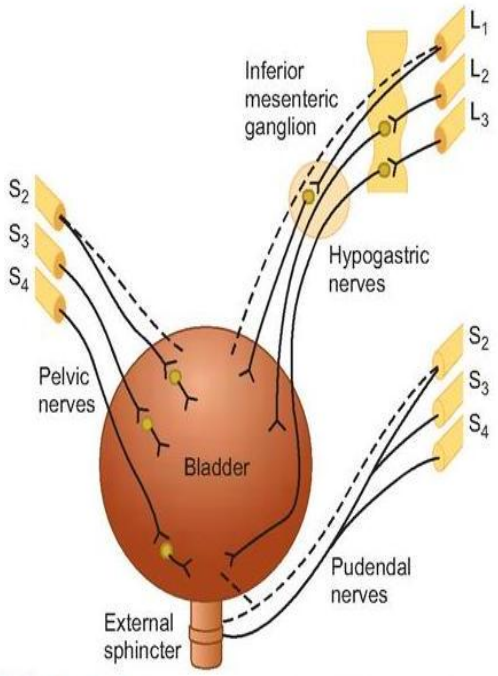
Somatic Innervation of the bladder

The Pudendal nerves

Origin: Anterior horn cells *(AHCs)* of *S2,S3,S4*

Its efferent fibers arise **AS** the parasympathetic nerves from the 2nd, 3rd and 4th sacral segments of the spinal cord *BUT from the AHCs.*

They supply and control the activity of the *External urethral sphincter*



{ The pudendal nerve arises from the same origin of parasympathetic but from the anterior horn cells. }

The Reservoir function of U.B

- Urine will enters the urinary bladder without producing much increase in *I.V.P.* till the bladder becomes well-filled.

I.V.P. : intravesicular pressure
"The pressure exerted on the contents of the urinary bladder, being the sum of the intraabdominal pressure from outside the bladder and the detrusor pressure exerted by the bladder wall musculature itself. Also called *bladder pressure, vesical pressure".*

- **Cystometry** Study the relationship between *intravesical volume* and *pressure.*
- Done by inserting catheter and emptying the bladder, then recording the pressure while bladder filled at **50 ml** increment of water.
- The plot of *I.V.P.* against the volume is called **"Cystometrogram".**

A cystometrogram allows us to assess how your bladder and sphincter behave while you store urine and when you pass urine. This test is done for people with urinary incontinence, people who have difficulty with urination, and in people with neurologic diseases that can affect bladder function. This test will measure your bladder capacity and pressure. By doing this we can identify problems such as a small capacity bladder, overactive bladder or high pressure bladder.

Cystometrogram “EXTRA”

- **Guyton corner :**

✓ **Filling of the Bladder and Bladder Wall Tone; the Cystometrogram :**

Figure 26-8 shows the approximate changes in intravesicular pressure as the bladder fills with urine. When there is no urine in the bladder, the intravesicular pressure is about 0, but by the time 30 to 50 milliliters of urine have collected, the pressure rises to 5 to 10 centimeters of water. Additional urine—200 to 300 milliliters—can collect with only a small additional rise in pressure; this constant level of pressure is caused by intrinsic tone of the bladder wall itself. Beyond 300 to 400 milliliters, collection of more urine in the bladder causes the pressure to rise rapidly. Superimposed on the tonic pressure changes during filling of the bladder are periodic acute increases in pressure that last from a few seconds to more than a minute. The pressure peaks may rise only a few centimeters of water or may rise to more than 100 centimeters of water. These pressure peaks are called *micturition waves* in the cystometrogram and are caused by the micturition reflex

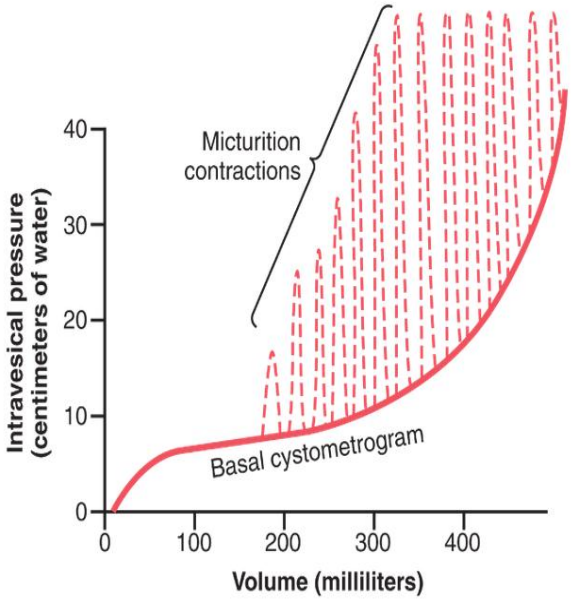


Figure 26-8

Cytometrogram is a graph that studies the relation between the pressure and urine volume, in order to study these 2 factors we should first empty the bladder and then in the lab they put 2 catheters. The 1st catheter fills the bladder gradually and the other one measures the pressure , as they increase the volume of urine the pressure increases. When the volume reaches to 50 ml the pressure increases to be 5 but they noticed that when the volume contuses increasing between 100 to 200 ml the pressure remains constant this is due to the bladder has feature called “ receptive relaxation “ i.e. : as it receives more urine it dilate more and more so the pressure remains constant and this dilation is due to presence of transitional epithelium “ this called Laplace law . The pressure will remain constant within limit but when it reaches 400 ml any further increase will increase the pressure and it won’t be constant anymore.

3 components (segments) of the plot

Stage Ia

Represent initial rise in I.V.P. (when the first increment in volume are produced) by about 10 cm H_2O when the first volume is produced by about 50 ml.

Stage Ib

It is a long, nearly flat segment produced by further ↑ in filling up to nearly 150 ml.

Stage II

a sudden, sharp rise in pressure as the micturition reflex is triggered

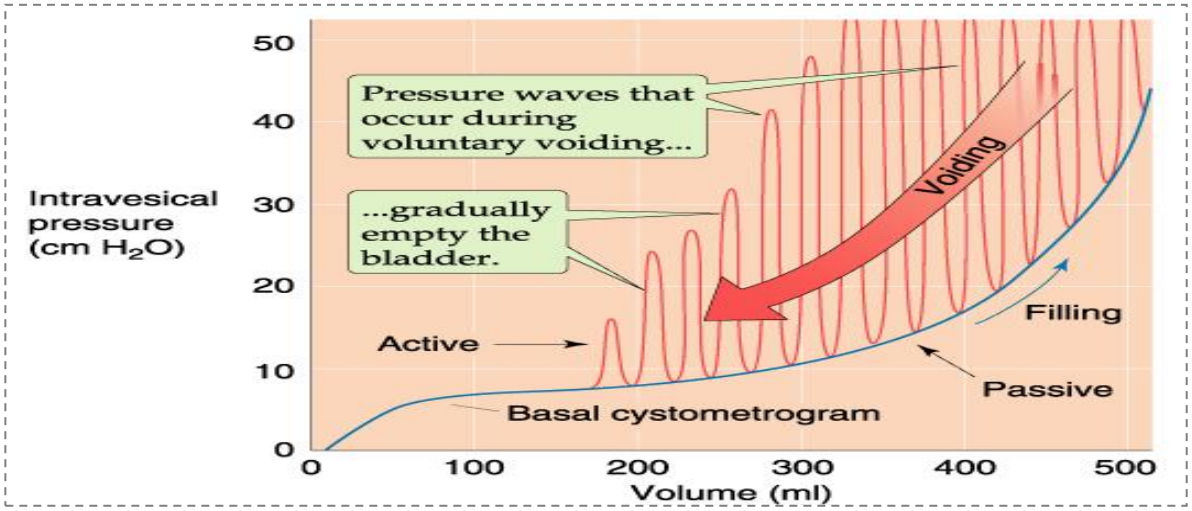
(sense of fullness and urge to void)

This segment is produced by further increment of volume

(150 – 400 ml)

The flatness of segment Ib is a manifestation of the *Laplace Law* which states that the pressure in the spherical viscus equal to *twice* the wall tension divided the radius { P = 2T / r }.

Cystometrogram



- In the urinary bladder ⇒ the *tension* on the wall *increases* as the volume increases & also the *Radius increases*, so there is little change in pressure until the organ is filled. (↑ tension = ↑ Volume = ↑ Radius)
- Any increase in volume after this will NOT be accommodated & is reflected by rapid rise of pressure.
- Superimposed on this curve, periodic acute increase in pressure which lasts very few seconds, & called "micturition waves" & are caused by micturition reflex.

Sensations from the U.B at different urine volumes

Stage 1 : U.V 150-300 ml 1ST urge to void urine

Micturition reflexes start to appear at this stage

Stage 2 : U.V 300-400 ml sense of fullness of U.B

Stage 3 : U.V 400-600 ml sense of discomfort

Stage 4 : U.V 600-700 ml sense of pain

Break point : U.V 700 ml micturition **can't** be suppressed

Sometimes when you don't want to urinate and your bladder is extremely filled it will inhibit the pudendal nerve which result in opening of external sphincter .

At the stages from (1 to 4)Micturition reflexes can be voluntarily suppressed.

** U.B means urinary bladder*
** U.V means urine volume by ml*

Control of Micturition reflex

- It is a *complete autonomic spinal reflex* to get urine outside the body, that is facilitated or inhibited by higher brain centers.

Infants as they born they don't have this control they develop control after 2 years that's why moms always need to change their diapers .

- **Guyton Corner :** *page 309 -310*

Once a micturition reflex begins, it is "self-regenerative." That is, initial contraction of the bladder activates the stretch receptors to cause a greater increase in sensory impulses from the bladder and posterior urethra, which causes a further increase in reflex contraction of the bladder; thus, the cycle is repeated again and again until the bladder has reached a strong degree of contraction. Then, after a few seconds to more than a minute, the self-regenerative reflex begins to fatigue and the regenerative cycle of the micturition reflex ceases, permitting the bladder to relax.
Thus, the micturition reflex is a single complete cycle of (1) progressive and rapid increase of pressure, (2) a period of sustained pressure, and (3) return of the pressure to the basal tone of the bladder. Once a micturition reflex has occurred but has not succeeded in emptying the bladder, the nervous elements of this reflex usually remain in an inhibited state for a few minutes to 1 hour or more before another micturition reflex occurs. As the bladder becomes more and more filled, micturition reflexesoccur more and more often and more and more powerfully.
Once the micturition reflex becomes powerful enough, it causes another reflex, which passes through the *pudendal nerves* to the *external sphincter* to inhibit it. If this inhibition is more potent in the brain than the voluntary constrictor signals to the external sphincter, urination will occur. If not, urination will not occur until the bladder fills still further and the micturition reflex becomes more powerful.

Micturition

Micturition	
Unconditioned (Automatic)	**Conditioned(Voluntary)**
In Infants • In infants ⇒ urination occurs through a series of spinal reflexes called "the micturition reflexes" which are automatic (not under voluntary control) because the nerve tracts are not yet myelinated in infants. • The stimulus that initiates these reflexes is rise of the IVP (which stimulates stretch receptors in the bladder wall)	In adult • In adults ⇒ the act of micturition occurs also through the micturition reflexes, but however, it can be voluntarily controlled by certain higher (or supra-spinal) centers in the brain, which include the following: • **Facilitatory** - In pontine area. - Posterior hypothalamus. - Other cortical centers • **Inhibitory** - In the midbrain

- **Guyton Corner :** *page 310*

Facilitation or Inhibition of Micturition by the Brain

The micturition reflex is an autonomic spinal cord reflex, but it can be inhibited or facilitated by centers in the brain. These centers include (1) strong *facilitative* and *inhibitory centers in the brain stem, located mainly in the pons,* and (2) several *centers located in the cerebral cortex* that are mainly inhibitory but can become excitatory.

The micturition reflex is the basic cause of micturition, but the higher centers normally exert final control of micturition as follows:

1.The higher centers keep the micturition reflex partially inhibited, except when micturition is desired.

2.The higher centers can prevent micturition, even if the micturition reflex occurs, by tonic contraction of the external bladder sphincter until a convenient time presents itself.

3.When it is time to urinate, the cortical centers can facilitate the sacral micturition centers to help initiate a micturition reflex and at the same time inhibit the external urinary sphincter so that urination can occur.

Voluntary urination is usually initiated in the following way: First, a person voluntarily contracts his or her abdominal muscles, which increases the pressure in the bladder and allows extra urine to enter the bladder neck and posterior urethra under pressure, thus stretching their walls. This stimulates the stretch receptors, which excites the micturition reflex and simultaneously inhibits the external urethral sphincter. Ordinarily, all the urine will be emptied, with rarely more than 5 to 10 milliliters left in the bladder.

"The Micturition Reflexes"

The micturition reflexes can be summarized as follows:

- Distention of the U.B. (as a result of ↑ I.V.P. & not by an ↑ in the bladder volume) produces reflex contraction of its wall & relaxation of the internal urethral sphincter & external urethral sphincter.

- The flow of urine in urethra will produce contraction of the U.B. wall & relaxation of both internal & external urethral sphincters.

Higher Centers Control Micturition :

1) Cerebral cortex:

Motor cortex exerts a *voluntary control of micturition* either stimulation or inhibition.

2) Hypothalamus:

There is *facilitatory area* in the hypothalamus.

3) Midbrain: *Inhibition.*

4) Pons: *Facilitation*

Mechanism of voluntary control of micturition

Filling of the bladder beyond 300 -400 ml

↓

Stretching of sensory stretch receptors

↓

These sensory signals stimulate *sacral segment* which is consciously appreciated by higher centers

↓

Condition is Favorable

The cortical centers facilitate micturition by discharging signals that leads to:

- Stimulation (+) of sacral micturition center.
- Inhibition (-) of pudendal nerves → relaxation of external urethral sphincter.
- Contraction of anterior abdominal muscle & diaphragm to increase intra-abdominal pressure → the intra-vesical pressure is increased. This intensifies the micturition reflex.

RESULTS IN :

Intensifies the micturition reflex → *Urination*

Condition is Unfavorable

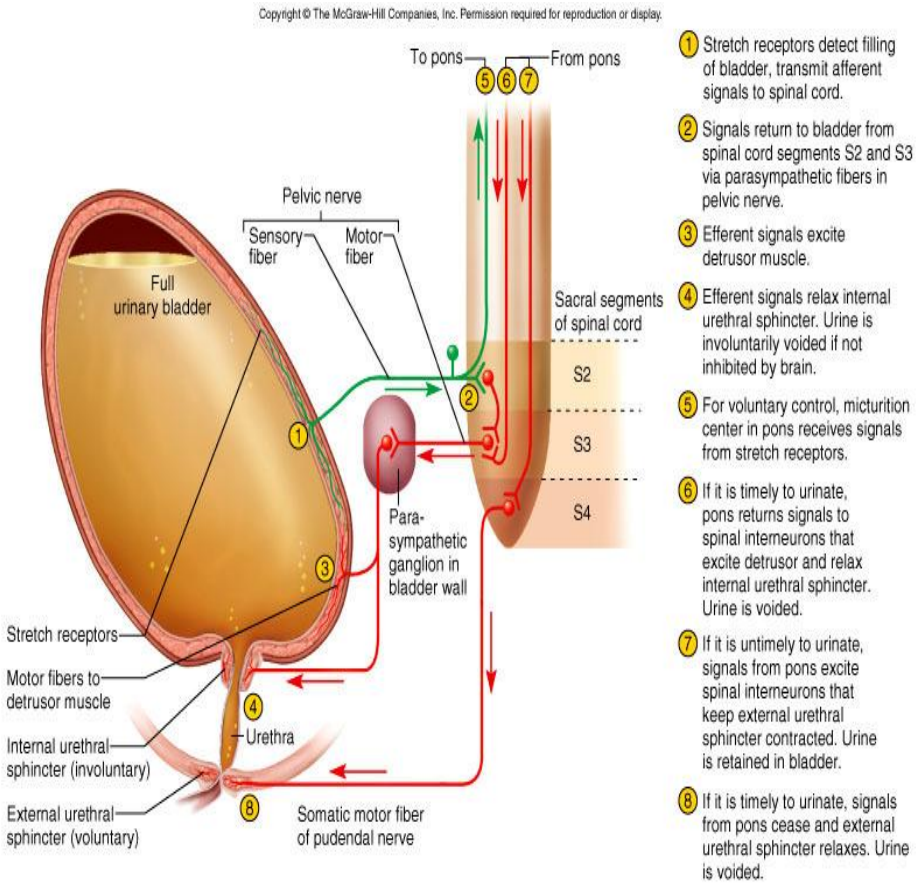
Higher centers will **inhibit** the micturition reflex

- Inhibition (-) of sacral micturition center
- Stimulation (+) of pudendal nerves → Contraction of external urethral sphincter

RESULTS IN :

Inhibit the micturition reflex → *No urination*

Mechanism of voluntary control of micturition



1) APs generated by stretch receptors
2) reflex arc generates APs that
3) stimulate smooth muscle lining bladder
4) relax internal urethral sphincter (IUS)
5) stretch receptors also send APs to Pons
6) if it is o.k. to urinate
   - –APs from Pons excite smooth muscle of bladder and relax IUS
   - –relax external urethral sphincter
7) if not o.k. :
   - –APs from Pons keep external urethral sphincter contracted

As the impulses flow through the afferent part of pelvic nerve other impulses travel to pons in the brain. if you want to urinate, pons will stimulate parasympathetic and inhibit pudendal nerve to open external sphincter. in the other hand, if you don't want to urinate pons will inhibit parasympathetic and stimulate pudendal nerve .

Disturbances (Abnormalities) of Micturition

Very important !

Denervation of the afferent supply only such as in : Tabes Dorsalis *(tabetic bladder)*	Denervation of both afferent and efferent nerve supply such as in tumor or injury to cauda equina
Loss of the U.B sensations and reflex micturition	Abolished (canceled) reflexes
↓	↓
Some intrinsic responses of the smooth muscle are retained	Increase in the intrinsic responses of the smooth muscles
↓	↓
The bladder becomes distended, thin walled & hypotonic (atonic bladder)	Hypertonic bladder due to denervation hypersensitivity because : → Decreased degradation of Ach by process of reuptake. → Decreased cholinesterase in the tissue → Increased number of cholinergic receptors
↓	
There is retention with overflow. i.e. *dribbling of urine* when the bladder becomes over filled.	This condition is associated with uncontrolled periodic micturition about 25 – 100 ml at a time

Disturbances (Abnormalities) of Micturition

- *Tabes dorsalis* is a late manifestation of untreated syphilis and is characterized by a triad of clinical symptoms namely gait unsteadiness, lightning pains and urinary incontinence. It occurs due to a slow and progressive degeneration of nerve cells and fibers in spinal cord. It is one of the forms of tertiary syphilis or neurosyphilis.

- If the afferent is denervated انقطعyou won't be able to feel the distention of your bladder, your bladder will continue filling and filling with no orders to contract this will result in dribbling of the excess urine ينقط يصير .
- The urinary bladder lost its ability to contract that's why they call it atonic bladder "no tone ".
- Afferent nerve is affected in 2 : diseases diabetes mellitus and tabes dorsalis "syphilis"
- If both afferent and efferent are damaged the muscle of UB will act as isolated muscles and contract by its self "hypertonic"
- Because the afferent and efferent parts of pelvic nerve are located at the end of spinal cord region called "cauda equina" any injury or tumor to this region will cause bladders' muscle to be hypertonic.

ABNORMALITIES OF MICTURITION

	ABNORMALITIES OF MICTURITION	
	ATONIC BLADDER	**AUTOMATIC BLADDER**
Lesion	Sensory nerve fibers from the bladder to the spinal cord are destroyed Crush injury to the sacral region of the spinal cord *and tabes dorsalis*	Spinal Cord Damage Above the Sacral Region resulting in Spinal shock
Feature	Bladder fills to capacity and overflows a few drops at a time through the urethra. This is called *overflow incontinence*.	return of excitability of micturition reflex until typical micturition reflexes returns & then, periodic (but unannounced) bladder emptying occurs which may be controlled by scratching or tickling

If the lesion is in the lumber region "above sacral region i.e. :sacral is intact parasympathetic works properly and micturition takes place " but it doesn't have higher control " the defect is in pons ,cerebral cortex and sympathetic supply" the impulses from pons and cerebral cortex won't reach the bladder and their control is stopped this person will urinate involuntarily as if he is a baby . (Very important).

This happen in people with Quadriplegia as result of car accident these patient at the 1st 2 wks will suffer from shock and urinate by dribbling (because parasympathetic in his body used to get impulses from pons and cerebral cortex in his situation no impulses are transmitted to parasympathetic) after shock has gone the parasympathetic will restore its function and he will urinate normally but without control.

نحن بالوضع الطبيعي لو أردنا التبول نستطيع التبول بزيادة الضغط على عضلات المعدة لكن في حالة مرضى الشلل الرباعي يقوم المريض بوخزات أو دفع التبول كطريقة للتحكم بالبول لتجنب الإحراج.

Cont. Disturbances of micturition

Spinal cord transaction (Above the sacral region)

- **The spinal cord transaction consist of 3 stages :**

Stage of spinal shock:

caused by a sudden separation of the spinal centers from the higher centers that control them, this will cause the spinal centers functionless for 2-6 weeks and the micturition reflex will be abolished

⇒ "retention with overflow" i.e. the bladder distends until the I.V.P. exceeds the urethral sphincter resistance & so, urine starts to dribble.

→

Stage of recovery:

Automatic micturition occurs as soon as the IVP pressure rises to 15-20 cm water, this will cause micturition reflex.

→

Stage of failure of recovery:

This stage is caused by toxins and bacterial infection and will lead to abolished micturition reflex then will lead to **"Retention with overflow"**.

Dr.Mannan mentioned this scenario at the beginning of lecture :

middle aged patient came to ER due to car accident on x-ray , they found that the patient has broken his lumber vertebrae L3 and L4 which result in paralysis . After 2-3 wks he suffered from involuntary urination.

IMPORTANT !!

- **Guyton Corner :** *page 310*

Abnormalities of Micturition

Atonic Bladder and Incontinence Caused by Destruction of Sensory Nerve Fibers.

Micturition reflex contraction cannot occur if the sensory nerve fibers from the bladder to the spinal cord are destroyed, thereby preventing transmission of stretch signals from the bladder. When this happens, a person loses bladder control, despite intact efferent fibers from the cord to the bladder and despite intact neurogenic connections within the brain. Instead of emptying periodically, the bladder fills to capacity and overflows a few drops at a time through the urethra. This is called *overflow incontinence.*

A common cause of atonic bladder is crush injury to the sacral region of the spinal cord. Certain diseases can also cause damage to the dorsal root nerve fibers that enter the spinal cord. For example, syphilis can cause constrictive fibrosis around the dorsal root nerve fibers, destroying them. This condition is called *tabes dorsalis,* and the resulting bladder condition is called *tabetic bladder.*

Automatic Bladder Caused by Spinal Cord Damage Above the Sacral Region.

If the spinal cord is damaged above the sacral region but the sacral cord segments are still intact, typical micturition reflexes can still occur. However, they are no longer controlled by the brain. During the first few days to several weeks after the damage to the cord has occurred, the micturition reflexes are suppressed because of the state of "spinal shock" caused by the sudden loss of facilitative impulses from the brain stem and cerebrum. However, if the bladder is emptied periodically by catheterization to prevent bladder injury caused by overstretching of the bladder, the excitability of the micturition reflex gradually increases until typical micturition reflexes return; then, periodic (but unannounced) bladder emptying occurs.

Some patients can still control urination in this condition by stimulating the skin (scratching or tickling) in the genital region, which sometimes elicits a micturition reflex.

Uninhibited Neurogenic Bladder Caused by Lack of Inhibitory Signals from the Brain.

Another abnormality of micturition is the so-called uninhibited neurogenic bladder, which results in frequent and relatively uncontrolled micturition. This condition derives from partial damage in the spinal cord or the brain stem that interrupts most of the inhibitory signals. Therefore, facilitative impulses passing continually down the cord keep the sacral centers so excitable that even a small quantity of urine elicits an uncontrollable micturition reflex, thereby promoting frequent urination.

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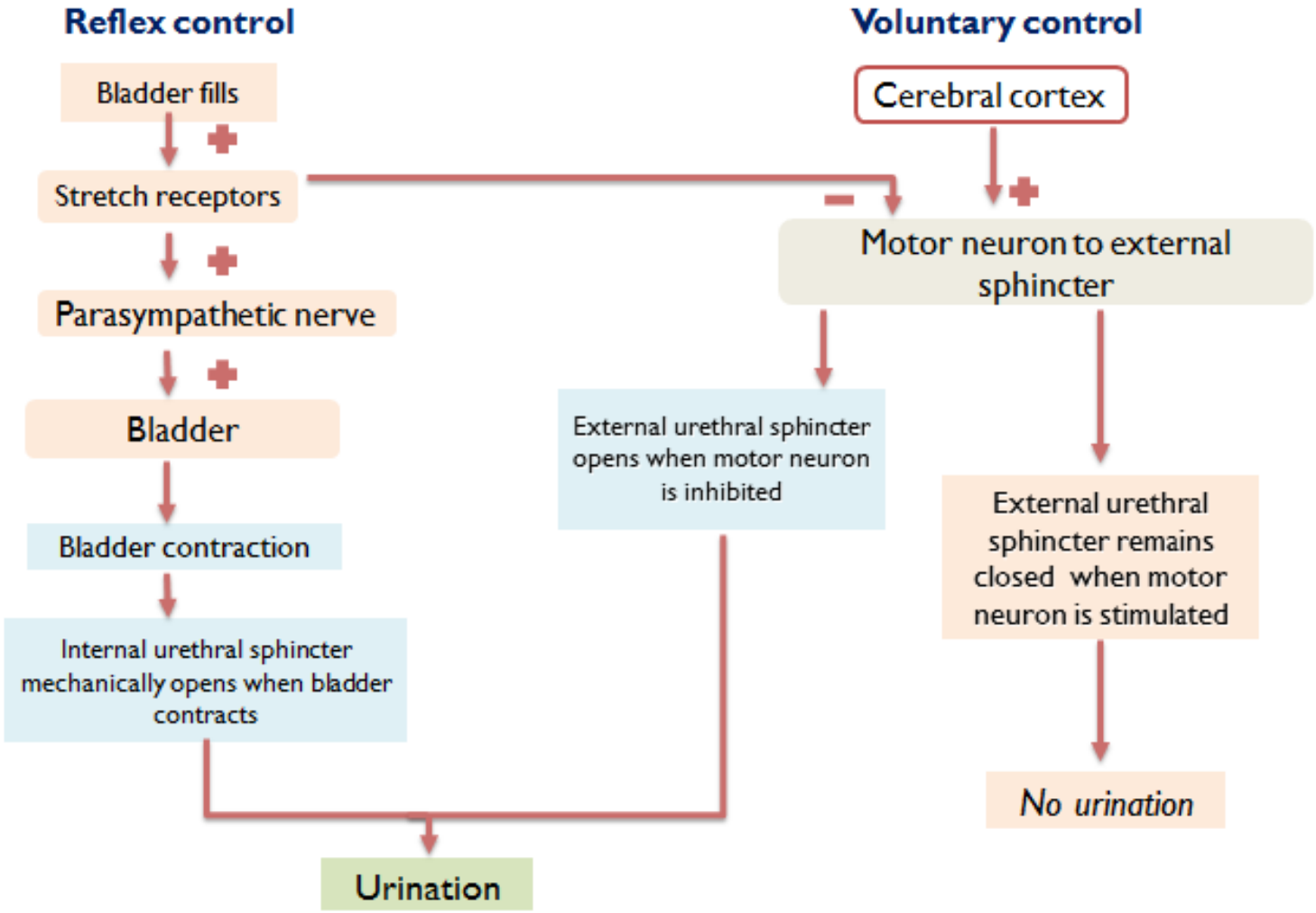
Essential functions and anatomy
The bladder has two functions – storage and voiding. Afferent pathways (T12–S4) respond to pressure within the bladder and sensation from the genitalia. As the bladder distends, continence is maintained by suppression of parasympathetic and reciprocal activation of sympathetic outflow. Both are under some voluntary control. Voiding takes place by parasympathetic activation of the detrusor, and relaxation of the internal sphincter (Table 21.18). Cortical awareness of bladder fullness is located in the
post-central gyrus, parasagittally, while initiation of micturition is in the pre-central gyrus. Voluntary control of micturition is located in the frontal cortex, parasagittally. Neurological disorders of micturition
Urogenital tract disease is dealt with largely by urologists. Incontinence is common and easy to recognize; neurological causes are sometimes not obvious. These are: Cortical:

- Post-central lesions cause loss of sense of bladder
- fullness.
- Pre-central lesions cause difficulty initiating micturition.
- Frontal lesions cause socially inappropriate micturition.

Spinal cord. Bilateral UMN lesions (pyramidal tracts) cause urinary frequency and incontinence. The bladder is small and hypertonic, i.e. sensitive to small changes in intravesical pressure. Frontal lesions can also cause a hypertonic bladder. LMN. Sacral lesions (conus medullaris, sacral root and pelvic nerve – bilateral) cause a flaccid, atonic bladder that overflows cauda equina, p. 1177), often unexpectedly.
Management. Assessment of both urological causes (e.g.
calculi, prostatism, gynaecological problems) and potential neurological causes of incontinence is necessary. Intermittent self-catheterization is used by many patients, with for example spinal cord lesions.

موجودة في هامش سلايدات د.منان

Reflex & voluntary control of micturition (*summary*)



[There are very helpful summaries in Students' work folder]

THE BLADDER

FILLING

The walls of the ureters contain smooth muscle arranged in spiral, longitudinal, and circular bundles, but distinct layers of muscle are not seen. Regular peristaltic contractions occurring one to five times per minute move the urine from the renal pelvis to the bladder, where it enters in spurts synchronous with each peristaltic wave. The ureters pass obliquely through the bladder wall and, although there are no ureteral sphincters as such, the oblique passage tends to keep the ureters closed except during peristaltic waves, preventing reflux of urine from the bladder.

EMPTYING

The smooth muscle of the bladder, like that of the ureters, is arranged in spiral, longitudinal, and circular bundles. Contraction of the circular muscle, which is called the **detrusor muscle,** is mainly responsible for emptying the bladder during urination (micturition). Muscle bundles pass on either side of the urethra, and these fibers are sometimes called the **internal urethral sphincter,** although they do not encircle the urethra. Farther along the urethra is a sphincter of skeletal muscle, the sphincter of the membranous urethra **(external urethral sphincter).** The bladder epithelium is made up of a superficial layer of flat cells and a deep layer of cuboidal cells. The innervation of the bladder is summarized in Figure 38–20.

The physiology of bladder emptying and the physiologic basis of its disorders are subjects about which there is much confusion. Micturition is fundamentally a spinal reflex facilitated and inhibited by higher brain centers and, like defecation, subject to voluntary facilitation and inhibition. Urine enters the bladder without producing much increase in intravesical pressure until the viscus is well filled. In addition, like other types of smooth muscle, the bladder muscle has the property of plasticity; when it is stretched, the tension initially produced is not maintained. The relation between intravesical pressure and volume can be studied by inserting a catheter and emptying the bladder, then recording the pressure while the bladder is filled with 50-mL increments of water or air **(cystometry).** A plot of intravesical pressure against the volume of fluid in the bladder is called a **cystometrogram** (Figure 38–21). The curve shows an initial slight rise in pressure when the first increments in volume are produced; a long, nearly flat segment as further increments are produced; and a sudden, sharp rise in pressure as the micturition reflex is triggered. These three components are sometimes called segments Ia, Ib, and II. The first urge to void is felt at a bladder volume of about 150 mL, and a marked sense of fullness at about 400 mL. The flatness of segment Ib is a manifestation of the law of Laplace. This law states that the pressure in a spherical viscus is equal to twice the wall tension divided by the radius. In the case of the bladder, the tension increases as the organ fills, but so does the radius. Therefore, the pressure increase is slight until the organ is relatively full.

During micturition, the perineal muscles and external urethral sphincter are relaxed, the detrusor muscle contracts, and urine passes out through the urethra. The bands of smooth muscle on either side of the urethra apparently play no role in micturition, and their main function in males is believed to be the prevention of reflux of semen into the bladder during ejaculation.

The mechanism by which voluntary urination is initiated remains unsettled. One of the initial events is relaxation of the muscles of the pelvic floor, and this may cause a sufficient

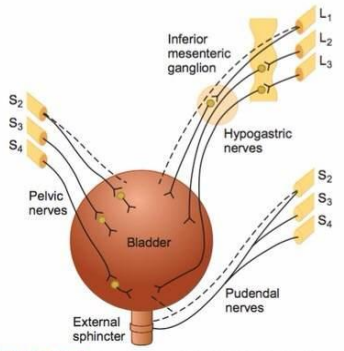


FIGURE 38–20 Innervation of the bladder. Dashed lines indicate sensory nerves. Parasympathetic innervation is shown at the left, sympathetic at the upper right, and somatic at the lower right.

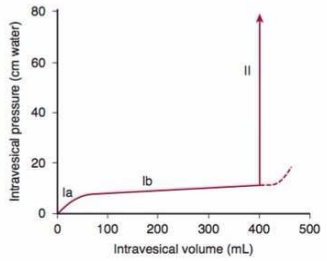


FIGURE 38–21 Cystometrogram in a normal human. The numerals identify the three components of the curve described in the text. The dashed line indicates the pressure–volume relations that would have been found had micturition not occurred and produced component II. (Modified and reproduced with permission from Tanagho EA, McAninch JW: *Smith's General Urology,* 15th ed. McGraw-Hill, 2000.)

downward tug on the detrusor muscle to initiate its contraction. The perineal muscles and external sphincter can be contracted voluntarily, preventing urine from passing down the urethra or interrupting the flow once urination has begun. It is through the learned ability to maintain the external sphincter in a contracted state that adults are able to delay urination until the opportunity to void presents itself. After urination, the female urethra empties by gravity. Urine remaining in the urethra of the male is expelled by several contractions of the bulbocavernosus muscle.

REFLEX CONTROL

The bladder smooth muscle has some inherent contractile activity; however, when its nerve supply is intact, stretch receptors in the bladder wall initiate a reflex contraction that has a lower threshold than the inherent contractile response of the muscle. Fibers in the pelvic nerves are the afferent limb of the voiding reflex, and the parasympathetic fibers to the bladder that constitute the efferent limb also travel in these nerves. The reflex is integrated in the sacral portion of the spinal cord. In the adult, the volume of urine in the bladder that normally initiates a reflex contraction is about 300 to 400 mL. The sympathetic nerves to the bladder play no part in micturition, but in males they do mediate the contraction of the bladder muscle that prevents semen from entering the bladder during ejaculation.

The stretch receptors in the bladder wall have no small motor nerve system. However, the threshold for the voiding reflex, like the stretch reflexes, is adjusted by the activity of facilitatory and inhibitory centers in the brainstem. There is a facilitatory area in the pontine region and an inhibitory area in the midbrain. After transection of the brain stem just above the pons, the threshold is lowered and less bladder filling is required to trigger it, whereas after transection at the top of the midbrain, the threshold for the reflex is essentially normal. There is another facilitatory area in the posterior hypothalamus. Humans with lesions in the superior frontal gyrus have a reduced desire to urinate and difficulty in stopping micturition once it has commenced. However, stimulation experiments in animals indicate that other cortical areas also affect the process. The bladder can be made to contract by voluntary facilitation of the spinal voiding reflex when it contains only a few milliliters of urine. Voluntary contraction of the abdominal muscles aids the expulsion of urine by increasing the intra-abdominal pressure, but voiding can be initiated without straining even when the bladder is nearly empty.

EFFECTS OF DEAFFERENTATION

When the sacral dorsal roots are cut in experimental animals or interrupted by diseases of the dorsal roots, such as **tabes dorsalis** in humans, all reflex contractions of the bladder are abolished. The bladder becomes distended, thin-walled, and hypotonic, but some contractions occur because of the intrinsic response of the smooth muscle to stretch.

CLINICAL BOX 38–4

Abnormalities of Micturition

Three major types of bladder dysfunction are due to neural lesions: (1) the type due to interruption of the afferent nerves from the bladder, (2) the type due to interruption of both afferent and efferent nerves, and (3) the type due to interruption of facilitatory and inhibitory pathways descending from the brain. In all three types the bladder contracts, but the contractions are generally not sufficient to empty the viscus completely, and residual urine is left in the bladder.

EFFECTS OF DENERVATION

When the afferent and efferent nerves are both destroyed, as they may be by tumors of the cauda equina or filum terminale, the bladder is flaccid and distended for a while. Gradually, however, the muscle of the "decentralized bladder" becomes active, with many contraction waves that expel dribbles of urine out of the urethra. The bladder becomes shrunken and the bladder wall hypertrophied. The reason for the difference between the small, hypertrophic bladder seen in this condition and the distended, hypotonic bladder seen when only the afferent nerves are interrupted is not known. The hyperactive state in the former condition suggests the development of denervation hypersensitization even though the neurons interrupted are preganglionic rather than postganglionic (see Clinical Box 38–4).

EFFECTS OF SPINAL CORD TRANSECTION

During spinal shock, the bladder is flaccid and unresponsive. It becomes overfilled, and urine dribbles through the sphincters **(overflow incontinence).** After spinal shock has passed, the voiding reflex returns, although there is, of course, no voluntary control and no inhibition or facilitation from higher centers when the spinal cord is transected. Some paraplegic patients train themselves to initiate voiding by pinching or stroking their thighs, provoking a mild mass reflex (see Chapter 16). In some instances, the voiding reflex becomes hyperactive, bladder capacity is reduced, and the wall becomes hypertrophied. This type of bladder is sometimes called the **spastic neurogenic bladder.** The reflex hyperactivity is made worse by, and may be caused by, infection in the bladder wall.

CHAPTER SUMMARY

- Plasma enters the kidneys and is filtered in the glomerulus. As the filtrate passes down the nephron and through the tubules its volume is reduced and water and solutes are removed (tubular reabsorption) and waste products are secreted (tubular secretion).

Dr. mannan recommended reading this chapter from Ganong's book.

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- Saad Almutairy