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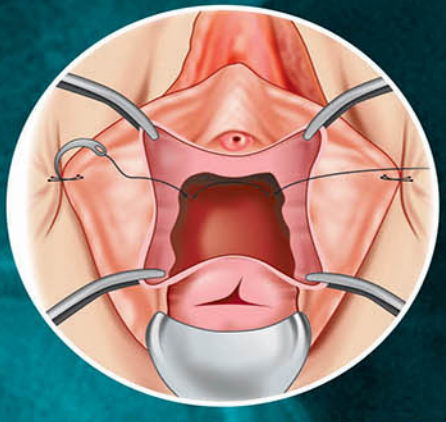

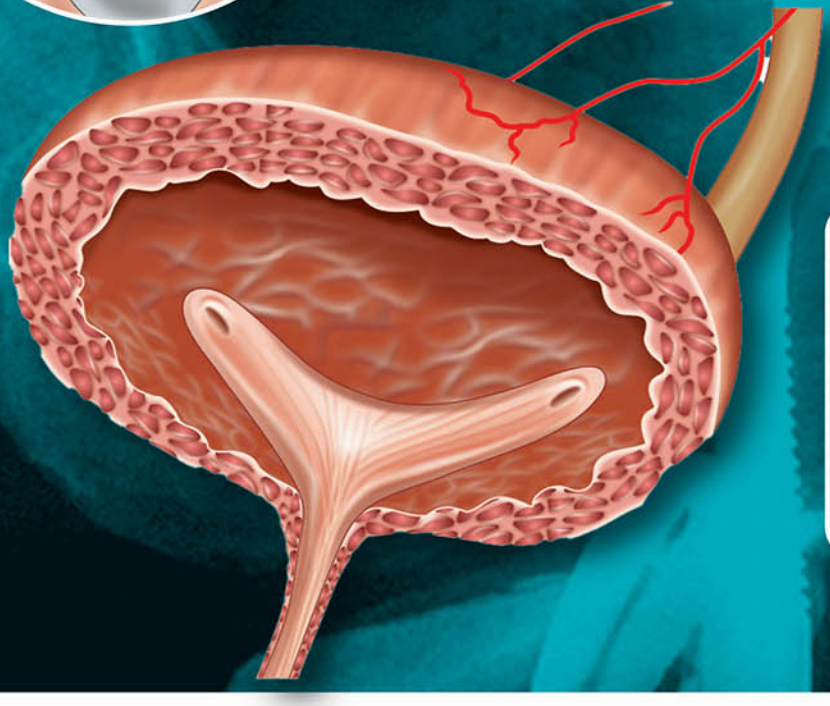



2nd Edition

Manidip Pal

Foreword
Paul Riss

JAYPEE

Contents

Section 1 BASIC

1. **Anatomy of the Pelvic Floor** 3
   Manidip Pal, Paul Riss
2. **Anatomy of Urinary Bladder, Urethra and Pelvic Ureter** 13
   Susmita Bhattacharya, L Ranjit Singh
3. **Anatomy of Retropubic Space** 20
   Manidip Pal
4. **Physiology of Micturition** 22
   Susmita Bhattacharya, L Ranjit Singh
5. **Nomenclature** 27
   Manidip Pal
6. **Pathophysiology of Stress Urinary Incontinence** 29
   Manidip Pal
7. **Pathophysiology of Overactive Bladder** 32
   K Bharathalaxmi, Manidip Pal
8. **Biomarkers in Overactive Bladder** 35
   Soma Bandyopadhyay, Manidip Pal
9. **Classification of Pelvic Organ Prolapse** 38
   Manidip Pal
10. **Pathophysiology of Pelvic Organ Prolapse** 44
    Manidip Pal
11. **Genomics of Prolapse (POP)** 46
    Manidip Pal

Section 2 DIAGNOSIS

12. **History and Examination of Patient** 53
    Soma Bandyopadhyay, Manidip Pal
13. **Neurological Evaluation of Pelvic Floor Disorder** 78
    Sudhir Adhikary, Sanjoy Kumar Bhattacharyya
14. **Ultrasonography in Urogynecology** 84
    Satyasis Ray, Saraswata Das, Manidip Pal
15. **Urodynamic Study** 90
    Manidip Pal

16. **Cystoscopy** 102
Manidip Pal

17. **Hysterosalpingography** 108
Manidip Pal

Section 3 TREATMENT

18. **Urinary Tract Infection** 115
N Nabakishore Singh

19. **Stress Urinary Incontinence: Conservative Management** 118
Chirom Pritam Kumar Singh, Manidip Pal

20. **Overactive Bladder: Management** 123
K Bharathalaxmi, Manidip Pal

21. **Underactive Bladder** 131
Manidip Pal

22. **Urinary Retention** 146
Manidip Pal

23. **Voiding Dysfunction** 149
Manidip Pal

24. **Interstitial Cystitis/Bladder Pain Syndrome** 152
Nagendra Nath Mishra

25. **Urethral Bulking Agents** 171
Manidip Pal

26. **Pediatric Nocturnal Enuresis** 175
Manidip Pal

27. **Chyluria** 180
Manidip Pal

28. **Pelvic Organ Prolapse: Conservative Management** 182
Manidip Pal

Section 4 RECTAL PROBLEM

29. **Rectal Prolapse** 189
Arshad Ahmad

30. **Fecal Incontinence** 195
Arshad Ahmad

Section 5 SURGICAL MANAGEMENT URINARY INCONTINENCE

31. **Anti-incontinence Surgery** 203
Manidip Pal

32. **Pubovaginal Sling** 206
Manidip Pal

33. Tension-free Vaginal Tape 212
Manidip Pal

34. Transobturator Tape 216
Manidip Pal

35. Burch Colposuspension: Conventional, Laparoscopic, and Robotic 222
Kiran Ashok

Section 6 VAGINAL WALL SURGERY FOR PELVIC ORGAN PROLAPSE

36. Anterior Colporrhaphy 235
Manidip Pal

37. Posterior Colporrhaphy/Colpoperineorrhaphy 241
Manidip Pal

38. Urethrocele Repair 249
Manidip Pal

39. Culdoplasty 252
Manidip Pal

Section 7 VAULT PROLAPSE

40. Sacrocolpopexy 259
Manidip Pal

41. Sacrospinous Colpopexy 264
Manidip Pal

42. Pectopexy 274
Manidip Pal

43. High Uterosacral Ligament Suspension: Intraperitoneal 278
Manidip Pal

44. Extraperitoneal High Uterosacral Ligament Suspension 282
Manidip Pal

45. Anterior Abdominal Wall Colpopexy 288
Manidip Pal

Section 8 UTERUS PRESERVING SURGERY

46. Manchester–Fothergill's Operation 293
Manidip Pal

47. Abdominal Cervicopexy for Prolapsed Uterus 298
Manidip Pal

48. Le Fort's Colpocleisis 303
Manidip Pal

Section 9 FISTULA

49. Overview of Pelvic Fistula 309
Manidip Pal

50. Vesicovaginal Fistula 313
Manidip Pal

51. Urethrovaginal Fistula 319
Manidip Pal

52. Ureterovaginal Fistula 323
Manidip Pal

53. Rectovaginal Fistula 329
Manidip Pal

54. Radiation Fistula 332
Manidip Pal

Section 10 CONGENITAL ANOMALY RECONSTRUCTIVE SURGERY

55. Scrotalization of Labia 337
Manidip Pal

56. Clitoromegaly 339
Manidip Pal

57. Vaginoplasty 343
Manidip Pal

58. Vaginal Septum 351
Manidip Pal

59. Urogenital Sinus 355
Manidip Pal

60. Uterine Anomaly 365
Manidip Pal

61. Tubal Reconstructive Surgery: Tubotubal Anastomosis 374
Ch Manglem Singh, N Jitendra Singh, Manidip Pal

62. Uterotubal Posterior Cornual Implantation 378
Ch Manglem Singh, N Jitendra Singh, Manidip Pal

Section 11 PREGNANCY

63. Pregnancy with Prolapse 387
Manidip Pal

64. Pregnancy with Urinary Incontinence 389
Manidip Pal

Section 12 MISCELLANEOUS

65. Mesh in Urogynecology 393
Manidip Pal

66. Choosing the Pelvic Organ Prolapse Surgery 397
Manidip Pal

67. Perineal Injuries 399
Manidip Pal

68. Distal Urethral Stenosis 406
Manidip Pal

69. Urethral Diverticulum 412
Manidip Pal

70. Uterine Inversion 418
Manidip Pal

71. Pelvic Organ Prolapse with Cervical Carcinoma 430
Manidip Pal

Index 437

Chapter 4

Physiology of Micturition

Susmita Bhattacharya, L Ranjit Singh

Chapter Outline

- Afferent nerve supply
- Autonomic
- Barrington's nucleus
- Conditioning reflex
- Efferent nerve supply
- Emptying phase
- Hypogastric nerves
- Laplace's law
- Nervi erigentes
- Onuf's nucleus
- Parasympathetic
- Pontine micturition center
- Pudendal nerve
- Somatic
- Storage phase
- Sympathetic
- α-adrenergic receptor
- β-adrenergic receptor

Abstract

Paracentral lobule at the cortex controls the micturition process through hypothalamic bladder center at reticular formation of Pons. This "Pontine Micturition Center" (PMC) receives afferent impulses and sends inhibitory or stimulatory efferent impulses to the lower spinal centers depending upon the surrounding circumstances. Normally, PMC sends inhibitory signals. Micturition is the process of withdrawal of this inhibitory signal. There are two phases of micturition—storage phase/filling phase and emptying phase/voiding phase: Storage (S) is facilitated by sympathetic (S) stimulation. S for S. Detrusor gets inhibited, smooth muscle sphincter gets contracted. Somatic stimulation causes striated sphincter contraction (voluntary control). Emptying is facilitated by parasympathetic stimulation, sympathetic inhibition. Detrusor gets contracted, smooth muscle sphincter gets relaxed causing opening of bladder neck. Somatic innervation causes striated sphincter relaxation (voluntary control).

MICTURITION

Kidneys produce urine and it propels down to urinary bladder via ureters. Urinary bladder is the store house of urine. During storage lower opening of the container (bladder), i.e., the internal urethral meatus remain closed. Once urine store reaches certain level bladder gives signal to evacuate it. Lower opening gets opened; bladder gets squeezed to evacuate the urine.[1]

Innervation of the Bladder[1]

Efferent Motor Nerve Supply

- *Autonomic:*
  - *Parasympathetic (S2 to S4):*
    - Motor to detrusor
    - Inhibitory to internal urethral sphincter
  - *Sympathetic (T10 to L2):*
    - Motor to sphincter
    - Inhibitory to detrusor
- *Somatic nerve (S2, S3, S4)*
- *Pudendal nerves*

The bladder is supplied by the following:

- *The nervi erigentes:* The parasympathetic fibers from the nuclei in the lateral horns of the second, third, and fourth sacral segments of the cord are carried by the nervi erigentes. These fibers end in the local ganglia of the detrusor and circular muscle of the bladder after passing via the hypogastric ganglia, but without relaying here. Moreover, these nerves transport sensory fibers from

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the bladder wall that carry touch, pain, and pressure sensations to the spinal cord via the posterior nerve roots and posterolateral columns before terminating in the post-Rolandic region of the cerebral cortex.[2]

- *Hypogastric nerves:* The first and second lumbar segments of the cord's preganglionic sympathetic fibers are carried by hypogastric nerves that emerge from the hypogastric plexus. It originates in the hypogastric ganglia, which are located behind the bladder, and ends in the internal sphincter and the detrusor muscle. Via the posterior nerve roots, this nerve also transmits the pain sensation associated with spasmodic contraction from the bladder up to at least the ninth thoracic segment of the cord.[2]
- *Pudendal nerve:* The external sphincter, whose closure is controlled voluntarily, is supplied by the pudendal nerve, which emerges from the first, second, third, and fourth sacral segments.[2]

Afferent Sensory Nerve Supply[1]

- *Bladder distension sensation:* Afferent impulses from detrusor travels via pelvic splanchnic nerve and afferent impulses from trigone and bladder neck travels via hypogastric nerve to reach spinal cord. From there via fasciculus gracilis (posterior column) impulses reach cortical centers.
- *Bladder pain sensation:* Afferent impulses travel mainly via hypogastric nerve to reach spinal cord; though few pain fibers are also present in pelvic splanchnic nerve. From there via lateral spinothalamic tract impulses reach cortical centers.
- *Urethral sensation:* Afferent impulses travels via pudendal nerve and then through the dorsal column.
- Afferent fibers axons are consisting of myelinated Aδ fiber responsive to passive distension and unmyelinated C fiber responsive to noxious chemicals and mechanical stimuli.

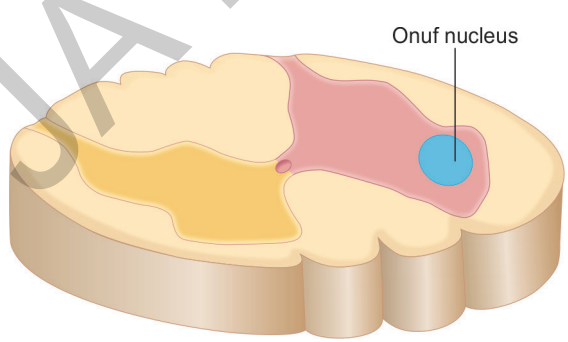

Fig. 1: Spinal cord segment controlling urination.
Source: Laiphrakpam Rashil Simon, Imphal.

Fibers in Spinal Cord to Control Urinary Bladder (Fig. 1)

These originates from the following:

- *Parasympathetic:* From intermediolateral gray horn
- *Sympathetic:* From intermediolateral gray horn
- *Somatic/Pudendal:* From nucleus of Onuf/sacral pudendal nucleus in ventral horn.

Bladder muscle, i.e., the detrusor smooth muscle has primarily β adrenergic receptors. These receptors are under the control of sympathetic and parasympathetic nervous system (autonomic nervous system). Sympathetic stimulation results storing of urine [S (sympathetic) for S (store)]. Parasympathetic stimulation results reverse action, i.e., emptying.

Bladder neck (internal urethral meatus/sphincter) has predominantly α adrenergic receptors. Sympathetic stimulation results closing of the meatus and parasympathetic stimulation results opening, i.e., the reverse action of the bladder **(Fig. 2)**.

INNERVATION OVERVIEW

Cortical Control

In cerebral cortex, mainly paracentral lobule controls the phenomenon through hypothalamic bladder center at reticular formation of Pons. This "pontine micturition center" receives afferent impulses and sends inhibitory or stimulatory efferent impulses to the lower spinal centers depending upon the surrounding circumstances. Normally pontine micturition center discharges inhibitory signals. Micturition is the process of withdrawal of this inhibitory signal.[1]

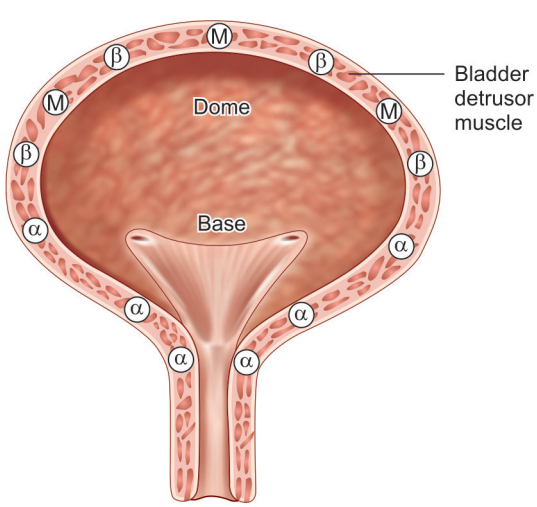

Fig. 2: Receptors of the bladder (α—alpha-adrenoceptors; β—beta-adrenoceptors; M—muscarinic receptors).
Source: Laiphrakpam Rashil Simon, Imphal

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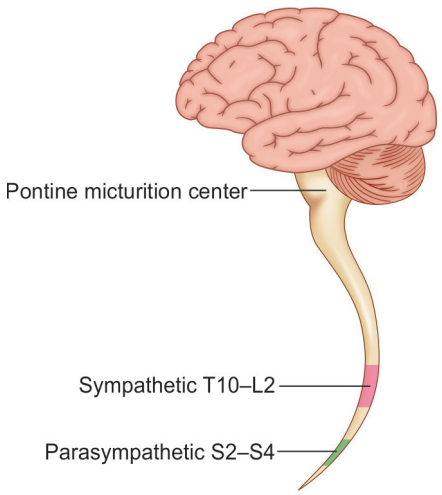

Fig. 3: Central mechanism controlling urination.
Source: Laiphrakpam Rashil Simon, Imphal.

Neural Reflex Arcs to Control Micturition (Fig. 3)

1. Spinal pathways
2. Pontine micturition center
3. Central pathways
4. Urethra to bladder reflexes.

These are discussed here.

1. *Spinal reflexes:* It suppresses urination during the filling phase, activates the striated sphincter (pudendal nerve), inhibits the detrusor muscle, and activates the smooth muscle sphincter through sympathetic nervous system activation.

   Afferent signals are produced by the bladder (when the bladder fills up more), the pelvic floor muscles, the vagina, the rectum, and other body parts.
2. *Pontine micturition center:* As the bladder fills up more, the brainstem's pontine micturition center becomes more active due to an increase in the bladder's afferent neuronal activity. The pontine micturition center (Barrington's nucleus) blocks spinal reflexes, keeping the detrusor muscle relaxed and inhibiting the urinary sphincter.
3. *Central pathways:* Micturition reflex is inhibited through central pathways. As the bladder reaches a specific point of fullness, the filling is consciously felt, and afferent signals are sent to the cortex as a result. The central routes can block the pontine micturition center throughout a certain range of the bladder filling, and the onset of micturition is voluntary.
4. *Urethra to bladder reflexes:* Urine flow or mechanical stretching of the urethra cause a stimulation of bladder contractions which is important function in complete bladder emptying. It provides an explanation for female urge and stress incontinence which is a detrusor contraction results from urine leaks into the urethra brought on by different types of stress.

Phases in Micturition

There are two functional phases in micturition:

1. Storage phase (filling of the bladder)
2. Emptying phase (micturition, voiding).

The *storage* is enabled by:

- Contraction of the striated sphincter (somatic innervation)
- Contraction of the smooth muscle sphincter (sympathetic innervation)
- Inhibition of detrusor activity (sympathetic innervation).

Voiding (micturition) is enabled by:

- Relaxation of the striated sphincter (somatic innervation)
- Relaxation of the smooth muscle sphincter and opening of the bladder neck (sympathetic innervation)
- Detrusor contraction (parasympathetic innervation).

Filling and Storage Phase

During this time detrusor pressure does not increase, if increases, then also not more than 15 cm of "H_2O". Sympathetic stimulation of β adrenergic receptors leads to relaxation of bladder wall detrusor muscle as well as sympathetic stimulation of α/α1 adrenergic receptors at bladder neck and internal urethral sphincter leads to constriction/contraction of the urethra. The external urethral sphincter also remains closed with the help of stimulus from pudendal nerve. Another mechanism that helps in continence is urethral mucosal coaptation. The urothelium with its connective tissue thrown into multiple folds within the urethral lumen. In this subepithelial layer, there is a rich capillary network which helps in mucosal approximation also known as coaptation. Now this water sealed closed quiescent bladder accumulate urine to some extent which is known as bladder capacity **(Fig. 4)**.

Initiation phase is the transition phase from the storage to the starting of voiding. Once a socially acceptable environment available the cortical inhibitory impulses stopped. Now the pelvic floor muscles relaxes, intrinsic striated muscle relaxes, fall in intraurethral pressure occurs. Next parasympathetic stimulation results detrusor contraction, rise of intravesical pressure, pulling of bladder neck to open it, shortening of urethra. Simultaneously inhibition of sympathetic impulses also occur which also

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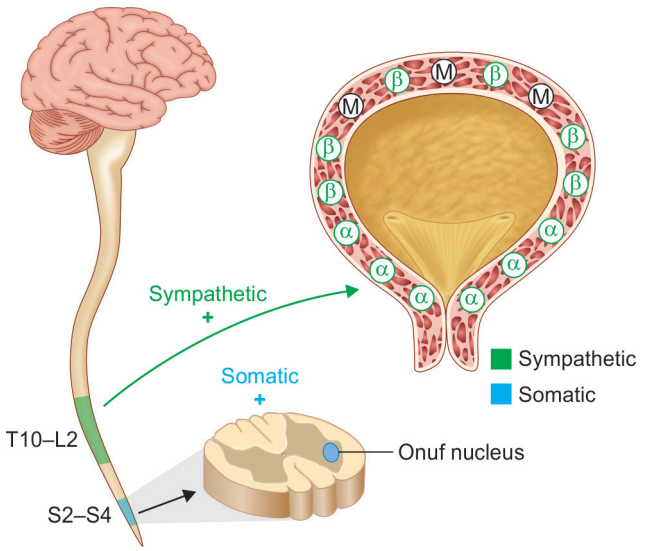

Fig. 4: Urine storage.

helps in detrusor contraction and urethral relaxation. Once the intravesical pressure equates intraurethral pressure urine flow starts.

Emptying Phase

Laplace's law: The wall tension of bladder depends on its:

- Internal pressure (Pves)
- Radius of the bladder (r)
- Bladder wall thickness (d)

$$\text{Wall tension} = (\text{Pves} \times r) / (2d)$$

The wall tension increases during urinary retention, although the bladder pressure remains relatively constant.

The wall tension increases because of the:

- Enlarged radius (r)
- Decrease in wall thickness (d).

A rhythmical contraction wave is created when the bladder wall distends as urine gradually builds up to a volume of around 200 cc under the inhibitory control of the local center in the sacral cord. Until this is involuntary. When a volume of 400 cc at about 15–18 cm of "H_2O" pressure promotes vesical contractions and achieves a threshold intensity, this subconscious process reaches the level of consciousness. The parasympathetic fibers of the nervi erigentes connect the posterior nerve roots of the S2-S4 segments to the distended wall's impulses, which then ascends through the cord's posterolateral columns to the sensory cortex in the post-Rolandic region. At random intervals, a vague pain in the anterior region of the perineum is felt radiating along the urethra. If the bladder expands more, then because of irritation of nerves of surrounding structures, there is localized pain in the hypogastrium which also gets referred down to the front of the thigh in its upper two-thirds.[2]

When it is convenient to empty the bladder, the excitatory signal descends from the motor cortex via the lateral columns to the lumbosacral center, increasing detrusor contractions while efferent impulses go down the nervi erigentes, therefore reciprocally relaxing the external sphincter. Pudendal nerve inhibition, that is voluntary, relaxes all perineal muscles. It permits a few trace amounts of urine to flow into the posterior upper urethra, causing the bladder wall to tighten reflexively more forcefully. Increasing the intra-abdominal pressure also helps. The perineal musculature regains its previous tone when the sphincter muscle contracts, forcing the final few drops out of the posterior urethra. [2]

Inhibitory impulses from the post-Rolandic area prevent vesical contractions from occurring when it is not practical to empty the bladder. The detrusor muscle relaxes as a result of the sympathetic fibers of the hypogastric nerves' inhibitory influence, which also causes the internal sphincter to close strongly. This allows the bladder to hold more urine.

The voluntary external sphincter, the perineal musculature, and adduction of the thigh muscles are needed to help prevent further urine accumulating. The pain becomes severe when the intravenous pressure exceeds 100 cm of "H_2O," and urine begins to force its way past the sphincters and drip slowly down the urethra until the effects of inhibition on the stretched musculature wear off. Later with the reinstatement of the vesical contractions, the rate of urine flow rises. The external sphincter can be forced to contract voluntarily, and after a certain amount of bladder evacuation, inhibitory signals from the higher centers cause the detrusor muscle to relax, stopping the flow. After the bladder empties, and the hypogastric pain subsides, but a vague nagging ache lasts for a few minutes.

The cerebral cortex, the reticular activating system (RAS), the locus ceruleus (LC), the hypothalamus, the pontine micturition center (PMC), the spinal cord, and the bladder are among the interrelated anatomic regions involved in arousal to the sense of a full or contracting bladder. The RAS regulates sleep depth, the LC regulates arousal, and the PMC starts a detrusor contraction on demand. Many neurotransmitters, including norepinephrine, serotonin, and antidiuretic hormone (ADH) are implicated **(Fig. 5)**.[1]

Conditioning of Reflexes

It takes time and education, which is typically acquired between the ages of 2.5 and 3 years old, for a youngster to develop this set of conditioned reflexes. A more rudimentary reflex evacuation with a lower volume that happens frequently occurs in smaller children. During sleep, the bladder may

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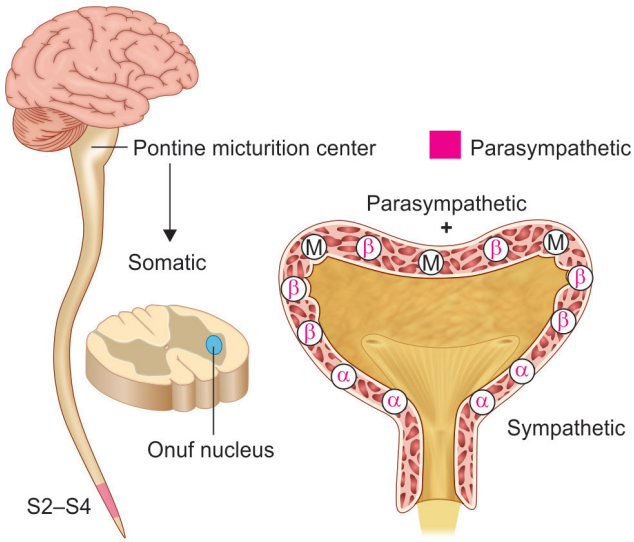

Fig. 5: Urine evacuation.

revert to its primordial irritation and empty unintentionally as the higher control is reduced. Larger volumes can be accommodated by the bladder with little discomfort, which is more typical in the case of women who often restrict micturition under unfavorable conditions due to social inhibition. On the other hand, "bladder consciousness" (concerning micturition) can result in vesical contractions and the willed evacuation of much less urine than is typically necessary to provide sufficient stimulation for evacuation.

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Urogynecology & Pelvic Reconstructive Surgery

Salient Features

- This is a textbook for MCh and Post-Doctoral Fellowship in Urogynecology & Pelvic Reconstructive Surgery, reference book for MS (Obstetrics and Gynecology) and practical book for Urogynecologists, Gynecologists, Vaginal Surgeons and Pelvic Reconstructive Surgeons
- The book encompasses two arenas—urogynecology and reconstructive pelvic surgery—together. Different operations and medical treatment of urinary incontinence, various surgeries for treatment and prevention of pelvic organ prolapse, reconstruction of different congenital anomalies of pelvic organs—all are described
- Minute detailing of all operative steps along with their pictorial representation to understand the respective operations in toto
- Labeling and legends of drawings and photographs are written in such a way that you need not to refer to the text again and again while you are going through the pictures
- Written in lucid language so that readers can understand the facts easily
- Basic science comprises anatomy, physiology, embryology along with clinical examination and their interpretations are described crystal clear
- Few new chapters are added, e.g. biomarkers of overactive bladder, underactive bladder, urethral diverticulum, pectopexy, uterine inversion, POP with cervical carcinoma, etc.
- Important points are incorporated in the box for ready reference.

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