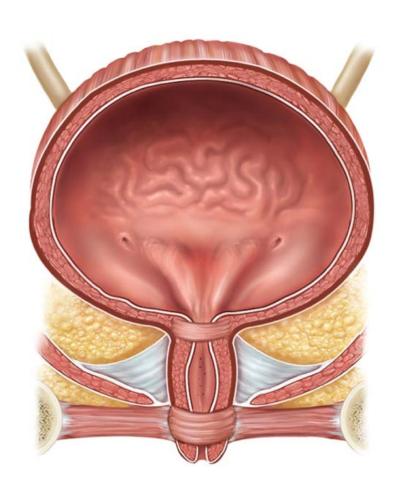
Chapter 23
Micturition and Renal Insufficiency



Voiding Urine

- Between acts of urination, the bladder is filling.
 - detrusor muscle relaxes
 - urethral sphincters are tightly closed
 - accomplished by sympathetic pathway from upper lumbar spinal cord
 - postganglionic fibers travel through the hypogastric nerve to the detrusor muscle (*relax*) and internal urethral sphincter (*excite*)
 - somatic motor fibers from upper sacral spinal cord through pudendal nerve stimulates the external sphincter to contract sphincter and give us voluntary control
- micturition is the act of urinating
- micturition reflex spinal reflex that <u>partly controls</u> urination

Voiding Urine – Micturition Reflex

- involuntary control (steps 1 4)
 - filling of the bladder to about 200 mL excites stretch receptors in the bladder wall
 - send sensory signals through fibers in pelvic nerve to sacral spinal cord (S2 or S3)
 - motor signals travel back from the spinal cord to the bladder by way of motor fibers in pelvic nerve and parasympathetic ganglion in bladder wall
 - excites detrusor muscle and relaxes internal urethral sphincter
 - results in emptying bladder
 - if there was no voluntary control over urination, this reflex would be the only means of control but we know we are able to exert control over urination (unlike new born infants!)

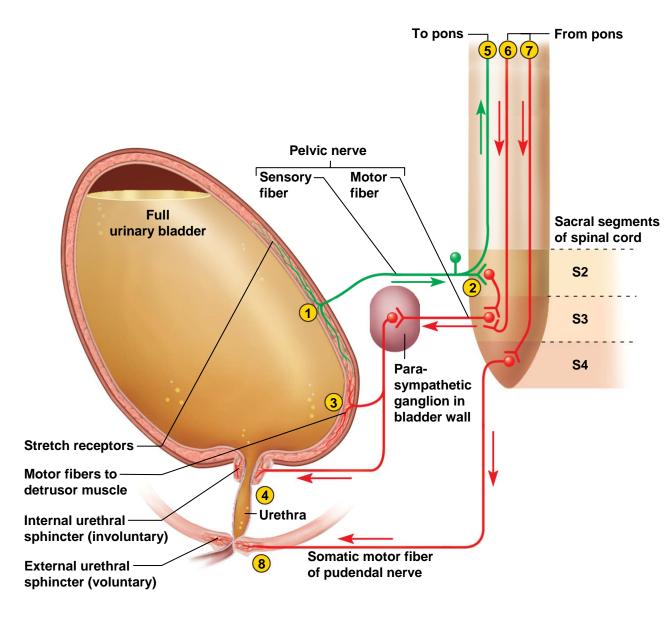
Voiding Urine – Micturition Reflex

- voluntary control (steps 5 8)
 - micturition center <u>nucleus in the pons</u> that receives some input from bladder stretch receptors that ascends the spinal cord
 - nucleus integrates information about bladder tension with information from other brain centers
 - urination can be prompted by fear
 - inhibited by knowledge that the circumstances are inappropriate for urination
 - fibers from micturition center descend the spinal cord
 - through reticulospinal tracts
 - some fibers inhibit sympathetic fibers than normally keep internal urethral sphincter contracted
 - others fibers descend farther to sacral spinal cord
 - excite parasympathetic neurons that stimulate the detrusor to contract and relax the internal urethral sphincter
 - initial detrusor contraction raises pressure in bladder, stimulate stretch receptors, bringing about more forceful contraction
 - external urethral sphincter receives nerve fibers from cerebral cortex by way of corticospinal tract
 - inhibit somatic motor neurons that normally keep that sphincter constricted

Voiding Urine – Micturition Reflex

- urge to urinate usually arises at an inconvenient time
 - one must suppress it
 - stretch receptors fatigue and stop firing
- as bladder tension increases
 - signals return with increasing frequency and persistence
- there are times when the bladder is not full enough to trigger the micturition reflex but one wishes to 'go' anyway
 - Valsalva maneuver used to compress bladder
 - excites stretch receptors early getting the reflex started

Neural Control of Micturition

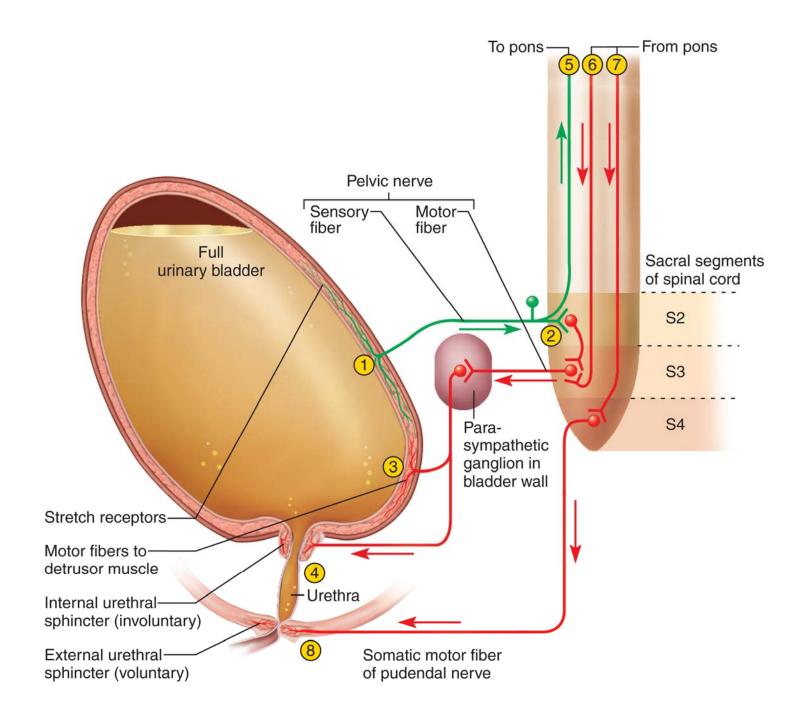


Involuntary micturition reflex

- 1 Stretch receptors detect filling of bladder, transmit afferent signals to spinal cord.
- 2 Signals return to bladder from spinal cord segments S2 and S3 via parasympathetic fibers in pelvic nerve.
- 3 Efferent signals excite detrusor muscle.
- 4 Efferent signals relax internal urethral sphincter. Urine is involuntarily voided if not inhibited by brain.

Voluntary control

- 5 For voluntary control, micturition center in pons receives signals from stretch receptors.
- 6 If it is timely to urinate, pons returns signals to spinal interneurons that excite detrusor and relax internal urethral sphincter. Urine is voided.
- 7 If it is untimely to urinate, signals from pons excite spinal interneurons that keep external urethral sphincter contracted. Urine is retained in bladder.
- 8 If it is timely to urinate, signals from pons cease and external urethral sphincter relaxes. Urine is voided.



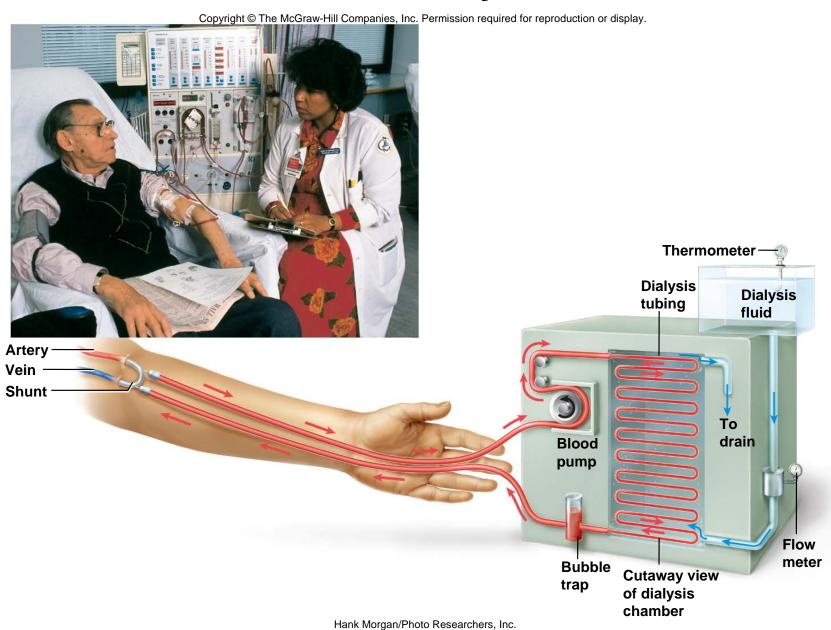
Renal Insufficiency & Hemodialysis

- renal insufficiency a state in which the kidneys <u>cannot maintain</u> <u>homeostasis</u> due to extensive destruction of their nephrons
- causes of nephron destruction
 - hypertension, chronic kidney infections, trauma, prolonged ischemia and hypoxia, poisoning by heavy metals or solvents, blockage of renal tubules in transfusion reaction, atherosclerosis, or glomerulonephritis
- nephrons can regenerate and restore kidney function after short-term injuries
 - others nephrons hypertrophy to compensate for lost kidney function
- can survive with one-third of one kidney
- when 75% of nephrons are lost and urine output of 30 mL/hr is insufficient (normal 50 -60 mL/hr) to maintain homeostasis
 - causes azotemia, acidosis, and uremia develops, also anemia

Hemodialysis

- procedure for artificially clearing wastes from the blood
- wastes leave bloodstream and enter the dialysis fluid as blood flows through a semipermeable cellophane tube
- also removes excess body water

Hemodialysis



Kidney Stones

- renal calculus (kidney stone) hard granule of calcium phosphate, calcium oxalate, uric acid, and/or a magnesium salt called struvite
- form in the renal pelvis
- usually small enough to pass unnoticed in the urine flow
 - large stones might block renal pelvis or ureter and can cause pressure build up in kidney which destroys nephrons
 - passage of large jagged stones is excruciatingly painful and may damage ureter causing hematuria
- causes include hypercalcemia, dehydration, pH imbalances, frequent urinary tract infections, or enlarged prostate gland causing urine retention
- treatment includes stone dissolving drugs, often surgery, or lithotripsy –nonsurgical technique that pulverizes stones with ultrasound

Urinary Tract Infection (UTI)

- cystitis infection of the urinary bladder
 - especially common in females due to short urethra
 - frequently triggered by sexual intercourse
 - can spread up the ureter causing pyelitis
- pyelitis infection of the renal pelvis
- pyelonephritis infection that reaches the cortex and the nephrons
 - can result from blood-borne bacteria