📖 **THE HIGHEST COMPLETE SCI LEVEL THAT CAN LIVE INDEPENDENTLY WITHOUT THE AID OF AN ATTENDANT IS A C6 COMPLETE TETRAPLEGIA.**

- This patient would have to be *extremely motivated*
- Feeding is accomplished with a universal cuff for utensils
- Transfers require stabilization of elbow extension with forces transmitted from shoulder musculature through a closed kinetic chain
- Bowel care is performed using a suppository insertion wand or other apparatus for digital stimulation
- Outcome studies of a subset of patients with motor and sensory complete C6 SCI revealed the following percentage of patients were independent for key self-care tasks:

  Feeding—16%
  Upper body dressing—13%
  Lower body dressing—3%
  Grooming—19%
  Bathing—9%
  Bowel Care—3%
  Transfers—6%
  Wheelchair propulsion—88%

📖 C7 level is the *usual* level for achieving independence.

■ MEDICAL COMPLICATIONS OF SCI

📖

Important Levels to Remember:
T6 and above: Individuals with SCI are considered to be at risk for
1. Autonomic Dysreflexia
2. Orthostatic Hypotension

T8: If lesion above T8, patient cannot regulate and maintain normal body temperature (Note: an easy way to remember this level is to spell the word ***temp eight ture.***)
Central temperature regulation in the brain is located in the hypothalamus.

ORTHOSTATIC HYPOTENSION (see Table 7-4) (Corbett, 1971)

State of transient reflex depression

Cause: Lack of sympathetic outflow, triggered by tilt of patient > 60 degrees

Lesion T6 or above
T1–L2 responsible for:
Tachycardia, vasoconstriction and increased arterial pressure
Heart and blood vessels supplied by T1–T7

Mechanism

- Upright position causes decrease in blood pressure (BP)
- Carotid body baroreceptors sense decrease in BP, which would usually increase sympathetic outflow.

- However brainstem is unable to send a message through the SC to cause sympathetic outflow and allow vasoconstriction of splanchnic bed to increase BP

Resultant Symptoms

1. Hypotension—loss of sympathetic tone (decreased systemic venous resistance, dilation of venous vessels) (decreased preload to the heart)
2. 🕮 Tachycardia—Carotid body responds to hypotension, no increase in sympathetic outflow, however, they can still inhibit parasympathetics, but the increase in heart rate is not sufficient enough to counterbalance decrease BP
3. Patient can lose consciousness

🕮 Treatment

1. Reposition—Trendelenburg/daily tilt table/recliner wheelchair
2. Elastic Stocking/Abdominal Binder/Ace wrap LE
3. Add Salt/Meds:
   Salt Tablets 1 gram QID
   Florinef® (mineralocorticoid): 0.05–0.1 mg QD
   Ephedrine (alpha agonist): 20–30 mg QD–QID
   Use caution: The same patient is at risk for autonomic dysreflexia
4. Fluid resuscitation: monitor for neurogenic pulmonary edema
5. Orthostasis lessens with time due to the development of spinal postural reflexes. This causes vasoconstriction due to improved autoregulation of cerebrovascular circulation in the presence of perfusion pressure

🕮 AUTONOMIC DYSREFLEXIA (see Table 7-4) (Braddom, 1991) (Lindan, 1980)

Onset: After spinal shock, usually within first 6 months–1 year

Incidence: 48%–85%

Cause: Noxious stimulus below the level of the lesion causing massive imbalanced sympathetic discharge, i.e., too much sympathetic outflow
Most commonly caused by distended, full bladder

Lesion: SCI patients with lesions T6 or above (complete lesions)

Mechanism: Syndrome of massive imbalanced reflex sympathetic discharge in patients with SCI above the splanchnic outflow
This is secondary to the loss of descending sympathetic control and hypersensitivity of receptors below the level of the lesion

Potential Symptoms: Noxious stimuli—Increases sympathetic reflex spinal release
Regional vasoconstriction (especially GI tract)
Increases peripheral vascular resistance—increases cardiac output, increases BP
Carotid body responds to HTN causing reflex bradycardia by the dorsal motor nucleus of the vagus nerve

Symptoms: Headache, Flushing
Piloerection
Sweating above level of SCI
Blurry vision (pupillary dilation)
Nasal Congestion

Note: The brainstem is unable to send message through SCI to decrease sympathetic outflow and allow vasodilation of splanchnic bed to decrease BP

Most common causes:

- Bladder—blocked catheter
- Bowel—fecal impaction
- Pressure ulcers
- Ingrown toenails
- Urinary tract infections
- Bladder stones
- Gastric ulcers
- Labor
- Abdominal emergency
- Fractures
- Orgasm
- Epididymitis
- Cholecystitis

📖 **Treatment:**

- Sit patient up
- Remove TEDS/Abdominal binder
- Identify and remove noxious stimulus
- Nitroglycerine—to control BP—1/150 sublingual or topical paste, which can be removed once noxious stimulus corrected
- Procardia®: 10 mg chew and swallow
- Hydralazine: 10–20 mg IM/IV
- Clonidine: 0.3–0.4 mg
- ICU - Nipride

Prevent Recurrence:

- Dibenzyline: 20–40 mg/day alpha blocker
- Minipress®: 0.5–1 TID alpha blocker
- Clonidine: 0.2 mg BID

Potential Complications of Autonomic Dysreflexia: If hypertensive episodes are not treated, complications can lead to: Retinal Hemorrhage, CVA, SAH, Seizure, Death
Autonomic Dysreflexia predisposes patient to atrial fibrillation by altering normal pattern of repolarization of the atria, making the heart susceptible to reentrant-type arrhythmias.

TABLE 7-4 Orthostatic Hypertension vs. Autonomic Dysreflexia

🕮 **Orthostatic Hypotension**	🕮 **Autonomic Dysreflexia (AD)**
Trigger: Tilt patient > 60 degrees	**Trigger:** Noxious stimulus: especially full bladder below level of lesion
Due to: Lack of sympathetic outflow **Lesion:** T6 or above	**Due to:** Too much sympathetic outflow, loss of descending control, hypersensitivity **Onset:** status post spinal shock usually within first six months **Lesion:** T6 or above
Symptoms: Hypotension due to being positioned in the upright position 🕮 Tachycardia: carotid body responds to hypotension Patient loses consciousness	**Symptoms:** Hypertension due to noxious stimulus. Bradycardia: carotid body responds to hypertension HA flushing Piloerection Sweating above level SCI Blurred vision, pupillary dilation Nasal congestion
Note: Upright position causes decrease in BP, carotid body Baroreceptors sense decrease BP, but brainstem is unable to send message through SC to cause sympathetic outflow and cause vasoconstriction of splanchnic bed to increase BP	**Note:** Noxious stimulus causes massive sympathetic output Carotid body senses increased BP, but brainstem is unable to send message through SC to cause decreased sympathetic outflow and allow for vasodilation of splanchnic bed to bring BP down
Tx: 1. Reposition: Trendelenburg 2. Elastic stockings 3. Abdominal binders 4. Increase salt 5. Fluid resuscitation: monitor neurogenic pulmonary edema **Meds:** Florinef® (Mineralocorticoid) Salt Tablets Ephedrine (Alpha Agonist)	**Tx:** 1. Sit patient up 2. Remove noxious stimulus (look for bladder distension, fecal impaction, etc.) 3. Treat hypertension • Consider temporary treatment with nitrates (transderm), hydralazine (parenteral), morphine (parenteral), captopril (oral), labetalol (oral or IV) • Decide need for intensive care and IV agents such as nitroglycerine, nitroprusside, spinal anesthesia It is estimated that 48%–85% of patients with high level SCI have symptoms of autonomic dysreflexia. Can lead to: 1. Retinal Hemorrhage 2. CVA 3. SAH, seizure, death AD may predispose patient to A. fib. by altering the normal. pattern of repolarization of the atria, making the heart susceptible to reentrant-type arrhythmias.

BLADDER DYSFUNCTION

Neuroanatomy and Neurophysiology of Voiding

Central Pathways

- *Corticopontine Mesencephalic Nuclei*–Frontal Lobe
  Inhibits parasympathetic sacral micturition center
  Allows bladder storage
- *Pontine Mesencephalic*
  Coordinates bladder contraction and opening of sphincter
- 📖 *Pelvic and Pudendal Nuclei*–Sacral Micturition
  Integrates stimuli from cephalic centers
  Mediates the parasympathetic S2–S4 sacral micturition reflex
- *Motor Cortex to Pudendal Nucleus*
  Voluntary control (contraction/inhibition) of the external urethral sphincter

Peripheral Pathways (Figure 7–25)

- 📖 *Parasympathetic Efferents*–S2–S4
  Travel through the pelvic nerve to parasympathetic receptors
  Allows contraction of the bladder and emptying
- 📖 *Sympathetic Efferents*–T11–L2
  Travel through hypogastric plexi to sympathetic receptors
  (Alpha 1 + Beta 2 adrenergics)
  Urine Storage
- *Somatic Efferents*–S2–S4
  Travel through pudendal nerve to innervate striated muscle of external urethral sphincter
  Prevents urine leakage or emptying
- *Afferent Fiber*
  Travel through pudendal and pelvic nerve through the hypogastric plexi to thoracolumbar SC
  Origin — detrusor muscle stretch receptors
  external anal and urethral sphincter
  perineum and genitalia
  When bladder becomes distended, afferent nerve becomes activated for parasympathetic stimulation, resulting in emptying of bladder

📖 Neurologic Innervation of the Bladder (Bladder Receptors) (Figure 7–24)

- Cholinergic Muscarinic–M2
  Located in the bladder wall, trigone, bladder neck, urethra
- Beta 2 Adrenergic
  Concentrated in the body of the bladder, neck
- Alpha adrenergic
  Located on the base of the bladder (neck and proximal urethra)
  (Note: Bladder wall does not have baroreceptors)

Note:
Alpha Adrenergic receptors respond to the appearance of norepinephrine with contraction

Beta adrenergic receptors respond to the appearance of norepinephrine with relaxation

Urethral Sphincter

Internal Sphincter:
- Innervated by T11–T12 sympathetic nerve
- Contracts sphincter for storage
- Smooth muscle

External Sphincter
- Innervated by S2–S4 pudendal nerve
- Prevents leakage or emptying
- Skeletal muscle, voluntary control

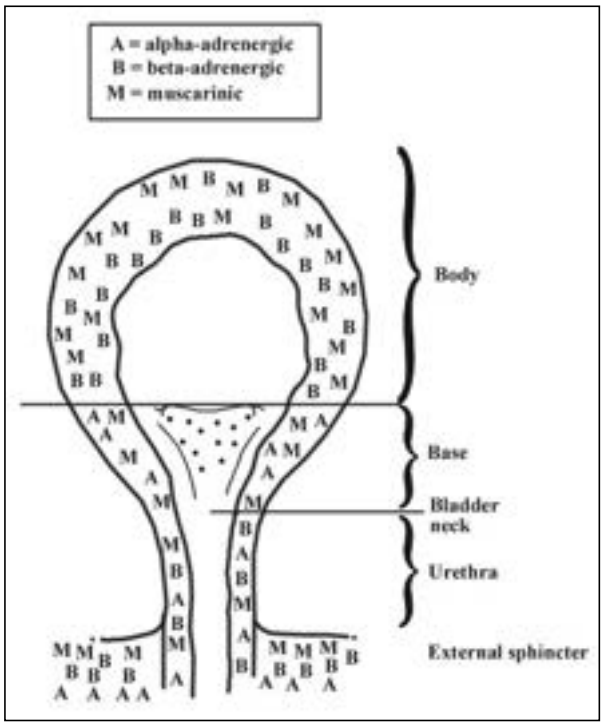

FIGURE 7–24. Bladder and proximal urethra distribution of autonomic receptors.

Storage

Sympathetic (Figure 7–25)
encouraged during fight, flight

T11–L2 sympathetic efferents
- Travel through the hypogastric nerve
- Causes the sphincter to contract and body to relax
- Urine is stored

📖 *Alpha1 Receptors Adrenergic*
- NE causes contraction of neck of bladder and prevents leakage
- Closes internal urethral sphincter and detrusor outlet, promoting storage

📖 *B2 Receptors Adrenergic*
- Located in body of bladder
- Activation causes relaxation of body of bladder to allow expansion
- Inhibitory when activated

Emptying

Parasympathetic (Figure 7–25)
encouraged during relaxation

Muscarinic (M2) cholinergic receptors are located in:
- The bladder wall
- Trigone
- Bladder Neck
- Urethra

Stimulation of pelvic nerve (parasympathetic)
- Allows contraction of bladder + therefore, emptying!

B2 Receptors Adrenergic
- Relaxation of the bladder neck on the initiation of voiding

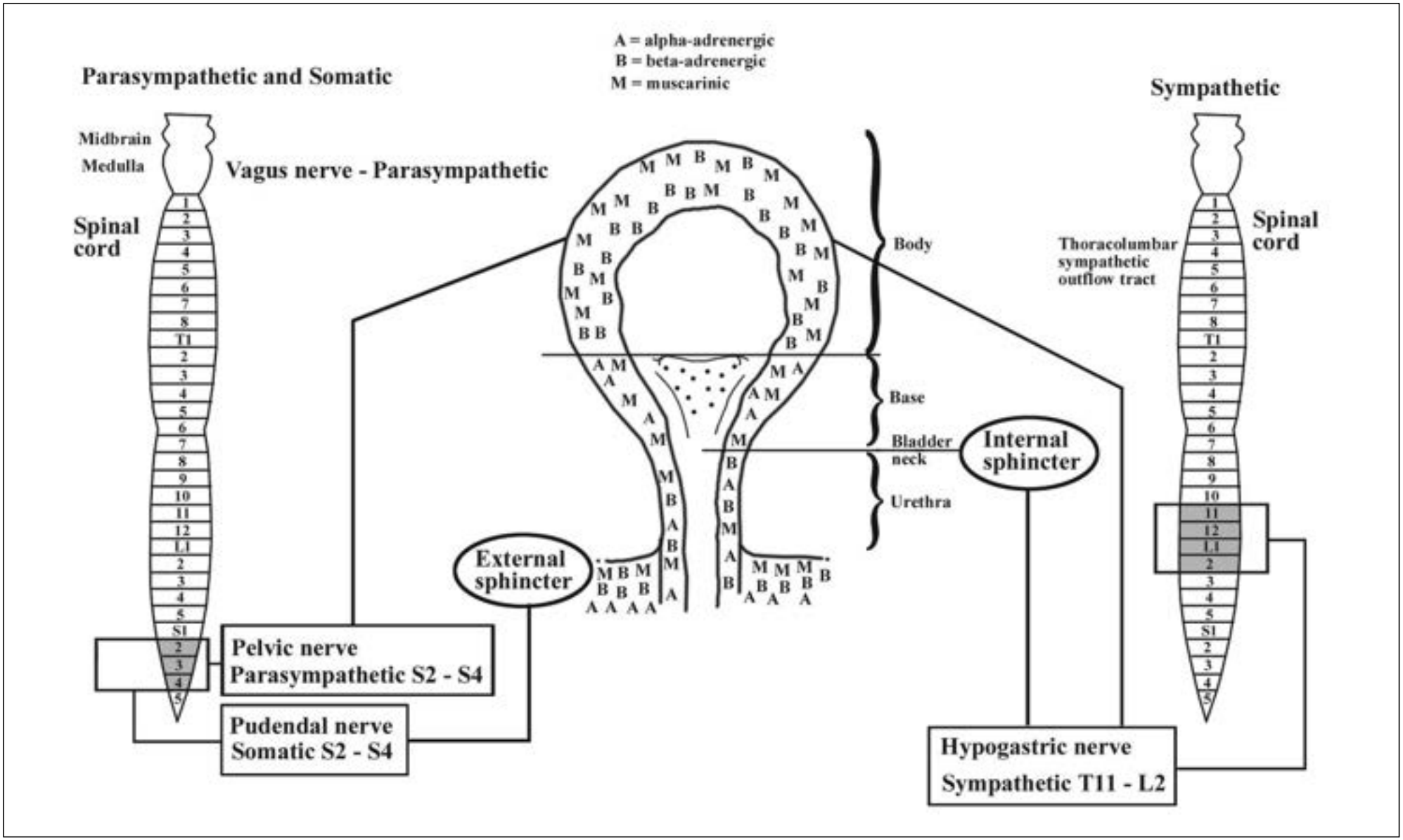

FIGURE 7–25. Neurologic innervation of the bladder.

Evaluation of Urinary Function: Cystometrogram and Pelvic Floor EMG

During cystometry: sensation, capacity, and the presence of involuntary detrusor activity are evaluated. A typical urodynamic study is depicted in Figure 7–26

- Sensations evaluated include:
  First sensation of bladder filling—occurs at approximately 50% of bladder capacity
  First urge to void—proprioceptive sensation
  Strong urge to void—proprioceptive sensation
- Accepted normal bladder capacity is 300–600ml
  Functional bladder capacity = voided volume + residual urine volume

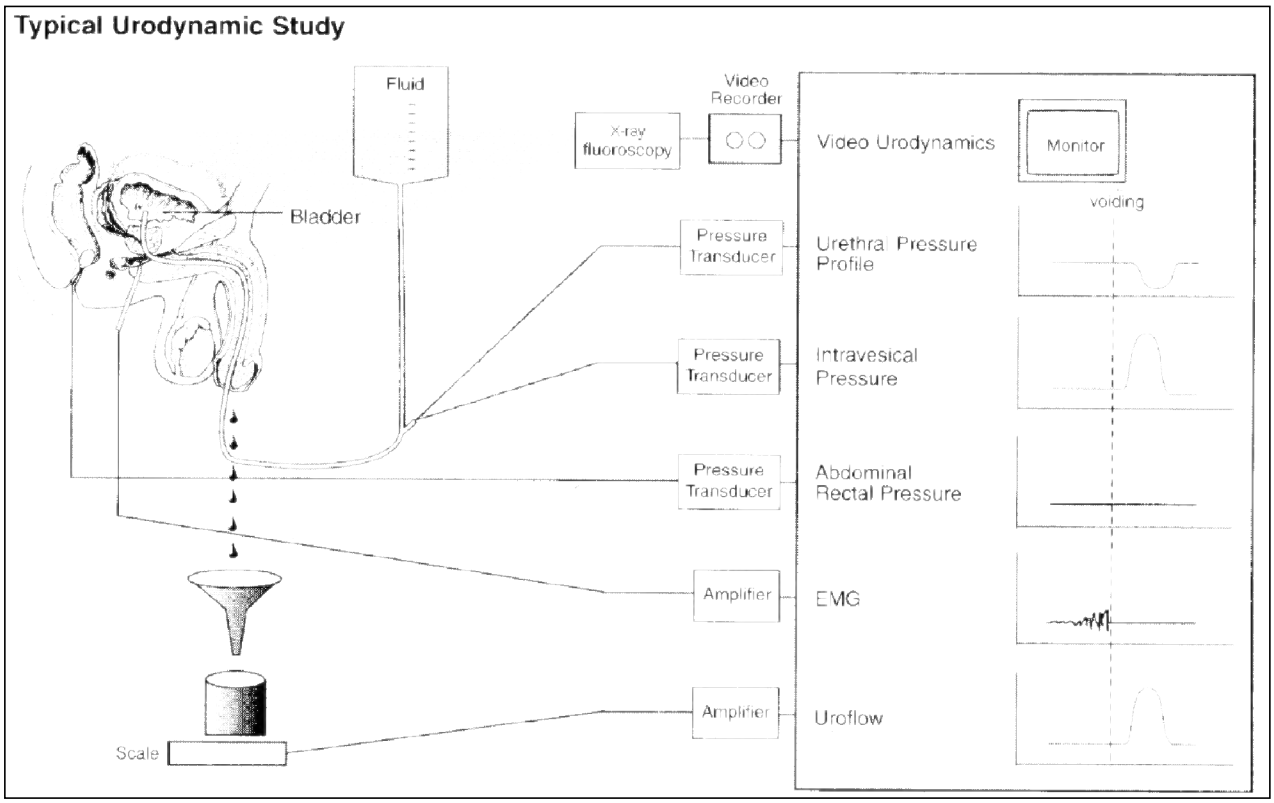

FIGURE 7–26. Instrumentation for urodynamic studies is not standardized. The illustration above uses radio-opaque fluid. Some physicians, however, prefer to use carbon dioxide. Normal bladder function can be divided into storage and voiding phases. The first sensation of bladder filling is between 100 cc and 200 cc. The patient experiences bladder fullness between 300 cc and 400 cc and the sense of urgency between 400 cc and 500 cc. Intravesical pressure does not increase significantly during the storage phase due to the vascoelasticity of the vesical wall. During the voiding phase, sphincter activity stops and the bladder contracts. During normal voiding, the EMG signal will be silent, intravesical pressure will increase, and urethral pressure will decrease. Fluoroscopy will qualitatively assess bladder contraction and document any potential vesioureteral reflux. (From Nesathurai S. The Rehabilitation of People With Spinal Cord Injury: A House Officers Guide. © Boston Medical Center for the New England Regional Spinal Cord Injury Center. Boston, MA: Arbuckle Academic Publishers, with permission).

Normal Detrusor Contraction
Detrusor Pressure cm H_2O and
EMG Pelvic Floor (Fig. 7-27)

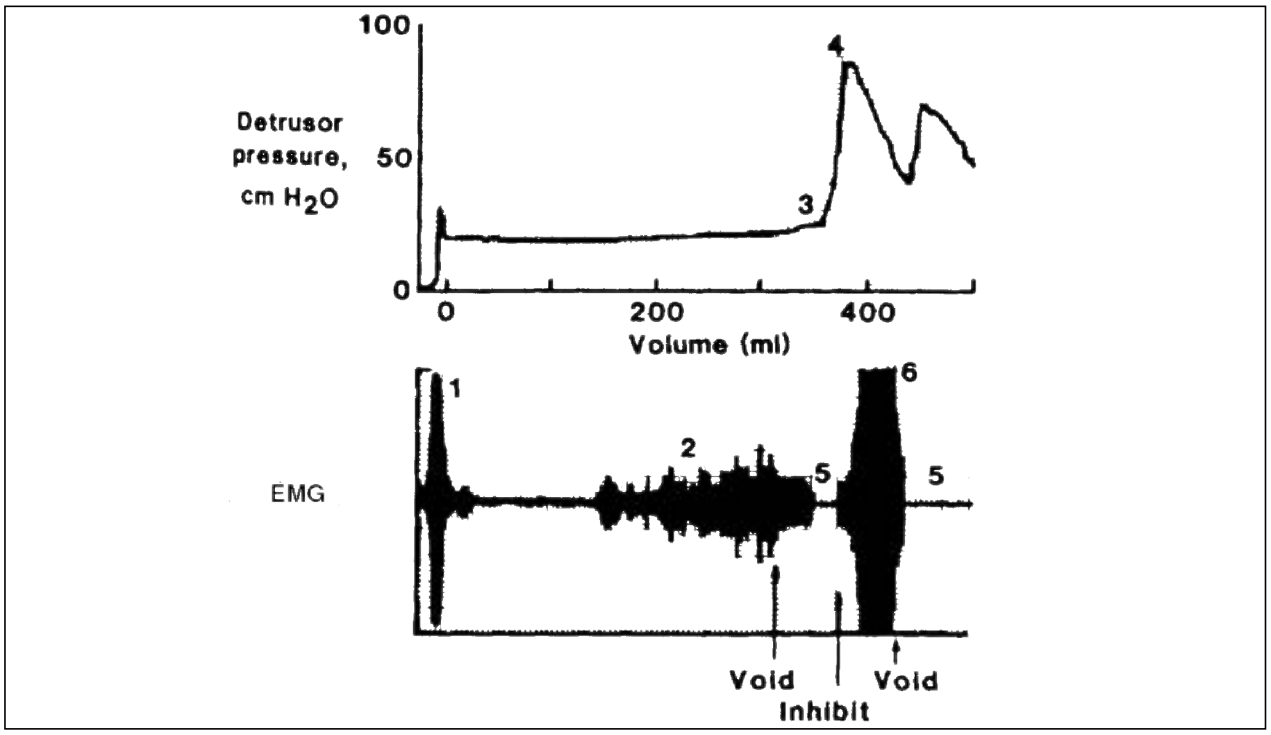

FIGURE 7–27. Normal Cystometrogram/Pelvic Floor EMG. 1. Bulbocavernosus reflex. 2. Contraction of pelvic floor muscles during later phase of filling (progressively increasing electrical activity) 3. Functional bladder capacity. 4. Detrusor contraction that occurs during voiding. 5. Electrical silence (abrupt) which occurs during voiding. 6. Electrical activity of pelvic floor muscles that occurs during voluntary inhibition.

Genitourinary Function and Management

During the acute period after injury, the bladder usually presents *areflexic*, i.e., spinal shock phase.

May initially manage the bladder with indwelling catheter, while intravenous body fluids are administered. An intermittent catheterization program should be established soon after, with fluid restriction of approx. 100 cc/hr.

📖 Volumes should always be monitored and maintained below 400–500 cc to avoid:

- Vesicoureteral reflux—caused by bladder hypertrophy and loss of the vesicoureteral angle (see previous page). This is normally prevented by the anatomy of the ureter, which penetrates the bladder obliquely through the trigone and courses several centimeters into the bladder epithelium.
- Overflow incontinence
- Hydro-ureter

Urodynamic studies should be performed to assess:

The bladder neck, the external sphincter, and the detrusor

Note: Bladder dysfunction is closely related to the level of injury, i.e., lower motor neuron vs. upper motor neuron.

TABLE 7-5 Lower Motor Neuron Bladder vs. Upper Motor Neuron Bladder

LMN Bladder **Failure to Empty (Fig. 7-28)**	**UMN Bladder** **Failure to Store (Fig. 7-29)**
Causes: • Spinal Shock: when reflex arc is not functioning due to initial trauma • Conus Medullaris Syndrome • Cauda Equina Syndrome • Tabes Dorsalis, Pernicious Anemia, Syringomyelia • Multiple Sclerosis	**Causes:** • SCI: when reflex arc returns after initial trauma to cord passes • CVA • Multiple Sclerosis
Lesion: Complete destruction of Sacral Micturition Center (S2–S4) at S2 or below Lesion involving exclusively the peripheral innervation of the bladder	**LESION:** Above Sacral Micturition Center (above S2)
Can Result in:	**Can Result in:**
Big Hypotonic Bladder (flaccid, areflexic bladder),*Tight Competent Sphincter* Results in: **Failure to Empty** TX: • Intermittent Catheter • Crede maneuver (suprapubic pressure) • Valsalva maneuver • Drugs to induce urination Urecholine: stimulate cholinergic receptors Minipress®: block alpha adrenergic receptors Dibenzyline: block alpha adrenergic receptors Hytrin®: block alpha adrenergic receptors Cardura®: block alpha adrenergic receptors	*Small Hyperreflexic, Overactive, Little Bladder* Results in: **Failure to Store** (Incontinence) TX: Ditropan: direct smooth muscle relaxer Pro-Banthine®, Detrol®: anticholinergic Tofranil®, ephedrine: stimulates alpha, beta receptors to allow storage

LMN Bladder (Figure 7–28)
Failure To Empty
Cystometrogram and EMG

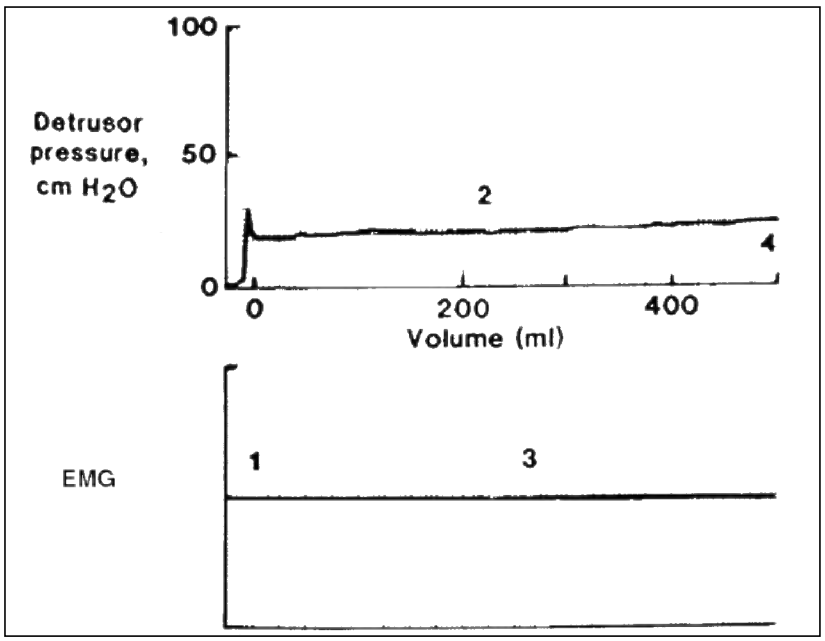

FIGURE 7–28. Cystometrogram and EMG in a patient with complete lower motor neuron bladder dysfunction. 1. Absent bulbocavernosus reflex. 2. Major detrusor contractions are absent. 3. No pelvic floor muscle activity (external urethral sphincter). 4. Large bladder capacity.

Note:
- Internal sphincter may have increased tension and prevent voiding

These patients usually only void by:
- *Overflow voiding* when bladder can no longer expand

UMN Bladder (Figure 7–29)
Failure to Store
Cystometrogram and EMG

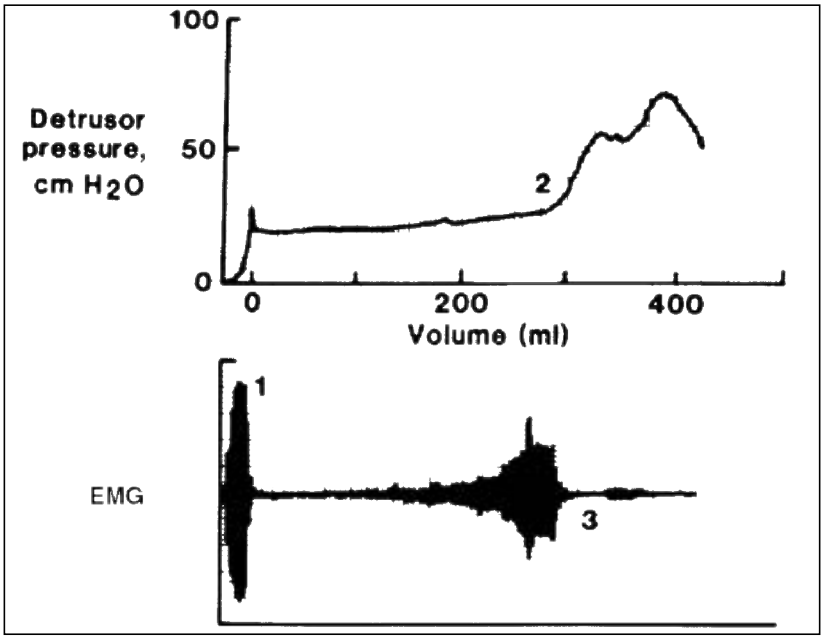

FIGURE 7–29. Cystometrogram and EMG in a patient with complete uninhibited neurogenic bladder dysfunction. 1. Brisk bulbocavernosus reflex. 2. Spontaneous detrusor contraction at reduced bladder capacity. 3. Silence of pelvic floor muscles (external urethral sphincter).

Due to upper motor neuron lesion, there is *no* suppression of micturition center, therefore, patient voids prematurely

Combination Type Bladder
(Figures 7–30, 7-31)
Many patients (as many as 85%) with SCI develop Detrusor Sphincter Dyssynergia (DSD)

Causes:
- Central Cord Syndrome
- MS
- Progression of SCI (UMN Lesion)

Lesion:
Neurologic injuries between the sacral (S2–S4) and pontine micturition center

Resultant Scenario:
Tight little bladder (Detrusor hyperreflexia)
Tight sphincter (Sphincter hyperactivity)

Result:
Failure to void

Risk if Not Treated:
- Infected urine travels up towards kidneys (Figure 7–30)
- Note: These patients frequently have frequency and urgency, but lack of coordination between bladder and sphincter. This prevents complete bladder emptying.
- Result: Increased residual volumes, urine becomes infected, patient then tries high-pressure voiding against closed sphincter, this sends infected urine up to kidneys

Treatment:
1. Anticholinergic Meds – to expand the detrusor to prevent infected urine from going up to the kidneys (intact sphincter is good for continence)
2. Intermittent catheterization
3. Antimuscarinic drugs (i.e., anticholinergic) to cause bladder relaxation
4. Alpha blocker—to open bladder neck
5. Sphincterotomy

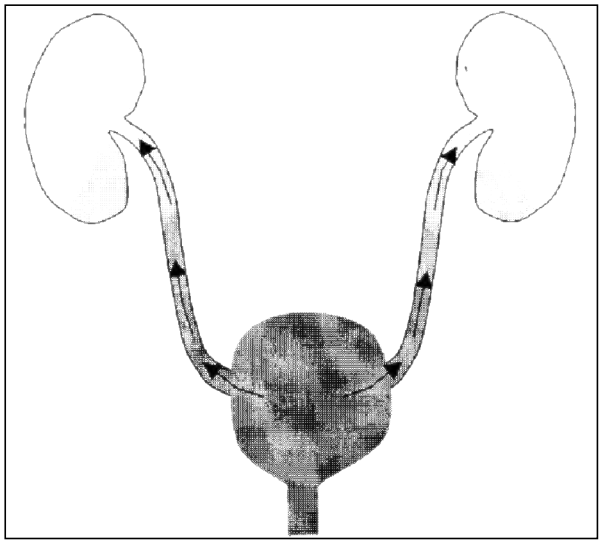

FIGURE 7–30. Reflux of infected urine backs up towards kidneys.

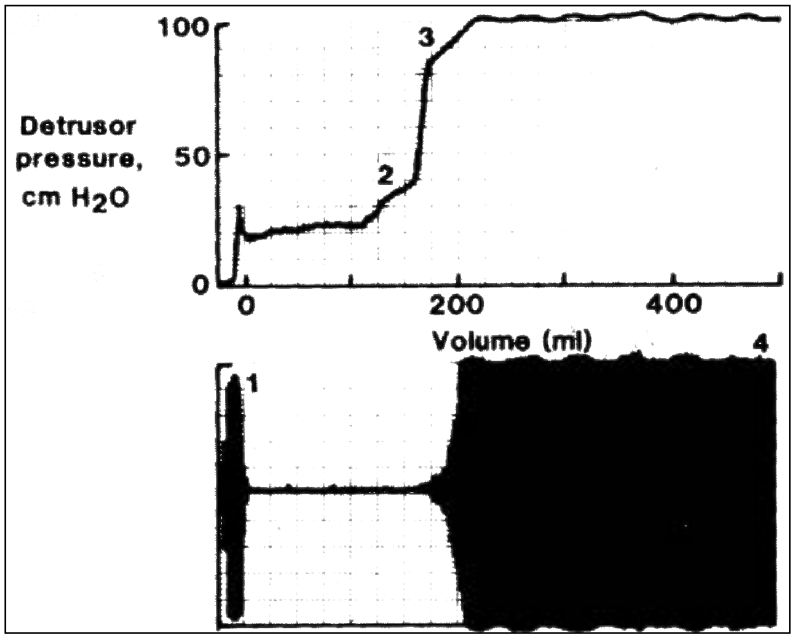

FIGURE 7–31. Cystometrogram and EMG in a patient with complete upper motor neuron bladder dysfunction shows: 1. Brisk bulbocavernosus reflex. 2. Bladder capacity is reduced. 3. High intravesical pressure during detrusor contraction. 4. Detrusor/external urethral sphincter dyssynergia with marked electrical activity of the pelvic floor muscles during detrusor contraction.

The normal bladder mechanism that prevents vesicoureteral reflux is described below:

Normal Anatomy (Figure 7–32A)

- The one-way valve mechanism can remain competent only as long as the oblique course of the ureter within the bladder wall is maintained
- During relaxation of the bladder, when urine is being stored, the ureter pumps urine into the bladder
- During bladder contraction the valve shuts closed. As a result urine cannot reenter the ureter and, therefore, the bladder is emptied with no reflux of urine into the ureters. (Figure A)

Bladder Wall Hypertrophy (Figure 7–32B)

- When bladder hypertrophy causes the course of the distal ureter to become progressively perpendicular to the inner surface of the bladder, the vesicoureteral function becomes incompetent, permitting vesicoureteral reflux
- During relaxation of the bladder, the ureter pumps urine into the bladder
- During bladder contraction, because the distal ureter becomes perpendicular to the inner surface of the bladder, the valve cannot close. Urine is forced up the ureter to the kidney and hydronephrosis can result. (Figure B)
- Reflux is further complicated by acute or chronic pyelonephritis with progressive renal failure.

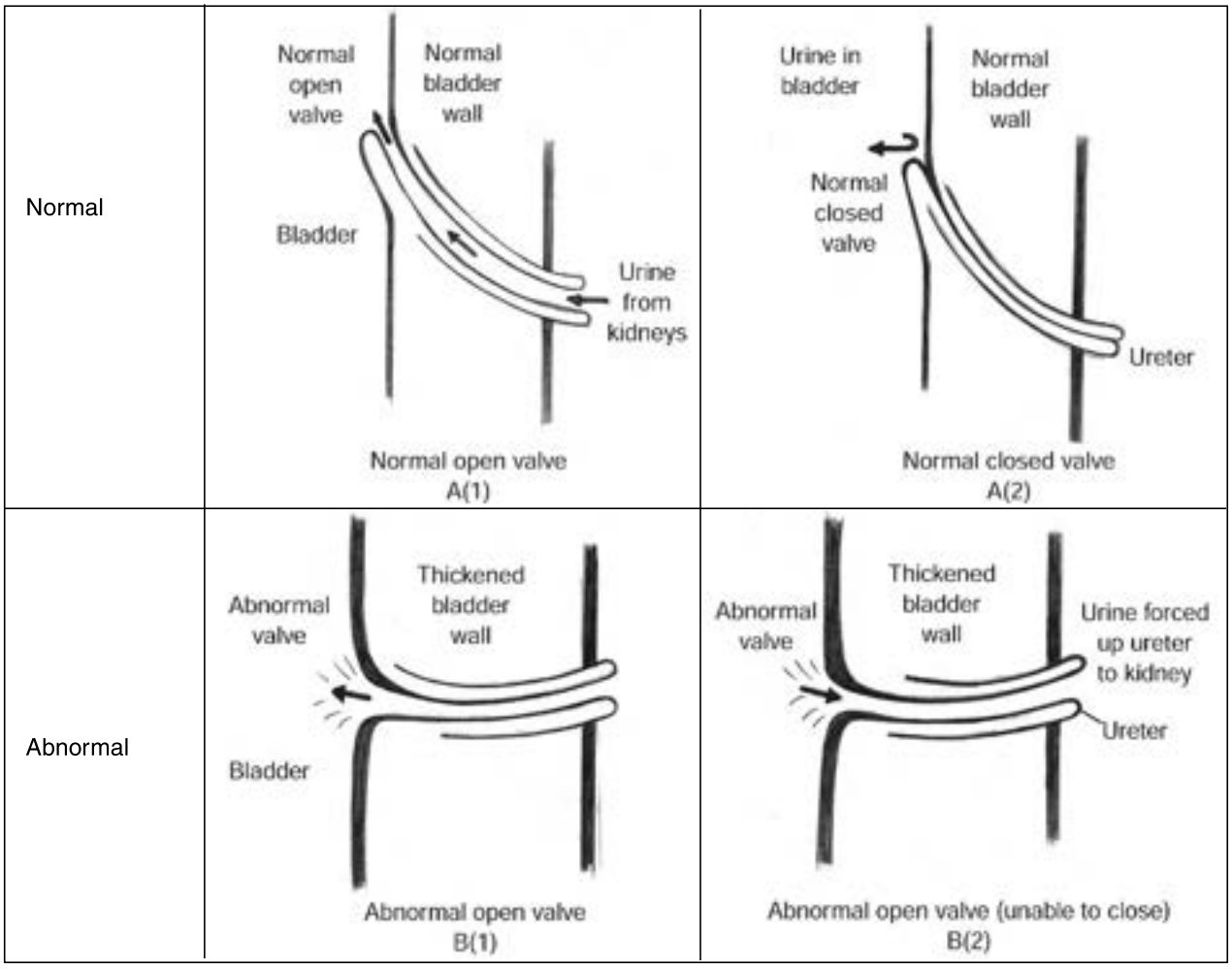

FIGURE 7–32. Vesicoureteral Junction. **A(1):** Normal Open Valve: The muscle of the ureter "milks" urine through the valve into the bladder. **(2):** Normal Closed Valve: When the bladder contracts, the valve is pressed shut. The normal valve prevents urine from flowing back into the ureter.
B(1): Abnormal Open Valve: The abnormal valve still allows urine to pass through the ureter into the bladder. **(2):** Abnormal Valve: The abnormal valve is unable to close. When the bladder contracts, urine is pushed back into the ureter and kidney.

🕮 Intermittent Catheterization (IC)

IC has reduced many of the associated complications of the indwelling catheter, which include epididymitis, penoscrotal abscess, fistula formation, renal and bladder calculi, and malignancy. IC has been shown to reduce the incidence of urinary tract infection.
Pathophysiology of Urinary Tract Infections (UTI):
UTIs are generally caused by the endogenous flora of the host overcoming the competing normal flora and host defense mechanism. The presence of the UTI is affected by:

- The virulence of the invading microorganism
- The condition of the urine as the culture medium
- The host defense mechanisms

In general:
An acidic concentrated urine inhibits microbial growth. UTIs are prevented by the washout effort of large volumes of urine. The large flow of fluid impedes the adherence of microorganisms and dilutes the concentration of microorganisms.

Management of UTIs

Asymptomatic UTIs
🕮 In SCI patients on an intermittent catheterization (IC) program recurrent asymptomatic bacteriuria (with less than 50 WBC per high power field) is generally not treated to avoid the development of resistant organisms.

(The exception to this is evidence of vesicoureteral reflux, hydronephrosis, or growth of urea splitting organisms.)

In general, asymptomatic UTI in individuals with indwelling catheters should not be treated.

Symptomatic UTIs
Symptomatic bacteriuria with fever leukocytosis or increased spasticity is treated and the catheterization is increased to reduce bacterial concentration and remove the urine that serves as a culture medium for bacterial growth. (A Foley may be necessary if volumes are too large for an IC program)

Most Common Urinary Tract Complications in Neurogenic Bladder

- Irregular, thickened bladder wall and small diverticuli—earliest changes
- Vesicoureteral reflux: 10%–30% of poorly managed bladders, leads to pyelonephritis, renal stones
- Hydronephrosis and hydroureters caused by outlet obstruction
- Overdistended areflexic bladder
- Bladder infections can lead to marked reduction in compliance of bladder

Prevention
All of these complications can be prevented by adequately draining the bladder at a pressure below 40 cm H_2O, either by intermittent catheterization along with the use of anticholinergic drugs or by timely surgical relief of the outflow obstruction

Prophylactic Treatment of UTIs in SCI patients

- Prophylactic antibiotic role is not fully established, but it is still used (i.e., Macrodantin® (nitrofurantoin))
- Vitamin C supplementation, cranberry juice, methenamine salts—overall used as acidifying agents

SEXUAL DYSFUNCTION

Physiology of Normal Sexual Act

Male Sexual Act (Bors, 1960)
Male erectile and ejaculatory function are complex physiologic activities that require interaction between vascular, nervous, and endocrine systems.
Erections are controlled by parasympathetic nervous system
Ejaculations are controlled by sympathetic nervous system

Erection:
Controlled by a reflex arc that is mediated in the sacral spinal cord
A reflex involves an afferent and an efferent limb

The Afferent Limb:
- Consists of somatic afferent fibers from the genital region that travel through the pudendal nerve into the sacral spinal cord

The Efferent Limb:
- Involves parasympathetic fibers that originate in the sacral spinal cord. These fibers travel through the cauda equina and exit via S2–S4 nerve roots.
- Postganglionic parasympathetic fibers secrete nitric oxide, which causes :
  - Relaxation of smooth muscle of the corpus cavernosum
  - Increases blood flow to the penile arteries—vascular sinusoids of the penis become engorged with blood
  - The result is an erection
  - This reflex is modulated by higher brainstem, subcortical, and cortical centers.

Ejaculation:
- Signals the culmination of the male sexual act, and is primarily controlled by the sympathetic nervous system
- Similar to sympathetic innervation to the bladder—these fibers originate in the thoracolumbar spinal cord (T11–L2), then travel through the hypogastric plexus to supply the vas deferens, seminal vesical, and ejaculatory ducts

Female Sexual Act
- Sexual excitation is the result of psychogenic and physical stimulation
- Stimulation of the genital region, including clitoris, labia majora, and labia minora, causes afferent signals to travel via the pudendal nerve into the S2–S4 segment of the spinal cord
- These fibers interact with the efferent parasympathetic fibers that project through the pelvic nerve
- The result is:
  - Dilation of arteries to perineal muscles and tightening of the introitus
  - Bartholin's glands secrete mucus, which aids in vaginal lubrication
- Female orgasm is characterized by the rhythmic contraction of the pelvic structures. Female orgasm also results in cervical dilation, which may aid in sperm transport and fertility.

Erectile Dysfunction

Men with SCI may obtain reflexogenic or psychogenic erections

Reflexogenic Erections
- Can occur independently of conscious awareness and supraspinal input (mediated by paraspinal division of ANS through sacral roots S2–S4)
- Are secondary to manual stimulation of the genital region (however, once stimulation has been removed, the erection may no longer be sustained)

Psychogenic Erections
- Involve supraspinal (above SC) effects, that are the result of erotic stimuli that result in cortical modulation of the sacral reflex arc
- Erection is mediated by central origin and psychological activated center

In general, erections are more likely with incomplete lesions (both UMN and LMN) than complete lesions. Many times, men with SCI can only maintain an erection while the penis is stimulated and the quality of the erection is insufficient for sexual satisfaction. As such, the erection must be augmented or induced.

Methods to Induce Erections
- Oral Therapy – Sildenafil, i.e., Viagra®
- Intracavernosal injection therapy – i.e. papaverine, alprostadil, phentolamine
- Penile vacuum device
- Transurethral devices – i.e., alprostadil
- Penile implants

Ejaculatory Dysfunction

In men with SCI, the ability to ejaculate is less than the ability to obtain an erection
The rate of ejaculation varies depending on the location and nature of the neurologic injury:
- Complete UMN lesions: ejaculation rate is estimated at 2%
- Incomplete UMN lesions: ejaculation rate is estimated at 32%
- Complete LMN lesions: ejaculation rate is estimated at 18%
- Incomplete LMN lesions: ejaculation rate is estimated at 70%

📖 Most SCI males are unable to ejaculate.
If they do, they are usually incomplete LMN lesions

Methods to Induce Ejaculation
- Intrathecal neostigmine
- Subcutaneous physostigmine
- Direct aspiration of sperm from vas deferens
- Vibratory stimulation – can be used at home
  - Increased incidence of autonomic dysreflexia
  - Males inseminate females with syringe
- Electroejaculation: most popular in USA
  - In incomplete lesion: very painful
  - If sensation is intact, patient cannot tolerate pain leading to heart rate and BP increase, and *autonomic dysreflexia* is a problem.

- Equipment used has been modified and it is possible to obtain the ejaculate through low-intensity constant repeatable current
- Patients need medical supervision
- Hospitals or office-based procedure: to evaluate through anoscopy before and after procedure to assess for injury to rectal mucosa
- Pretreat with Nifedipine (has lowered risk of autonomic dysreflexia)

Direct Stimulation of Hypogastric Nerve

- Ejaculate is obtained through the use of an implanted hypogastric nerve stimulator
- Surgical procedure—not appropriate for patients with intact pelvic pain sensitivity, since hypogastric nerve stimulation causes severe pain.

Sexual function might not return for 6–24 months. 80% experience return within 1 years of injury, 5% in 2 years

Infertility in Males With SCI (Linsenmeyer and Perkash, 1991)

Fertility in many paraplegic and tetraplegic men after SCI is severely impaired. Two major causes are (already discussed), *ejaculatory dysfunction* and *poor semen quality*

Poor semen quality is secondary to

- Stasis of prostatic fluid
- Testicular hyperthermia
- Recurrent UTI
- Abnormal testicular histology
- Changes in hypothalamic-pituitary-testicular axis
- Possible sperm antibodies
- Type of bladder management
- Chronic—Long-term use of various medications

Stasis of prostatic fluid

- Decreases sperm motility
- Studies have shown that in patients who did not have spontaneous ejaculations, there was an improvement in semen quality after 2–4 electroejaculations

Testicular Hyperthermia

Studies have shown higher deep scrotal temperatures (average = 0.9°C higher) in men with paraplegia who were seated when compared to noninjured control subjects who were seated. Men with SCI often sit with their legs close together, in contrast to nonimpaired men.

Sperm Counts and Motility Indices

- Sperm counts are lower in men who were having prostatic inflammation compared to those who were not
- Leukocytes (WBC > 10^6) in the spermatic fluid reduced total sperm count 41%, sperm velocity 12%, and total motile sperm 66%
- The single worst predictive factor for immobility to penetrate an ovum was leukocyte concentration in the semen
- Postinfective changes may affect fertility, such as atrophy of the testicles or obstruction of epididymal ducts.

Most Common Finding Noted on Biopsy is Atrophy of the Seminiferous Tubules

No investigations have found a significant correlation among biopsy finding, level of injury, length of injury, hormonal changes, or number of UTIs

Testosterone
Appears to remain normal or slightly above or below normal

Antisperm Antibodies
- Inhibit cervical mucous penetration
- Despite studies, immunologic-mediated infertility remains controversial
- Infertility due to antibodies is often not an absolute condition; additional time may be required but pregnancy can occur
- Two factors associated with antibody formation include: obstruction of the genital tract and UTIs

Female Infertility

Immediately postinjury 44%–58% of women suffer from temporary amenorrhea.
Menstruation returns within six months post injury.
Most women with SCI are fertile.

Birth Control
Can be problematic for SCI women:
- Condoms—provide protection
- Diaphragm—need adequate hand dexterity
- Oral Contraceptives—associated with increased risk of thromboembolism
- IUD—can increase risk of pelvic inflammatory disease, which may lead to autonomic dysreflexia

Pregnancy
The likelihood of pregnancy after spinal cord injury is unchanged, since fertility is unimpaired.
Pregnant women with SCI have an increased risk of:
- UTIs
- Leg edema
- Autonomic dysreflexia
- Constipation
- Thromboembolism
- Premature birth

Uterine innervation arises from T10–T12 level
Patients with lesions above T10 may not be able to perceive uterine contractions.
Pre-eclampsia may be difficult to differentiate from autonomic dysreflexia.
Autonomic dysreflexia may be the only clinical manifestation of labor.

GASTROINTESTINAL COMPLICATIONS AND BOWEL MANAGEMENT

Innervation of Bowel—Review of Anatomy and Neuroregulatory Control (Figure 7–33)

- The colon is a closed tube bound proximally by the ileocecal valve and distally by the anal sphincter
- The colon is composed of smooth muscle oriented in an inner circular and outer longitudinal layer
- The lower colon and anorectal region receive innervation by the sympathetic, parasympathetic, and somatic pathways
- In addition, the intrinsic enteric nervous system (ENS), composed of the Auerbach's (myenteric) plexus and the Meissner's (submucosal) plexus, coordinates the function of each segment of the bowel
- Auerbach's plexus is primarily motor; Meissner's plexus is primarily sensory. Both these plexi lie between the walls of smooth muscle mentioned earlier.
- The parasympathetic and sympathetic nervous systems modulate the activity of the ENS, which in turn inhibits the inherent automaticity of the bowel's smooth muscle

The Parasympathetic Nervous System
- Increases upper GI tract motility
- Enhances colonic motility
- Stimulation is provided by the action of the vagus nerve, which innervates proximal to mid. transverse colon, and by the splanchnic nerves (pelvic nerve), which originate from the S2–S4 region, which innervate the descending colon and rectal region

The Sympathetic Nervous System
- Stimulation inhibits colonic contractions, and relaxes the internal anal sphincter favoring the function of storage
- Innervation projects through the hypogastric nerve via superior mesenteric, inferior mesenteric, and celiac ganglia

The Somatic Nervous System
- Increases external anal sphincter tone to promote continence
- The external sphincter (EAS) consists of a circular band of striated muscle that is part of the pelvic floor

Anal Region

The Internal Anal Sphincter
- Composed of smooth muscle under the influence of the sympathetic system (T11–L2)
- Surrounds the anus proximally.
- In patients without SCI the sphincter normally relaxes with filling of the rectum.

The External Anal Sphincter
- Composed of skeletal muscle
- Helps to maintain continence by increasing its tone
- It acts under volitional control, learned by maturation, and reflex activity.
- It is innervated by the pudendal nerve (roots S2–S4). Higher cortical centers and the pontine defecation center send stimulus for EAS relaxation, allowing defecation.

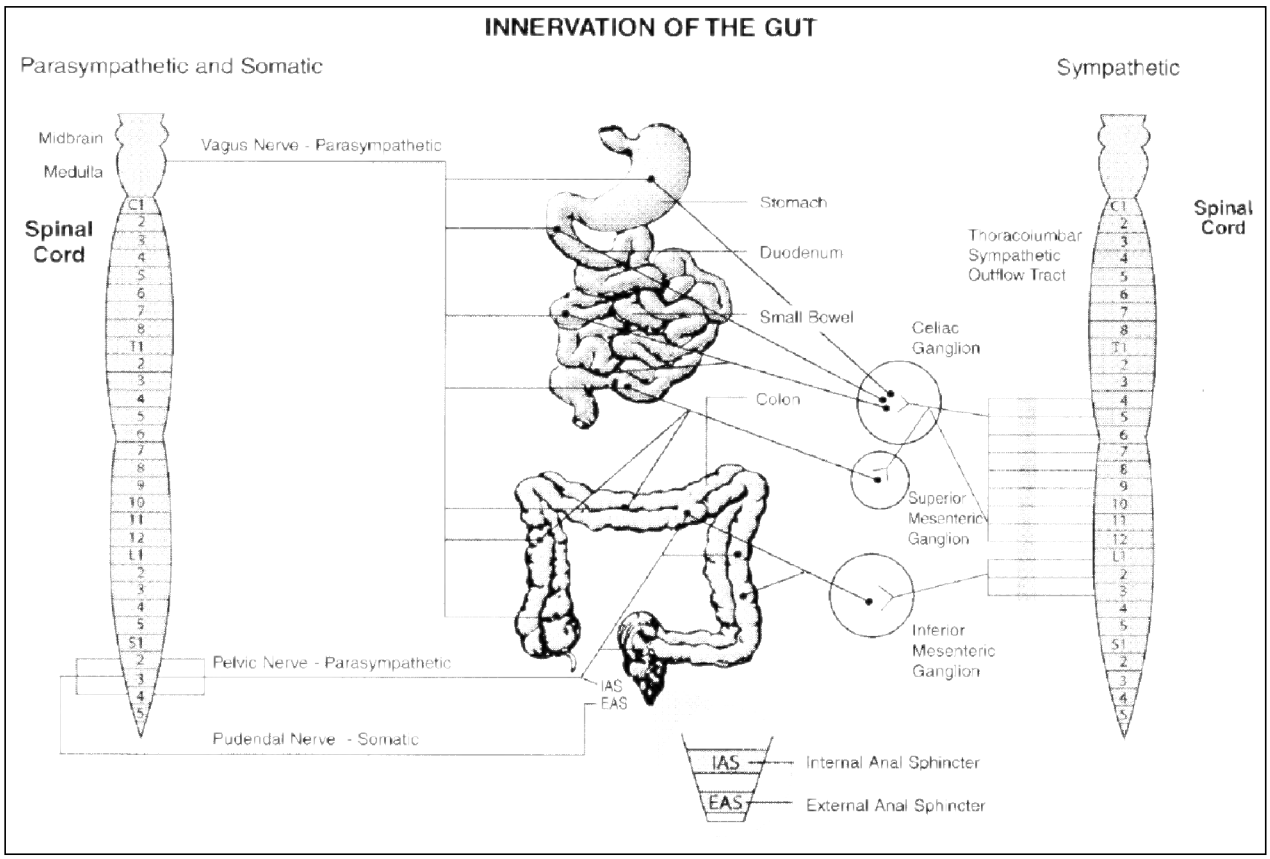

FIGURE 7–33. Innervation of the Gut. (From Nesathurai S. The Rehabilitation of People with Spinal Cord Injury: A House Officer's Guide. © Boston Medical Center for the New England Regional Spinal Cord Injury Center. Boston, MA: Arbuckle Academic Publishers, with permission).

Storage and Defecation in the Neurologically Intact Individual

Storage

- The internal anal sphincter, is sympathetically activated (T11–L2) allowing for relaxation This occurs with filling of the rectum in patients without SCI
- External anal sphincter (EAS) tone increases, secondary to spinal cord reflexes and modulated action of higher cortical regions, maintaining continence

Defecation

- Rectosigmoid distention causes reflex internal anal sphincter relaxation
- Volitional cortical activity sends signal to pontine defecation center. Volitional contraction of the levator ani muscle occurs, opening the proximal canal, relaxing the external anal sphincter and puborectalis muscles
- Reflexive rectal propulsive contractions take place resulting in expulsion of the stool bolus

Defecation in the Spinal Cord Injured Patient

Upper Motor Neuron Lesions (Hyperreflexic Bowel)

- Cortical control is disrupted, with decreased ability to sense the urge to defecate
- EAS cannot be voluntarily relaxed and pelvic floor muscles become spastic. However, nerve connections between the spinal cord and colon, as well as the myenteric (Auerbach's) plexus remain intact and the stool can be propelled by reflex activity.

Lower Motor Neuron Lesions (Areflexic Bowel)

- Spinal shock or lesion below conus medullaris
- Reflex defecation is absent

- Myenteric (Auerbach's) plexus coordinates the movement of stool, but movement is slow
- Overall, constipation may result (most common result)

Note: Attenuated or absent external anal sphincter contractions may result in fecal smearing or fecal incontinence

📖 In SCI the GI system can be affected by loss of sympathetic and parasympathetic input at the transverse and descending colon, resulting in decreased fecal movement. In SCI, fecal impaction and constipation is the most common complication during recovery.
To help with defecation, the physician may take advantage of two reflexes:

📖 *The Gastrocolic Reflex*
Increased colonic activity occurs in the first 30 to 60 minutes after a meal (usually within 15 minutes). Therefore, place the SCI patient on the commode within one hour subsequent to a meal.

📖 *The Anorectal Reflex (Rectocolic Reflex)*
Occurs when the rectal contents stretch the bowel wall reflexively, relaxing the internal anal sphincter. Suppositories and digestive stimulation cause the bowel wall to stretch and take advantage of this reflex.
Note this reflex
Can be manipulated by digital stimulation of the rectum.
Digital stimulation is accomplished by gently inserting a lubricated finger into the rectum, and slowly moving the digit in a clockwise manner.

Management of Bowel Dysfunction in SCI

Acute Phase

📖 **Gastric Atony and Ileus**
- After a significant SCI, the patient is at high risk for the development of gastric *Atony* and *Ileus*, which may cause vomiting and aspiration
- Stomach decompression by nasogastric tube should be considered in all acutely SCI patients.
- The ileus onset can be delayed for 24–48 hrs.
- The ileus usually lasts from 3–4 days up to 7 days.

Gastric Atony and Ileus (or Adynamic Ileus) occurs in 63% of patients with SCI
- Results from spinal shock and reflex depression.

Management
- NG suction to prevent GI dilation and respiratory compromise
- IV fluids
- Abdominal Massage—TENS to stimulate peristalsis of gut
- Injections of neostigmine methylsulfate (Prostigmine) 3–5 hrs.
- As soon as bowel sounds appear, start clear liquid diet
- If persists—may use Reglan

Chronic Phase
- **Colonic distention:** problems with small bowel motility and gastric emptying
- **Pseudo obstruction:** no evidence of obstruction on radiographic studies.
- Abdominal distention, nausea, vomiting, constipation.
- **Secondary causes:** electrolyte imbalance and medications (narcotics, anticholinergics)

Management
- NG suction
- Remove offending agent
- If cecum is dilated >12 cm., surgical decompression or colonoscopy

Constipation
Long-Term Management
- Defecation done using bedside commode (sitting facilitates emptying)
- Maintain adequate fluid intake, medications that decrease bowel motility such as narcotics, tricyclic agents, and anticholinergics should be minimized

Diet: high fiber
Meds
Bulk Cathartics: promotes evacuation by retaining or pulling H_2O into colon
- Metamucil®: dietary fiber increase

Irritants
- Castor oil, irritates bowel—AVOID castor oil

Fecal Softener
- Colace®: increases fluid accumulation in GI tract, AVOID Peri-Colace®—causes cramping

Oral Stimulants
- Senokot®: stimulates peristalsis by acting on Auerbach's plexus

Suppositories: placed high against rectal wall
- Glycerine: draws water into stool/stretches rectal wall
- Dulcolax®: stimulates peristalsis. Stimulates sensory nerve endings

Bowel Program

Initially Aims for Bowel Movement Daily
Surgical Intervention—Bowel diversion done when incontinence becomes a problem
Bowel Program consists of:
- Glycerin supp. (or Dulcolax®)
- Encourage patient to have BM at same time QD
- Use Gastrocolic Reflex—Q1hr post breakfast or dinner

If meds. are used, start with:
1. Dulcolax® Q daily after meal - dinner or breakfast
2. Stool softener (Colace®): 100 mg. TID
3. Senokot®: PO q daily at noon

(Spasticity of external anal sphincter may signify interference with bowel care) complications of neurogenic bowel:
Fecal Incontinence: Skin breakdown, ulcerations, UTI
Fecal Impaction: Autonomic hyperreflexia
Must use Lidocaine® Gel during digital extraction
📖 Anticholinergic meds used for failure-to-store bladder can cause severe constipation
Bowel dysfunction affects the patient's community integration—socially, vocationally and psychologically

Other Gastrointestinal Complications

Gastroesophageal reflux
- Avoid prolonged recumbency; elevate the head of the bed
- Avoid smoking
- Avoid medications: Ca^+ channel blockers, Valium®, nitrates, anticholinergics

Treatment:
Provide antacids for mild to moderate symptoms:
H_2 antagonists, Metoclopramide 10 mg tid, Omeprazole 20 mg QD

Gastroesophageal bleeding
- Most frequently secondary to perforating and bleeding ulcers
- Stress ulcers secondary to interruption of sympathetic vasoconstrictors (vasodilatation and mucosal hemorrhage)
- Steroid use
- Increased gastric secretion

Treatment:
Provide prophylaxis with:
- Antacids
- H_2 blockers—Cimetidine, Ranitidine, Famotidine
- Sucralfate—stimulates local prostaglandin synthesis

Endoscopy is the diagnostic method of choice
With active GI bleeding—maintain BP, correct coagulation deficits, consult GI/Surgical service

Cholecystitis
- Most common cause of emergency abdominal surgery in SCI patients
- Increased risk: 3x > in SCI
- Possible causes: abnormal gallbladder motility in lesions above T10, abnormal biliary secretion, abnormal enterohepatic circulation

Treatment:
Observe, May opt for surgical removal or dissolution

Pancreatitis
- Most common in the first month post injury.
- May be related to steroid use—increased viscosity of pancreatic secretions
- May suspect when adynamic ileus doesn't improve.

Evaluate
- Radiographs
- CT
- Ultrasonogram
- Labs: amylase, lipase

📖 **Superior Mesenteric Artery (SMA) Syndrome**
Condition in which the third portion of the duodenum is intermittently compressed by overlying SMA resulting in GI obstruction (Figure 7–34) (Roth, 1991)

Predisposing factors include:
- Rapid weight loss (decrease in protective fatty layer)
- Prolonged supine position
- Spinal orthosis
- Flaccid abdominal wall causes hyperextension of the back

Exacerbated by:
- Supine positioning
- Tetraplegic patient with abdominal and cervical orthosis

Symptoms:
- Postprandial nausea and vomiting
- Bloating
- Abdominal pain

Diagnosis:
UGI Series: demonstrates abrupt duodenal obstruction to barium flow

Treatment:

Conservative
- Eat small, frequent meals in an upright position
- Lie in the left lateral decubitus position after eating
- Metoclopramide (Reglan®): stimulates motility of UGI tract

Rarely requires surgery
- If conservative treatment fails, surgical duodenojejunostomy (DJ ostomy)

Remember:
Any condition that decreases the normal distance between the SMA and aorta (weight loss, supine position, halo, flaccid abdominal wall) may result in compression of the duodenum described as the nutcracker effect

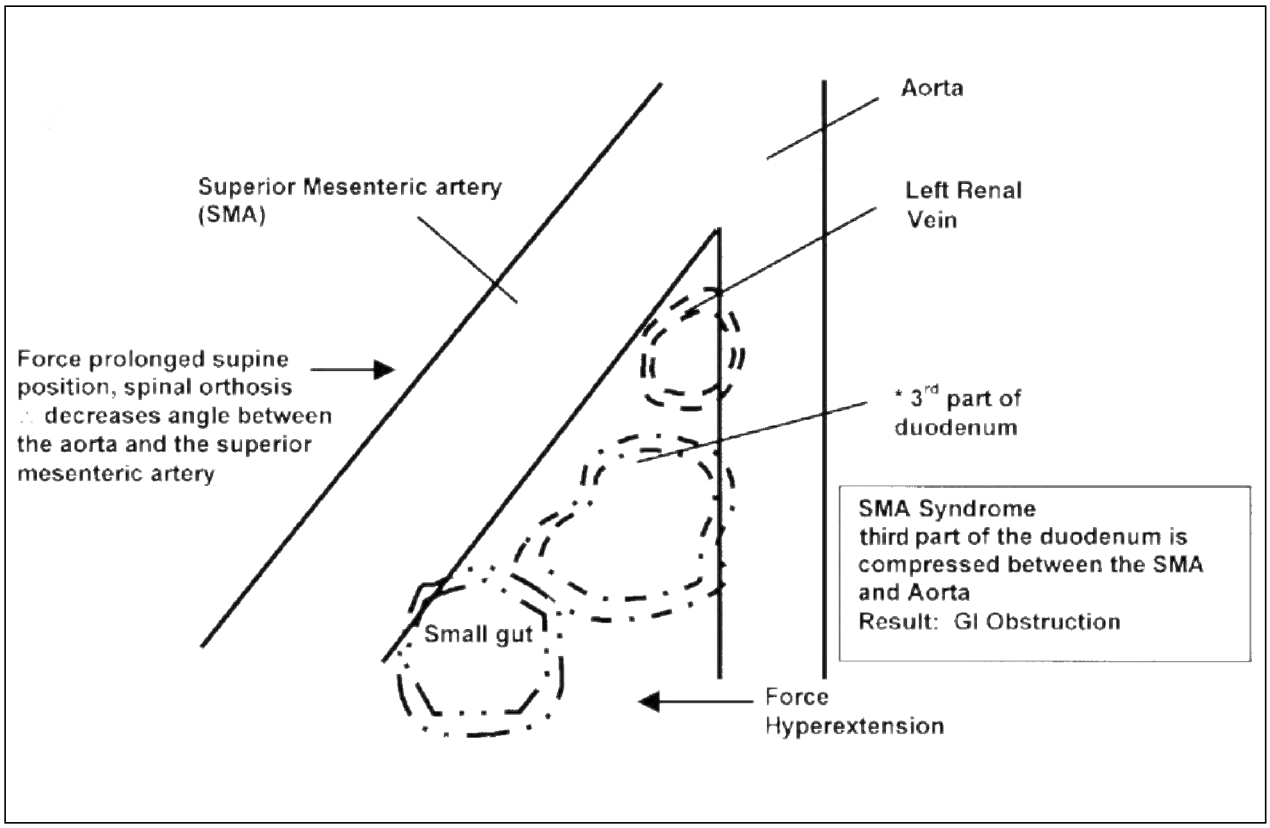

FIGURE 7–34. Lateral view through duodenum and left renal vein.

🕮 METABOLIC COMPLICATIONS

Hypercalciuria

Immobilization, decreased weight bearing promotes bone resorption.
Patients become hypercalciuric—this may continue for 18 months.
Vitamin D, parathyroid hormone are not involved in the process.

Hypercalcemia

Patients with hypercalciuria can, in rare cases, develop hypercalcemia
Symptoms: nausea, vomiting, dehydration, decreased renal function, decreased mental status, abdominal discomfort, anorexia, malaise
Symptoms of hypercalcemia can be remembered with the mnemonic *stones, bones, and abdominal groans.*
Patients are most commonly young tetraplegic males.

Treatment: (Merli, 1984)
- Hydration—IV normal saline solution, furosemide
- Mobilize the patient: tilt table, weight bearing activities
- Decrease GI absorption of calcium: give steroids, decrease vitamin D
- Didronel®, Calcitonin

Osteoporosis

Secondary to disuse—localizes below the level of the lesion
Calcium excretion also increased (increased bone resorption)
Approx. 22% bone loss 3 months post injury (Claus-Walker, 1975)
Increased risk of fracture

Treatment
- Weight bearing
- FES cycling—demonstrated to decrease the rate of bone resorption in acute SCI, effect remains after discontinuation
- Pharmacologic agents have not been proven to reduce osteoporosis in SCI.

Hyperglycemia

Up to 70% of the patients show insulin resistance, with abnormal response to glucose load.
Rarely require treatment (Duckworth, 1983)

MUSCULOSKELETAL COMPLICATIONS

Upper Extremity Problems

Shoulder is the most commonly affected joint. Frequently secondary to weight bearing and overuse.

Causes
- Impingement syndrome
- Overuse
- DJD
- Rotator cuff tear/bicipital tendinitis
- Subacromial bursitis
- Capsulitis
- Myofascial pain
- Disuse
- Cervical radiculopathy

Diagnosis
- Consider possible causes such as heterotopic ossification, syrinx
- Perform complete physical exam, including functional assessment, ROM, flexibility, and sensation

Treatment
- Rest, pharmacotherapy for pain treatment
- Compensatory techniques to be used for daily function
- Treat the condition
- Educate the patient regarding posture, weight bearing

Compression neuropathies
Have been noted to increase with the length of time from injury.
27% incidence of carpal tunnel syndrome in SCI patients seen 1–10 years post-injury
54% incidence of carpal tunnel syndrome in SCI patients seen 11–30 years post-injury
90% incidence of carpal tunnel syndrome in SCI patients seen more than 31 years post-injury
(Ditunno, 1992)

PULMONARY COMPLICATIONS OF SCI AND MANAGEMENT

Incidence

Respiratory complications occur in 50% of patients. During the first month post injury, if the initial rehabilitation period is included, the incidence increased to 67% of SCI patients.
Atelectasis/pneumonia have an onset within the first 24 days post injury

Pulmonary complications are more common in high cervical injuries (C1—C4)
The *most frequent complications* are pneumonia, atelectasis, and ventilatory failure.
In low cervical (C5—C8) and thoracic (T11—T12) complications are equally frequent.
Thoracic injuries present with: *pleural effusion, atelectasis, pneumothorax, hemothorax, or both.*
(Fishburn, 1990; Jackson, 1994; Langis, 1992)

Pulmonary Dysfunction: Occurs for several reasons following SCI

1. Paralysis of some or all respiratory muscles to varying degrees
2. Loss of ability to cough secondary to varying levels of abdominal muscle paralysis
3. Injury to chest—e.g. rib fracture
4. Pulmonary injury—e.g. lung contusion

Predisposing Factors for Pulmonary Complications Include
Older Age
Obesity—restrictive respiratory deficits
Hx—COPD
Hx—Smoking

📖 *Pneumonia is the leading cause of death among long-term SCI patients.*
Patients tend to retain secretions in the lower lung fields due to:
- Difficulty in achieving postural drainage positions during the acute SCI period
- Altered ventilatory pattern after SCI (reduced airflow to lower lobes leads to atelectasis)
- Decreased ability to clear secretions independently, decreased effective cough

Left-sided respiratory complications are more common among hospitalized SCI patients. This is due to the following:
- The left mainstem bronchus takes off as a 40°–50° angle from vertical, making routine suctioning more difficult.
- In addition to this, there is a tendency to retain secretions in the lower fields.

Respiratory function may be affected to different degrees depending on the level of injury.

Pulmonary Compromise Related to Level of Injury

- Head trauma: May knock out respiratory drive
- Lesions above C3 (and incomplete lesions initially):
  - Initially they require ventilatory support
  - Later they will fall into two groups
  1. No damage to phrenic nucleus
  2. Damage to phrenic nucleus

No Damage to Phrenic Nucleus	**Damage to Phrenic Nucleus**
• C3,4,5 • Determined by EMG of phrenic nerve • Can stimulate phrenic nerve nucleus therefore, the patient will benefit from phrenic pacing (i.e., C1,2, and incomplete lesions)	• C3,4,5 • Determined by EMG of phrenic nerve • Cannot stimulate phrenic nerve nucleus, therefore, will not benefit from phrenic pacing (i.e., lesions of phrenic nucleus causing irreparable damage*) *Continue to require ventilatory support* *Note: Intercostal nerve grafts are being attempted

EMG of diaphragm is necessary to rule out damage to phrenic nerve nucleus

- C3: Respiratory failure secondary to disruption of diaphragmatic innervation, requiring mechanical ventilation
- C4: Generally the highest level of injury at which spontaneous ventilation can be sustained
- Injuries above C8: Loss of all abdominal and intercostal muscles, impairment of inspiration and expiration
- T1 through T5: Intercostal volitional function is lost
- T5 through T12: Progressive loss of abdominal motor function, impairing forceful expiration or cough
- Injuries below T12: Few complications if there is lung injury (e.g. trauma) otherwise, no respiratory dysfunction

Phrenic Pacing (Lee, 1989)

- Phrenic pacing has reduced the need for mechanical ventilation in tetraplegic patients with respiratory failure since its introduction in 1972
- The technique involves the electrical stimulation of intact phrenic nerves via surgically implanted electrodes to contract the diaphragm
- Induces artificial ventilation through electric stimulation of the phrenic nerve, which causes the diaphragm to contract
- Used successfully in patients with COPD, central hypoventilation, and high tetraplegia
- Treatment option in patients with respiratory paralysis after cervical injury above the origin of the phrenic neurons
- Option in patients who do not have significant impairment of the phrenic nerves, lungs, or diaphragm

📖 **Contraindications to Phrenic Pacing**

- Denervated diaphragm (determined in EMG)
- Denervated—nonviable anterior horn cells C3, C4, C5
- Placement of phrenic pacer prior to 6 months post injury is contraindicated
  From 0–6 months–the chest is too FLAIL and flaccid
  You need some rigidity of chest wall to allow pacer to work
- Significant lung impairment

Major Complications of Phrenic Pacemaker

📖 *Signs of Failure of Pacemaker*

1. Sharp chest pain
2. SOB—Shortness of Breath
3. Absence of breath
4. Erratic pacing
5. Must maintain adequate ventilation via manual resuscitation bag

📖 *Causes of Phrenic Pacing Failure*

1. Diaphragmatic failure—due to overly aggressive pacing schedule
2. Infection of lung and/or phrenic nerve
3. Meds: including sedatives, tranquilizers, and narcotics
4. Upper airway obstruction—tracheal aspiration
5. Phrenic nerve damage from overstimulation or surgery

📖 *Benefits of Phrenic Pacing*

- Increased arterial oxygenation despite decreased alveolar ventilation
- Longer survival in patients with SCI
- Increased daily function secondary to conditioning of the diaphragm from nocturnal pacing

Physiology of Lung:

Inspiratory Muscles

- Accessory muscles
- Diaphragm—main respiratory muscle, main muscle during quiet breathing (75% of volume change) Contracts at inspiration, relaxes at expiration
- External intercostals

Expiratory Muscles

- Abdominal muscles—primarily active during forceful expiration and in producing cough (contract); push the relaxed diaphgram toward the chest cavity
- Internal intercostals

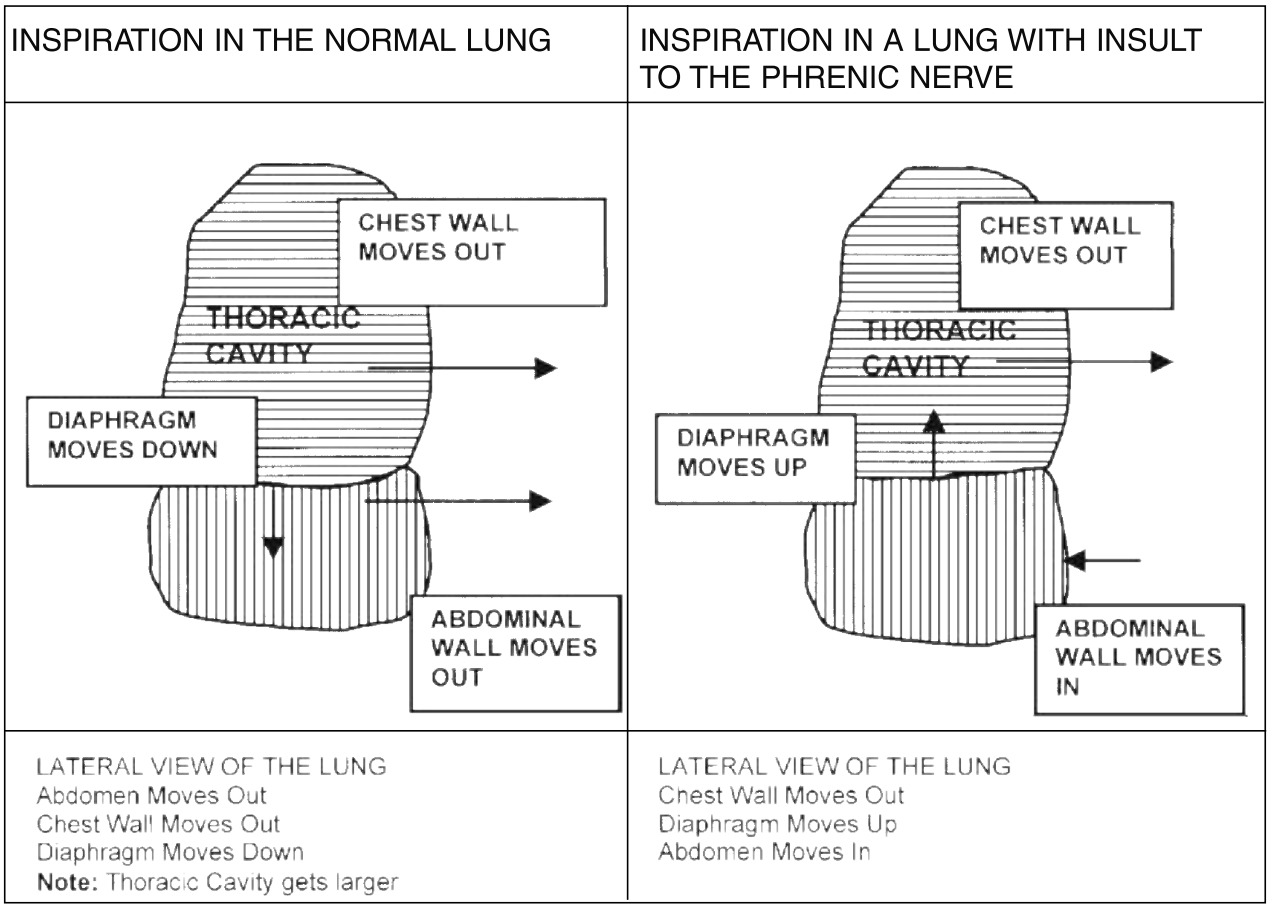

FIGURE 7–35. Inspiration in the Normal Lung and in a Lung with Insult to Phrenic Nerve

Pulmonary Function In SCI—Restrictive Respiratory Changes (Langis, 1992)

- *Forced Vital Capacity* (FVC) during the acute phase of cervical injury is noted to decrease 24%–31% when compared to the normal values secondary to paradoxical respirations
- With development of intercostal and abdominal spasticity *FVC can improve to 50–60%* of predicted normal value
- Tetraplegics develop *restrictive lung patterns*
  All volumes shrink (except residual volume)
  If VC < 1 liter: consider ventilation mechanically
  If VC < 600: must have ventilation mechanically
  If VC > 1.5 get off ventilator
- Signs of impending respiratory failure
  Increased respiratory rate with decreased tidal volume
  Decreased FVC < 15 cc/Kg body weight
  Decreased inspiratory force <20 mm H_2O
  Neurological level C3 or higher
  Patient can't count to 15 slowly

Mechanically ventilate when:
VC < 1 liter
📖 ABG show increasing PCO_2 or decreasing PO_2 levels
- PO_2 < 50
- PCO_2 > 50

Severe atelectasis

Prevention of Respiratory Complications
- Use of incentive spirometer
- Monitor CO_2 levels with ABGs
- Monitor vital capacity
- Cough assist—placing the hands on each side of the pts. upper abdomen and applying intermittent pressure, coordinated with the initiation of cough by the patient—helps produce forceful cough
- Suctioning—remember—tracheal suctioning may cause increased vagal tone with SA node suppression and brady-arrhythmia, leading to cardiac arrest (only suction as you withdraw catheter)
- Chest physical therapy—(see pulmonary chapter)
- Strengthening of pectoralis major muscle, clavicular portion, in tetraplegic patients
- Glossopharyngeal breathing—stroking maneuver to force air into the lungs by the use of the lips, soft palate, mouth, tongue, pharynx, and larynx, followed by passive exhalation.
- Pneumobelt—helps with exhalation. Inflatable, compresses the abdominal wall, diaphragm rises, and active respiration is produced.

HETEROTOPIC OSSIFICATION

Definition
Formation of mature lamellar bone indistinguishable from normal bone in soft tissues, most frequently deposited around a joint
As bone matures it becomes encapsulated, not connected to periosteum

Causes
- Possibly due to alteration in neuronal control over the differentiation of mesenchymal cells into osteoblasts which form new bone *or*
- A decrease in tissue oxygenation or induces changes in multipotential connective tissue cells in which new bone forms in planes between connective tissue layers
- No definitive explanation established.

Incidence: Heterotopic Ossification (HO) has been reported to occur in 16%–53% of patients following SCI.
Clinically significant HO: (resulting in significant limitation of joint range) affects 10%–20% of SCI patients.
Occurs below the level of neurological injury (only in the area of paralysis, unless other factors are present such as TBI or burn)
📖 Most common joints involved in SCI (in orderof occurrence): hip/knee/shoulder/elbow

📖 **Onset**
1–4 months status post injury most common, but can present after first 6 months

Symptoms: Early clinical findings include heat and soft tissue swelling
Swelling progresses to more localized and firm area over several days, may present as ROM in joint decreases
- Heat
- Localized soft tissue swelling—may look like DVT
- Decreased ROM of a joint
- Joint erythema/joint effusion
- Low grade fever

Risk Factors
- Spasticity
- Completeness of injuries
- Trauma or prior surgery to joint
- Age
- Pressure ulcer in proximity of joint

Diagnosis: Can be seen one week from onset in static bone scan or/triple phase bone scan precedes X-ray by at least 7–10 days
Plain film detects HO in 7–10 days after clinical signs are observed
Bone Scan returns to normal as HO matures in 6–18 months post injury
Serum Alkaline Phosphatase: Increases at 2 weeks—exceeds normal levels at 3 weeks—peaks at 10 weeks—returns to normal after HO matures
Not specific for HO

Treatment:
- 📖 Didronel® (etidronate disodium): 20 mg/Kg/day for 2 weeks then 10 mg/Kg/day for 10 weeks
  Does not change overall incidence, but less HO is laid down overall
- Indocin®—Not commonly used in acute SCI

- ROM—Maintain function while HO matures—The goal is to maintain functional range. The affected joint should be gently moved through functional range—vigorous force should not be used as this may lead to further ectopic bone formation
- Surgery—Used when HO severely limits ROM impairing function—should only be planned after bone is mature: 12–18 months post injury. Bone scan must be back to baseline and alkaline phosphatase should be back to normal

Complications
Peripheral nerve entrapment
Decreased ROM/loss of function/ankylosing
HO overlying a bony prominence will directly predispose to pressure ulcer/skin breakdown secondary to poor positioning

DEEP VENOUS THROMBOEMBOLISM (DVT)/ PULMONARY EMBOLISM (PE) IN SCI

Deep Venous Thrombosis (DVT)

Predisposing Factors
Virchow's Triad: Venous stasis/intimal injury/hypercoagulability
LE fractures
Obesity
Hx of previous DVT
DM
Arterial vascular disease
Immobility
Malignancy

Incidence
Ranges 47%–100%
- Varies Widely depending on the method of detection and number of cases evaluated in the study

More common in neurologically complete patients
More common in tetraplegic patients
10 times more frequent in plegic leg
20% of calf vein thrombi extend proximally

Onset
Most common during first 2 weeks after SCI
Greatest incidence decreases after 8–12 weeks post SCI

Diagnosis
Venogram is the Gold Standard
Venous Doppler is used as a screening test for lower extremity DVTs
Impedance Plethysmography—accurate in assessment of DVTs above the calf
Sensitivity—95%, Specificity—98%
Used to look for occlusions in the thigh, place cuff around the thigh and listen for flow

Complications: 🕮 Pulmonary Embolism (PE) leading cause of death in acute LCI

Pulmonary Embolism (Fluter, 1993; Goldhaber, 1998)

Symptoms
Pleuritic chest pain
Dyspnea

Fever, hemoptysis
Tachycardia
Hypoxemia

Physical Examination
1. Increased S2 sound: severe pulmonary HTN → cor pulmonale. → Right heart failure
2. Dullness at bases of lungs

Incidence
1%–7% of SCI patients (Hull)

Diagnosis (PE)
EKG: R. Axis Deviation
Right Bundle Branch Block (RBBB):—if massive PE
ABG:—decreased PO_2 (PO_2 drops severely)
Chest X-ray:
  wedge shaped opacity
  fluid
  vascularity
Perfusion lung scan: VQ mismatched
📖 *Gold Standard:* Pulmonary arteriogram

Treatment (PE)
O_2
Heparin
Vasopressor to treat shock

Surgical Treatment
Embolectomy

Other Complications of DVT

- **Postphlebitic syndrome (late complication of DVT)**
  - distal venous hypertension (residual obstruction of outflow—incompetent valve)
  - swelling
  - exercise induced pain
  - pigmentation
  - ulceration
  - long standing autonomic dysreflexia

Prophylaxis Treatment and Prevention of DVT

Prophylaxis for DVT (Merli, 1988)
- External intermittent pneumatic compression devices
  - increases LE venous return
  - decreases stasis and stimulates fibrinolysis
  - if delays > 72 hours—need to do duplex prior to initiation
- Enoxaparin—low molecular weight heparin (LMWH)
  - Dose 30 mg SQ BID
  - *Best intervention to prevent DVT if no contraindications*
  - Not used in patients with active bleeding, TBI or coagulopathy
- Thigh-high graded compression stockings (TEDS)—alone, not prophylaxis
- Coumadin®
- Minidose subcutaneous unfractionated Heparin
- Greenfield filter (may be indicated in selected cases, high risk, or failed prophylaxis)

Treatment DVT:
- Heparin—if not contraindicated
  - standard: 5,000 units IV bolus; followed by a constant infusion of 1,000 units/ (25,000 units in 250 cc D5W at 10 cc/hr)
  - maintain PTT 1.5-2 times normal
  - at least 5–10 days of anticoagulant prior to mobilization
- Warfarin started once PTT therapeutic (approximately three days after Heparin started); takes 5 days to load; target INR 3.0
  Coumadin for 3 months in case with DVT
  Coumadin for 3–6 months in case w/PE
  (Note: Heparin can be discontinued once coumadin is 1/2 times normal for 48 hrs.)
- No ROM in involved extremity. With small popliteal clots, patients may transfer to bedside chair in 1–2 days. If clot is in proximal veins or with PE, immobilization 5–10 days.
- If anticoagulation is contraindicated, then an IVC filter is necessary

Prevention:
- Recommended that patients receive both a method of mechanical prophylaxis as well as anticoagulant prophylaxis
- Pneumatic compression stockings or device should be applied to the legs of all patients during the first two weeks following injury
  If this is delayed for more than 72 hours after injury, test to exclude the presence of clots should be performed
- Anticoagulant prophylaxis—LMW heparin or adjusted unfractionated heparin should be initiated within 72 hours after injury if there is no hemorrhage or risk of bleeding
  LMWH: 30 SQ BID

🕮 Functional Electrical Stimulation (FES) in SCI has two general uses

- As exercise to avoid complications of muscle inactivity
- As a means of producing extremity motion for functional activities
  🕮 *FES can be used to*
  - Provide a cardiovascular conditioning program
  - Increase muscle bulk strength and endurance
  - Attempt to decrease risk of DVT
  - Produce extremity motion for standing and ambulation

🕮 PAIN IN THE SCI PATIENT

Incidence of chronic pain in SCI population is estimated between 20%–50%
Pain may be musculoskeletal, neuropathic, or visceral

MUSCULOSKELETAL PAIN

Upper Extremity Pain: common in the SCI patient

Patients with SCI load joints that do not normally bear weight (shoulder, elbow, wrist)
This predisposes them to painful UE conditions
These conditions include
- Carpal tunnel syndrome (which is present in up to 90% of SCI pts. at 31 years post injury)
- Rotator cuff tendonitis
- Rotator cuff tears
- Subacromial bursitis