Department of
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Veterans
Health
Initiative

Medical Care of Persons with Spinal Cord Injury

Independent Study Course
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MESSAGE FROM THE UNDER SECRETARY OF HEALTH

Dear Colleagues in Quality Health Care:

I am very pleased to present the enclosed Veterans Health Initiative (VHI) independent study guide on basics of caring for the veteran with spinal cord injury (SCI). "Medical Care of Persons with Spinal Cord Injury" has been designed and written by VA SCI physicians for all VA clinicians. To ensure that our veterans get the right care, at the right time, in the right place, at the right cost, it is important that all clinicians be aware of the specific conditions that may confront individuals with spinal cord injury and disorders. What may be a relatively minor symptom or problem in the person without SCI may herald a grave and even life-threatening problem for the individual with SCI. Greater general awareness of the specialized health issues facing persons with SCI is needed to assure therapeutically appropriate clinical processes.

The Education Contact at your medical center has the necessary information so you can receive ten hours of continuing medical education credits for studying this book and successfully completing the accompanying test. It is my expectation that every practitioner in the VA system will complete this course. I hope that you will keep this book available for reference when you have the opportunity to provide care for veterans with SCI in the future. This is one way that we can ensure provision of quality health care across the continuum of acute care, rehabilitative care, to extended care. VA sees veterans with spinal cord injury and disorders in a variety of health care settings, and they are counting on you to provide the best care possible. We owe them nothing less.

Thomas L. Garthwaite, M.D.

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Independent Study Outline

Purpose

This program is designed to familiarize you with the unique aspects of SCI medicine. Its purpose is to help you gain knowledge so that any clinical encounter is more comfortable and informational for both you and the veteran served. This is not an attempt to make you a specialist, but to provide information so you can manage the patient until a subspecialist is available.

Objectives

Upon completion of this self-study program, participants should be able to:

1. delineate the most common conditions affecting persons with SCI;

2. identify the changes in physiology, compensatory mechanisms, common disorders, physical functioning, and general health care needs of individuals with spinal cord injury;

3. specify the unique aspects of SCI medicine so that clinical encounters are more informative and comfortable for clinicians and the veterans served; and

4. describe the various treatments for persons with SCI.

Outcome

As a result of this program, clinicians will have a broader base of knowledge with which to provide effective care to patients with SCI and a better understanding of patients who experience this condition.

Target Audience

This independent study is designed for all VA physicians.

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INTRODUCTION

Margaret C. Hammond, MD

Spinal cord injury medicine evolved over the last fifty years and recently achieved recognition from the American Board of Medical Specialties. Due to the unique needs of persons with spinal cord injury (SCI) or disorders, care is typically provided in specialized centers in VA as well as in the private sector. However, with changes in health care delivery and the need to provide greater access to care, more practitioners are treating this population. Many physicians have had little exposure through either training or practice to individuals with SCI. Each organ system of the body is affected by SCI, and the changes in physiology, compensatory mechanisms, common disorders, physical functioning, and general health care needs of such individuals are unfamiliar to most practitioners. Being uninformed about these issues can have profound consequences for individuals with SCI.

This manual is intended to familiarize you with the unique aspects of SCI medicine. Its purpose is to help you gain knowledge so that any clinical encounter is better informed and more comfortable for both you and the person served. This is not an attempt to make you a specialist, but to provide information so you can manage the patient until a subspecialist is available.

Each chapter includes learner objectives and follow-up questions for self-study. An effort has been made to emphasize what is different about the presentation and treatment of SCI problems, not to repeat what is standard patient care for the able-bodied population. Although the term "injury" is used throughout, the principles apply to many etiologies of myelopathy.

The SCI Network of care is extensive within VA, and specialists are available at every SCI Center for ready consultation. Designated SCI primary care teams at most facilities are experienced with the issues described in this manual. Each facility has an SCI coordinator who tracks patients and assists with contacts and transfers to an SCI Center. Contact should be made with any of these individuals in order to facilitate care for persons with SCI.

Appropriate care of this population requires a comprehensive interdisciplinary team assessment on an annual basis, the scope of which is beyond this text. The goals are the delivery of preventive health services, as for any person, and a systemic evaluation of impairments and the detection of SCI-related complications. Such evaluations include appropriate diagnostic tests for the organ systems at particular risk. A focus on function,

equipment needs, and psychosocial and vocational status completes the review of impairment, disability, handicap, patient satisfaction and maintenance of wellness. Such evaluations are provided by the SCI Centers or selected other trained clinics.

I would like to extend a special note of thanks and appreciation to Dr. Robert Hendricks for his thoughtful and diligent work in support of this project.

Margaret C. Hammond, M.D.
Chief Consultant, Spinal Cord Injury and Disorders Strategic Healthcare Group
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Editor

1 PATHOPHYSIOLOGY AND CLASSIFICATION

Stephen P. Burns, MD

Objectives for the Learner

1. Identify the important tracts of the spinal cord and their location and functions.

2. Identify components of the neurologic examination of a person with SCI.

3. Describe findings of upper vs. lower motor neuron injury and their clinical significance in SCI.

4. Conduct motor and sensory evaluations that will provide accurate American Spinal Injury Association (ASIA) neurologic classification, along with identification of neurologic level, completeness of impairment, and clinical syndromes.

5. Know the five clinical syndromes of incomplete SCI.

6. Describe changes in autonomic nervous system function following SCI, including the effects secondary to impaired control.

Overview

The spinal cord is the primary pathway for transmission of information between the brain and the peripheral nervous system. Injury to the spinal cord causes disruption of movement, sensation, and autonomic nervous system function. Knowledge of the relevant neuroanatomy and the types of information carried by spinal cord tracts allows a more complete understanding of the consequences of SCI.

Anatomy and Physiology

Gray matter is centrally located in the cord and is the site of all nerve cell bodies, including those of the lower motor neurons that innervate muscle fibers. White matter contains groupings of axons called tracts. The tracts of primary importance for understanding loss of neurologic function after SCI are shown in Table 1-1.

TABLE 1-1.
MAJOR SPINAL CORD TRACTS AND THEIR FUNCTIONS

Tract	Location Within Cord	Direction of Signals	Function
Corticospinal	Lateral & anterior	Descending	Motor: Precise control of movement
Spinothalamic	Anterolateral	Ascending	Sensory: pain and temperature
Dorsal Columns	Posterior	Ascending	Sensory: proprioception and vibration

Pathophysiology of Spinal Cord Injury

Immediate events in acute injury include damage to tracts and motor neurons due to:

- Fracture of vertebral body with cord impingement from bony fragments

- Dislocation of vertebral bodies with loss of normal spinal canal diameter

- Transient narrowing of spinal canal diameter without bony fracture

- Traction on cord with disruption of neurologic structures

Only rarely is the complete cord transected. Subacute and secondary effects include cord edema, hemorrhage, ischemia, and onset of the inflammatory response.

Nontraumatic spinal cord conditions include multiple sclerosis, spondylotic myelopathy, syringomyelia, epidural abscess or hemorrhage, spinal cord tumor, virus-related transverse myelitis, spinal cord infarction, vitamin B12 deficiency, HIV-related myelopathy, and familial and degenerative spinal cord disease. Examples include epidural tumor metastasis and cervical spondylotic myelopathy (degenerative changes and ligament hypertrophy in the cervical spine with loss of canal diameter). Vascular compromise results when there is occlusion of critical branches of either the anterior or posterior spinal arteries resulting in cord ischemia and necrosis.

Upper vs. Lower Motor Neuron Injury

Upper motor neuron injury results from damage to the descending tracts with preservation of reflex spinal activity below the site of the lesion. Findings on neurologic examination include:

- Increased muscle tone (spasticity)

- Brisk reflexes

- Involuntary muscle spasms

- Relative preservation of muscle bulk

- Weakness or paralysis

- Extensor plantar responses

Lower motor neuron injury results from damage to the cell bodies or axons of the lower motor neurons. Findings include:

- Decreased muscle tone (flaccidity)

- Absent reflexes

- Marked muscle atrophy

- Weakness or paralysis

The spinal cord ends at about the level of the L1 vertebral body. The most caudal portion of the spinal cord is termed the conus medullaris. Most injuries above the conus medullaris will result in upper motor neuron findings in the lower extremities. An injury to the conus will damage both the terminal portion of the cord and multiple nerve roots, resulting in primarily lower motor neuron findings involving the low lumbar and sacral levels. The continuing bundle of nerve roots below the conus medullaris is named the cauda equina. Injury at this level will result in lower motor neuron findings exclusively. An occasional individual will have injury to the blood supply of the spinal cord (infarction) and show unexpected flaccidity and atrophy below the level of infarction, indicating an injury to anterior horn cells.

Some individuals with long-standing injuries will show gradual change from upper to lower motor neuron findings. Although this change is common in those who are 20 or 30 years from time of injury, it could indicate development of new cord pathology. For additional information, see Chapter 10.

Neurologic Examination

Accurate neurologic examination is required in treating both acute and chronic SCI. A standardized system for classification of neurologic function in SCI has been developed by the American Spinal Injury Association (ASIA)[1, 2]. Use of a standardized system allows:

- Determination that neurologic function is remaining stable over time, even with different examiners

- Effective communication between physicians regarding patients

- Prediction of an expected level of function for a patient based on motor level[3]; for example, persons with C5 ASIA A tetraplegia will require assist for transfers[4]

- Assessment of the effectiveness of interventions to treat SCI

The ASIA classification system uses findings from the motor and sensory examination to determine the level of injury and degree of preserved neurologic function. Additional components of the examination, as described below, allow further characterization of impairment.

The motor examination for ASIA classification involves manual muscle testing of ten key muscles bilaterally (Table 1-2). Strength is graded on a 0-5 scale: 0=absent; 1=trace; 2=less than antigravity; 3=antigravity; 4=less than normal; 5=normal. The examination also includes assessment of voluntary anal contraction, graded as either present or absent.

The sensory examination required for ASIA classification requires testing of light touch sensation and sharp/dull discrimination in each dermatome from C2 through S4-S5 (tested as a single dermatome). The presence or absence of sensation on rectal examination is also recorded (see Dermatone Chart from ASIA Standards).

TABLE 1-2. KEY MUSCLES USED IN NEUROLOGIC TESTING		
Root	**Movement**	**Muscle(s)**
C5	Elbow Flexion	Biceps, brachialis
C6	Wrist Extension	Extensor carpi radialis longus and brevis
C7	Elbow Extension	Triceps
C8	Finger Flexion	Flexor digitorum profundus to middle finger
T1	Finger Abduction	Abductor digiti minimi
L2	Hip Flexion	Iliopsoas
L3	Knee Extension	Quadriceps
L4	Ankle Dorsiflexion	Tibialis anterior
L5	Long Toe Extension	Extensor hallucis longus
S1	Ankle Plantarflexion	Gastrocnemius and soleus

Additional examination components

Although not required for ASIA classification, the following are essential components of a comprehensive neurologic examination.

- Motor: Depending on the neurologic status of the patient, additional muscles to test may include the diaphragm (assessed with either fluoroscopy or measurement of vital capacity), deltoids, hip extensors, and hip abductors.

- Sensory: Assessment of vibratory sensation and proprioception tests the function of the dorsal columns.

- Deep tendon reflexes (DTR) and spasticity: These give information on degree of upper vs. lower motor neuron involvement. In the first few weeks following SCI, there is a depression or loss of DTRs, which in the case of upper motor neuron injury is followed by development of brisk reflexes and spasticity. In the case of lower motor neuron injury, reflexes remain absent and there is no development of spasticity.

STANDARD NEUROLOGICAL CLASSIFICATION OF SPINAL CORD INJURY

MOTOR

KEY MUSCLES

R L

	C2
	C3
	C4
Elbow flexors	C5
Wrist extensors	C6
Elbow extensors	C7
Finger flexors (distal phalanx of middle finger)	C8
Finger abductors (little finger)	T1
	T2
	T3
	T4
	T5
	T6
	T7
	T8
	T9
	T10
	T11
	T12
	L1
Hip flexors	L2
Knee extensors	L3
Ankle dorsiflexors	L4
Long toe extensors	L5
Ankle plantar flexors	S1
	S2
	S3
	S4-5

0 = total paralysis
1 = palpable or visible contraction
2 = active movement, gravity eliminated
3 = active movement, against gravity
4 = active movement, against some resistance
5 = active movement, against full resistance
NT = not testable

Voluntary anal contraction (Yes/No)

MOTOR SCORE

TOTALS + =
(MAXIMUM) (50) (50) (100)

SENSORY
KEY SENSORY POINTS

Dermatome Chart (from ASIA Standards)

LIGHT TOUCH — PIN PRICK

R L R L

| C2 | C3 | C4 | C5 | C6 | C7 | C8 | T1 | T2 | T3 | T4 | T5 | T6 | T7 | T8 | T9 | T10 | T11 | T12 | L1 | L2 | L3 | L4 | L5 | S1 | S2 | S3 | S4-5 |

0 = absent
1 = impaired
2 = normal
NT = not testable

Any anal sensation (Yes/No)

TOTALS + + = **PIN PRICK SCORE** (max: 112)
= **LIGHT TOUCH SCORE** (max: 112)
(MAXIMUM) (56) (56) (56) (56)

NEUROLOGICAL LEVELS
The most caudal segment with normal function

R L
SENSORY
MOTOR

COMPLETE OR INCOMPLETE?
Incomplete = Any sensory or motor function in S4-S5

ASIA IMPAIRMENT SCALE

ZONE OF PARTIAL PRESERVATION
Partially innervated segments

R L
SENSORY
MOTOR

Reprinted with permission of the American Spinal Injury Association, from International Standards for Neurological and Functional Classification of Spinal Cord Injury (revised, 1996)

Neurologic Classification

Neurologic level

By convention, the neurologic level refers to the most caudal level with normal function, rather than the first level with abnormal function. For sensation, the neurologic level is defined as the most caudal level with normal light touch and sharp/dull discrimination, provided all rostral levels have normal sensory function. For motor function, the neurologic level is defined as the most caudal key muscle with at least 3/5 strength, provided that all rostral levels have normal motor function.

The level is determined separately for motor and sensory functions, as well as for right and left sides, since frequently there is asymmetry or a lack of correspondence between motor and sensory level. For example, a level of C7 motor, C6 sensory would indicate a patient with at least 3/5 strength in the C7 key muscle (triceps) bilaterally with normal strength in all rostral groups, and with sensory function intact in the C6 dermatome and in all rostral dermatomes.

ASIA Impairment Scale

The ASIA Impairment Scale defines complete and incomplete injuries and categorizes the incomplete injuries as shown in Table 1-3. A complete injury is defined as an absence of motor and sensory function in the S4-S5 dermatome or sensation on rectal examination, and an incomplete injury is defined as a preservation of sensation in the S4-S5 dermatome or sensation on rectal examination[5].

The ASIA Impairment Scale is a modification of a previous scale, the Frankel Scale, and there is a general agreement between the two scales, such that an ASIA C is similar to a Frankel C. Many individuals with chronic SCI remain classified with this older terminology.

TABLE 1-3.
ASIA IMPAIRMENT SCALE (ADAPTED FROM ASIA DEFINITIONS)

A= Motor complete, sensory complete:
No motor or sensory function is preserved in the sacral segments S4-S5.

B= Motor complete, sensory incomplete:
Sensory but not motor function is present below NL* and includes the S4-S5 dermatome.

C= Motor incomplete, sensory incomplete:
Motor function is preserved below NL, and the majority of key muscles below the NL are less than grade 3.

D= Motor incomplete, sensory incomplete:
Motor function is preserved below NL, and at least half of key muscles below NL are grade 3 or more.

E= Normal:
Motor and sensory function are normal.

*Neurologic level

Clinical Syndromes

Distinct patterns of neurologic deficit are recognized with incomplete SCI. It is common for certain pathways to be relatively spared from injury, and this results in recognizable syndromes.

Central cord syndrome

- Occurs with cervical level injuries, often in older individuals with preexisting cervical spinal canal narrowing

- Results from preferential damage to upper limb corticospinal tracts

- Greater weakness in upper limbs than in lower limbs

Brown-Sequard syndrome

- Results from asymmetric cord lesion, classically thought of as a cord hemisection, although a true hemisection is uncommon with trauma

- Relatively greater proprioceptive and motor loss ipsilateral to the lesion, with contralateral loss of sensitivity to pain and temperature

Anterior cord syndrome

- Results from occlusion of anterior spinal artery

- Variable loss of motor function and of sensitivity to pain and temperature with preservation of proprioception

Conus medullaris syndrome

- Injury to the sacral cord and lumbar nerve roots within the spinal canal

- Usually results in lower motor neuron findings, although sacral reflexes may occasionally be preserved

Cauda equina syndrome

- Injury to the lumbosacral nerve roots within the neural canal resulting in areflexic bladder, bowel, and lower limbs

- A purely lower motor neuron injury

Alteration of Autonomic Nervous System Function

Loss of motor and sensory function is obvious in a person with SCI. Alterations of autonomic function are not as visible, although they have significant consequences for the individual. The sympathetic supply to the entire body, including the head, leaves the spinal cord through the roots between T1 and L2. Parasympathetic supply to the body is derived from the vagus nerve (usually spared in SCI) and sacral roots. Varying degrees of dysfunction result based on the level of the injury. Changes in autonomic function are in fact a source of many medical complications in persons with SCI.

- A complete cervical-level lesion, with interruption of all sympathetic input to the body, may result in unopposed vagal input to the heart causing marked bradycardia.

- Impairment of vasoconstriction may result in a baseline low blood pressure as well as orthostatic hypotension for an individual with tetraplegia[6].

- Autonomic dysreflexia results from loss of descending sympathetic control of responses to noxious stimuli (see Chapter 3) [7].

- Impaired ability to modulate blood flow to the skin and control of sweat gland secretion alters the control of thermoregulation. The normal temperature of a person with SCI may be 1-2 degrees Fahrenheit cooler than for a neurologically intact person. Thus, a relatively mild temperature elevation may actually indicate a significant infection. Persons with SCI are also predisposed to development of body temperature elevation in a warm environment.

- Neurogenic bladder dysfunction results from impaired storage and emptying.

- Neurogenic bowel dysfunction results from impaired peristalsis and evacuation.

- Sexual dysfunction derives from impaired sensation, erection, ejaculation, or vaginal lubrication.

Self-Study Review

1. *What are the important tracts of the spinal cord? Where are they located? What direction do they travel? What are their functions?*

2. *What are the clinical signs and implications for upper motor neuron injury vs. lower motor neuron injury? Where are these types of injury most likely to occur? Which types of SCI are likely to occur immediately? Which types of injury are more likely to be subacute or secondary? What are some types of nontraumatic injury?*

3. *List the key muscles to be tested for motor function along with the movement and corresponding neurologic root.*

4. *How are the sensory and motor neurologic levels defined?*

5. *What are the characteristics of the five severity levels on the ASIA Impairment Scale?*

6. *What are the differential diagnostic features of five SCI syndromes?*

7. *Describe the effects of autonomic dysfunction on several organ systems and the corresponding impairment of control.*

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2   REVIEW OF SYSTEMS

Stephen P. Burns, MD

Objectives for the Learner

1. Understand why serious medical illness may present with unusual or nonspecific symptoms and signs in an individual with SCI.

2. Understand the common factors that make the assessment process more difficult in a person with SCI.

3. Describe how the neurologic classification (level and completeness) of a person's injury influences the symptoms experienced with illness.

4. Recognize the variety of altered sensory patterns that can result following an SCI.

5. Understand the assessment implications of upper vs. lower motor neuron injuries.

6. Know how to alter the review of systems for persons with SCI.

7. Relate specific signs and symptoms in SCI to diagnostic concerns.

Overview

In a person with SCI, evaluation of a medical illness is much more difficult than in a neurologically intact person. Making a correct diagnosis in a timely fashion can be a challenge for physicians who are experienced with SCI, and it is even more difficult for those who only infrequently treat these individuals. A better understanding of signs and symptoms as well as knowledge of certain medical complications and common disorders in SCI, enables physicians to provide appropriate care.

Assessment

Factors that make assessment difficult include:

- Physicians may lack an understanding of alterations in basic physiology after SCI, such as an expected baseline low blood pressure in persons with tetraplegia.

- Most physicians do not have experience with medical disorders that occur almost exclusively in persons with SCI, such as autonomic dysreflexia.

- Loss of sensory function may mask the expected symptoms with illness, such as abdominal pain with cholecystitis.

- Persons with partial sparing of somatic or visceral sensation after SCI frequently have only vague and nonspecific symptoms when presenting with serious medical illnesses and may have difficulty in localizing the source of pain.

- Persons with SCI frequently experience neuropathic pain. It is sometimes difficult to determine whether the person's pain is solely neuropathic, or whether it is a symptom of internal pathology.

- Many persons with SCI already have painful conditions (shoulder, abdominal pain), and careful assessment of exacerbations or change in pain is needed.

Key steps in assessment:

- Determine the neurologic level and whether the injury is complete or incomplete (see Chapter 1). Most people with long-standing injuries can provide this information.

- Consider degree of sparing of sensory function. This provides clues to suggest whether a person will perceive symptoms of the pathology. Sparing of somatic and visceral sensation after SCI is variable and cannot be predicted solely by knowing whether the injury is complete or incomplete.

- Evaluate persons with complete (ASIA A) injuries who may have:
  - Complete loss of all sensory function below the lesion, including visceral sensation
  - Complete loss of sensory function as determined on ASIA neurologic examination, but with sparing of some vague, nondiscriminative sensation, or sparing of some visceral sensation below the neurologic level

- Evaluate persons with incomplete (ASIA B, C, or D) injuries who may have:
  - Incomplete loss of sensory function, with preservation of some degree of light touch or pinprick below the neurologic level, as well as some visceral sensation
  - Sensory function that is present but not normal

- Determine whether the injury is primarily upper or lower motor neuron. Increases in spasticity can occur with medical illness. For example, spasticity frequently increases with urinary tract infections. However, spasticity is absent in lower motor neuron injuries, so it is a useful symptom only in upper motor neuron injuries. Patients will generally be aware of changes in degree of spasticity.

Signs and Symptoms

SCI results in alteration of normal physiology and also increases the incidence of specific medical disorders, some of which are rare in persons without SCI. Knowing the level and type of SCI is essential in assessing symptomatology. All of the following signs and symptoms relate to a person with tetraplegia. The person with a mid-thoracic or lower neurologic level will generally have less risk for pulmonary or cardiovascular problems (except pulmonary embolism). They are also typically not at risk for autonomic dysreflexia. Signs or symptoms often give rise to a differential diagnosis dissimilar to that for a neuro-logically intact person (Table 2-1).

TABLE 2-1.
SIGNS AND SYMPTOMS AND DIAGNOSTIC CONCERNS

General

Fatigue/feeling lousy	Nonspecific but may be only complaint
	Sleep apnea (Ch. 5)
	Chronic alveolar hypoventilation with CO_2 retention
	Cardiac disease (Ch. 6)
Fever	Usually increased with infection
	Acute abdomen (Ch. 7)
	Deep venous thrombosis
	Urinary tract infection
	Abscess with pressure sore
	Pneumonia (Ch. 5)
	Environmental change
Weight gain	Not unusual in first years after SCI

Skin

Breakdown	Pressure ulcer (Ch. 4)
	Trauma
Sweating, flushing	Autonomic dysreflexia (Ch. 3)

Pulmonary

Cough	Pneumonia
Increased sputum production	Pneumonia
Shortness of breath after meals	Abdominal distention
Sleep apnea symptoms	Sleep apnea
Dyspnea	Pulmonary embolism
	Mucus plugging

Cardiovascular

Orthostatic hypotension	Common in acute SCI
	Can be normal finding
	Postprandial shunting
	Syringomyelia (Ch. 10)
Bradycardia	Can be an expected finding
Hypertension with tachycardia or bradycardia	Autonomic dysreflexia
Lower extremity swelling	Deep venous thrombosis (Ch. 6)
	Heterotopic ossification (Ch. 9)
	Occult fracture (Ch. 9)

TABLE 2-1. *(continued)*

Gastrointestinal

Changes in bowel program	Altered bowel management (Ch. 7)
	Colorectal malignancy
Stool incontinence	Altered bowel management
Diarrhea	Impaction
Rectal bleeding	Traumatic bowel care, hemorrhoids
Early satiety	Abdominal distention
Abdominal swelling/distention	Altered bowel management

Genitourinary

Urinary tract infections	Neurogenic bladder (Ch. 8)
Sexual dysfunction	Common with SCI

Musculoskeletal

Shoulder pain	Degenerative changes (Ch. 9)
Spine pain	Instability (Ch. 9)
Limited joint range	Contracture

Neurologic

Headache, facial flushing	Autonomic dysreflexia (Ch. 3)
Increased spasticity	Urinary tract infection (Ch. 8)
	Bowel impaction (Ch. 7)
	Pressure ulcer (Ch. 4)
	Ingrown toenail
	Any noxious stimulus
	Deep venous thrombosis (Ch. 6)
New loss of sensation or motor function or change in spasticity	Syringomyelia and related disorders (Ch. 10)
Hand numbness	Carpal tunnel syndrome (Ch. 10)
	Ulnar neuropathy

Self-Study Review

1. *What features complicate the assessment process in a person with SCI?*

2. *Explain the reason for nonspecific symptoms and signs in the presentation of illnesses for persons with SCI.*

3. *What is the differential effect on spasticity from upper vs. lower motor injury?*

4. *How will knowledge regarding level and completeness of injury provide information regarding pulmonary function, sensory findings, autonomic dysreflexia, and accuracy of self-report regarding a new onset of symptoms?*

5. *What are key signs and symptoms in persons with SCI that need to be addressed in a history and review of symptoms?*

3 AUTONOMIC DYSREFLEXIA

Lance L. Goetz, MD

Objectives for the Learner

1. Describe the relationship between autonomic dysreflexia (AD) and neurologic level, neurologic completeness, and time since injury.

2. Understand the pathophysiological mechanisms that underlie AD.

3. Describe the most common stimuli that can trigger an acute episode of AD.

4. Accurately and promptly recognize and diagnose AD to allow for rapid treatment and prevention of life-threatening complications.

5. Know the monitoring approaches, conservative interventions, pharmacologic interventions, and prevention/long-term management strategies for AD.

Overview

AD is a potentially life-threatening condition in which noxious visceral or cutaneous stimuli cause a sudden, massive, uninhibited reflex sympathetic discharge in individuals with high-level SCI. Prompt recognition of symptoms and rapid treatment are critical in the prevention of life-threatening complications.

Incidence

One or more episodes of AD occurs in up to 85% of persons with SCI at or above the sixth thoracic neurologic level[1].

Pathophysiology

AD is an exaggerated increase in blood pressure in response to a noxious stimulus arising from a source below the level of the SCI. It occurs when spinal shock resolves and spinal-mediated sympathetic vasomotor reflexes return. The accepted mechanism for AD involves complex neurovascular processes[2]. The normal sympathetic response to a painful stimulus is not balanced by supraspinal inhibitory control because descending impulses are blocked by the SCI. The resultant tachycardia and vasoconstriction produce blood pressure increases that trigger sensory information from the carotid bulb and atrial baroreceptor to the brainstem. The efferent vagal response may control tachycardia (often producing bradycardia) but has no effect on vasoconstriction and blood pressure. Signs and symptoms are due to both exaggerated systemic sympathetic activity and inhibited local sympathetic activity above the level of injury where descending influences are intact. Of foremost concern is a patient complaint of headache and the need to rule out a hypertensive crisis.

There are milder manifestations of AD with blood pressure elevations to a lesser degree. Because the baseline blood pressure for an individual with tetraplegia is often 90-110/60 mm Hg, a blood pressure of 140/90 mm Hg likely represents moderate AD.

AD occurs in persons with neurologic levels at or above the sixth thoracic level because such lesions disrupt control over the major sympathetic outflow (thoracolumbar outflow, from approximately T6-L2). AD is more likely to occur in persons with complete injuries but has been seen in persons with incomplete injuries as well.

The most common noxious stimuli are distention of hollow viscera such as the bladder (80% of episodes) or the bowel[1,2]. The causes can be as benign as an ingrown toenail or as serious as an acute abdomen. Table 3-1 lists common noxious stimuli below the level of the SCI.

TABLE 3-1.
AUTONOMIC DYSREFLEXIA: PARTIAL LISTING OF COMMON CAUSES

Genitourinary
  Bladder distention, e.g., catheter plugging, obstructed voiding, urodynamics
  Urethral distention, e.g., inflation of Foley catheter in the urethra
  Manipulation, e.g., traction on catheter, cystoscopy
  Urinary tract infections, epididymitis, orchitis, testicular torsion
  Renal calculi
  Genital stimulation, intercourse
  Gynecological examination
  Uterine contraction, e.g., parturition, menses

Gastrointestinal
  Bowel distention, fecal impaction
  Manipulation, e.g., digital exam, suppository insertion, digital rectal stimulation
     for bowel care
  Acute abdomen
  Peptic ulcer disease
  Gastroesophageal reflux
  Biliary colic

Dermatologic
  Pressure sores
  Cutaneous stimulation, e.g., tight clothing, sunburn, cold exposure, lacerations, bruises
  Ingrown or infected toenails

Musculoskeletal
  Fractures, fracture manipulation
  Range of motion with stretch of soft tissues or spastic muscles
  Heterotopic ossification (see Chapter 9)

Cardiovascular/Pulmonary
  Deep venous thrombosis
  Pulmonary emboli
  Angina/myocardial infarction
  Pneumonia

Other
  Medications: digoxin, intravenous norepinephrine
  Radiologic procedures

Signs and Symptoms

- Blood pressure can exceed 260 mm Hg systolic and 220 mm Hg diastolic.

- Not all of the following symptoms may be present, especially with mild or moderate AD.

- With long-standing SCI, patients typically have AD with minimal to no symptoms and may just report that "something is not right."

- Any complaint of headache in this population requires an evaluation of blood pressure.

TABLE 3-2.
AUTONOMIC DYSREFLEXIA: SYMPTOMS[3]

Symptom	Cause
Headache	Elevated blood pressure
Visual changes (blurred vision, spots)	Elevated blood pressure
Altered heart rate (bradycardia, arrhythmia)	Vagal response to elevated blood pressure
Flushing, diaphoresis, piloerection above injury level	Excessive vasodilation in response to elevated blood pressure
Nasal congestion	Excessive vasodilation in response to elevated blood pressure

Diagnosis

Diagnosis depends on an understanding of symptoms, prompt recognition of signs, and measurement of blood pressure.

Treatment[4]

Search for inciting causes because alleviation will quickly result in resolution of the episode of AD and return of blood pressure to normal.

- Check the blood pressure and pulse and monitor them every two to five minutes.

- Sit the patient upright to lower blood pressure by vascular pooling.

- Loosen or remove tight clothing, including belts, leg drainage bag straps, and shoes.

- Quickly identify and alleviate any obvious causes of noxious stimuli, beginning with the urinary system.

- Evaluate urinary drainage appliances for kinks, tightness, and placement.

- If a catheter is present and appears blocked, gently irrigate it. Caution must be used because this may aggravate the rise in blood pressure. If it does not drain, remove and replace it after instilling 2% lidocaine in the urethra.

- If no catheter is present, catheterize the patient after instilling 2% lidocaine in the urethra.

- If these measures do not relieve the patient's symptoms and lower the blood pressure, the rectal vault should be gently checked for stool using a gloved finger, again lubricated with lidocaine jelly. If stool is present, lidocaine jelly should be applied to the perianal area and allowed to anesthetize it for five minutes. The stool should then be gently removed digitally.

- If neither the bladder nor the bowel appears to be the source, examination should be made for other causes as listed in Table 3-2, with special attention to examination of the skin for lesions or other signs of trauma.

- Urine culture should be sent if indicated.

- There are occasions when a cause is not readily found.

Pharmacologic intervention may be required for severe AD and can be instituted at any time during an episode. The timing of medication use depends on the degree of blood pressure elevation and the likelihood of quickly alleviating the inciting cause. As soon as the cause is removed, blood pressure will quickly return to normal. It is generally accepted

that blood pressure elevations above 150-180 mm Hg systolic warrant medication use. Also consider medications before any noxious stimulus that may aggravate the rise in blood pressure. Should hypotension occur because of medication use and removal of the noxious stimulus, place the person supine and elevate the feet. If the cause of AD cannot be immediately removed, medication management may need to continue until resolution of the event. Many patients have their own supply of medication and are often familiar with their needs regarding usage.

- If the patient's blood pressure is greater than 180 mm Hg systolic, give nifedipine 10 mg (immediate release form). The patient is instructed to bite through the capsule and swallow it. The dosage may be repeated after 15 minutes if necessary.

- Alternative treatments include 2% nitroglycerin ointment, 1 inch to the upper chest or back. An additional inch may be given after 15 minutes if needed. This medication has the advantage of easy removal if the blood pressure falls precipitously.

- For severe episodes not responding to the above measures, transfer cases to medical intensive care and give an intravenous drip of sodium nitroprusside.

- Systolic blood pressure can be safely lowered to 90-110 mm Hg, which is a typical baseline for a person with tetraplegia.

Blood pressure monitoring every 15-30 minutes for 4 hours after the episode is important because of the sensitivity to recurrence at this time. Especially monitor blood pressure during a positional change back to supine. Reduction in blood pressure may have been due to medications rather than resolution of the noxious stimulus. In addition, some patients exhibit rebound hypotension.

Prevention and long-term management

- Ensure low pressure bladder drainage and regular bowel care.

- Persons who are at risk for AD are instructed to carry a medical alert card in their wallet which describes the symptoms and treatment of this syndrome.

- Chronic prophylaxis with medications is sometimes warranted.

- Surgery usually requires use of local, regional, or general anesthetic in preventing AD, even in persons without sensation.

Self-Study Review

1. *Why should AD always be regarded as a medical emergency?*

2. *Explain the pathophysiological mechanisms underlying AD.*

3. *What is the relationship of AD to neurologic level, neurologic completeness, and time since injury?*

4. *Explain which symptoms of AD are related to elevated blood pressure, vagal response, or excessive vasodilation.*

5. *What conditions result in the majority of episodes of AD?*

6. *What are the sequential steps in the treatment of AD?*

7. *What are the indications for pharmacologic intervention?*

8. *How often should blood pressure be monitored during AD? How long should blood pressure be monitored following an episode?*

9. *List at least four strategies for the prevention and/or long-term management of AD.*

10. *Rank order six classes of stimuli having potential for triggering AD in order of their likelihood as an exacerbating source.*

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4   NEUROGENIC SKIN AND PRESSURE ULCERS

Barry Goldstein, MD, PhD

Objectives for the Learner

1.   Appreciate the costs and human suffering that are associated with pressure ulcers as a common secondary complication of SCI.

2.   Accurately classify the four stages of pressure ulcers by their clinical presentations.

3.   Know the key components of an appropriate evaluation for a person with SCI presenting with a pressure ulcer.

4.   Create a treatment plan for an individual with a pressure ulcer that optimizes wound healing.

5.   Know the components necessary to develop a pressure ulcer prevention program for individuals with SCI.

Overview

Following SCI, areas of insensate, denervated (neurogenic) skin undergo physiological changes, making it more susceptible to breakdown. The perception of the need to change body position after being in one position is often absent. The functional need to physically shift weight to relieve pressure is difficult to practice without the sensory stimulus. This maneuver is called a "pressure relief."

Pressure ulcers are a diverse group of pathologic states that affect one or more of the body wall tissues. They include minor reversible changes in the most superficial epidermis to major invasive ulcers that extend to bone or joint cavity.

Management of pressure ulcers and skin breakdown are among the most common and challenging problems faced by physicians who see individuals with SCI. Abundant clinical and research-based literature have demonstrated staggering costs and human suffering from these chronic wounds[1, 2]. The average cost of treatment for a single pressure ulcer episode has been estimated to be between $30,000-$120,000; the total annual cost of treating pressure ulcers in the United States is approximately $6.5 billion[3].

Incidence

Pressure ulcer prevalence and incidence is extremely high in this population, and more than 50% of individuals with chronic SCI experience some breakdown in their lifetime.

Pathophysiology

Excessive and/or prolonged tissue pressure that exceeds capillary pressure results in tissue ischemia and skin breakdown. Whereas direct pressure may be greatest at the bone-soft tissue interface and contribute to initial deep tissue loss that extends to the surface, shear forces and friction also contribute to superficial breakdown.

Signs and Symptoms

- Signs and symptoms are usually local although systemic symptoms do occur (fever, chills, anorexia, weight loss).

- A careful history includes overall health, mental and psychological status, condition of equipment, and social circumstances.

- Establishing an etiology (change in routine such as travel on an airplane; trauma even as minor as a scrape during a transfer; fewer "pressure reliefs"; changes in seating, cushions, and/or mattress) is critical to future prevention.

- Attend to reports of "just a scrape" or "a pimple" or "a boil," as the onset may appear minor.

Diagnosis

- Define characteristics of the wound (e.g., location, duration, appearance, odor, exudate).

- Determine the extent of the ulcer, based on the tissue level involved and the wound dimensions. Pressure ulcers are commonly minimized in the initial assessment because the visible superficial changes are insignificant in contrast to deeper changes (Table 4-1).

- Describe the ulcer and surrounding tissue with respect to location, erythema, odor, color, necrosis, undermining and tracts, capillary refill, and warmth.

- Rule out osteomyelitis, bacteremia, and cellulitis since these complications occur in association with pressure ulcers[4].

- Obtain ESR, CBC, and plain film X-rays to help in the initial assessment for complications; blood cultures should be obtained if sepsis is suspected.

- Perform a nutritional assessment in the initial evaluation[5,6]. Clinically significant malnutrition is diagnosed if body weight has decreased more than 15% or if serum albumin is less than 3.5 mg/dL. Malnutrition might result from, rather than be the cause of, a pressure ulcer since significant protein loss might result from an open wound.

TABLE 4-1.
METHODS OF DETERMINING WOUND EXTENT[7]

Parameter	Noninvasive	Invasive
Level	Visual, ultrasound	Surgical debridement, biopsy
Area	Linear measurement, acetate tracing, planimetry	
Volume	Linear measurement, MRI, ultrasound	Liquid capacity, gel molds

Classification

A uniform assessment tool is essential for communication among health care practitioners. The following staging system has been recommended by the U.S. Department of Health and Human Services, Treatment of Pressure Ulcers Guideline Panel[8]:

Stage I: Nonblanchable erythema of intact skin, the heralding lesion of skin ulceration. In individuals with darker skin, discoloration of the skin, warmth, edema, induration, or hardness may also be indicators.

Stage II: Partial-thickness skin loss involving epidermis, dermis, or both. The ulcer is superficial and presents clinically as an abrasion, blister, or shallow crater.

Stage III: Full-thickness skin loss involving damage to or necrosis of subcutaneous tissue that may extend down to, but not through, underlying fascia. The ulcer presents clinically as a deep crater with or without undermining of adjacent tissue.

Stage IV: Full-thickness skin loss with extensive destruction, tissue necrosis, or damage to muscle, bone, or supporting structures (e.g., tendon, joint capsule). Undermining and sinus tracts may also be associated with Stage IV pressure ulcers.

Treatment

Improve the individual's general health[9] and minimize factors that interfere with wound healing.

- Provide aggressive nutritional support and rehydration. Approximately 30-35 calories/kg/day and 1.25-1.50 grams of protein/kg/day intake are recommended while an individual is healing a pressure ulcer.

- Correct nutritional deficiencies (vitamin C, vitamin K, and zinc supplementation are recommended if deficiencies are suspected).

- Correct anemia.

- Treat co-morbid conditions that interfere with wound healing.

- Improve oxygenation by improving pulmonary disease, cardiac disease, anemia, fever, and malignancy.

- Minimize exogenous factors (anticoagulants, antimetabolites, corticosteroids).

Optimize wound healing by eliminating all mechanical forces applied to the wound in bed or wheelchair. Unfortunately, this means loss of independence due to the contraindication to sit for meals, bowel programs, and usual activities of daily living if the wound is located on a sitting surface.

- Use a special mattress or mattress overlay to reduce tissue interface pressure.

- Use positioning devices to remove all pressure on the wound, such as pillows to elevate the calf if there is a heel ulcer. Ring devices ("donuts") should not be used.

- Assist with turns in bed to avoid shear forces.

- Evaluate and avoid trauma to wound during transfers.

- Confirm that there is no pressure on wound while in the wheelchair.

- Check and change wheelchair cushion, if necessary.

Debride necrotic tissue.

- Sharp debridement rapidly removes devitalized, infected tissue and is the preferred technique for thick, fibrous, adherent tissue. Sharp debridement is done with sterile instruments, and clean, dry dressings should be applied for 24 hours following the procedure.

- Wet-to-dry dressings are also used to debride necrotic tissue.

- Enzymatic debridement is effective for relatively small amounts of fibrous tissue within the ulcer.

- Many types of hydrotherapy are used to clean pressure ulcers.

- Consider use of analgesics (many individuals with SCI have sensation) because of the risk for autonomic dysreflexia (in individuals with neurologic levels at T6 and above).

Optimize the local wound environment.

- Keep the wound clean.
  - Irrigate wounds gently with saline solution at each dressing change.
  - Avoid antiseptic agents and cleansers such as Betadine that are cytotoxic to new granulation tissue.
  - Protect the wound from contamination (e.g., feces, other wounds, other patients).

- Keep the wound moist, yet avoid moisture on the surrounding intact tissue.

- Choose a dressing that:
  - controls exudate
  - maintains a moist wound and avoids dessication
  - minimizes caregiver or nursing time (some occlusive dressings remain undisturbed for days while moist saline gauze must be changed frequently each day)

- In applying the dressing:
  - fill cavities and tracts with packing material
  - avoid over-packing the wound by not using pressure during the packing

Surgical repair is often indicated for a Stage III or worse pressure ulcer and should be performed in an SCI center. Postoperative treatment requires attention to joint range, careful mobilization and safe transfers, progressive sitting times, and appropriate equipment.

Prevention[10]

It is unreasonable to expect that superficial breakdown will never occur (Stages I and II). Rather, the major objective of a prevention program is early detection and the appropriate response (immediate non-weight-bearing on the affected area) to avoid Stages III and IV.

Daily inspection is the cornerstone of a successful prevention program and involves careful examination of areas at risk twice a day. Whether this is done by the individual or an attendant/family member, it is mandatory that the examination include both visual and palpatory inspection (edema, warmth, induration) since skin breakdown often occurs in deeper layers and is not always detectable by visual inspection.

Review "pressure reliefs" to ensure complete removal of sitting pressure on weight-bearing surfaces by doing a wheelchair push-up at least every 15 minutes. Frequent turns distribute and relieve pressure when in bed.

Educational programs should emphasize

- Meticulous skin care and proper skin hygiene with daily bathing with nonirritating, nonalkaline soaps

- Close vigilance

- Good nutrition with avoidance of tobacco and obesity

- Avoidance of excessive moisture (sweating, incontinence). Excessive sweating, particularly in the region of the groin, is a significant problem and can be treated with underpads or briefs, topical skin barriers (e.g., ointments and pastes), and antiperspirant deodorants.

- Limited sun exposure and use of sunscreens

- Avoidance of dry skin, including use of moisturizers to prevent cracking

- Maintenance of equipment and cushions

- Increasing time since SCI and aging are important risk factors[11].

- Review "pressure reliefs" when function declines with aging and during illness.

Minimizing tissue loads is imperative for long-term prevention of skin problems[12]. Extrinsic factors relate to interface materials (cushions and mattresses), positioning, and equipment. Individual factors relate to personal habits and biological condition.

- Maintain wheelchair cushions (neglect often leads to the development of a pressure ulcer).

- New situations, such as unfamiliar surroundings, trips (prolonged sitting in a vehicle, sitting in an airplane seat, not having the usual equipment in a bathroom), and changes in daily schedules warrant evaluation.

- Pressure reliefs and turns at night remain the most effective preventative approach, yet many individuals do not, or are unable to, incorporate this into their lives.

Self-Study Review

1. *What is the incidence of pressure ulcers in the SCI population?*

2. *Preventing one episode of a skin pressure ulcer could potentially achieve what cost savings?*

3. *What is the clinical presentation for each pressure ulcer stage? Why is this important?*

4. *What other essential components should be included in the appropriate evaluation of skin pressure ulcers beyond describing the characteristics, etiology, and extent of the wound?*

5. *Describe several interventions that could improve the person's general health while facilitating healing of the pressure ulcer.*

6. *What components should be included in a successful pressure ulcer prevention program?*

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5 PULMONARY SYSTEM

James W. Little, MD, PhD

Objectives for the Learner

1. Identify the neurologic levels that increase the risk of failed cough, failed ventilation, and respiratory complications after SCI.

2. Recognize the signs and symptoms of atelectasis, pneumonia, aspiration, mucous plugging, and failed ventilation after SCI.

3. List medical treatments for atelectasis, pneumonia, aspiration, mucous plugging, and failed ventilation.

4. Identify and structure prevention strategies for potential respiratory complications after SCI by focusing on both ventilation and cough.

5. Know appropriate treatment recommendations for deep venous thrombosis and pulmonary embolism after SCI.

Overview

Common pulmonary complications of SCI are impaired cough leading to pneumonia, impaired ventilation sometimes requiring use of a mechanical ventilator, and deep venous thrombosis with pulmonary embolism.

Anatomy and Pathophysiology

Ventilation is primarily due to diaphragmatic function (innervated by C3,4,5) and secondarily due to the sternocleidomastoid, scalenes, trapezius, and external intercostals. Cough is primarily due to function of the abdominal muscles (innervated T6-T12) and internal intercostals.

• Ventilatory failure due to upper cervical cord lesions (e.g., C1-C3) requires mechanical ventilation. Unilateral or bilateral diaphragm paralysis (partial or complete) also predisposes to atelectasis and pneumonia.

- Persons with higher levels of SCI show restrictive pulmonary function with low forced vital capacity (FVC). Chest wall expansion is deficient causing reduced lung and chest wall compliance. Those with FVC < 1.0 liter often require mechanical ventilation. The low FVC results from diaphragm weakness and from inefficient paradoxical breathing (i.e. no chest wall expansion during inspiration due to nonfunctioning intercostals, T1-T11).

- Inadequate cough due to cervical or upper thoracic cord lesions (T8 or higher) may require assisted cough ("quad cough") to clear secretions. Ineffective cough leads to retained secretions, mucous plugging, and pneumonia. A quad cough is the delivery of a forceful abdominal compression directed at the diaphragm and synchronized with the person's attempt to cough.

- The parasympathetic imbalance, due to absent sympathetic outflow, leads to bronchoconstriction and to increased mucous secretion.

- Vocal cord paralysis, gastroesophageal reflux, and ileus can lead to recurrent aspiration.

- Baseline respiratory rate in a person with tetraplegia is often 14-18.

- Baseline blood gases are usually normal.

PNEUMONIA

Incidence

The incidence of pneumonia can vary from 6.0% to 37.8% following acute SCI, but at annual follow-ups, the incidence varies from 1% to 6.4%, depending on the neurologic level and completeness of injury.[1]

Signs and Symptoms

Atelectasis, pneumonia, and recurrent aspiration may initially present with an increased respiratory rate as the only objective finding. Additional manifestations (as in the able-bodied) include fever, dyspnea, purulent sputum, and decreased breath sounds. Physical examination can be challenging due to the inability to inspire deeply.

Diagnosis

In addition to the usual studies, consider a swallowing study to identify the potential for aspiration and a diaphragm evaluation with fluoroscopy or ultrasound to identify diaphragm paralysis.

Treatment

In addition to antibiotics for pneumonia, therapy includes incentive spirometry, chest clapping and postural drainage, quad cough, intermittent positive pressure breathing (IPPB), bronchodilators (either beta-agonists such as metaproterenol or anticholinergics such as ipratropium), and aggressive use of bronchoscopy. Mechanical ventilation may be required. Some SCI centers use the insufflator-exsufflator (a device which provides positive pressure inflation of the lungs followed by a sudden shift to negative pressure exhalation, yielding high air-flow comparable to a cough that can clear bronchi and bronchioles[2]).

Prevention

Education in early recognition and treatment of respiratory infections is essential. Persons with SCI should avoid smoking, and at the earliest symptom of congestion or infection, increase use of incentive spirometry, increase fluid intake and humidification, and perform frequent quad coughs and postural drainage. One-time pneumococcal and yearly influenza vaccinations may reduce respiratory infections.

FAILED OR IMPAIRED VENTILATION

Incidence

High-level tetraplegia may require tracheal intubation and mechanical ventilation initially after SCI. Many individuals are subsequently weaned from the ventilator as their diaphragm function improves. A minority will require lifelong mechanical ventilation[3] or may experience late-onset ventilatory failure.

Pathophysiology

Factors that contribute to late-onset ventilatory failure include:

- Late neurologic decline (e.g., cervical stenosis with compression, posttraumatic syringomyelia)

- Obesity

- Recurrent atelectasis and pneumonia

- Progressive kyphosis or scoliosis

- Loss of diaphragm motoneurons with aging

Signs and Symptoms

Suspect late ventilatory failure when there is:

- Tachypnea

- Dyspnea

- Daytime drowsiness

- Fluctuating mental alertness

- Unexplained erythrocytosis

- Increased positional influences on breathing. Marginal diaphragm function may be most evident in certain positions. Sitting usually compromises marginal ventilation more than lying supine. Side-lying with the most functional diaphragm positioned down is often more compromising than with the most functional diaphragm up.

- Respiratory muscle fatigue

- Sleep apnea (central or obstructive)

Diagnosis

Arterial blood gas, pulmonary function tests, oximetry and a sleep study[4] can confirm ventilatory failure or obstructive sleep apnea.

Treatment

Interventions can include:

- Stopping cigarette use

- Continuous positive air-way pressure (CPAP) or bilevel positive airway pressure (BIPAP) at night

- Oxygen supplementation

- Noninvasive mouth-mask ventilation[5]

- Tracheostomy for obstructive apnea

- Mechanical ventilation

Previously ventilated, high-level quadriplegics (i.e., C3-C5), who are at greatest risk for late-onset ventilatory failure, need a preventive home program of:

- Daily postural drainage, incentive spirometry, and assisted coughing

- Glossopharyngeal breathing (the process of increasing vital capacity by a learned technique of forcing air from the oropharyngeal cavity into the lungs) to increase tidal volume

DEEP VENOUS THROMBOSIS AND PULMONARY EMBOLISM

Incidence

Deep venous thrombosis (DVT) is common and can lead to fatal pulmonary embolism (PE) in persons with SCI. The highest risk is in the first 3 months after acute SCI.

Pathophysiology

Increased risk after acute injury is due to hypercoagulability, venous stasis, and vessel wall injury. In chronic SCI, DVT/PE risk is increased such as after major surgery, with prolonged immobilization, and after lower limb fractures.

Signs and Symptoms

- Signs such as calf swelling, venous distension, or venous cords are unreliable. The physician must have a high suspicion, especially of an unexplained fever, and use sensitive diagnostic tests (e.g., venous Duplex examination).

- Persons with chronic SCI and a history of DVT who have new lower limb swelling are challenging. A venous Duplex examination is often positive but cannot distinguish new versus old DVT. Full anticoagulation for possible recurrent DVT may be given without a definite diagnosis.

Diagnosis

- Pulmonary embolism is often misdiagnosed as pneumonitis in this population.

- Symptoms of PE (dyspnea, cough, hemoptysis) require a ventilation-perfusion lung scan.

Treatment

- Treatment for DVT/PE is the same as for the able-bodied. There is evidence that recanalization of lower extremity veins takes longer.

- If anticoagulation fails (i.e., continuing DVT or PE, bleeding complication), vena caval interruption may be needed (e.g., Greenfield filter). However, "quad coughs," which are essential to facilitate mobilization of secretions, are contraindicated in individuals with a vena caval filter because the filter can be displaced and migrate.

Self-Study Review

1. *Why is inadequate cough more common in individuals with injuries at T8 or higher?*

2. *List the treatment for atelectasis, pneumonia, aspiration, and mucous plugging that is unique to persons with SCI.*

3. *If you were going to design a pulmonary complications prevention program for individuals with SCI, which elements would you be sure to include?*

4. *What are the signs and symptoms that would lead you to suspect late ventilatory failure?*

5. *How is diagnosis of deep venous thrombosis and pulmonary embolism complicated in individuals with SCI and prior DVT?*

6. *What is unique about the treatment for DVT and PE in persons with SCI?*

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6 CARDIOVASCULAR SYSTEM

Steven A. Stiens, MD, MS

Objectives for the Learner

1. Explain how the altered anatomy and physiology affect the cardiovascular system and contribute to the extreme hypotension and hypertension under various conditions after SCI.

2. Identify modifications in the interpretation of diagnostic procedures in persons with SCI.

3. Increase awareness of several cardiac arrhythmias that are common in persons with SCI.

4. Know the risk factors for coronary artery disease potentiated by SCI.

Overview

The primary effect of SCI on cardiovascular control is a loss of coordination between sympathetic response and conditions demanding changes in heart rate and vascular tone[1].

Anatomy and Physiology

Effective control of the cardiovascular system requires an integrated network consisting of receptors, central modulators, and effectors[2]. The vagus nerve originates from the medulla and innervates the sinoatrial node, atrioventricular node, and ventricles, and remains intact in persons with SCI. Sympathetic innervation of the heart originates from T1 through T5 and reaches the heart via branches from the sympathetic chain. After SCI at or above T6, autonomic imbalance occurs with intact parasympathetic innervation and either a reduced or an exaggerated (hyperreflexic) sympathetic response to stimuli[3]. Loss of the pump effect of muscular contraction in the legs after SCI results in diminished venous return, smaller heart chamber sizes, and reduced cardiac output. The adaptive capacity to exercise through an increased heart rate, contractility, and arteriolar and venoconstriction[4] is impaired in persons with higher levels of injury.

Humoral mechanisms of cardiovascular compensation are also affected by SCI because of impaired sympathetic modulation of the adrenal medulla[5]. With reduced central inhibition of spinal sympathetic outflow, plasma renin activity, angiotensin II, and aldosterone are all elevated. The baseline plasma renin activity is elevated in tetraplegia and rises more than expected compared to non-SCI controls on rapidly lifting the head of the bed[6].

Blood pressure is commonly 90/60 mm Hg (range 90-110/56-70 mm Hg) in persons with tetraplegia, whereas blood pressure is more likely to be normal in individuals with lower levels of injury. Baseline heart rate may be as low as 50-60 in persons with high-level injury. Such individuals are also at risk for silent (unperceived) ischemia due to impaired sensation.

Diagnostic Procedures and SCI

Chest radiography: Artifacts of suboptimal inspiration may occur on chest X-ray. Small cardiac silhouettes may be due to a decrease in myocardial muscle mass and diminished systemic venous return. Atelectasis and pneumonia commonly occur following SCI, and the radiographic changes accompanying these disorders make it more difficult to diagnose congestive heart failure.

Electrocardiography: Abnormalities include sinus bradycardia, supraventricular arrhythmias, and on occasion primary cardiac arrest (particularly with acute high cervical SCI). Many persons with long-standing SCI exhibit nonspecific ST-segment and T-wave changes. Ambulatory electrocardiographic (Holter) monitoring may be needed to diagnose ischemic and/or arrhythmic cardiac disease.

Echocardiography: Diminished left ventricular mass and contractility may be present. Endocarditis and amyloidosis can occasionally occur as sequelae of chronic infections (e.g., decubitus ulcers, osteomyelitis, or urinary tract infections) that often accompany SCI. Exercise echocardiography may aid in the evaluation of symptomatic hypotension in individuals with SCI through the imaging of cardiac volume deficits or valvular dysfunction.

Blood work analysis: SCI by itself is not associated with increases in serum enzyme levels so these are useful in detecting ischemic heart problems.

Oximetry: Baseline oxygenation is likely to be normal.

Exercise stress testing: Arm ergometry using either wheelchairs or upper extremity ergometers is the most valid and reliable exercise mode for these individuals. However, in contrast to leg exercise, exhaustive arm exercise typically produces lower peak heart rates, making detection of significant heart disease difficult[7]. ECG abnormalities also may be harder to detect due to artifactual changes from upper extremity use in arm ergometry. An additional problem with exercise testing in persons with SCI is the frequent occurrence of abnormal systolic blood pressure responses (usually hypotension) that may masquerade as a primary cardiac abnormality. Many individuals with tetraplegia may not be able to perform adequate exercise, making "pharmacologic" stress testing the best option.

Radionuclide imaging: Myocardial perfusion imaging in conjunction with the administration of a pharmacologic stressor may be needed to demonstrate ischemic CAD in persons with SCI[8]. Dipyridamole-induced hypotension is readily reversible with theophylline. Adenosine has a much shorter functional half-life than dipyridamole and hence it may have less propensity to provoke prolonged hypotension in persons with SCI.

Invasive modalities: It is important to assess renal function and to ensure an adequate blood volume to maintain high renal perfusion pressures before and after angiography since many individuals with SCI have underlying renal insufficiency.

CARDIAC ARRHYTHMIAS

Persons with acute high-level SCI are predisposed to cardiac disturbances due to unopposed vagal tone, a high risk of developing hypoxia, hypotension, and fluid and electrolyte imbalances. Cardiac arrhythmias may be harmless or may progress to fatal complications.

Incidence

During the initial rehabilitation stay for SCI, 4.9% of persons experienced cardiopulmonary arrest. The incidence increases with advanced age and complete tetraplegia as compared to paraplegia or incomplete SCI[9 -11].

Pathophysiology

Bradycardia after acute injury is due to unopposed vagal tone and is exacerbated by suctioning or changes in position, especially during the first 12 weeks following injury[12]. Sympathetic discharges can also trigger malignant arrhythmias. Arrhythmias do not seem to play a significant role in persons with chronic SCI. No life-threatening arrhythmias were found on telemetry or 2-year follow-up chart review in 47 persons with SCI suspected of having a rhythm disturbance[13].

Signs and Symptoms

Symptoms may be minimal.

Treatment

- Minimize risk conditions: prevent hypoxia, minimize cardiac workload, maintain desirable blood volume and electrolytes.

- Minimize stimulating effects of increased catecholamines by alleviating pain and promoting rest.

- Avoid vagal stimulation and pre-oxygenate before maneuvers that are known to cause vagal stimulation (suctioning). Consider use of atropine before such stimulation.

- Cardiopulmonary resuscitation requires awareness of neck stability and general avoidance of neck extension for intubation.

CORONARY ARTERY DISEASE

Incidence

As the SCI population ages, the management of coronary artery disease (CAD) becomes an increasingly significant issue. Long-term survival after SCI is approaching that of people without SCI. As people with SCI age, the risk of death from circulatory disease increases to 32%[14-16].

Pathophysiology

A thorough family history and review of systems is essential because specific risks for CAD may be magnified in persons with SCI. Contributing risk factors include physical inactivity, low high-density lipoprotein levels (HDL), proportionally increased body fat, and an increased incidence of glucose intolerance. HDL cholesterol concentrations have been demonstrated to be significantly lower in chronically inactive SCI persons as compared with able-bodied controls and Olympic-caliber SCI wheelchair athletes[17]. The specific mechanisms and epidemiology of hypertension after SCI and correlation with SCI level need further study.

Signs and Symptoms

Individuals with lesions above T5 are at increased risk for unperceived cardiac ischemia because of the interruption of ascending spinal cord pain afferents. Symptoms of angina after SCI may include chest pain, but also consider nausea, palpitations, unexplained autonomic dysreflexia, jaw pain, episodic dyspnea, syncope, and changes in spasticity. Just as autonomic dysreflexia can be the result of ischemia, it also can cause coronary vasospasm and should be carefully evaluated. Symptomatic gastroesophageal reflux is common with SCI and may masquerade as cardiac angina.

Diagnosis

The diagnostic evaluation is similar to that employed in non-SCI individuals, although a pharmacologic stress test may be needed because exercise testing is difficult.

Treatment

Management protocols for the risk factors and for CAD in individuals with SCI need further investigation[18].

- Minimize changes in blood pressure, since fluctuations in blood pressure due to autonomic dysreflexia may contribute to damage.

- Monitor for hypertension and treat hypertension and hypertensive episodes appropriately.

- Maintain near ideal body weight.

- Smoking cessation is particularly pertinent to persons with SCI due to the increased risk of pulmonary and cardiac disease.

- Recommend diets low in animal fat and dietary changes to increase HDL.

- Encourage exercise. Increased activity in previously sedentary SCI individuals can raise HDL levels[17, 19].

- Promote peripheral vascular circulation by avoiding circumstances giving rise to peripheral edema, postural hypotension, and autonomic dysreflexia.

- Teach patients to recognize symptoms that may be associated with cardiac ischemia.

- The role of cardioprotective antianginal medications requires further exploration in persons with SCI.

DEPENDENT EDEMA

Incidence

Dependent edema is a common finding in both chronic and acute SCI.

Pathophysiology

Lack of the muscle pump contributes to dependent edema.

Signs and Symptoms

Dependent edema may extend to the knee (as in non-SCI patients).

Diagnosis

Evaluation is the same as in the non-SCI population.

Treatment

Minimize edema with leg elevation and compressive stockings. Use requires careful skin evaluation for excessive pressure. Diuretics are generally not indicated due to low baseline blood pressures.

EXERCISE INTOLERANCE

Pathophysiology

The reduced exercise capacity accompanying SCI is due to multiple factors, including diminished sympathetic outflow with exertion; smaller cardiac chamber size; decreased voluntary muscle mass; decreased venous return with blood pooling in the legs and lower abdomen diminishing the blood volume available for circulation; and the physiologic consequences of inactivity.

Signs and Symptoms

Fatigue may occur. Deficits in circulatory modulation contribute to a reduced exercise capacity and frequent complaints of exhaustion after minimal exertion[18].

Treatment

• Educate the person regarding the undesirable effects of immobilization on the body's organs.

• Even modest exercise can achieve health benefits, although many individuals are not capable of prolonged and highly stressful cardiovascular exercise.

• Encourage participation in therapeutic recreational and avocational activities.

• Evaluate whether ergometry would be useful for patients who cannot use their wheelchairs for aerobic training (due to poor weather, hills, etc.)[20, 21].

ORTHOSTATIC HYPOTENSION

Spinal shock, which occurs immediately after SCI, can include hypotension and extreme orthostasis due to absent sympathetic reflex responses. Orthostasis can also occur in chronic SCI from bed rest, decreased fluid intake, or an inflammatory illness causing vasodilation. Adaptation includes a modest increase in heart rate mediated by the withdrawal of vagal tone[2]. Compensation also occurs through renal mechanisms with exaggerated renin secretion in response to positional challenges[6]. Nevertheless, baseline blood pressure in a person with tetraplegia is typically 90-110/56-70 mm Hg.

Signs and Symptoms

Findings may include weakness, dizziness, pallor, blurred vision, tachycardia, excessive sweating above the level of lesion, or fainting during positional change.

Treatment

- Preventive measures include increased salt intake, use of an abdominal binder, and elastic stockings.

- Change position gradually with slow mobilization to partial recumbency and then transfer to wheelchair. Use supportive reassurance to ameliorate anxiety.

- Pharmacologic adjuncts could include oral ephedrine (alpha-agonist) 30 minutes before sitting. If needed, fludrocortisone (a mineralocorticoid) can be administered daily to expand the intravascular volume via sodium retention.

Self-Study Review

1. *Which diagnostic procedures might need modified interpretation for individuals with SCI?*

2. *What major risk factors for coronary artery disease are often present in the SCI population?*

3. *List several parameters that should guide the management of silent cardiac ischemia in persons with SCI.*

4. *Describe several cardiac arrhythmias that are common in persons with acute SCI.*

5. *How could you best treat or prevent orthostatic episodes in an individual with SCI?*

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7 GASTROINTESTINAL SYSTEM

Steven A. Stiens, MD, MS

Objectives for the Learner

1. Describe the pathophysiologic effects of SCI on gastrointestinal function.

2. Describe routine management of the neurogenic bowel.

3. Describe common gastrointestinal complications after SCI.

4. Describe unique aspects of managing acute abdomen in this population.

Overview

Neurogenic bowel refers to colonic dysfunction (constipation, incontinence, and difficulty with evacuation) due to lack of nervous control. SCI can produce loss of coordination throughout the alimentary canal. The resultant changes in autonomic function may produce slow gastric emptying, increased acid secretion, stress ulceration, predisposition to ileus, and altered colonic motility, depending on the level of injury. Even after the acute phase of injury with potential gastrointestinal complications, colonic dysfunction can be a source of inconvenience, frustration, and expense especially for persons with high-level injuries who require attendant support[1].

Incidence

All persons with complete SCI have neurogenic bowel. Most persons with incomplete SCI have some manifestation of dysfunction.

Functional Anatomy and Pathophysiology

Innervation to the gastrointestinal system is both autonomic and somatic.

- Parasympathetic innervation is via the vagus nerve to the stomach, small intestine, ascending colon, and transverse colon, and via the pelvic splanchnic nerve (from the conus medullaris, sacral levels 2, 3, 4) to the descending colon and rectum. Innervation produces propulsive peristalsis.

- The sympathetic greater splanchnic (T5-9), lesser splanchnic (T10-12), and hypogastric (L1-3) nerves innervate the stomach and small and large intestines.

- The somatic pelvic nerve (sacral levels 2, 3, 4) innervates the pelvic floor and external anal sphincter.

- The enteric nervous system of the gut includes the submucosal Meissner's plexus and the intramuscular Auerbach's plexus with reflex connections that coordinate local peristalsis.

The effect of SCI on gastrointestinal function is dependent on the neurologic level and completeness of SCI (Table 7-1), and whether the type of neurologic deficit is upper or lower motor neuron.

TABLE 7-1.
EFFECT OF SCI ON COLON PATHOPHYSIOLOGY

Injury	Level of Lesion	Physiological Effect
Upper motor neuron (UMN)	Lesion above conus medullaris	Underactive propulsive peristalsis Fecal distention of the colon Overactive local peristalsis Increased fecal retention due to external anal sphincter constriction
Lower motor neuron (LMN)	Lesion affecting parasympathetic cell bodies within the conus medullaris or their axons in the cauda equina and pelvic nerve	Slow stool propulsion accomplished with segmental peristalsis Anal sphincter has low tone

Diagnosis

As with the neurogenic bladder, neurologic examination aids in predicting the type of dysfunction. The rectal examination allows assessment of external anal sphincter tone. The person's sensation of the examining finger should be assessed, and voluntary contraction should be requested in persons with incomplete injuries. Perineal sensation should be evaluated with light touch and pin prick. Reflex assessment includes the bulbocavernosus reflex (anal sphincter contraction in response to clitoral or penile glans compression) and anocutaneous reflex (anal sphincter contraction in response to pin prick).

Methods of Bowel Management

The goals of a bowel program are to achieve effective and efficient colonic evacuation, prevent incontinence, and prevent the secondary complications of distension, impaction and diverticuli. Bowel care is the process of facilitated reflex defecation or manual fecal evacuation.

UMN bowel. The external anal sphincter muscle, normally under voluntary control, remains tight due to spasticity of the pelvic floor. Stool accumulates unless reflex defecation is triggered. Without the ability to feel stool in the rectum or to easily initiate reflex defecation, a person with SCI must regularly assume the need for bowel movements.

- A mechanical or chemical stimulus is required to trigger reflex defecation at regular intervals of 1 to 3 days.

- The reflex peristalsis is stimulated with an appropriate rectally administered medication and/or manually with a finger (or assistive device) inserted into the rectum (digital stimulation)[1].

- The chemical stimulus is typically a suppository or mini-enema, which produces a mucosal contact stimulus that initiates conus-mediated reflex peristalsis. The chemical trigger is squirted or digitally inserted against the mucosa in the upper rectum. Insertion is followed by a waiting period as the active ingredients dissolve, disperse, and act.

- Digital stimulation relaxes and opens the external anal sphincter, activates coordinated peristalsis by the myenteric plexus, and stimulates conus-mediated reflex peristalsis[1]. The technique is as follows:

  - Gently insert entire lubricated gloved finger into rectum to open the external anal sphincter and provide a stretch stimulus to trigger peristalsis. Movement of the finger in a cone configuration dilates the rectum with minimal anal stretch.

  - Maintain mucosal contact with the finger exerting gentle pressure against the rectal vault.

  - Continue rotation until relaxation of the bowel wall is felt, flatus passes, stool advances, or the internal sphincter constricts.

  - Digital stimulation ideally should require no longer than 1 minute at a time to generate peristalsis.

  - Stool flow begins and is augmented as necessary with digital stimulations, repeated every 10 minutes if no stool passes in the interim.

Factors to consider in individualizing a bowel program include:

- Dietary modulation of stool consistency with fiber and liquid intake

- Increased physical activity

- Regular scheduling of bowel care time and frequency

- Equipment (commode chair, digital stimulation devices)

- Oral medications

- Rectal medications

LMN Bowel tends to be flaccid because no spinal-cord-mediated reflex peristalsis occurs and the anal sphincter is flaccid.

- The absence of spinal reflex peristalsis and low anal sphincter tone often mean that the rectum must be cleared of stool more frequently. This is required one or more times per day, to prevent incontinence from stool that cannot be retained by the patulous external sphincter.

- The LMN bowel care procedure usually consists of removing stool with the finger and using digital stimulation to increase peristalsis.

- Continence is improved by modulation of stool consistency with a high-fiber diet. Plant fibers such as psyllium hydromucilloid may be useful to produce uniform stool viscosity by absorbing excess water in the fecal matter.

- Some people with LMN bowel wear tight underwear or bicycle pants to support the pelvic floor and help retain stool.

COMMON GASTROINTESTINAL PROBLEMS

Incidence

The risk of gastrointestinal complications is much higher in certain subpopulations of SCI; for example, early gastrointestinal bleeding is more common in individuals with complete injuries above the T5 level, and this risk is increased by other variables such as artificial ventilation or heparinization. Among individuals with SCI or related disorders, 54% rank

bowel and bladder dysfunction as a major life-limiting problem[1, 2]; 41% of the Stockholm SCI study rated neurogenic bowel management as a moderate to severe life-limiting problem[3].

People with SCI may present with an increased frequency of certain symptoms related to GI dysfunction or, more often, an absence of specific symptoms that otherwise pinpoint the pathology. The following recurring symptoms and frequencies have been reported in persons with chronic SCI:

- Esophagitis, 45%

- Difficulty with bowel evacuation, 20-54%

- Intermittent abdominal distention, 50%

- Autonomic dysreflexia due to GI causes, 43%[4] (see Chapter 3)

- Hemorrhoids, 74%

Signs and Symptoms

The physician must be attentive to new symptoms and judiciously evaluate them to be sure they do not represent significant underlying pathology. Most difficult to evaluate is a change in or magnification of a chronic symptom pattern.

- Anorexia may indicate fecal impaction.

- Nausea may indicate gastric distention, excess acid secretion, or fecal impaction.

- Abdominal fullness or distention may indicate postprandial problems (gastroduodenal obstruction or hypokinesis) or fecal impaction.

- Leakage of mucus from the rectum may be secondary to fecal impaction or retained chemical stimulant used to trigger defecation.

- Hard stools may indicate inadequate fluid intake (which may be due to reduced inter-mittent catheterization frequency), infrequent bowel care, insufficient fiber content of stool, or too small a dose of stool softener.

- Hemorrhoids may cause dysreflexia if irritated by digital stimulation or if the anus is poorly supported in a shower chair.

- Diarrhea may indicate excessive oral laxatives, antibiotics, dairy products, or fecal impaction.

- Incontinence of stool is not unusual with antibiotic use.

- Symptoms of autonomic dysreflexia (see Chapter 3; common GI-mediated causes are acid irritation of esophagus, colonic distention, and hemorrhoidal irritation).

Pharynx and esophagus: dysphagia, aspiration, and reflux esophagitis

- The pharynx and esophagus are seldom affected by SCI, but severe cervical trauma, extensive internal fixation surgery, and osteophytes can affect the mechanical function of the esophagus anterior to the cervical spine.

- Dysphagia is a common complaint during the first few months with tetraplegia.

- Tracheostomy tubes may tether the pharynx, limiting upward excursion in swallowing.

- Esophageal reflux disease is more likely after SCI due to predisposing factors such as increased acid secretion, reduced gastroesophageal sphincter tone, delayed gastric emptying, medications, recumbency, and immobilization[2].

- Persons with SCI should eat in the upright seated position to facilitate swallowing. Swallowing evaluation by a speech therapist should be pursued if dysphagia is present or aspiration is suspected. Videofluoroscopy to visualize aspiration may be needed to plan compensation techniques or therapy.

Stomach: ulcers, reduced motility, and outlet obstruction

- During the first few months after SCI there is excess gastric acid and pepsin secretion due to intact gastric vagal innervation and loss of sympathetic inhibition. The resulting high incidence (3.0% - 22%) of gastroduodenal ulcer formation warrants prophylaxis for the first 3 months after injury.

- Persons with tetraplegia often have prolonged gastric emptying, which improves with time after the injury[2].

- Delayed gastric emptying may respond to metoclopramide 10 mg orally or intravenously every 6 to 8 hours.

- Superior mesenteric artery syndrome (SMAS) arises from intermittent duodenal compression by the superior mesenteric artery. Individuals with tetraplegia are more likely to have SMAS, presenting with epigastric pain, vomiting, and duodenal cut-off, all of which diminish with upright position as confirmed with abdominal radiographs. Use of a lumbosacral corset, postprandial head elevation, and replacement of weight lost is usually effective. Occasionally these symptoms occur due to the abdominal pressure of a thoracic-lumbar-sacral orthosis which needs to be revised to allow more space for the abdomen.

Gallbladder and small intestine: cholelithiasis and ileus

- Asymptomatic cholelithiasis is more prevalent in persons with SCI than in non-SCI populations. The prevalence of gallstones after SCI as determined in cross-sectional studies has been estimated to be as high as 30%, making it up to three times more likely than in the general population[5]. Additional risk factors for cholelithiasis in this population include decreased intestinal transit rate, which delays the enterohepatic circulation of bile salts; catabolic changes after SCI that could mobilize protein, calcium, and cholesterol into bile; and gallbladder stasis from impaired neural and hormonal control[5].

- Annual ultrasound surveillance for stone formation and evidence of inflammation, along with renal scanning, is recommended. Prophylactic cholecystectomy has been recommended in selected individuals.

- Ileus is a complication observed in persons with new SCI but usually clears with conservative measurements such as nasogastric suction.

- Adynamic ileus can occur in the setting of colonic impaction, systemic illness, or inactivity.

Large intestine

With LMN bowel dysfunction, rectal prolapse can occur from excessive straining.

Impaction

- If no bowel movement has occurred for 4 or 5 days, perform a rectal examination with liberal lubrication, and gently break up and digitally remove any stool in the rectum. Complete a routine bowel care procedure, and repeat at least daily until resolution of distension.

- Mineral oil or bisacodyl enemas may be used to allow hard stools to pass, but caution must be used not to precipitate autonomic dysreflexia because of distending the rectal vault.

- One may use osmotic laxatives such as magnesium citrate or oral stimulants to evacuate the bowel.

- Once the bowel is empty, review for circumstances that may have contributed to the difficulty:

  – Change in exercise

  – Medication changes

  – Emotional stress

  – Change in diet

  – Change in fluid consumption

  – Change in bowel care regimen

- Adjust the entire bowel program accordingly. Advise the individual to eat foods higher in fiber, increase fluid consumption as tolerated by the individual's bladder program, increase activity, take bulk-forming agents, ensure privacy during bowel care, etc.[4].

Diarrhea

- Increase the frequency of bowel care to prevent incontinence.

- If diarrhea is suspected to be infectious, treat as in the able-bodied population but also increase frequency of bowel care with digital stimulation to predictably eliminate liquid stool.

- If diarrhea is believed to be due to fecal impaction, clear the impaction as described previously.

- Modify diet to include more stool-hardening foods, e.g., cheese and starches[6].

- Discontinue exacerbating factors (spicy foods, caffeine, antibiotics, laxatives, stress).

- Judiciously use antidiarrheal agents: kaolin pectin, diphenoxylate/atropine.

Hemorrhoids

All efforts should be made to prevent hemorrhoids by adherence to an appropriate program of bowel management.

- Limit maximum commode or shower chair time to 1 hour.

- Proceed very gently with bowel care procedures such as digital stimulation.

- Use anti-inflammatory suppositories, topical ointments, lidocaine jelly, and comfortable positioning as needed to limit discomfort and prevent autonomic dysreflexia.

ACUTE ABDOMEN

Overview and Incidence

Persons with SCI are at higher risk for late presentation of abdominal emergencies because of their sensory deficits, functional abnormalities, and imbalance of parasympathetic and sympathetic innervation of the gut.

Prognosis

Rapid evaluation with laboratory (CBC, SGOT, SGPT, amylase, urinalysis) and imaging studies is warranted to reduce delays in diagnosis and to reduce the 10-15% mortality rate.

Anatomy and Physiology

Dull visceral pain is transmitted by the vagus and sympathetic nerves, which travel par-avertebrally to enter the brainstem and spinal cord. Abdominal wall pain is mediated by segmental mixed nerves and is carried by the spinothalamic tracts in the spinal cord. If the neurologic level is

- above T6, the individual may experience autonomic dysreflexia, increased spasticity, rigid abdomen, and vague, nonlocalized abdominal discomfort.

- between T6 and T12, there may be some reflex responses and localization depending on the specific organ involved.

- lower than T12, sympathetic splanchnic outflow is spared, allowing sensory responses as would be expected for those without SCI.

Signs and Symptoms

It may be helpful to elicit a history of what the person with SCI routinely feels to gauge what sensory pathways may be intact after the injury. Routine awareness of hunger, abdominal gas, bladder or rectal fullness, and the need to evacuate should be asked about. It is important to distinguish direct sensations from associated symptoms such as the occurrence of sweating with the need to void. A high index of suspicion is needed to promptly diagnose abdominal emergencies, which often are identified only by subtle and minimal symptoms accompanied by poorly localized findings.

- Anorexia, if present, is often the first symptom and is very nonspecific.

- Abdominal pain may be atypical in location, intensity, and description.
  - Visceral pain is deep-seated, dull, and poorly localized.
  - Somatic pain is localized and occurs as parietal epithelium becomes inflamed.

- Vomiting is vagally mediated and may be precipitated by vagal and sympathetic afferents within the abdomen.

- Spinal reflex sweating above or below the lesion is frequently associated with visceral inflammation or bowel distention.

- Referred pain to the shoulder may still occur in many individuals with SCI.

- A rigid abdomen would be expected in a person with an upper motor neuron lesion and intact reflex pathways.

Diagnosis

- The most prominent objective finding may be as seemingly insignificant as an elevated pulse rate. Conversely, bradycardia may be present as an adaptive response to elevated blood pressure from autonomic dysreflexia.

- Fever may be present.

- Orthostatic hypotension may be present in persons with chronic tetraplegia.

- In order to examine the abdomen, spasticity can be lessened by supporting the flexed limbs with pillows under the knees.

- Abdominal palpation should include assessment for colonic and bladder distention.

Treatment

Management of the causes of the acute abdomen are generally the same as in persons without SCI. Succinylcholine or other depolarizing agents for use during anesthesia are contraindicated because of uncontrolled hyperkalemia. Consideration must be given to the use of baclofen in those who cannot have oral intake because side effects can occur with sudden withdrawal. One must be careful in postlaparotomy mobilization of the individual as abdominal wall support is impaired and dehiscence may occur. Lack of muscular tone requires use of an abdominal binder. Transfers with full assistance are recommended at first to prevent trauma, Valsalva, and excessive increase in intra-abdominal pressure. Progression to independent transfers may require 3-6 weeks.

Self-Study Review

1. *What are several key differences in a bowel program for individuals with upper vs. lower motor neuron dysfunction patterns?*

2. *Describe reasons for the increased prevalence of cholelithiasis in persons with SCI.*

3. *List several key symptoms and signs associated with common GI problems in this population.*

4. *Describe aspects of managing the acute abdomen that differs in this population.*

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8   GENITOURINARY SYSTEM

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Objectives for the Learner

1. Describe the neurologic findings that distinguish upper from lower motor neuron bladder dysfunction.

2. List the relative advantages and disadvantages of different methods of bladder management after SCI.

3. Identify the effects and contraindications of medications in persons with neurogenic bladder.

4. Describe common complications of the genitourinary system after SCI and their management.

5. Describe a program of surveillance for urinary tract complications after SCI.

Overview

Before WWII, SCI was nearly always fatal, and urosepsis was the leading cause of death. Although this is no longer the case due to improved methods of bladder management, genitourinary problems are the most common secondary complications of SCI.

Incidence

- Approximately one third of persons with incomplete SCI have voluntary micturition at the time of discharge. The remainder, and those with complete injuries, require individualized bladder management.

- The rate of urinary tract infection is 50-80% in the first year post-injury[1].

- Renal calculi are the most common upper tract complication, with a prevalence of 8% by 10 years post-injury.

Functional Anatomy and Pathophysiology

Innervation to the lower urinary tract is both somatic and autonomic.

- Somatic innervation via the pudendal nerve (S2-4) provides volitional control to the striated muscle of the external urethral sphincter. Although under volitional control, its function is closely linked to that of bladder outlet smooth muscle.

- Parasympathetic cholinergic innervation via the pelvic splanchnic nerves (S2-4) to the urinary detrusor and bladder outlet provides detrusor contraction and bladder outlet relaxation.

- Sympathetic innervation is from preganglionic cell bodies in the intermediolateral gray of the spinal cord (T11-L2) and postganglionic nerves of the hypogastric plexus. Alpha-adrenergic receptors are concentrated in the bladder outlet and proximal urethra, while more beta-adrenergic receptors are found in the detrusor. In general, smooth muscle cells possessing alpha receptors contract in the presence of norepinephrine, while cells with beta receptors relax. The overall effect of sympathetic stimulation is therefore to cause bladder outlet contraction and detrusor relaxation.

- Urinary tract dysfunction after SCI can be divided into two basic patterns, upper motor neuron (UMN) and lower motor neuron (LMN).

Signs and Symptoms

Abnormal voiding may be revealed in a careful history. Persons with complete SCI and UMN bladders may have incontinence due to reflex bladder contractions. Persons with incomplete SCI who can void may have hesitancy, frequency, urgency, difficulty stopping the flow, and incontinence due to an UMN bladder. Facilitation of voiding may require gentle tapping over the bladder to stimulate a reflex contraction. Persons with LMN bladders may report overflow or stress incontinence.

Diagnosis

The results of a careful history and neurologic examination aid in predicting the type of dysfunction (Table 8-1). Diagnosis can be confirmed with urodynamic testing.

TABLE 8-1.
COMPARISON OF UPPER AND LOWER MOTOR NEURON DYSFUNCTION IN LOWER URINARY TRACT DYSFUNCTION

	Upper Motor Neuron	Lower Motor Neuron
Reflex detrusor contraction with bladder filling	Yes	No
Lesion	Cervical, thoracic, or lumbar spinal cord segments (T11 or higher spine fracture)	Sacral spinal cord involving conus medullaris or cauda equina (usually T12 or lower spine fracture)
Neurologic examination	Intact bulbocavernosus reflex, elevated anal sphincter tone, increased tendon reflexes, increased tone of extremities*	Absent bulbocavernosus reflex, absent anal sphincter tone*
Risk	High detrusor pressure, detrusor-sphincter dyssynergia, vesiculo-ureteral reflux, hydronephrosis, and renal failure	Lower risk for upper tract deterioration; however, manual pressure (e.g., Credé maneuver) can result in vesicoureteral reflux and hydronephrosis

*Voluntary control of anal sphincter will be absent in complete injury but may be present in incomplete injury.

Management

- After SCI, the bladder is initially managed with an indwelling catheter. This is continued until medical stability is achieved and fluid can be restricted to a daily output of 1.5-2L.

- The goals of long-term bladder management are low intravesical pressure, complete emptying, avoidance of indwelling catheters, and no incontinence.

- The method depends on the individual's neurologic level, type of bladder dysfunction, gender, motivation, and other factors. Commonly used methods are intermittent catheterization, condom catheters, and indwelling or suprapubic catheters. Less common methods include assisted voiding with stimulation of reflex contraction via suprapubic tapping, anal sphincter stretch to promote relaxation of the external urethral sphincter, and detrusor compression via Credé maneuver.

Intermittent catheterization (IC) has contributed to a reduction in morbidity from renal failure after SCI.

- The individual must have adequate hand function or an attendant, ability to empty the bladder every 4-6 hours, adequate storage of urine between catheterizations, and sufficient cognition and motivation to adjust fluid intake to keep urine volumes less than 500 ml per catheterization.

- Anatomical considerations make IC more difficult for women with SCI.

- Most individuals do well with "clean" technique although sterile technique may be necessary for individuals with frequent infections.

- Anticholinergic medication is often necessary to lower intravesical pressure in order to prevent incontinence due to reflex detrusor contractions in persons with UMN bladders.

- Individuals on IC may still develop upper tract deterioration if vesicoureteral reflux is present or obstruction at the vesicoureteral junction occurs from bladder trabeculation (thickening from high pressures or recurrent infections).

External or condom catheters are the most practical method for men with high-level tetraplegia and limited hand function.

- The individual must have reflex voiding at safe intravesical pressures and with low residual volumes as determined by urodynamics.

- Detrusor sphincter dyssynergia can cause high intravesical pressure and usually requires medications or sphincterotomy to reduce outlet resistance.

- Individuals often also need to perform suprapubic tapping or the Credé maneuver every 4-6 hours to decrease residual urine if reflex emptying is incomplete.

- Condom catheters should generally be changed daily and the penile skin allowed to air dry. Overly tight condoms can cause erosion of penile skin, urethral damage, or vesicoureteral reflux and hydronephrosis from back-pressure.

- Obese individuals or those with a small, retractile penis may have difficulty retaining a condom. Penile prostheses are occasionally implanted to facilitate use of a condom catheter.

- UTIs may be related to high bacterial concentrations in stagnant urine around the urethral meatus and glans penis.

- The incidence of upper tract deterioration with external catheter management is similar to clean intermittent catheterization.

- Surveillance with urodynamic studies to ensure safe emptying pressures is necessary after injury and periodically due to potential long-term complications.

Indwelling catheters may be either urethral or suprapubic.

- Individuals are advised to drink large volumes of liquid to maintain a dilute urine.

- Bedside or leg drainage bags should be cleaned daily with commercially available agents such as sodium hypochlorite solution (bleach) or dilute phosphoric acid (Urolux). Some leg bags have a nonreflux valve which may be helpful in preventing UTIs.

- The urethral or suprapubic catheter should be changed at least monthly. Traction, kinking, or plugging should be avoided. The catheter may be secured by taping to the abdomen or by use of a Velcro thigh strap.

- Potential complications of indwelling urethral catheters include prostatitis, epididymitis, dislodgement, erosion of the urethra, and accidental inflation of the catheter balloon in the urethra. Urethral catheters should generally not be used in the presence of a penile prosthesis due to the risk of erosion.

- The degree of increased risk for bladder carcinoma is controversial. Individuals should have cystoscopy after 10 years of continued indwelling catheter use or 5 years if at high risk (smoker, age>40, recurrent UTIs)[2].

- Although only used if other options are not feasible, many individuals tolerate them well and do not have a significant increase in upper tract deterioration compared with other methods[3]. This may be attributed to improvements in catheter materials and design as well as better patient care and education.

- Suprapubic catheters are generally used only if other methods of management are unsuccessful as a result of complications.

Pharmacologic intervention

Persons with UMN bladders who use intermittent catheterization commonly require an anticholinergic drug to inhibit reflex contractions and to increase bladder storage. Commonly used anticholinergic medications include oxybutynin, propantheline, and imipramine. Conversely, they would be contraindicated in someone attempting to void by reflex contraction and using a condom catheter.

- Persons with dyssynergia may require alpha-adrenergic antagonists (or external sphincterotomy). Alpha-adrenergic antagonists include prazosin, terazosin, and doxazosin.

- Many commonly used medications for general medical conditions, such as antihistamines, have autonomic actions or side effects that can compromise voiding in a person with a neurogenic bladder. When possible, locally acting drugs rather than systemic drugs should be used. Use of any systemic drug with known autonomic effects requires careful monitoring of bladder function.

- Cholinergic agonists, alpha-adrenergic agonists, beta-adrenergic agonists and beta-adrenergic antagonists have no proven usefulness.

Surgical management

Surgical techniques are designed to address either a failure to store or failure to empty urine and may be aimed at the bladder, nerves, or outlet. Procedures for a failure to store include augmentation cystoplasty, denervation procedures, artificial urinary sphincter, and Teflon periurethral injections. A failure to empty may require sphincterotomy, electrical stimulation, or urinary diversion. Referral to a urologist is indicated for individuals who fail traditional management methods or have chronic infections, outlet obstruction, or upper tract deterioration.

Urinary Tract Complications

Urinary tract infections (UTI). Symptoms may be limited to increased spasticity, autonomic dysreflexia, and new or increased urinary incontinence, or may include the usual fever, chills, cloudy or malodorous urine, and malaise. Due to the high frequency of resistant organisms, urine gram stain, culture, and sensitivity should be obtained prior to initiating therapy whenever possible.

• Check the prostate and scrotal contents for abscesses.

• Treatment of mildly to moderately ill individuals with suspected lower or upper UTI can be started empirically with oral agents such as a fluoroquinolone or trimethoprim-sulfamethoxazole, then modified according to sensitivity results. Severely ill individuals require parenteral broad-spectrum coverage such as ampicillin or a third-generation cephalosporin plus an aminoglycoside, or imipenem-cilastatin, or as recommended by an infectious disease consultant.

• Be careful not to confuse the normally low baseline blood pressure of a person with tetraplegia with urosepsis.

• Duration of treatment has not been rigorously investigated as in the able-bodied, therefore treatment should continue for 7-14 days.

• Individuals with known or suspected renal stones or recurrent UTIs may require an immediate ultrasound and KUB to rule out a complicated UTI. Certainly, if there is failure to improve after 48 hours, consider an abdominal CT or intravenous pyelography to rule out perinephric abscess or obstruction.

• Bacterial suppression with antibiotics or acidifiers is controversial but may be warranted in individuals with recurrent UTIs. Suppressive agents include methenamine mandelate which must be taken with high-dose Vitamin C (e.g., 1 gm BID), or methenamine hippurate. Methenamine drugs should be stopped when antibiotics are started due to efficacy and potential complications. Antibiotic choices for suppression include nitrofurantoin and trimethoprim-sulfamethoxazole.

Asymptomatic bacteriuria: Even if there are greater than 10,000 colonies/ml of urine, this condition does not require treatment in persons with SCI unless there is a urease-producing organism (e.g., Proteus, Providencia). Injudicious use of antibiotics results in high frequency of resistant organisms.

<u>Difficulty passing a catheter</u> may be due to urethral stricture, spasticity of the external urethral sphincter or pelvic floor musculature, or a false passage.

- Do not force a catheter if resistance is encountered.

- Initial catheterization may be attempted with the introduction of 5 ml of water-soluble lubricant into the urethra with a syringe, or use of 2% lidocaine jelly.

- A curved tip (Coudé) or stiffer catheter, along with steady gentle pressure at the level of the sphincter can be effective if spasticity is suspected.

- False passage from urethral trauma requires an indwelling relatively large-bore catheter to allow healing.

<u>Bladder overdistention</u> from obstruction, if severe, warrants use of prophylactic antibiotics due to the risk of urosepsis.

<u>Autonomic dysreflexia</u> is commonly caused by genitourinary disease (see Chapter 3).

<u>Calculi</u> are more common in individuals with indwelling catheters and recurrent infections (struvite stones). Important landmarks are the ureteropelvic junction and the vesicoureteral junction as calculi may commonly obstruct flow of urine at these points.

- Renal calyceal stones can initially be followed conservatively, but many eventually require treatment. Enlarging stones or those in the renal pelvis are at risk for causing obstruction and should be treated. Extracorporeal shock wave lithotripsy is generally used unless the stone is greater than 3 cm, which requires percutaneous removal.

- Ureteral stones should be observed closely for passage. They can be abnormally silent (painless) in persons with SCI even in the face of severe obstruction.

- Obstruction may frequently occur with infection in the SCI population and may require urgent drainage procedures.

- Bladder stones are treated with probe lithotripsy, followed by dissolution of remaining particles with bladder instillations with hemiacidrin solution (Renacidin). This solution can be used prophylactically in selected individuals, but should be avoided in individuals with known vesicoureteral reflux due to potential nephrotoxicity.

- Hypercalciuria may occur following acute SCI and is responsible for early development of calculi.

Ureteral reflux and upper tract dilatation are commonly caused by chronically elevated detrusor pressure. Bladder trabeculation and hypertrophy alters the normal antireflux capability of the vesicoureteral junction. Diverticuli at this location may also allow reflux. Control of bladder pressures with medications and control of catheterization volumes is the primary goal. Ureteral reimplantation, bladder augmentation, or sphincterotomy may be required if conservative treatment fails.

Metabolic abnormalities

Dilutional hyponatremia, salt-losing nephropathy, and the syndrome of inappropriate antidiuretic hormone secretion are common after SCI.

Urinary Tract Surveillance

Routine surveillance has contributed to the reduction in mortality after SCI.

- At the time of initial injury, baseline studies include urinalysis, renal ultrasound, post-void residual tests (if individual voids voluntarily or reflexly), urodynamic testing, and 24-hour creatinine clearance.

- Annual evaluations typically include urodynamic testing (as indicated), renal ultrasound, KUB X-rays, 24-hour urine for creatinine clearance, urinalysis, postvoid residuals if the individual voids, and other tests of renal function as needed (renal radionuclide scan).

- Regular intravenous pyelograms are not performed routinely and should be used as indicated by other test results.

- Cystoscopy is generally performed in individuals after use of an indwelling catheter for >10 years or if the individual is at high risk (smoker, frequent UTI).

Self-Study Review

1. *What are the etiologic, neurologic, and high-risk outcomes that are differentially associated with upper vs. lower motor neuron dysfunction?*

2. *Compare and contrast the relative advantages and disadvantages of intermittent catheterization, indwelling (urethral or suprapubic) catheters, or condom drainage as methods of bladder management after SCI.*

3. *Why must caution be exercised when using any systemic drug with autonomic side effects?*

4. *What are the standard treatment approaches used to manage urinary tract infections, autonomic dysreflexia, calculi, ureteral reflux, and upper tract dilatation?*

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9 MUSCULOSKELETAL SYSTEM

Barry Goldstein, MD, PhD

Objectives for the Learner

1. Learn how to diagnose and treat lower extremity fractures in individuals with SCI.

2. Anticipate common secondary complications of fracture in persons with SCI.

3. Develop diagnostic skills for differentiating contractures vs. heterotopic ossification as etiologies for decreased range of motion at the hip.

4. Develop proficiency in distinguishing shoulder pathologies from neck pathologies and assessing their functional significance.

5. Learn how to develop a treatment plan for chronic shoulder pain.

6. Increase skill in developing assessment and treatment plans for elbow, wrist, and hand pain for individuals with SCI.

7. Improve diagnostic planning skills for evaluating spine pain or new vertebral deformities.

Overview

Musculoskeletal problems in persons with SCI can lead to significant secondary impairments and additional disabilities. Long bones can fracture from relatively minor impact or during normal range of motion. Heterotopic ossification can lead to joint contracture and functional problems in seating and posture. Joint pain, especially in the neck and upper extremity, is extremely common. At any time after vertebral column injury, spine instability and neurologic deterioration can occur and must be treated immediately.

FRACTURES

Incidence

The incidence of long bone fractures in the lower extremities has been estimated to be 4-7%[1].

Pathophysiology

Fractures following SCI are related to the completeness of injury, being approximately 10 times more common in complete injuries. The onset of osteoporosis occurs within a few weeks or months after SCI, with bone loss occurring below the level of neurologic impairment[2]; i.e., individuals with tetraplegia lose significant bone mass in the upper extremities and upper axial skeleton in addition to the lower body, while those with paraplegia lose bone only from the pelvis, lower extremity, and axial skeleton below the level of SCI. Pathologic fractures accompany the appearance of osteoporosis. Although remarkably minor forces sometimes result in fracture (e.g., range of motion), the most common etiology in the SCI population is falls. Fall prevention, therefore, is important. Most fractures occur in the long bones of the lower extremity; supracondylar fractures are the most common, with femoral shaft and tibial fractures also commonly seen.

Signs and Symptoms

- Swelling, warmth, and some deformity of the affected area and deformity and shortening of the affected limb are the classic signs of a new fracture.

- There is often no discomfort in the setting of a complete SCI.

- Potential complications include autonomic dysreflexia, skin breakdown, increased spasticity, and deep venous thrombosis (DVT).

Diagnosis

Establish the location and type of fracture. Since comminuted, displaced fractures of the distal femur and proximal tibia are common, vascular studies should be obtained if DVT or arterial compromise is suspected.

Treatment

Although a high incidence of delayed union, malunion, and nonunion have been reported, the physiological and functional consequences are essentially unknown. Internal fixation is not recommended in cases of severe osteoporosis. Contact an SCI Center if surgery is being considered or if there are questions about treatment.

Initial assessment and treatment

- Immobilize the extremity with a soft, well-padded splint, and do not use a circular cast. If a circular cast is used after the period of edema, it should be bivalved, well padded, and wrapped with an elastic bandage. Skin should be checked twice per day, in the morning and evening.

- Elevate the affected extremity to minimize edema and prevent further complications.

- Bed rest should be minimized to avoid skin breakdown and deconditioning. Do not use skeletal or skin traction.

- Meticulous skin care and inspection should follow a fracture; superficial skin breakdown should be treated immediately to avoid deeper pressure ulcers.

- Increased spasmolytic medications and the use of special mattresses or cushions are often necessary to avoid further skin trauma from increased spasticity.

Functional evaluation

- Evaluate the functional implications of immobilization prior to discharge home.

- Evaluation of the functional cause of the fracture may be necessary.

- Some individuals require hospitalization to learn transfers, pressure reliefs, lower extremity dressing, bathing, and lower extremity management after a splint or cast has been applied.

- Seating and wheelchair changes are frequently necessary.

- Assess accessibility at home and in the community because lower extremity immobilization will increase accessibility needs.

Outcome

- Nonunion, malunion, or delayed union should be anticipated because factors known to retard healing such as denervation, osteoporosis, age, diabetes, chronic renal failure, and anemia are common in this population.

- With long-standing complete paralysis, healing may not occur for several months.

HETEROTOPIC OSSIFICATION

Heterotopic ossification (HO) is the development of ectopic bone within the soft tissues surrounding peripheral joints and is a well-known complication after SCI. Although HO can occur in any joint below the level of injury, it develops most commonly about the hips (the primary site is the anterior hip), knees (the distal quadriceps is the second most common site), shoulders, and elbows. Onset is typically within the first few weeks or months following SCI, although it might continue to develop for 1-2 years or in the presence of precipitating factors such as a fracture. The etiology is unknown. The initial inflammatory process resolves, leaving the ectopic bony mass.

Incidence

The prevalence is thought to be approximately 20-30%, although only about half of those cases are clinically significant. Between 3-8% go on to total ankylosis.

Signs and Symptoms

- Decreased joint range of motion

- Localized swelling, warmth, erythema

- Fever

- Increased spasticity

- Pain, unless the person is insensate

Diagnosis

Initial findings include an elevated ESR, elevated serum alkaline phosphatase (might remain elevated for up to 2 years) and presence of new bone mass in the periarticular soft tissues on X-ray (in later stages, the bone mass is well-defined). Radionuclide bone imaging is used for early diagnosis and to determine the activity of HO mass. The differential diagnosis includes DVT, septic arthritis, avascular necrosis and contracture. Complications include decreased range of motion and the potential for pressure ulcers, venous compression (e.g., femoral vein), and DVT.

Treatment

The mainstay of treatment for HO has been pharmacologic with use of Didronel (etidronate disodium) and/or nonsteroidal anti-inflammatories (ibuprofen, indomethacin, and naproxen).

Specialists may consider low-dose radiation, although the indications for it remain controversial, or surgical resection in cases of major functional limitations from HO[3, 4]. The timing is important in that the heterotopic bone should be mature. Significant risks with surgery include bleeding, fracture, and recurrence of HO.

Functional implications depend on the amount and location of the ectopic bone.

- At the hip, HO may affect posture, seating, lower extremity dressing, and hygiene.

- Skin breakdown is frequently a problem from either direct compression over a large mass of heterotopic bone or secondary to poor posture and seating.

- At the shoulder, HO might further impair upper extremity movement in the individual with tetraplegia.

UPPER EXTREMITY PAIN AND DYSFUNCTION

Neck and Shoulder Pain

Since shoulder and neck problems are common following SCI, a number of orthopedic and neurologic considerations should be entertained, depending on the specific symptoms and signs present. In most cases, the history and physical examination will be helpful in recognizing the problem as being a primary shoulder or neck problem. Shoulder and neck pain are related most frequently to mechanical problems, but rheumatologic and neurologic conditions also occur, resulting in diverse clinical presentations and a broad differential diagnosis.

Incidence

The exact incidence and prevalence of chronic neck and shoulder pain after SCI are unknown. Several authors have reported that 30-50% of persons with SCI have significant chronic shoulder pain that interferes with daily activities[5, 6].

Signs and Symptoms

- Shoulder pain can present unilaterally or bilaterally.

- Symptoms may begin quite rapidly or gradually over several weeks or months.

- Frequently, there is no antecedent trauma, or the onset of pain occurred with a routine activity such as a pressure relief.

- Pain may present as a discrete, self-limiting event or remain as a chronic, persistent problem.

- Individuals may report no functional limitations or severe limitations, such as problems with pressure reliefs and transfers.

- Sleep is frequently disrupted, and finding a comfortable position in bed is often a problem.

Diagnosis

Evaluation and directed treatment of acute shoulder pain are similar to that in the able-bodied population, although it is important to maintain a high index of suspicion for neurologic etiologies (e.g., syringomyelia, cervical radiculopathy) and referred pain from the cervical spine. However, the effects of shoulder pain are quite different, and the ensuing secondary disabilities resulting from shoulder pain must be considered. One should rule-out systemic disease (e.g., rheumatoid arthritis) and neurologic etiology (e.g., syrinx, radiculopathy).

Differentiate between neck and shoulder pain (adapted from [7])

- Mechanical shoulder pain is exacerbated by shoulder movement.

- The symptom pattern in the shoulder frequently involves stiffness and instability.

- Establish a working diagnosis for shoulder pathology by distinguishing between musculoskeletal entities (e.g., subacromial bursitis, impingement syndrome, acromioclavicular degenerative joint disease, biceps tendonitis, rotator cuff tendonitis, and small rotator cuff tears) that can occur concomitantly in individuals with chronic mechanical shoulder pain.

- It is not uncommon to have coexisting neck and shoulder problems.

- Mechanical neck pain is aggravated by neck movement.

- Neck problems often refer to both shoulders.

- If the predominant symptom is not pain but weakness or sensory change, a neurologic lesion should be considered.

- If the predominant symptom is not mechanical pain, or if there is pain at rest that wakes the person at night, look for neoplasms, infections, and other less common diagnoses.

- Functional problems that are not consistent with the underlying disease process may indicate a nonorganic process.

Conduct a functional assessment to establish the functional impairment (restricted range of motion, weakness, instability) and handicap since treatment is often directed at the functional impairment rather than the pathophysiologic process.

- Evaluate passive and active range of motion for restrictions of motion and movements that induce pain.

- Perform resisted and provocative tests to assess forces that exacerbate pain.

- Evaluate posture.

- Evaluate the environment.

- Assess pressure reliefs, wheelchair mobility, transfers, bed mobility, and ambulation.

Effects of shoulder pain that are unique to persons with SCI

- Relative rest is often not possible.

- "Bad habits" and "damaging patterns of use" are frequently related to environment and accessibility issues.

- Pain often interferes with basic activities of daily living and mobility.

- Secondary problems (e.g., skin breakdown) are frequent complications from shoulder pain.

- Psychosocial problems and depression are common with the onset of secondary disabilities caused by shoulder pain.

Treatment

Most chronic mechanical shoulder problems in the SCI population are best treated nonsurgically, according to the following conservative principles:

- Proper biomechanics are essential to avoid injury to the shoulder.

- Normal alignment of the shoulder, head, and spine should be maintained.

- Early diagnosis and aggressive treatment are the mainstay of an effective program.

- The main objectives of early medical management are:
  - Relief of pain
  - Reduction of inflammation
  - Cessation of deleterious activities

- The main objectives of early physical therapy intervention are to recommend changes in:
  - Functional activities (transfers, wheelchair propulsion)
  - Balancing anterior and posterior muscle strength
  - Posture
  - Equipment
  - Home and work environments

- Avoid injury and overuse to the asymptomatic shoulder.

- Avoid skin breakdown at all costs.

- Provide an educational and home program to help each individual:
  - Correct impairments (e.g., restricted range, weakness)
  - Integrate changes in home and work environments
  - Return to a productive life

- Social and psychological services should be offered to any individual who has major secondary problems as a result of shoulder pain.

- A weight-loss program should be instituted when appropriate.

- Orthopedic referral for surgery should be reserved for those individuals who fail a 6-month program of conservative therapy.

- Make functional and equipment changes to minimize exacerbating factors.

Elbow, Wrist, and Hand Pain

Incidence

Overuse and repetitive motion disorders of the soft tissues and joints of the upper extremity are prevalent in persons with SCI[6, 8]. Around the elbow, olecranon bursitis, medial epicondylitis, and lateral epicondylitis occur frequently although the exact prevalence is not known; osteoarthritis of the elbow is seen occasionally. Several investigations have found that symptoms related to carpal tunnel syndrome are among the most common complaints of upper extremity pain[8].

Pathophysiology

Since the upper extremities are used for weight-bearing and frequent reaching, there are increased stresses on the more distal aspects of the arm as well as the shoulder. It is important to remember that individuals with tetraplegia are positioning the upper extremities in unique postures prior to weight-bearing. Someone with C6 tetraplegia, for example, will externally rotate the upper extremities and lock the elbows to use their innervated muscles for support and movement.

The wrist is forcefully extended and loaded during many weight-bearing activities (e.g., transfers, crutch use). Overuse problems of the wrist and hand include entrapment of the ulnar nerve within Guyon's canal, and osteoarthritis of the wrist, the trapeziometacarpal joint, and the proximal and distal interphalangeal joints. Loss of range of motion at the wrist and hand is common and often leads to compensation and subsequent problems at more proximal joints.

Diagnosis

Diagnosis of these disorders is similar to that in the able-bodied population, but treatment is not.

Treatment

- Relative rest is often difficult for anyone who uses a wheelchair for ambulation.

- Frequent pressure reliefs are imperative to avoid pressure ulcers, as are good technique and adequate lift in transfers to avoid shearing.

- Aggressive treatment is appropriate and includes the following:
  - Treat pain.
  - Treat chronic inflammation.
  - Do not immobilize.
  - Re-establish range of motion.
  - Evaluate the entire upper extremity, neck, and posture.
  - Minimize mechanical loads, correct poor technique.
  - Optimize equipment.
  - Rule out neurologic change.
  - Consider therapeutic modalities (ultrasound, friction massage, etc.) as recommended by a physiatrist.

Vertebral Column Pain

Incidence

Neck, mid-back, and low back pain are all common in the SCI population. Although secondary disorders and degenerative changes due to aging are not well described, one would expect an increased incidence of mechanical problems following fractures, dislocations, fusion, and instrumentation of the vertebral column.

Pathophysiology

Frequently, the most mobile segments of the vertebral column (e.g., C5-C7, T12-L1) are fixed, and motion occurs above and below the level of a fusion or instrumentation. Also, deformities are common following traumatic SCI (e.g., healing of the cervical spine in a flexed position, gibbous deformity, or scoliosis of the thoracolumbar spine), and compensatory intervertebral postures are assumed to offset these malformations.

Diagnosis

It is essential to recognize any neurologic change associated with spine pain because the differential diagnosis includes many degenerative and neurologic conditions that might cause neurologic decline (e.g., posttraumatic syringomyelia, spinal stenosis, herniated nucleus pulposus).

Evaluation of spine pain is difficult, and a comparison of findings with past examinations and radiographs is essential.

- Frequently, instrumentation is the cause of discomfort, either from loosening, broken hardware, or infection. Multiple views are necessary to inspect screws, rods, plates, and/or wires.

- Instability is high on the differential diagnosis.

- Flexion-extension films should be obtained to evaluate stability of the joint. For further delineation of osseous and soft-tissue changes, a CT scan is often obtained.

Neuropathic arthropathies of the intervertebral joints are rare but may develop[9, 10].

- Progressive degeneration and changes in articular cartilage and subchondral bone eventually lead to a grossly unstable joint.

- The individual may complain of a change in posture, a new deformity, pain, or a sensation of grinding, popping, or clunking.

- Instability may also lead to a loss in sitting balance.

- Radiographs reveal degenerative changes that are increasingly severe until there is finally no recognizable joint, only parosteal bone and a pseudarthrosis.

- Nuclear medicine studies and biopsy should be performed to differentiate between acute or chronic infections from Charcot arthropathy.

Treatment

Most chronic mechanical neck and low back problems in a person with a SCI are best treated nonsurgically. Similar principles as discussed with shoulder pain should be followed:

- Establish optimal posture in the wheelchair and in bed.

- Normal alignment of the head, shoulder, and spine should be maintained, with lordosis of the cervical and lumbar curves, if possible.

- Judicious use of relative rest for short periods of time may be effective.

- Short courses of Tylenol and NSAID are frequently of benefit.

- Modalities (e.g., superficial heat) and massage (short applications) are often used but remain controversial. Since extreme caution should be exercised in using deep heating, manipulation, and traction in the setting of spinal instrumentation and fusion, this decision is best left to a clinician with considerable experience.

- Exercise and modification of activities are the mainstay of treatment. Educational programs to avoid poor habits and proper biomechanics are fundamental.

- Occasionally, orthoses (e.g., cervical collars) are used for pain management and as a postural reminder (e.g., to avoid extreme range of motion).

- Social, vocational, and psychological services should be available to any individual who has major secondary impairments and disabilities as a result of new vertebral column pain.

- Treatment of an unstable spine and/or neurologic deterioration requires immediate referral to a SCI center.

Self-Study Review

1. *In the absence of discomfort due to a complete SCI, what signs, symptoms, and complications should alert the practitioner to a possible new fracture?*

2. *List several functional complications besides the many medical complications that can result from a fracture in an individual with SCI.*

3. *Describe the appropriate treatment plan components for a fracture in a person with SCI.*

4. *List several features that might be useful in differentiating shoulder pain from neck pain in an individual with SCI.*

5. *Why are the effects of shoulder pain unique in the SCI population?*

6. *Enumerate several nonsurgical treatment principles and components for shoulder pain in persons with SCI.*

7. *Explain why the diagnostic evaluation of elbow, wrist, and hand pain is similar in the SCI and non-SCI populations, but treatment approaches are very different.*

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10 NEUROLOGIC SYSTEM

James W. Little, MD, PhD

Objectives for the Learner

1. Appreciate the common neurologic complications secondary to SCI and their effects on the person with SCI.

2. Learn how to differentiate musculoskeletal pain from central neuropathic pain.

3. Gain a greater understanding of the range of interventions that are available to facilitate the management of chronic central neuropathic pain.

4. Learn appropriate treatments for spasticity and hypertonus.

5. Enhance skills for the early recognition of signs and symptoms of posttraumatic syringomyelia so that prompt treatment can be initiated to prevent further cord damage as a result of syrinx enlargement.

Overview

Common neurologic complications of SCI include chronic pain, which is often central neuropathic pain, spasticity, and late neurologic decline.

ACUTE SPINAL CORD INJURY

Immediate management is beyond the scope of this text. What is essential, however, is the need to start methylprednisolone within the first 8 hours of injury. Consult with a specialist for advice.

CHRONIC PAIN

Incidence

Chronic pain after SCI is common, with 5-44% of persons reporting that it is severe enough to impair daily activities. It is often either central neuropathic or musculoskeletal in origin.

Signs and Symptoms

The clinical history and physical examination may allow one to distinguish between central neuropathic pain and musculoskeletal pain (Table 10-1). Both may be present together.

TABLE 10-1. CENTRAL VERSUS MUSCULOSKELETAL PAIN[1-4]		
Type of Pain	**Symptoms**	**Signs**
Musculoskeletal	Aching, grinding, jabbing pain; worse with activity. Common at neck, shoulders, elbows, low back, hips, knees.	Pain worsens with palpation or joint motion.
Central neuropathic	Burning, tingling, stabbing, shooting pain.Continuous or episodic but often not related to activity. Dermatomal at zone of SCI, perianal, abdominal, or distal lower limb pain.	Pain does not worsen with palpation or joint motion. Patient may show hyperesthesia. Pain in an anesthetic area.

Diagnosis

Diagnosis consists of a clinical history and physical examination that may allow one to distinguish between central neuropathic pain, musculoskeletal pain, and other etiologies. Secondary conditions aggravating pain should be identified. Identification of other neurologic causes (syrinx, spinal stenosis, disc herniation, spasms, nerve entrapment, musculoskeletal causes, and medical factors) will allow appropriate treatment.

Treatment

Musculoskeletal pain can often be improved and may be curable. Treatments include:

• Mild analgesics

• Nonsteroidal anti-inflammatory medications

• Passive stretching

• Muscle strengthening

• Trigger point injections

• Improved posture

Chronic neuropathic pain has no known cure. Interventions often require a multi-disciplinary team (e.g., psychologist, social worker, recreation therapist, physical therapist, occupation therapist). However, many interventions yield little benefit exceeding placebo effect[4]. Practical goals:

• Treat secondary conditions aggravating pain[3]. Identify and treat neurologic causes (syrinx, spinal stenosis, disc herniation, spasms, nerve entrapment); musculoskeletal causes (tendonitis, overuse, osteoarthritis, contracture); and medical factors (constipation, UTI, hemorrhoids, pressure sore).

• Increase physical and social functioning by encouraging activities such as work, school, recreation.

• Psychosocial interventions include treatment for depression, drug/alcohol abuse, smoking, and anxiety. Patient education, reassurance, psychological support, relaxation techniques, self-hypnosis, and biofeedback are useful.

• Physical modalities include range of motion, stretching, strengthening, functional training, and desensitization. Superficial heat, ultrasound, and massage may be useful.

• Medications to use in a stepwise approach include analgesics (acetaminophen, aspirin, ibuprofen); antidepressants (amitriptyline, desipramine, nortriptyline); antiseizure medications (carbamazepine, phenytoin, gabapentin); lesser narcotics (codeine, oxycodone); greater narcotics[2] (methadone, slow-release morphine); and muscle relaxants (diazepam, lorazepam).

- Narcotic medications are reported by 10-20% of SCI patients to be of benefit in reducing central pain, but their use is fraught with complications (constipation, tolerance, addiction, illegal sale). Complications can be minimized by careful management, such as:

  1. Contract with individual for collaborative goals:
     - Partial pain relief; complete pain relief is not expected
     - Improved physical and social functioning
     - No escalation of dose and no early refills
     - No alcohol or illegal drug use
     - No narcotic or muscle relaxant from another provider

  2. Monthly clinic visits:
     - Adjust dose of narcotic medication to achieve above goals. Rarely exceed 20 mg/day of methadone.
     - Consider using a liquid pain cocktail containing methadone and possibly Vistaril and acetaminophen.
     - Refill medications contingent on monthly or bimonthly clinic visits.
     - Refill contingent on compliance with contract.
     - Refill contingent on achieving pain improvement, physical and social goals.

  3. If individual deviates from contract:
     - First error — reminder of contract
     - Second error — warning about termination of narcotic use and shorten the duration of prescription
     - Third error — taper off narcotics and institute non-narcotic treatments; may need hospitalization to withdraw from narcotic

- Surgical techniques undertaken in an SCI center may include dorsal root entry zone procedure, deep brain stimulation, or other options.

SPASTICITY AND SPASMS

Incidence

About 35% of individuals have severe spasms requiring treatment.

Pathophysiology

Hyperactive spinal reflexes of SCI cause flexor and extensor spasms, muscle hypertonicity, and clonus. Such spasms can impair voluntary movement, balance, function, skin integrity, and sleep.

Signs and Symptoms

Increased spasms may indicate new pathology, such as urinary tract infection, acute abdomen, inflamed hemorrhoids, and paronychia or any noxious stimulus.

Treatment

• Elimination of noxious input causing spasms

• Range of motion and static stretching

• Oral spasmolytic medication

The following medications are used (Table 10-2). There is no role for acute muscle relaxants. A gradual taper-off medication is recommended, especially with baclofen, due to central nervous system withdrawal effects.

TABLE 10-2.
MEDICATIONS FOR SPASTICITY AND SPASMS

Medication	Indications	Common Side Effects
Baclofen	1st line for spasms, clonus	Withdrawal hallucinations and seizures
Clonidine	2nd line for spasms, clonus	Sedation, hypotension
Diazepam	2nd line for spasms, clonus	Sedation, depression, addiction
Tizanidine	3rd line for spasms, clonus	Sedation, hypotension
Dantrolene	3rd line for spasms	Hepatotoxicity, weakness

Treatment best undertaken in an SCI Center includes

• Neurolytic blocks with ethyl alcohol, phenol, or botulinum toxin

• Tenotomy or tendon lengthening

• Intrathecal baclofen infusion

• Myelotomy (incising the lumbar cord) or dorsal rhizotomy (cutting the dorsal roots).

NEUROLOGIC DECLINE

Overview

Late neurologic decline is often subtle and responds best to early treatment.

Incidence

About 15% will experience late neurologic decline, either due to peripheral or central neurologic complications. Posttraumatic syringomyelia (PTS), a fluid-filled syrinx that develops at the SCI site and extends rostrally or caudally[5], occurs in over 3% of persons with SCI.

Pathophysiology

The etiology of PTS is unknown. Cord damage results as the syrinx enlarges. Syrinxes often ascend unilaterally, causing ascending loss of pain sensation and reflexes unilaterally. Peripheral nerve entrapment, often at the carpal tunnel or ulnar groove, may accompany SCI. Spinal stenosis, either cervical or lumbar, and disc herniation, either cervical, thoracic, or lumbar, can also compromise neurologic function in persons with SCI.

Signs and Symptoms

The symptoms of peripheral nerve entrapment, often at the carpal tunnel or ulnar groove, may be similar to PTS, but the following symptoms pertain especially to PTS.

- Pain

- Increased diaphoresis

- Increased or decreased spasticity

- Worsening orthostatic hypotension

- Increased sensory loss or weakness

Signs, either above or below the SCI level, include

- Sensory loss (particularly pain and temperature sense)

- Weakness

- Reflex loss

Diagnosis

Neurologic examinations must be conducted regularly after SCI and documented in enough detail to identify neurologic decline. Much of the necessary diagnostic evaluation is best accomplished in an SCI Center. Serial measurement of voluntary strength can also help confirm neurologic decline; pinch meters, grip meters, handheld myometers, and isokinetic dynamometers are useful for quantitating strength. Additional tests include electromyography, nerve conduction studies (peripheral nerve, segmental H-reflexes and F-waves, and long-tract somatosensory evoked potentials and motor evoked potentials); spinal cord magnetic resonance imaging; and CT myelography.

Treatment

- PTS is treated by avoidance of Valsalva maneuver and percutaneous CT-guided drainage and surgical shunting.

- Spinal stenosis or disc herniation is treated by surgical decompression, if there is significant neurologic decline.

- Peripheral nerve entrapment may be managed with splinting or nerve protection but may require surgical decompression.

OTHER NEUROLOGIC ISSUES

- Seizures and stroke may be less evident in persons with SCI because the neurologic signs are obscured by the SCI neurologic deficits.

- Extensor spasms and clonus can be mistaken for tonic-clonic seizures. Associated traumatic brain injury (TBI) with residual cognitive impairment is found in about one third of persons with SCI. This may result in subtle cognitive deficits, including impaired judgment.

- Cerebrospinal fluid (CSF) analysis is not routinely undertaken in a fever workup unless there are central nervous system signs and symptoms. After SCI, there may be an obstruction to CSF flow, with mildly elevated white blood cells and elevated protein in the CSF.

Self-Study Review

1. *What are the common neurologic complications in SCI?*

2. *List features that might be helpful in differentiating central neuropathic pain from musculoskeletal pain.*

3. *Describe one chronic pain intervention that could be contributed by each member of a multidisciplinary team.*

4. *What general parameters would you include in policies regarding opioid medication use for chronic pain that could minimize complications?*

5. *Construct a hierarchy of indicated medications for spasticity treatment going from first-line to third-line agents.*

6. *List several symptoms and signs that would raise your diagnostic suspicion of the presence of PTS.*

7. *What battery of neurologic diagnostic assessments would provide adequate detail to detect and identify neurologic decline in individuals with SCI?*

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Medical Care of Persons with Spinal Cord Injury

Independent Study Test Questions for CME Credit

1. **Which of the following statements is likely <u>NOT</u> true regarding a lower motor neuron injury?**

   a) Decreased muscle tone, absent reflexes, and muscle atrophy often characterize a lower motor neuron injury.

   b) A person with a longstanding injury of more than thirty years is likely to show gradual change from upper motor neuron injury to lower motor neuron injury findings.

   c) Spasticity is a common complication of lower motor neuron injury.

   d) A person with a cervical SCI who exhibits flaccidity and lower extremity atrophy should be evaluated to rule-out neurologic loss or lower cord vascular infarction.

2. **A person with SCI who had greater proprioceptive and motor loss ipsilateral to their lesion and contralateral loss of sensitivity to pain and temperature is most likely to have which one of the following SCI syndromes?**

   a) Central Cord Syndrome.

   b) Conus Medullaris Syndrome.

   c) Anterior Cord Syndrome.

   d) Brown-Sequard Syndrome.

3. **Which of the following most accurately defines the B level of impairment on the ASIA Impairment Scale?**

   a) Motor complete, sensory incomplete below the neurologic level.

   b) Motor incomplete, sensory complete below the neurologic level.

   c) Motor complete, sensory complete below the neurologic level.

   d) Motor incomplete, sensory incomplete below the neurologic level.

4. **Which of the following could be common normal findings among individuals with SCI?**

   a) Orthostatic Hypotension.

   b) New Loss of Sensation and/or Function.

   c) Bradycardia.

   d) A and C, but NOT B.

5. **Which of the following does <u>NOT</u> complicate assessment in persons with SCI?**

   a) Altered sensory function and vague symptoms mask expected symptoms of illness.

   b) Persons with chronic injuries are unlikely to accurately report their level and completeness of SCI.

   c) Persons with SCI have unique medical syndromes (e.g., autonomic dysreflexia) that may be unfamiliar to most healthcare practitioners.

   d) Alterations in basic physiology following SCI may contribute to nonstandard findings or vital signs.

6. **Which of the following statements is <u>FALSE</u> regarding altered sensation following SCI?**

   a) Higher level injuries usually have impaired or absent sensation from intra-abdominal pathology.

   b) Differentiating neuropathic pain from symptomatic exacerbations due to new pathology is often difficult.

   c) Having altered sensation or neuropathic pain below their level of injury is impossible for an individual with a complete SCI (ASIA A).

   d) Persons with SCI are likely to have greater difficulty in localizing sources of pain.

7. **Which statement is <u>NOT</u> true?**

   a) Autonomic dysreflexia occurs in 85% of susceptible individuals.

   b) Autonomic dysreflexia usually occurs in individuals who have conus medullaris syndrome.

   c) Individuals may have the clinical signs of autonomic dysreflexia but be asymptomatic.

   d) Autonomic dysreflexia is less likely to occur during periods of spinal shock.

8. **Which are the two most common triggers for autonomic dysreflexia episodes?**

   a) Genitourinary and gastrointestinal.

   b) Dermatologic and musculoskeletal.

   c) Pulmonary and cardiovascular.

   d) Medications and radiologic procedures.

9. **Which of the following is <u>NOT</u> a treatment for autonomic dysreflexia?**

   a) Sitting the person upright.

   b) Checking the bowel and bladder to alleviate noxious stimuli.

   c) Encouraging the patient to lie down until his or her headache ameliorates.

   d) Pharmacologic intervention when the systolic blood pressure is greater than 180 mm Hg.

10. **A pressure ulcer involving full-thickness skin loss and involving damage to or necrosis of subcutaneous tissue that may extend down to, but not through, underlying fascia and clinically presents as a deep crater with or without undermining of adjacent tissue would be most appropriately classified as which stage?**

    a) Stage I.

    b) Stage II.

    c) Stage III.

    d) Stage IV.

11. **Nutritional assessment could raise significant concerns regarding prognosis for skin healing if:**

   a) Anemia is not present and no co-morbid conditions are noted.

   b) There has been body weight loss of greater than 15% and serum albumin levels are less than 3.5 mg/dL.

   c) The individual is well nourished, well hydrated, and consumes a diet providing all recommended daily allowances.

   d) Zinc and Vitamin C deficiency is not suspected.

12. **If an individual with SCI presents with a stage III or greater skin pressure sore, the need for analgesia should be evaluated:**

   a) Always.

   b) Only when requested by the patient.

   c) Only in individuals with ASIA Impairment Scale Scores of E.

   d) Only in persons with pre-existing neuropathic pain syndromes.

13. **Assisted cough to clear secretions may be indicated with which types of injury?**

   a) Only with cervical injuries.

   b) Only with individuals having high level cervical injuries (C1-5).

   c) Injuries at T8 or higher with inadequate coughs.

   d) Conus Medullaris Syndrome.

14. **Which factor is <u>NOT</u> likely to contribute to late onset ventilatory failure?**

   a) Late neurologic decline.

   b) Postural drainage.

   c) Progressive kyphosis or scoliosis.

   d) Aging with accompanying loss of diaphragm motoneurons.

15. **Deep venous thrombosis is related to which of the following?**

    a) Hypercoagulability.

    b) Venous stasis.

    c) Vessel wall injury.

    d) All of the above.

16. **With reduced central inhibition of spinal sympathetic outflow, which of the following is true?**

    a) Plasma renin activity is diminished.

    b) Angiotensin II is decreased.

    c) Aldosterone is reduced.

    d) None of the above.

17. **Which of the following is generally <u>NOT</u> recommended as a treatment for orthostasis in individuals with SCI?**

    a) Diuretics.

    b) Fludrocortisone (mineralocorticoid) administered daily to expand the intravascular volume via sodium retention.

    c) Minimize dependent edema with elevation and compression with elastic stockings.

    d) Abdominal binders.

18. **Which of the following are risk factors for cardiac arrhythmias in persons with SCI?**

    a) Hypoxia.

    b) Hypotension.

    c) Fluid/Electrolyte Imbalances.

    d) All of the above.

19. **Which of the following increases the risk for cholelithiasis in individuals with SCI?**

    a) Increased intestinal transit time with rapid circulation of bile salts.

    b) Anabolic changes after SCI.

    c) Gallbladder stasis from impaired neural and hormonal control.

    d) Increased risk of Type I diabetes mellitus.

20. **Which of the following is a common characteristic of a lower motor neuron pattern of neurogenic bowel dysfunction?**

    a) The anal sphincter has low tone.

    b) External anal sphincter constriction secondary to a hyperactive holding reflex.

    c) Overactive segmental peristalsis.

    d) Fecal distention of the colon leading to megacolon.

21. **Besides appropriate oral and rectal medications, proper design of the bowel program should include consideration of:**

    a) Dietary modulation of fiber and liquid intake.

    b) Scheduling of bowel care time and frequency.

    c) Prescription of appropriate equipment.

    d) All of the above.

22. **Which of the following statements is <u>FALSE</u> regarding an upper motor neuron type of neurogenic bladder dysfunction?**

    a) Increased risk for high detrusor pressure is common.

    b) There is a lower risk of upper tract deterioration in this type of dysfunction.

    c) Detrussor-sphincter dyssynergia can lead to vesicoureteral reflux even without Crede maneuvers.

    d) The bulbocavernosus reflex is usually intact in this type of dysfunction.

23. **When would anticholinergics be contraindicated?**

   a) Trying to reduce detrusor contractility.

   b) In promoting storage of urine.

   c) In individuals with upper motor neuron bladder dysfunction who are managed with intermittent catheterization.

   d) In a person with condom drainage who relies on reflex bladder contraction for bladder drainage and is at significant risk for UTIs, bladder over-distention, and vesicoureteral reflux.

24. **Which of the following statements regarding calculi is <u>FALSE</u>?**

   a) Hypercalciuria is more closely associated with the chronic phase while struvite stones are more common in acute phases.

   b) Ureteral stones may be asymptomatic even in the face of severe obstruction and renal damage.

   c) Extracorporeal shock wave lithotripsy is generally used for calculi of less than three cm.

   d) Particles remaining after probe lithotripsy may be dissolved with hemiacidrin solution (Renacidin).

25. **If immobilization is indicated for a fracture in an individual with SCI, which of the following approaches should <u>NOT</u> be used?**

   a) Soft, well-padded splints.

   b) Bivalved cast wrapped with an elastic bandage.

   c) Full circular casts.

   d) Bivalved cast with appropriate changes in wheelchair and seating.

26. **Which of the following might be an evaluation or laboratory result consistent with the diagnosis of heterotopic ossification?**

   a) Decreased ESR.

   b) Fever.

   c) Decreased serum alkaline phosphatase.

   d) Negative radionuclide bone imaging for activity.

27. **Which of the following should be regarded as a treatment to receive first line consideration for neck, shoulder, or low back pain in a person with SCI?**

    a) Surgery.

    b) Proper biomechanics and normal alignment.

    c) Traction.

    d) Complete immobilization.

28. **Which of the following clinical features is <u>unlikely</u> to help in the differentiation of central neuropathic pain from musculoskeletal pain?**

    a) Adjectival sensation descriptions.

    b) Dermatome distributions.

    c) Intensity.

    d) Exacerbation by motion.

29. **Appropriate evaluation and treatment of spasticity is important for all of the following reasons except which one?**

    a) Spasms can impair balance.

    b) Spasms usually facilitate voluntary movement.

    c) Spasms may be indicative of new pathology (e.g., syringomyelia, urinary tract infections, acute abdomen, inflamed hemorrhoids, and paronychia).

    d) Spasms can impair skin integrity and sleep.

30. **Which of the following symptoms could suggest the new onset of post-traumatic syringomyelia?**

    a) Increased pain complaints.

    b) Decreased diaphoresis.

    c) Fewer episodes of orthostatic hypotension.

    d) Greater strength.