Basic Athletic Training
Course Pack D

CHAPTER

30

Athletes with Physical Disabilities

STUDENT OUTCOMES

1. Identify the disability groupings of athletes participating in Paralympic sports.
2. Define spinal cord injury (SCI) in terms of complete or incomplete.
3. Utilize the American Spinal Injury Association (ASIA) Impairment Scale level to describe spinal cord injury by level of neurological impairment.
4. Explain tetraplegia, thoracic paraplegia, and lumbar paraplegia and implications for participation in activities of daily living and sports.
5. Describe why persons with SCI are at higher risk for

developing urinary tract infections, neurogenic bowel, pressure sores, blood clots, and respiratory complications and provide strategies for preventing, identifying, and treating the condition.

6. Define autonomic dysreflexia.
7. Describe specificity in terms of cause, type, and complications.
8. Explain effects of spinal cord injury on exercise.
9. Educate others regarding the pathophysiology and impairments experienced by an individual with cerebral palsy (CP).
10. Provide examples of how using the tools for classifying cerebral palsy assist the athletic trainer in caring for athletes with CP.
11. Explain the effects of CP on exercise and the evaluation and treatment of sport-related injury.
12. Educate others regarding the causes of amputation and pathophysiology and impairments experienced by individuals with amputation.
13. Explain the effects of amputation on exercise and the evaluation and treatment of sport-related injury.

INTRODUCTION

Sports participation by athletes with disabilities originated as a key component of rehabilitation programs designed for those who had been severely injured and impaired in World War II. By 1960, the first Paralympic Games took place, with 400 athletes from 23 countries participating.[1] It is estimated that in the 2016 Paralympic Games, more than 4,350 athletes will compete at this elite international competition.[2] Athletes participating in Paralympic sports are grouped based on disability: physical impairment, visual impairment, and intellectual impairment.[3] Physical impairment includes impaired muscle

power, impaired passive range of motion, loss of limb or limb deficit, leg length difference, short stature, hypertonia, ataxia, and athetosis.[3] Today, about 15% of the world's population experiences some sort of disability.[4] In the United States, there are 26 organizations for persons with disabilities who desire to participate in competitive and recreational sports (**Table 30.1**).[5] For athletes participating at the highest level, the Paralympic Games, injury rates are similar to those found for able-bodied (AB) participants competing in similar elite level events.[6] Overuse injuries are more prevalent than acute injuries, although acute injuries are frequently seen in ball-handling sports.[6] Wheelchair athletes have the highest risk of sustaining upper extremity injuries, whereas athletes with spinal cord injury are at risk for sustaining lower leg fractures due to poor bone density.[6] Due to altered biomechanics and issues related to prosthetics, amputee athletes appear to have the highest incident of injury and pain. Athletes with cerebral palsy experience the highest number of musculoskeletal soft-tissue injury.[6] This chapter will cover the most common physical disability groups represented in the Paralympic Games: spinal cord injury, cerebral palsy, and amputation. Information regarding classification, typing, complications, and exercise and treatment concerns for athletes with physical disabilities within each of these categories is presented.

TABLE 30.1 Disability Sports

ORGANIZATION	CONTACT INFORMATION
Adaptive snow sports	
a. Alpine skiing	a. http://www.disabledsportsusa.org/adaptive-skiing-alpine/
b. Sit skiing	b. http://www.spinalcord.org/access-sit-ski-solutions/
c. Adaptive snowboarding	c. http://www.disabledsportsusa.org/snowboarding/
d. Cross country skiing	d. http://www.disabledsportsusa.org/cross-country-skiing/
e. Sledge hockey	e. http://www.usahockey.com/sledhockey
Blind sports	
a. Alpine skiing	http://www.disabledsportsusa.org/adaptive-skiing-alpine/
b. Archery	
c. Athletics	
d. Chess	
e. Football	
f. Goalball	
g. Judo	
h. Nine-pin bowling	
i. Cycling	
j. Shooting	
k. Showdown	
l. Swimming	
Wheelchair sports	
a. Basketball	http://www.wheelchairsportsfederation.org/
b. Curling	
c. Racing	
d. Rugby	
e. Tennis	
f. Dance	
g. Fencing	
h. Power hockey	
i. Netball	
j. Soccer	
Adaptive water sports	
a. Sailing	http://wnyadaptivewatersports.org/wp/
b. Scuba diving	
c. Skiing	
d. Fishing	

SPINAL CORD IN JURY

? Mary is about to conduct an assessment of an athlete with a spinal cord injury. During the history portion of the examination, what information will be most useful in helping her to obtain a comprehensive picture of his injury? After obtaining the history, what potential complications might this patient experience based on his level of spinal cord injury? Could these complications impact the physical examination?

Spinal cord injury (SCI) can be defined as a lesion to the neural elements within the spinal canal (i.e., the cervical, thoracic, lumbar, and sacral segments of the spinal cord, including the conus medullaris and caudal equina). These

injuries can occur due to trauma or congenital in nature, as in spina bifida. For the purpose of this chapter, traumatic and congenital SCI will be treated in the same manner. Estimates of the number of individuals with SCI currently living in the United States have been reported as high as 400,000 with approximately 12,000 new injuries each year.[7] The most common cause of traumatic SCIs is motor vehicle accidents, representing approximately 42% of the injuries in the United States. The majority of persons living with SCI are male (82%).[7] Common sports for athletes with SCI include track and field, basketball, and rugby. Athletes with SCI utilize wheelchairs specifically designed for their chosen sport, with the exception being swimming.

Types of Spinal Cord Injury

SCI can be classified into two categories: complete and incomplete. Individuals who have sustained a complete SCI have no sensory or motor function below the level where the injury occurred (**Fig. 30.1**). Individuals who have sustained an incomplete SCI have partial sensory and motor function below the level where the injury occurred. SCI is also described by the *Neurological Level of Injury*, or the lowest or most caudal, fully intact and functioning spinal nerve segment, for both sensory and motor function, after SCI.

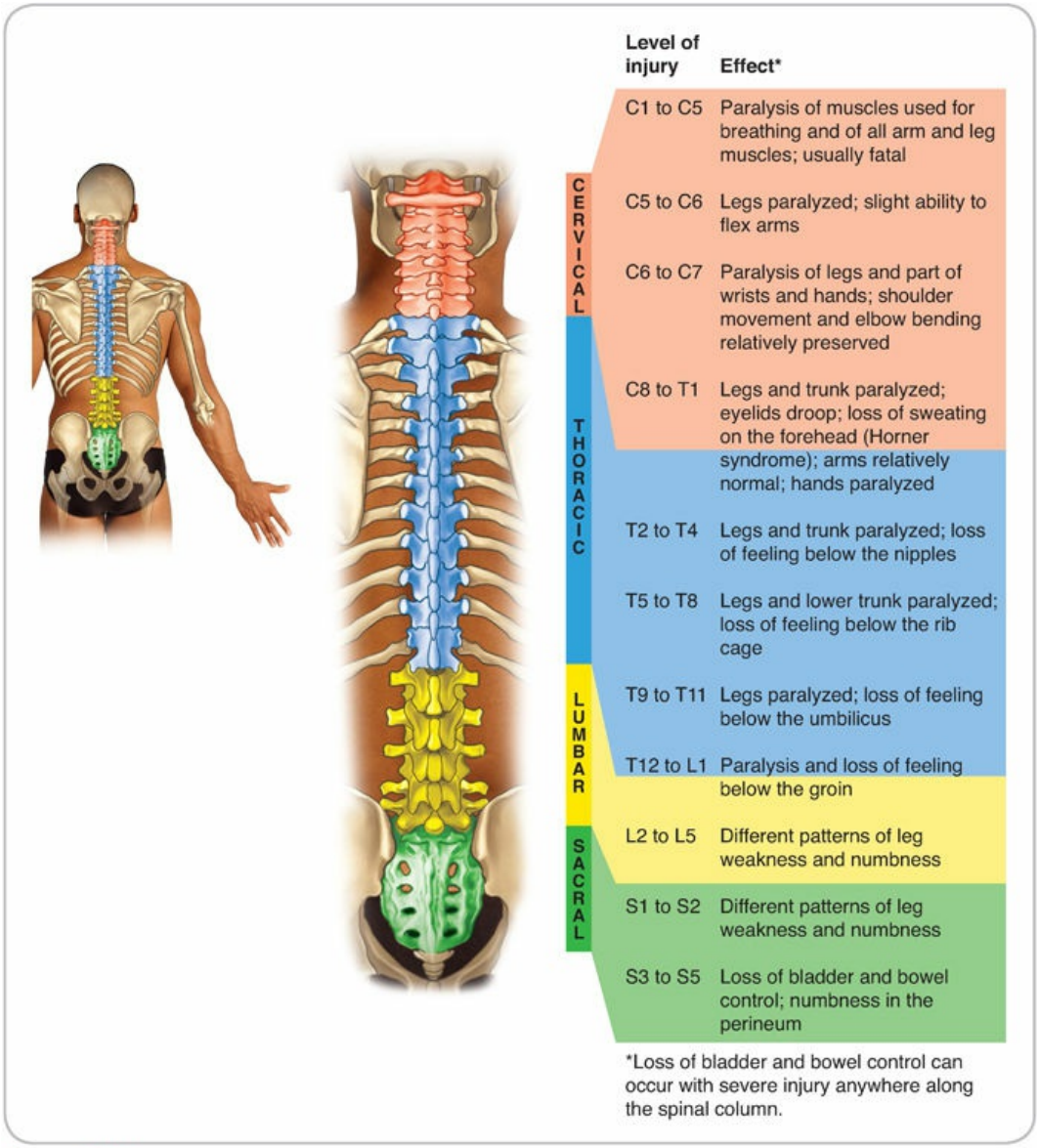

Figure 30.1. Neurological levels of impairments.

Neurological Level of Injury

The American Spinal Injury Association (ASIA) Impairment Scale describes the neurological levels of injury based on the resulting functional impairments due to SCI.[8] The classifications include the following:

A = Complete: no motor or sensory function in the lowest sacral segment (S4 to S5)

B = Sensory incomplete: some sensory function below neurological level and in S4 to S5

C = **Motor incomplete:** There is voluntary anal sphincter contraction, with some motor function to three levels below the motor level of the injury, but most of the key muscles are weak.

D = Motor incomplete: the same as C, with the exception that most of the key muscles are fairly strong

E = Normal: Although hyperreflexia may be present, normal sensory and motor recovery are exhibited.

Complete Spinal Cord Injury (Tetraplegia)

Tetraplegia results in impairment of both motor and sensory function of the arms, trunk, legs, and pelvic organs and is caused by complete SCI within the cervical region. Previously, tetraplegia was known as quadriplegia, but both refer to the same condition.[7] Complete SCI at the C1 to C3 level results in the most significant amount of impairment. A person who has sustained a complete SCI between C1 and C3 will need a ventilator in order to breath and will have no motor or sensory function of any of the four extremities. Complete SCI below C3 may require initial use of a ventilator, but eventually, the patient will regain control over lung function, as the lungs are innervated from the C4 nerve root. Patients sustaining complete SCI at the C1 to C4 level are able to control power wheelchairs by manipulating the control stick with the chin or mouth.[8]

Complete SCI at the C5 level results in the ability to flex elbows and the possibility of using hand controls to steer a power wheelchair. It is possible that a person with C5-level SCI may be able to utilize a manually powered wheelchair but will still need assistance with transferring from the wheelchair to another chair or bed.[9]

Persons with complete SCI at the C6 level are able to flex the elbows, flex and extend the wrists, are able to use assistive devices to grasp objects, and provide assistance during transfers. It is also possible for individuals with C6 SCI to drive adaptive vehicles designed with acceleration and breaking controls on the steering wheel.[9]

Persons with complete SCI at the C7/C8 level have the greatest ability to engage in independent living because wrist extension (C7) and finger flexion (C8) is still possible. Assistive devices enable the patient to engage in

activities of daily living, including personal care, driving, and even typing.[9]

Incomplete Spinal Cord Injury

Individuals with incomplete SCI will have some degree of sensory and motor function below the point of injury. However, the extent of damage is not known until 6 to 8 weeks after the initial injury. Depending on how the cord has been damaged, an individual may have some sensory function and limited to no motor function, or may have limited to no sensory function but retain motor function. There are five categories of incomplete SCI, and each is named for the portion of the spinal cord that has been damaged (**Box 30.1**).

BOX 30.1 Classification of Incomplete Spinal Cord Injuries

- **Anterior cord syndrome:** results in an impaired ability to correctly detect temperature, touch, and pain sensations below the point of injury. Eventually, some movement may later be recovered.
- **Central cord syndrome**: results in loss of motor function in the arms but some leg movement is retained. Some recovery is possible.
- **Posterior cord syndrome**: The patient will retain good muscle power, pain, and temperature sensation but have poor coordination.
- **Brown-Sequard syndrome**: results in opposing bilateral function because the damage is to the lateral aspect of the spinal cord. The patient will have unilateral impaired loss of movement but preserved sensation on one side of the body and preserved movement and loss of sensation on the other side of the body.
- **Cauda equina lesion**: results in loss of full or partial sensation below L1 and L2. It is possible that function may eventually be restored.

Adapted from BrainAndSpinalCord.org. Spinal cord injury. http://www.brainandspinalcord.org/spinal-cord-injury.html. Accessed November 24, 2015.

Thoracic Paraplegia

The majority of individuals with SCI who participate in recreational and competitive sports are thoracic paraplegics, meaning they have full motor and sensory function of the upper extremities and some of the torso. Individuals with SCI damage between T1 and T6 may obtain a high degree of independency with regard to self-care and wheelchair control. Individuals with damage between T6 and T12 are able to balance and engage quite successfully in recreational and competitive sports and may even walk with the assistance of canes, crutches, or walkers.

Lumbar Paraplegia

Individuals with lumbar paraplegia will have full motor and sensory function of the torso and upper extremities but have varying degrees of motor and sensory deficits in the lower extremities. People who have lumbar and/or sacral spinal cord injuries can obtain full independence, ambulate using walking assistive devices, and even drive. However, spinal cord damage at any level may result in loss of bladder, bowel, and sexual function.

Complications Associated with Spinal Cord Injury

Patients with tetraplegia need varying levels of assistance with feeding, personal hygiene, dressing, toiletry needs, transportation/mobilization, and transfers. Patients with paraplegia may be functionally independent and encounter fewer medical complications associated with their SCI. Depending on the level within the spinal cord where the impairment occurs, attention should be focused on preventing secondary complications from occurring. From a prevention, recognition, possible treatment, and referral perspective, the athletic trainer, as part of the health care team, needs to be aware of several complications associated with SCI.

Urinary Tract Infection and Neurogenic Bowel

Depending on the level of injury, people with tetraplegia have differing levels of bowel and bladder control. **Urinary tract infections** (UTIs) are common among individuals with SCI and may be life threatening if not recognized early

and treated.[9] A UTI describes any infection that begins in the urinary system. Although many cases are simply painful and annoying, the condition can become a serious health problem if the infection spreads to the kidneys. Although any structure in the genitourinary system can be infected, most infections occur in the lower tract. **Cystitis** (inflammation of the bladder) and **urethritis** (inflammation of the urethra) are the most common basis for UTIs. Most cases are caused by *Escherichia coli*, which ascend the urinary tract from the opening in the urethra. The bacteria also may be introduced during urinary tract catheterization. Elite athletes competing with complete SCI catheterize an average of four times a day and often reuse the same catheter multiple times, increasing the risk of developing UTIs.[10] Some individuals may present with very few symptoms, yet have significant **bacteriuria**. An individual with cystitis may complain of pain during urination (**dysuria**), urinary frequency and urgency, and pain superior to the pubic region. However, in the athlete with SCI, the patient has no or limited sensory function, so may be unable to feel the pain or the need to void. Therefore, care should be taken to inspect the urine for cloudiness, blood, or a foul-smelling odor. Other symptoms, such as high fever, shaking, chills, nausea, and vomiting, may indicate a simultaneous upper UTI of the kidney (**acute pyelonephritis**).

Following complete SCI, **neurogenic bowel** occurs due to impaired sphincter muscle control and disruption of normal ambulation impacting normal bowel motility. As a result, constipation or fecal incontinence may occur, posing serious health concerns for the individual with SCI.[10] Clinically, neurogenic bowel presents one of two ways, depending on the level of spinal cord impairment. The ability to voluntarily control the external anal sphincter muscle is impaired, resulting in the rectum being constantly constricted, and an inability to voluntarily expel fecal matter. This is due to injury to the spinal cord above the conus medullaris (lower end of the spinal cord between T12 and L1) and is referred to as **upper motor neuron (UMN) bowel syndrome**. In order for the stools to be evacuated, the rectum must be externally stimulated. Injury to the spinal cord below the conus medullaris and cauda equina causes a loss in control over anal muscles that control the opening to the rectum and is referred to as **lower motor neuron (LMN) bowel syndrome**.

LMN bowel syndrome is characterized by both constipation and fecal incontinence.[11]

Pressure Sores

Pressure sores are also referred to as bedsores or decubitus ulcers. As the name implies, the lesions are caused by constant pressure applied over a period time, most often in areas of boney prominences. Pressure sores are frequently found over the ischial tuberosity, greater trochanter, medial epicondyle of the femurs, and the posterior aspect of the calcaneus and elbows. The skin is able to withstand pressure for short periods of time before the tissue begins to break down. Because of the sensory deficits experienced by individuals with SCI, the brain does not receive the pressure or pain impulses, nor can the individual redistribute the weight in order to relieve the pressure on the affected area. The rate of blood flow is decreased as well, and as a result, the healing process is impaired.[12] The skin begins to break down after about 15 minutes of constant pressure, with the first sign being redness. Tissue break down occurs in four stages (**Fig. 30.2**). **Application Strategy 30.1** describes the signs of pressure sores as well as the management of each stage. If left untreated, bedsores can be life threatening, as the infection may spread to the vital organs. Other causes of bedsores include prolonged exposure to wet skin from sweat, stool or urine, and bruises, burns, or scrapes that go untreated.[12]

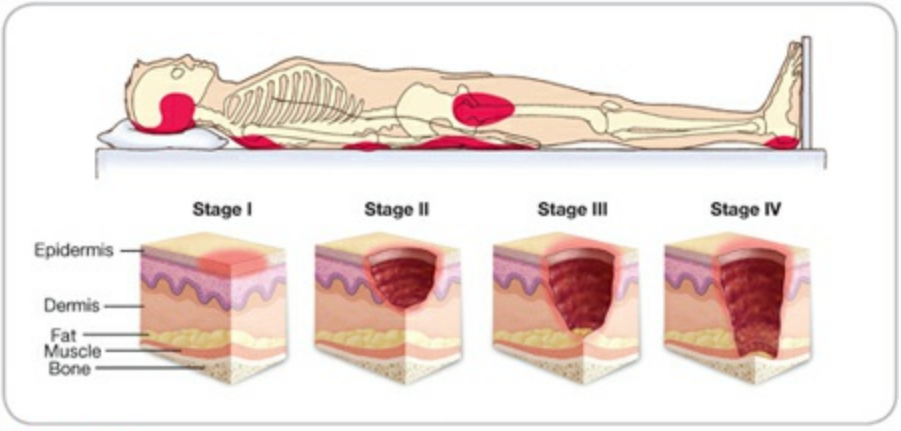

Figure 30.2. Classification of pressure sores by stage.

APPLICATION STRATEGY 30.1

Stages of Pressure Sore Progression and Management Strategies

Stage 1

Signs

- Redness or discoloration appearing within 15 minutes that does not resolve or fade within 30 minutes

Management

- If area is wet, clean with mild soap and water, patting the skin dry (not wiping) with a soft cloth. Ensure that clothing and padding are also dry.
- Continue to monitor the area. If redness/discoloration does not resolve within 2 days, refer the patient for further medical evaluation.

Prevention

- Keep the area clean and dry.
- Check for sources of friction, such as worn out padding and replace as needed. Inspect any protective equipment, seat cushions, and straps and replace as needed.
- Immediately attend to bruises, scraps, and burns.

Stage 2

Signs

- The epidermis appears broken, and a wound begins to appear. Drainage may or may not be present.

Management

- The wound should be cleaned with saline and patted dry using a soft, sterile cloth. If little to no drainage is present, use a bio-occlusive, self-adherent hydrocolloid dressing to prevent infection. This may be left in place for up to 5 days, but the patient should be inspected frequently for signs of infection.

Prevention

- Consult frequently with a medical director during this period and immediately refer the patient if the condition deteriorates. Keep the area dry and reduce the amount of pressure placed on the area if possible.

Stage 3

Signs

- The depth of the wound extends into the dermis and underlying tissues.

Management

- Contact a medical director for instructions on special cleaning and debriding techniques. The wound may need to be packed and antibiotics administered.

Prevention

- May be necessary to utilize a mechanical pneumatic mattress or chair seats that constantly change the pressure of the surface in contact with the affected area. Continue to monitor, keeping the area dry and clean.

Stage 4

Signs

- Tissue damage extends into the muscle and the bone may be visible. Dead and damaged tissue is clearly seen and may be accompanied by a foul smell.

Treatment

- Consult your medical director immediately as surgery is needed.

Blood Clots

Individuals with SCI are at risk for developing a blood clot, or **deep vein thrombosis** (DVT), due to the lack of muscle contraction and decreased blood flow, especially during the acute phase of an injury and when bedridden.[13,14]

Preventive measures include the use of compression stockings and blood thinners. Blood clots form most often in the veins of the thigh and lower leg. If the clot becomes dislodged, it may travel through the body to the lungs, resulting in a pulmonary embolism. DVTs may present with or without symptoms. In the SCI population, DVTs may be even more difficult to recognize because the patient may not experience the pain and tenderness that can be present with this condition. The affected area may become swollen and red in the absence of any known trauma.[15] If a DVT is suspected, refer the patient to the nearest medical facility immediately.

Respiratory Considerations

As mentioned in the discussion on DVTs, individuals with SCI are at risk for developing a **pulmonary embolism** (PE) secondary to a DVT. Again, the risk is highest during the acute phase of the initial injury but should always be a concern in any SCI patient. A patient experiencing a PE may present with difficulty breathing, arrhythmia, tachycardia, chest pain or discomfort, excessive coughing or coughing up blood, light-headedness, low blood pressure, and fainting.[15] For patients with SCI, signs are dependent on the level at which the spine has been damaged. A more common respiratory complication is **pneumonia**. Preventative measures include working with a respiratory therapist on breathing exercises. Please refer to Chapter 26 for a full discussion on the causes, sign and symptoms, complications, and management options of pneumonia.

Autonomic Dysreflexia

Autonomic dysreflexia is a life-threatening condition seen in both the acute and chronic phases of injury for individuals having sustained SCI above the T6 level. Pain or irritation due to abnormal conditions below the damaged spinal segment attempt to send a signal to the brain that is interrupted and/or interpreted incorrectly. The most common trigger is irritation of the bladder or colon.[16] In response, the blood pressure rises and heart rate may increase or decrease. The patient may have no symptoms or may complain of a headache and mild discomfort. However, if the blood pressure continues to rise and

causes increasing intracranial pressure, the condition may become a life-threatening emergency. Potential outcomes include intracranial hemorrhage, retinal detachment, seizures, and death.[16] Two important factors that influence the severity and occurrence are the level of injury and the completeness of a SCI. There is a greater cardiovascular response at higher levels of injury. About 27% of tetraplegics with incomplete SCI will present with autonomic dysreflexia as compared to 91% of tetraplegics with complete lesions. One way to prevent an occurrence of autonomic dysreflexia is eliminating the cause of the irritation. Because a common cause of the condition is from bowel or bladder irritation, it is imperative that anticholinergic drugs and clean intermittent catheterization techniques are utilized.[10]

Spastic Muscles

In the months following the initial SCI, the patient may experience muscle **hypertonus**, **hyperreflexia**, **clonus**, and muscle spasm.[17] In fact, 80% of people who have suffered SCI develop muscle spasticity. Acute prolonged spasmodic episodes may be triggered by superficial cutaneous stimulation, exposure to heat or cold, and bladder distention. Episodes may be so violent and forceful that the patient may be thrown out of his or her wheelchair or fracture bones. Spasticity eventually results in permanent muscle contraction and shortening. Management is rendered in the form of physical, pharmaceutical, surgical, and electrical interventions.

Diminished, Absent, or Altered Pain Response

A diminished or absent pain response[18] may contribute to the development of pressure sores and the failure to notice the presence of wounds, injuries, infections, and other conditions for which pain is the first indication. However, persons with SCI also may develop chronic pain as well as experience pain arising from different areas within the body that is manifested differently than before the injury occurred. **Table 30.2** describes the different types of pain individuals with SCI most often experience. Patients may describe a burning, tingling, or stabbing pain arising from areas of their body that no longer have sensory innervation. This type of pain is called **neurogenic pain** and is a result

of a failure of the brain to receive all the needed information to properly interpret the origin and cause of the pain. This type of pain is difficult to treat because the origin is unknown. Onset of neurogenic pain years after the initial injury is cause for alarm, because it may indicate the onset of a new pathology.

TABLE 30.2 Pain Experienced by Individual with Spinal Cord Injury

TYPE	DESCRIPTION/CAUSE	TREATMENT
Neurogenic pain	Burning, tingling, or stabbing pain arising from areas of the body that no longer have sensory innervation. This results as a failure of the brain to receive all the needed information to properly interpret the origin and cause of the pain.	Neurogenic pain is difficult to treat as the origin is unknown. Onset of neurogenic pain years after the initial injury is cause for alarm, because it may indicate the onset of a new pathology.
Musculoskeletal pain	Aching, dull throbbing pain that increases with activity and is relieved by rest. Pain can usually be traced to repetitive motions such as using the upper extremity to propel a wheelchair, throwing motions in sports, and so forth.	Musculoskeletal pain is treated through activity modification, ergonomic corrections to equipment, as well as therapeutic pain management interventions.
Visceral or referred pain	Cramping, aching, stabbing pain located in the stomach or abdomen and low to mid back may indicate complications with the digestive system. The pain may not be experienced over the organ that is involved but referred to another part of the viscera.	Onset of referred pain warrants patient referral for further medical assessment.
Chronic pain	Pain that is constant, lasting for months and even years in areas that have normal sensation, as well as noninnervated areas is considered chronic pain.	Chronic pain has a psychological component, often contributing to depression. Work in consultation with a medical director and mental health provider to treat the patient's chronic pain.

Effects on Exercise

There are several physiological changes which may occur following SCI that can affect exercise training and testing. Many changes occur due to associated damage to the sympathetic nervous system. The heart may be affected due to sympathetic outflow from the medulla to the SA node being partially interrupted between T1 and T4 nerve roots and completely interrupted above T1. With these individuals, increases in heart rate will occur due to the vagus nerve and the Frank-Starling mechanism being intact.[19] Heart rate, blood pressure, and blood vessel constriction may also be affected due to the separation of the adrenal medullae from normal innervation. SCI above T5 completely separates the adrenal medullae from normal innervation and SCI between T5 and T9 results in partial innervation. Leg vasomotor control can also be compromised; normally, vasoconstriction facilitates shunting of blood to active muscles during exercise and venoconstriction aids venous return from the legs to the heart when upright. SCI above L1 results in complete separation of the leg vasculature from the central nervous system (CNS), and SCI between L1 and L2 can lead to partial separation. The accumulated effects of this is

often referred to as **sympathetic decentralization** which can result in venous pooling, orthostatic and exercise hypotension, edema, and thermoregulation issues.[19] Due to these issues, most adaptations during exercise with SCI individuals are more peripheral in nature (e.g., increased size, strength, endurance, aerobic power, anaerobic power, blood flow, enzymes, and glycogen) as opposed to central adaptations (e.g., increased stroke volume, cardiac output, blood volume, oxygen delivery, and high-density lipoprotein cholesterol). Medications, such as muscle relaxants, anticonvulsants, and antibiotics, may also have an effect on one's response to exercise.[19]

Injury Diagnosis, Treatment, and Rehabilitation Considerations

Sensory dysfunctions can make the SCI individual more susceptible to easy burning of the skin in areas of sensory impairments. When stimuli are applied to sensory-compromised areas, decubitus ulcers, pain, and dysesthesia may also occur. Motor dysfunction can lead to muscle atrophy and deterioration, flaccid or spastic paralysis, cardiovascular deconditioning, and osteoporosis in areas where motor control has been compromised. Taking these into account, it is very difficult to diagnose and treat injuries that occur in a sensory-compromised area. *The use of heat and cold modalities must be monitored very closely*. The use of the upper extremity for ambulation by individuals with SCI must be a concern when designing exercise treatment and rest protocols. **Table 30.3** contains the most common injuries seen in individuals with SCI.[19]

TABLE 30.3 Common Injuries Sustained by Athletes with Spinal Cord Injury

OVERUSE INJURIES	OVEREXERTION INJURIES	SOFT TISSUE INJURIES	LOW BACK PAIN
Shoulder impingement	Muscle strains	Blisters	Soft tissue: muscle or ligament
Tendinitis and bursitis	Sprains	Abrasions	Mechanical or biomechanical: joint
Carpal tunnel syndrome	Contusions	Lacerations	Neurological: nerve root impingement

There are several considerations that must be made during the rehabilitation process due to the sympathetic nervous system, sensory, and motor deficits. Many individuals with SCI will have pronounced orthostatic

and postexercise hypotension.[19] All exercise sessions must take place in a safe environment taking into account compromised balance due to motor dysfunction. Autonomic dysreflexia may also be a concern for individuals with quadriplegia. Many individuals may have a compromised thermoregulatory system that can result in impaired perspiration mechanisms; therefore, exercise environment temperature will be a concern. Surgical issues such as fusions, internal fixation devices, and shunts must be addressed when designing a rehabilitation program.[19]

During the history portion of the examination process, Mary should ask if the patient had a complete or incomplete SCI and if the patient knows his ASIA neurological level of impairment. If the patient responds that he has an incomplete SCI, Mary can then determine the classification of the incomplete SCI. Individuals with SCI are at a higher risk than AB individuals for developing UTIs and neurogenic bowel. During the history, Mary should ask specific questions about the patient's bladder and bowel function as well as perform abdominal percussion and auscultation. Urinalysis and temperature assessment will provide useful information regarding these conditions. Lung auscultations and percussions will assist in assessing for the presence of respiratory complications. A vital sign and cranial nerve assessment should be performed to rule out possible autonomic dysreflexia. Mary should also inquire about potential hot spots that may develop into pressure sores and inspect the skin in areas that are more susceptible to developing these sores. Finally, a musculoskeletal assessment that includes range of motion and manual muscle testing along with myotome, dermatome, and deep tendon reflexes would be beneficial for assessing spasticity associated with SCI. Depending on the patient's level of neurological impairment, the area needing treatment may have sensory and motor deficits. In patents with sensory deficit, care should be taken to avoid using modalities that may burn the skin because the patient will be unable to detect the painful stimuli. If working on core stabilization or dynamic strength in a rehabilitation

program, the patient needs to be supported and secured to prevent falling. Another concern is orthostatic hypertension, so it is imperative to slowly progress the patient through positional changes in posture.

CEREBRAL PALSY

Tyrese is providing athletic training services at a cerebral palsy (CP) race walking event. A coach has asked him to look at one of her athletes. Tyrese confirms that this is not an emergent situation with the athlete and asks the coach what type of CP the athlete has. How will the knowledge of the type of CP that the athlete has impact the history taking and physical examination of the athlete?

Athletes with CP will compete as ambulatory athletes or utilize wheelchairs specifically designed for their sports. Common sports for athletes with CP include 7-a-side football (soccer), race running, athletics, snow skiing, and table cricket.[20] In race running, participants use three-wheeled tricycles designed to support the upper body, yet allow appropriate space for using the legs to run (**Fig. 30.3**). The different stresses on the body during training and competition must be considered when working with athletes with CP.

Figure 30.3. Race walking with assistance from a modified wheel chair. (Used with permission, Hannah Dines/ Matthew Hamilton [photographer].)

Pathophysiology, Common Causes, and Risk Factors

CP is defined as a nonprogressive lesion or malformation of the brain that interferes with normal brain development before, during, or immediately after birth.[20] The condition damages areas of the brain that control muscle tone and spinal reflexes and results in limited ability to move and maintain posture and balance. CP is not one disease with a single cause, but a group of disorders that are related. About 85% of CP cases occur before birth and it occurs in as many as 5 out of 1,000 live births.

CP has several suspected causes: infections during pregnancy, such as German measles or rubella, which can cause damage to the developing nervous system; Rh incompatibility in which the mother's body produces immune cells (antibodies) that destroy the fetus's blood cells leading to jaundice, which can damage brain cells and lead to severe oxygen shortage in the brain; or trauma to the head during labor and delivery. Medical mistakes are also commonly blamed for cases of CP. Impairments may include

cognitive, visual, hearing, speech, and swallowing difficulties. Factors that increase the risk for CP include breech presentation, complicated labor and delivery, low Apgar score (heart rate, breathing, muscle tone, reflexes, and skin color), low birth weight, multiple births, nervous system malformations, such as microcephaly, and maternal bleeding, or severe proteinuria late in pregnancy.

Types of Cerebral Palsy

CP may be described by the body part that has been impacted by the condition, how a person's movements are affected, and by the severity of impairments (Fig 30.4).[21] **Quadriplegia CP** is used to describe bilateral involvement of both the upper and lower extremity, as well as the muscles of the trunk, face, and mouth.[20] **Diplegia CP** describes involvement of both legs, with some involvement of the arms. When the arm and leg on the same side are affected, the term used to describe the condition is **hemiplegia**. Spastic, dyskinetic, and ataxic are terms used to describe CP by how movement is impaired.

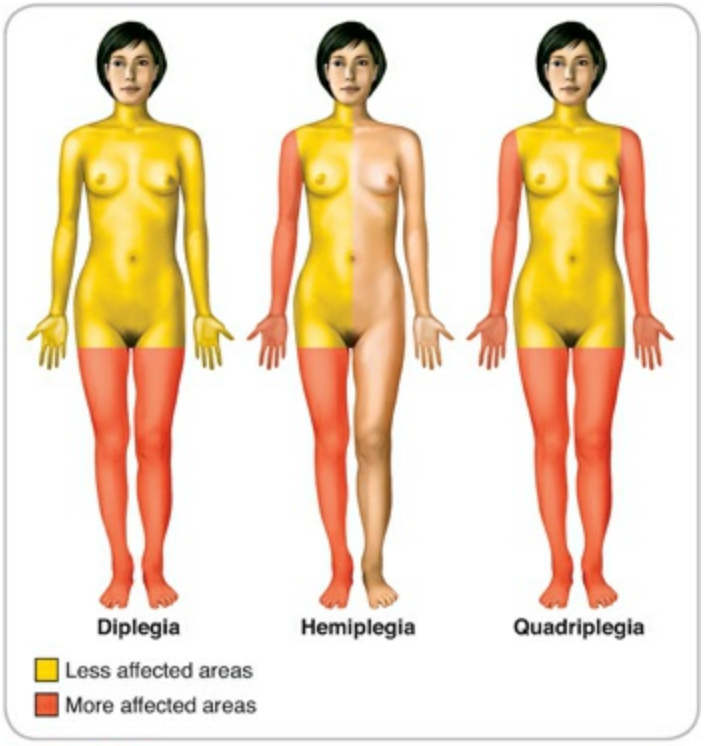

Figure 30.4. Types of cerebral palsy.

Spastic CP occurs when there is damage to the motor cortex of the brain and accounts for 70% to 80% of all CP.[22] Spasticity is an abnormal increase in muscle tone or stiffness, limiting range of motion and normal muscle length and function. A person with spastic CP may present with increased muscle tone (*hypertonicity*), rapid, serial muscle contractions (*clonus*), involuntary crossing of the legs (*scissoring*), or fixed joints due to sustained muscle *contracture*. Additional impairment may include muscle spasm and exaggerated deep tendon reflexes.[23] Spasticity may result in permanent flexion contractures of the elbows, wrists, and fingers in the upper extremity and of the hip and knees in the lower extremity. As the gastrocnemius–soleus complex shortens, an equinovarus foot posture may occur. Overtime, specificity will result in atrophy of the soft tissue, joint contractures, and skeletal deformity and pain.[22]

Dyskinetic CP occurs in about 6% of persons with CP and is caused by damage to the basal ganglia of the brain.[24] A person with dyskinetic CP presents with involuntary motion that occurs when the person is attempting voluntary movement. Slow twisting movements or repetitive movements are referred to as **dystonia** or dyskinesia. **Athetosis** is alternating episodes of hypotonus and extreme motion. Athetosis is sometimes described as slow, writhing, restless movements. Individuals with **chorea**, a third form of dyskinesia, present with brief, abrupt, irregular, and unpredictable movements that may at times appear wild and violent.[24]

Ataxic CP results when the centers within the cerebellum that control posture have been damaged.[25] A person with ataxic CP will appear clumsy, uncoordinated, shaky, and unsteady. Hand tremors, imprecise movement, and difficulty grasping small objects are commonly seen in the upper extremity, whereas a wide-base gait, staggering, and frequent falling is seen in the lower extremity. Ataxic CP will also affect speech, swallowing, and eye movement.

Knowing the type of CP an individual has should provide the athletic trainer with a better understanding of the potential complications and concerns the person may encounter during athletic and recreational activities. Understanding these classification systems, in particular, knowing the impact on gross motor function, fine motor function, and communication function, will

allow for appropriate modifications to sports and protective equipment based on the needs of the individual.[21]

The Gross Motor Function Classification System–Expanded & Revised

This classification system uses the ability to self-initiate mobility with regard to standing, walking, and wheeled conveyance to assess gross motor function and ability. Testing is conducted in the individual's own environment (home, school, work, community) making this a true functional assessment.[26] The Gross Motor Function Classification System–Expanded & Revised (GMFCS–E&R) has five distinct classifications that can serve to clarify the appropriate assistive technologies the individual needs. Although this tool is designed to assess persons in age bands 6 to 12 years old and 12 to 18 years old, the GMFCS–E&R classification rarely changes older than 5 years. Therefore, it is likely that once the initial needed assistive devices are identified, a person with CP will most likely need the same assistance/adaptations throughout the lifespan.[21] The GMFCS–E&R classifications are presented in **Table 30.4**.

TABLE 30.4 Gross Motor Function Classification System–Extended & Revised

FOR CHILDREN/YOUTH BETWEEN THEIR 6TH AND 18TH BIRTHDAY

LEVEL	DESCRIPTORS
1	Can walk indoors and outdoors and climb stairs without using hands for support Can perform usual activities such as running and jumping Has decreased speed, balance, and coordination
2	Has the ability to walk indoors and outdoors and climb stairs with a railing Has difficulty with uneven surfaces, inclines, or in crowds Has only minimal ability to run or jump
3	Walks with assistive mobility devices indoors and outdoors on level surfaces May be able to climb stairs using a railing May propel a manual wheelchair (may require assistance for long distances or uneven surfaces)
4	Walking ability is severely limited even with assistive devices. Uses wheelchairs most of the time and may propel their own power wheelchair May participate in standing transfers
5	Has physical impairments that restrict voluntary control of movement and the ability to maintain head and neck position against gravity Is impaired in all areas of motor function Cannot sit or stand independently, even with adaptive equipment Cannot independently walk although may be able to use powered mobility

Adapted from Figoni S. Spinal cord disabilities: paraplegia and tetraplegia. In: Durstine JL, Moore GE, eds. *ACSM's Exercise Management for Persons with Chronic Diseases and Disabilities*. Champaign, IL: Human Kinetics; 2009; Cerebral Palsy International Sports and Recreation Association. Sports. http://cpisra.org/dir/sports/. Accessed December 10, 2015.

The Manual Ability Classification System

The Manual Ability Classification System (MACS) is a classification system for children with CP ages 4 to 18 years and examines fine motor skills when engaging in activities of daily living.[27] The system includes five categories describing how the child attempts to self-initiate manipulation of everyday objects and their most frequently used fine motor skills. See Table 30.5 for MACS classification descriptors.

TABLE 30.5 Manual Abilities Classification System

LEVEL	DESCRIPTOR
1	Objects are handled easily and successfully.
2	Handles most objects but with some reduced quality and/or speed
3	Handles objects with difficulty—the child will need help to prepare and/or modify activities.
4	Handles a limited selection of easily managed objects and always requires some help from others
5	The child is not able to handle objects or to complete even simple actions with their hands.

Adapted from Figoni S. Spinal cord disabilities: paraplegia and tetraplegia. In: Durstine JL, Moore GE, eds. *ACSM's Exercise Management for Persons with Chronic Diseases and Disabilities*. Champaign, IL: Human Kinetics; 2009; Cerebral Palsy Alliance. Types of cerebral palsy. https://www.cerebralpalsy.org.au/what-is-cerebral-palsy/types-of-cerebral-palsy/. Accessed December 10, 2015.

The Communication Function Classification System

This classification system is used to classify everyday communication needs based on five areas of effective communication.[28] Consistent with the MACS and GMFCS, the Communication Function Classification System (CFCS) also incorporates activity and participation levels as described in the World Health Organization's (WHO) International Classification of Functioning, Disability, and Health (ICF). CFCS classification levels can be viewed in Table 30.6.

TABLE 30.6 Communication Function Classification System

LEVEL	DESCRIPTOR
1	A person independently and effectively alternates between being a sender and receiver of information with most people in most environments
2	A person independently alternates between being a sender and receiver with most people in most environments but the conversation may be slower
3	A person usually communicates effectively with familiar communication partners, but not unfamiliar partners, in most environments
4	The person is not always consistent at communicating with familiar communication partners.
5	A person is seldom able to communicate effectively even with familiar people.

Adapted from Figoni S. Spinal cord disabilities: paraplegia and tetraplegia. In: Durstine JL, Moore GE, eds. *ACSM's Exercise Management for Persons with Chronic Diseases and Disabilities*. Champaign, IL: Human Kinetics; 2009; Cerebral Palsy Alliance. Spastic cerebral palsy. https://www.cerebralpalsy.org.au/what-is-cerebral-palsy/types-of-cerebral-palsy/spastic-cerebral-palsy/. Accessed December 12, 2015.

Effects on Exercise

There has been very little research involving CP and exercise due to the fact that individuals with CP historically didn't participate in exercise.[29] With spasticity and inefficient mobility present, higher than expected exercise response values are seen. Individuals with CP will have higher heart rate, blood pressure, expired air, and blood lactate levels than AB individuals at equal work rates. Reduced mechanical efficiency will also be seen due to the extra energy required to overcome muscle tonus in spastic CP. Maximal physiological responses to exercise have been shown to be 10% to 20% lower than AB individuals with physical work capacity as much as 50% lower.[29] This low work capacity and maximal responses may be related to low fitness levels and poor exercise habits. However, they may also be presented due to difficulty performing movements, muscle imbalances, and poor functional strength. Considering the nature of disability, there is no reason not to expect benefits from exercise. The limited research has observed some benefits, such as increased heart and lung efficiency and increased strength, flexibility, mobility, and coordination. Exercise can also be expected to help maintain bone mineral density, assist in weight control, and reduce the risk of chronic diseases as seen in AB individuals.

Injury Diagnosis, Treatment, and Rehabilitation Considerations

Injury diagnosis can be very difficult with individuals with CP due to the irregularities of muscle tone and movement. Range of motion and manual muscle testing can be of little use when diagnosing an injured individual with CP. Palpation can often be difficult due to excessive neuromuscular excitability seen in many individuals. Therefore, the athletic trainer must often rely almost exclusively on the history, mechanism of injury, and visual observation when diagnosing the injury.[29] The aforementioned irregularities will also have an effect on the treatment of individuals. The application of modalities may trigger episodes of spasticity and neuromuscular excitability. Additionally, these irregularities may inhibit the athlete's ability to keep body parts in certain positions for extended periods of time.

Athletes with CP may compete in wheelchair activities or ambulatory events depending on the severity of their movement dysfunction. Athletes utilizing wheelchairs will experience many of the injuries common to athletes with SCI, whereas ambulatory athletes will commonly incur many of the injuries seen with AB athletes.[29]

The irregularities of muscle tone and movement must be considered when designing an injury treatment protocol for athletes with CP. Modes of exercise will be very dependent on ability. Leg cycle, wheelchair, and arm crank ergometers may be used for aerobic conditioning, as well as traditional ambulatory methods. Muscular strength and endurance can be increased using traditional methods, such as free weight and weight machines, and have also been found to have some positive effect on specificity.[29] Many athletes with CP may have compromised balance which must be considered at all times. Rehabilitation should focus on flexibility to prevent joint contractures. Traditional therapies such as massage, stretching, hydrotherapy, cryotherapy, thermotherapy, and neuromuscular inhabitation may be useful.[30] Keep in mind that many athletes with CP may be taking antiseizure, as well as muscle relaxant medications, which may slow the physiological responses to exercise.[29]

By knowing if the athlete has quadriplegia, hemiplegia, or diplegia CP, Tyrese will have an expectation of where he should see impairment. Likewise, knowing if the individual has spastic, dyskinetic, or ataxic CP provides Tyrese with baseline information on the athlete's gross motor function, fine motor function, and communication function. Individuals with CP often have irregularities in muscle tone and movement, as well as hyper- or hyponeurological responses. Because range of motion, manual muscle testing, and palpation will be of little use when diagnosing an injured individual with CP, the athletic trainer must rely almost exclusively on the history, mechanism of injury, and visual observation when diagnosing the injury.

AMPUTATION

? What biomechanical factors can increase the risk of injury for an amputee?

Within the United States, there are approximately 2 million people who have experienced amputation of a limb. Common causes of amputations include vascular disease, trauma, and cancer.[31] Athletes with amputation may compete using a wheelchair or with or without prosthetics. Common sports for athletes with amputation included track and field, swimming, basketball, volleyball, and cycling. When working with individuals with amputations, having a shared language is important for increased communication. Commonly used terms are presented in Table 30.7.

TABLE 30.7 Common Terminology in Amputee Sports

TERM	DEFINITION
Acquired amputation	Surgical removal of a limb (i.e., traumatic, elective, disease)
AE	Above the elbow amputation; transhumeral
AK	Above the knee amputation; transfemoral
Amputee/limb loss athlete	At least one major joint or part of an extremity missing
BE	Below the elbow amputation; transradial
BK	Below the knee amputation; transtibial
Blade runners	Term for amputee sprinters
Congenital amputation	Born missing a limb, "limb deficient"
Day leg	Prosthetic designed for everyday activity, occasionally worn for some of the field events
DBK	Double below knee amputation (bilateral)
Intact limb	Nonamputated limb
Phantom limb	Sensation that the missing limb is still attached
Phantom pain	Pain sensation arising from missing limb
Residual limb	Portion of limb remaining after amputation (i.e., stump or residuum)

Adapted from Hetzler T, Smith AE, Rempe D. Amputee athletes, part I: foundational knowledge. *Int J Athl Ther Train.* 2014;19(2):33–38.

Pathophysiology, Common Causes, and Risk Factors

Amputations may be a surgical amputation and removal of any part of the body, or a congenital amputation where an individual is born without a limb or

limbs. The more common causes for surgical amputation are diabetes (50%), injuries, infections, tumors, and insufficient blood supply.[32] Congenital amputation occurs in approximately 1 in 2,000 newborns, and the cause is rarely known. Modern technology has significantly helped individuals who have incurred an amputation through improved rehabilitation of badly damaged limbs and the increased technology of prosthetic devices.

Effects on Exercise

Physiological adaptations to exercise training are similar to AB individuals. The effect on exercise training and testing in individuals with amputation is very individualized depending on where the amputation has occurred, therefore individualization is the key.[33] Testing and training should be focused on mobilization of all available muscle mass. Depending on the area of amputation, testing and training can be conducted using an arm crank or wheelchair ergometer, bicycle, or treadmill. Depending on the area and level of the amputation, scar tissue in the knee, hip, shoulder, or elbow may be a limiting factor to exercise with the affected area.

Injury Diagnosis, Treatment, and Rehabilitation Considerations

The biomechanics of individuals with amputations will be altered placing the individual at greater risk for sustaining chronic, overuse, and stress-related injury. There are three primary reasons why biomechanical function is a major factor for amputees and injury. First, the intact limb (if one exists) and core must compensate for loss of power, control, endurance, and force no longer generated by the lost limb.[34] The individual must develop and refine new/altered patterns of movement as he or she learn how to compete at increasing levels of competition. Second, depending on the prosthetic worn, changes to the individual's center of gravity may occur, as well as alteration in the running gait.[35] Stress will be generated and transmitted differently, to which the body must adapt. If increased stress and adaptation do not occur together or are not addressed through appropriate preventative and treatment measures, the

individual is at greater risk for developing stress response or overuse injury such as low back pain, chronic hamstring strains, and lateral hip pain for athletes with lower limb amputation.[34,35] Athletes with upper extremity amputations have a higher incidence of shoulder, cervical, and thoracic conditions.[34] Finally, the skin should be inspected frequently for deterioration associated with constant pressure applied by the prosthetic and/or securing straps. Pressure sores, addressed earlier in this chapter, as well as blisters, rashes, and wounds frequently occur in areas where a constant load is applied. Individuals will attempt to alter biomechanics in order to relieve the pressure being applied to the involved area.

Injury rehabilitation considerations are similar to AB individuals with the exception of the increased incidence of scar tissue at the distal end of the limb. Muscle imbalances, flexibility, and proper gait mechanics must be addressed. Core stabilization exercises, increasing proprioception, and improving balance also decrease risk of injury.[33–35] Phantom limb pain may be present in some athletes with amputations. This is due to a reorganization of the cerebral cortex in which the area of the cortex serving the amputated limb enlarges and the neurons cause pain. Although phantom pain is commonly experienced by individuals with amputations, the pain should not be dismissed but instead, requires the clinician to rule out other causes. Multiple treatment options are available to treat phantom pain, ranging from basic physical therapy to pharmaceutical interventions, although none have been found to be more successful than another.

With an amputation, the intact limb and core must compensate for loss of power, control, endurance, and force no longer generated by the lost limb, thereby making it necessary to develop and refine new/altered patterns of movement as they learn how to compete at increasing levels of competition. Depending on the prosthetic worn, changes to the individual's center of gravity may also occur, as well as alterations in the running gait leading to stress being generated and transmitted differently throughout the body. If increased stress and adaptation do not occur together or are not addressed through

appropriate preventative and treatment measures, the individual is at greater risk for developing stress response or overuse injuries. Finally, constant pressure applied by the prosthetic and/or securing straps may lead to pressure sores, blisters, rashes, and wounds. Individuals may then attempt to alter biomechanics in order to relieve the pressure being applied to the involved area, thus increasing the risk for further injury.

SUMMARY

1. Athletes participating in Paralympic sports are grouped based on disability: physical impairment, visual impairment, and intellectual impairment. Physical impairment includes impaired muscle power, impaired passive range of motion, loss of limb or limb deficit, leg length difference, short stature, hypertonia, ataxia, and athetosis.
2. Individuals who have sustained a complete SCI have no sensory or motor function below the level where the injury occurred. Individuals who have sustained an incomplete SCI have partial sensory and motor function below the level where the injury occurred.
3. The ASIA Impairment Scale describes the neurological level of injury based on the resulting functional impairments due to the SCI.

   A = Complete: no motor or sensory function in the lowest sacral segment (S4 to S5)
   B = Sensory incomplete: some sensory function below neurological level and in S4 to S5
   C = Motor incomplete: There is voluntary anal sphincter contraction, with some motor function to three levels below the motor level of the injury, but most of the key muscles are weak.
   D = Motor incomplete: the same as C, with the exception that most of the key muscles are fairly strong
   E = Normal: Although hyperreflexia may be present, normal sensory and

motor recovery are exhibited.

4. Tetraplegia results in impairment of both motor and sensory function of the arms, trunk, legs, and pelvic organs and is caused by complete SCI within the cervical region. Persons with tetraplegia need assistance with most personal care needs and activities of daily living.
5. The majority of individual with SCI who participate in recreational and competitive sports are thoracic paraplegics, meaning they have full motor and sensory function of the upper extremities and some of the torso.
6. Individuals with lumbar paraplegia will have full motor and sensory function of the torso and upper extremities but have varying degrees of motor and sensory deficits in the lower extremities.
7. Because of limited mobility and impaired innervation to the muscles controlling bladder and bowel function, persons with SCI are at greater risk for developing UTIs and neurogenic bowel.
8. Lack of mobility and impaired innervation in SCI individuals contribute to the increased risk for developing pressure sores, blood clots, DVT, and potential PEs.
9. Autonomic dysreflexia is a life-threatening condition seen in both the acute and chronic phases of injury for individuals having sustained SCI above the T6 level. Inability to receive and/or accurately analyze pain originating from below the injury causes an exaggerated increase in blood pressure and heart rate, thus increasing intracranial pressure that may lead to death.
10. Spasticity is an abnormal increase in muscle tone or stiffness, limiting range of motion and normal muscle length and function. A person with spastic CP may present with increased muscle tone (hypertonicity); rapid, serial muscle contractions (clonus); involuntary crossing of the legs (scissoring); fixed joints due to sustained muscle contracture; and exaggerated deep tendon reflexes. Overtime, specificity will result in atrophy of the soft tissue, joint contractures and skeletal deformity, and

pain.

11. Physiological changes in the SCI patient due to associated damage to the sympathetic nervous system, referred to as sympathetic decentralization, can result in venous pooling, orthostatic and exercise hypotension, edema, and thermoregulation issues.
12. CP is defined as a nonprogressive lesion or malformation of the brain that interferes with normal brain development before, during, or immediately after birth. The condition damages areas of the brain that control muscle tone and spinal reflexes and results in limited ability to move and maintain posture and balance.
13. The ability to make appropriate modifications to sports and protective equipment based on the needs of the individual requires the athletic trainer to understand CP classification systems relating to gross motor function, fine motor function, and communication function. The three primary classifications systems are GMFCS–E&R, the MACS, and the CFCS.
14. Due to spasticity and inefficient mobility present, higher than expected exercise response values are seen in individuals with CP, such as a higher heart rate, blood pressure, expired air, and blood lactate levels than AB individuals at equal work rates.
15. Athletic trainers must rely almost exclusively on history, mechanism, and visual observation when diagnosing an injury in CP patients.
16. Common causes of amputations include vascular disease, trauma, cancer, and congenital factors. Amputations are classified by body part and proximity to the nearest joint.
17. The biomechanics of individuals with amputations will be altered and, therefore, place the individual at greater risk for sustaining chronic, overuse, and stress-related injury, such as low back pain, chronic hamstring strains, and lateral hip pain for athletes with lower limb amputation. Upper extremity amputees experience shoulder, thoracic, and cervical pathology.

18. The skin at the site of the amputation should be inspected frequently for deterioration associated with constant pressure applied by the prosthetic and/or securing straps. Pressure sores, blisters, rashes, and wounds frequently occur in areas where a constant load is applied.

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